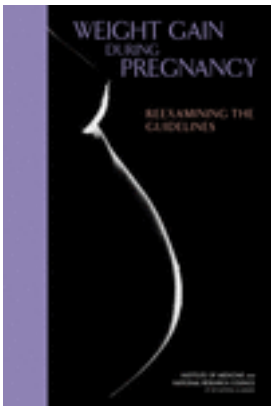

Weight Gain During Pregnancy: Reexamining the Guidelines

Kathleen M. Rasmussen and Ann L. Yaktine, Editors; Committee to Reexamine IOM Pregnancy Weight Guidelines; Institute of Medicine; National Research Council

ISBN: 0-309-13114-6, 336 pages, 6 x 9, (2009)

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Weight Gain During Pregnancy: Reexamining the Guidelines

Kathleen M. Rasmussen and Ann L. Yaktine, *Editors*

Committee to Reexamine IOM Pregnancy Weight Guidelines
Food and Nutrition Board and Board on Children, Youth, and Families

INSTITUTE OF MEDICINE *AND*
NATIONAL RESEARCH COUNCIL
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This study was supported by Contract No. HHSH250200446009I TO HHSH240G5806 between the National Academy of Sciences and U.S. Department of Health and Human Services Health Resources and Services Administration; Contract No. 200-2007-M-21619 between the National Academy of Sciences and Centers for Disease Control and Prevention Division of Nutrition and Physical Activity and Obesity; Contract No. N01-OD-4-2139 TO 192 between the National Academy of Sciences and National Institutes of Health Institute of Child Health and Human Development; Contract No. N01-OD-4-2139 TO 192 between the National Academy of Sciences and National Institutes of Health National Institute of Diabetes and Digestive and Kidney Diseases; Contract No. HHSP23300700522P between the National Academy of Sciences and U.S. Department of Health and Human Services Office on Women's Health; Contract No. HHSP23320070071P between the National Academy of Sciences and U.S. Department of Health and Human Services Office of Disease Prevention and Health Promotion; and Contract No. 21-FY07-576 between the National Academy of Sciences and March of Dimes. Additional support came from U.S. Department of Health and Human Services Office of Minority Health and the National Minority AIDS Council. Any opinions, findings, conclusions, or recommendations expressed in this publication are those of the author(s) and do not necessarily reflect the view of the organizations or agencies that provided support for this project.

International Standard Book Number 0-309-XXXXX-X (Book)
International Standard Book Number 0-309- XXXXX -X (PDF)
Library of Congress Control Number: 00 XXXXXX

Additional copies of this report are available from the National Academies Press, 500 Fifth Street, N.W., Lockbox 285, Washington, DC 20055; (800) 624-6242 or (202) 334-3313 (in the Washington metropolitan area); Internet, http://www.nap.edu.

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The serpent has been a symbol of long life, healing, and knowledge among almost all cultures and religions since the beginning of recorded history. The serpent adopted as a logotype by the Institute of Medicine is a relief carving from ancient Greece, now held by the Staatliche Museen in Berlin.

Suggested citation: IOM (Institute of Medicine). 2009. *Weight Gain During Pregnancy: Reexamining the Guidelines.* Washington, DC: The National Academies Press.

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"Knowing is not enough; we must apply.
Willing is not enough; we must do."
—Goethe

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Reviewers

This report has been reviewed in draft form by individuals chosen for their diverse perspectives and technical expertise, in accordance with procedures approved by the National Research Council's (NRC's) Report Review Committee. The purpose of this independent review is to provide candid and critical comments that will assist the institution in making its published report as sound as possible and to ensure that the report meets institutional standards for objectivity, evidence, and responsiveness to the study charge. The review comments and draft manuscript remain confidential to protect the integrity of the deliberative process. We wish to thank the following individuals for their review of this report:

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Although the reviewers listed above have provided many constructive comments and suggestions, they were not asked to endorse the conclusions or recommendations nor did they see the final draft of the report before its release. The review of this report was overseen by **Neal A. Vanselow,** Tulane University, Professor Emeritus and **Nancy E. Adler,** Departments of Psychiatry and Pediatrics and Center for Health and Community, University of California–San Francisco.

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Appointed by the NRC and Institute of Medicine, they were responsible for making certain that an independent examination of this report was carried out in accordance with institutional procedures and that all review comments were carefully considered. Responsibility for the final content of this report rests entirely with the authoring committee and the institution.

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Preface

In the last century, many answers have been given by health professionals to the question "how much weight should I gain while I am pregnant?" In the early 1900's, the answer was often only 15-20 pounds. Between 1970 and 1990, the guideline for weight gain during pregnancy was higher, 20-25 pounds, and in 1990, with the publication of *Nutrition During Pregnancy*, it went higher still for some groups of women. This most recent guideline reflected new knowledge about the importance of maternal body fatness before conception, as measured by body mass index, for the outcome of pregnancy. It had become clear that heavier women could gain less weight and still deliver an infant of good size. Since that time, the obesity epidemic has not spared women of reproductive age. In our population today, more women of reproductive age are severely obese (obesity class III; 8 percent) than are underweight (3 percent) and their short- and long-term health has become a concern in addition to the size of the infant at birth. Clearly the time had come to reexamine the guidelines for weight gain during pregnancy.

To prepare for this possibility, the National Research Council and the Institute of Medicine held a workshop in 2006 to evaluate the availability of data that could be used reexamine the current guidelines. Based on the outcome of this workshop, numerous federal agencies (Department of Health and Human Services: Health Resources Services Administration; Centers for Disease Control and Prevention Division of Nutrition, Physical Activity, and Obesity; National Institutes of Health: The Eunice Kennedy Shriver National Institute of Child Health and Development; National Institute of Diabetes, Digestive and Kidney Diseases, Division of Nutrition Research Coordination Office on Women's Health; Office on Disease Prevention and Health Promotion; and Office on Minority Health; as well as the March of Dimes) agreed to sponsor the work of this committee.

The committee was asked to review the determinants and a wide range of short- and long-term consequences of variation in weight gain during pregnancy for both the mother and her infant. Based on the outcome of this review, the committee was asked to recommend revisions to the current guidelines if this was deemed to be necessary. In addition, the committee was asked to consider the approaches that might be necessary to promote appropriate weight gain and to identify gaps in knowledge and make recommendations about priorities for future research.

Although many studies relevant to the committee's charge have been published since 1990 and the Agency for Healthcare Research and Quality (AHRQ) completed their report *Outcomes of Maternal Weight Gain* while the committee was gathering data, many gaps in knowledge remained. To address this problem, the committee held a public session with project sponsors, and two workshops. We are grateful to those who participated in these sessions for sharing their experience and wisdom. We are also grateful to a number of individuals who supplied data to the committee: Alan Ryan, Aimin Chen, Andrea Sharma, Joyce Martin, Sharon Kirmeyer, K.S. Joseph, Marie Cedergren, and Raul Artal, with special thanks to Patricia Dietz. The committee also commissioned additional analyses of data from both Denmark and the United States. We thank our consultants, Ellen Aagaard Nohr, Amy Herring, and Cheryl Stein for these analyses

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and for their contributions to the committee's work. The committee also felt that it was important to understand what would be involved in analyzing the trade-off between mother and infant in risk of adverse outcomes of variation in weight gain during pregnancy. To accomplish this, we commissioned such an analysis based on the data at hand. We thank our consultant, James Hammitt, for conducting these analyses and for his contribution to the committee's work.

The committee's 14 members gave freely of their expertise and volunteered their time and energy in all aspects of the preparation of this report, from developing its intellectual framework, writing the text and deliberating about the recommendations and conclusions of the report. Their efforts merit our sincere gratitude.

The committee received excellent staff support from Ann Yaktine, Study Director, Heather Del Valle, Research Associate and Jennifer Datiles, Senior Program Assistant. Their effort on our behalf is sincerely appreciated. Both the Director of the Food and Nutrition Board, Linda Meyers, and the Director of the Board on Children, Youth and Families, Rosemary Chalk, contributed their wisdom and support to this effort and we thank them for it.

Kathleen M. Rasmussen, *Chair*
Committee to Reexamine IOM Pregnancy Weight Guidelines

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Summary

Since 1990, the last time the Institute of Medicine (IOM) released guidelines for weight gain during pregnancy, many key aspects of the health of women of childbearing age have changed. This population now includes a higher proportion of women from racial/ethnic subgroups and prepregnancy body mass index (BMI) and gestational weight gain (GWG) have increased among all population subgroups. Moreover, high rates of overweight and obesity are common in the population subgroups that are at risk for poor maternal and child health outcomes. Finally, women are also becoming pregnant at an older age and, as a result, are entering pregnancy more commonly with chronic conditions such as hypertension or diabetes, which put them at risk for pregnancy complications and may lead to increased morbidity during their post-pregnancy years. These and other factors suggested a need to reexamine the IOM (1990) guidelines for weight gain during pregnancy and consider whether revision might be warranted.

In response to these concerns, sponsors[1] asked the Food and Nutrition Board of the IOM and the Board on Children, Youth, and Families in the Division of Behavioral and Social Sciences and Education of the National Research Council to review the IOM (1990) recommendations for weight gain during pregnancy. Specifically, the committee was asked to review evidence on relationships between weight gain patterns before, during, and after pregnancy, and maternal and child health outcomes; consider factors within a life-stage framework associated with outcomes such as lactation performance, postpartum weight retention, cardiovascular and other chronic diseases; and recommend revisions to existing guidelines where necessary. Finally, the committee was asked to recommend ways to encourage the adoption of the weight gain guidelines through consumer education, strategies to assist practitioners, and public health strategies.

GUIDELINES FOR WEIGHT GAIN DURING PREGNANCY

The new guidelines for GWG that are shown in Table S-1 are formulated as a range for each category of prepregnancy BMI. This approach reflects the imprecision of the estimates on which the recommendations are based, the reality that good outcomes are achieved within a range of weight gains, and the many additional factors that may need to be considered for an individual woman. It is important to note that these guidelines are intended for use among women in the United States. They may be applicable to women in other developed countries. However, they are not intended for use in areas of the world where women are substantially shorter or thinner than American women or where adequate obstetric services are unavailable.

The new guidelines differ from those issued in 1990 in two ways. First, they are based on the World Health Organization (WHO) cutoff points for the BMI categories instead of the previous ones, which were based on categories derived from the Metropolitan Life Insurance tables. Second, and more importantly, the new guidelines include a specific, relatively narrow range of recommended gain for obese women.

[1]Sponsors include: Health Resources and Services Administration, Department of Health and Human Services; Centers for Disease Control and Prevention, Division of Nutrition, Physical Activity, and Obesity; National Institutes of Health, National Institute of Child Health and Human Development, National Institute of Diabetes and Digestive and Kidney Diseases, Division of Nutrition Research Coordination; Office on Women's Health; Office on Disease Prevention and Health Promotion; Office on Minority Health; and the March of Dimes.

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TABLE S-1 New Recommendations for Total and Rate of Weight Gain during Pregnancy, by Prepregnancy BMI

	Total Weight Gain		Rates of Weight Gain* 2nd and 3rd Trimester	
Prepregnancy BMI	**Range in kg**	**Range in lbs**	**Mean (range) in kg/week**	**Mean (range) in lbs/week**
Underweight (< 18.5 kg/m^2)	12.5–18	28–40	0.51 (0.44–0.58)	1 (1–1.3)
Normal weight (18.5–24.9 kg/m^2)	11.5–16	25–35	0.42 (0.35–0.50)	1 (0.8–1)
Overweight (25.0–29.9 kg/m^2)	7–11.5	15–25	0.28 (0.23–0.33)	0.6 (0.5–0.7)
Obese (≥ 30.0 kg/m^2)	5–9	11–20	0.22 (0.17–0.27)	0.5 (0.4–0.6)

* Calculations assume a 0.5–2 kg (1.1– 4.4 lbs) weight gain in the first trimester (based on Siega-Riz et al., 1994; Abrams et al., 1995; Carmichael et al., 1997)

These new guidelines should be considered in the context of data on women's reported GWG. Data from several large groups of women indicate that the mean gains of underweight women fall within the new guidelines, but some normal weight women may exceed these new guidelines and a majority of overweight or obese women will likely exceed them. These data provide a strong reason to assume that interventions will be needed to assist women, particularly those who are overweight or obese at the time of conception, in meeting the guidelines. These interventions may need to occur at both the individual and community levels and may need to include components related to both improved dietary intake and increased physical activity.

The committee intends that the guidelines shown in Table S-1 be used in concert with good clinical judgment as well as a discussion between the woman and her care provider about diet and exercise. If a woman's GWG is not within the proposed guidelines, clinicians should consider other relevant clinical evidence, modifiable factors that might be causing excessive or inadequate gain, and information on the nature of excess GWG (e.g. fat or edema) as well as both the adequacy and consistency of fetal growth before suggesting that a woman modify her pattern of weight gain.

Special Populations

Women of Short Stature

The IOM (1990) report recommended that women of short stature (< 157 cm) gain at the lower end of the range for their prepregnant BMI. The committee was unable to identify evidence sufficient to continue to support a modification of GWG guidelines for women of short stature. Although women of short stature had an increased risk of emergency cesarean delivery, this risk was not modified by GWG. Women of short stature did not have an increased risk of having an small-for-gestational age (SGA) or large-for-gestational age (LGA) infant or of excessive postpartum weight retention over taller women.

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Pregnant Adolescents

Evidence available since the IOM (1990) report is also insufficient to continue to support a modification of the GWG guidelines for adolescents (< 20 years old) during pregnancy. The committee also determined that prepregnancy BMI could be adequately categorized in adolescents by using the WHO cutoff points for adults, in part because of the impracticality of using pediatric growth charts in obstetric practices. Adolescents who follow adult BMI cutoff points will likely be categorized in a lighter group and thus advised to gain more; however, younger adolescents often need to gain more to improve birth outcomes.

Racial or Ethnic Groups

Although an increasing proportion of pregnant U.S. women are members of racial or ethnic minority groups, the limited data available to the committee from commissioned analyses suggested that membership in one of these groups did not modify the association between GWG and the outcome of pregnancy. As a result, the committee concluded that its recommendations should be generally applicable to the various racial or ethnic subgroups that make up the American population although additional research is needed to confirm this approach.

Women with Multiple Fetuses

Recent data suggest that the weight gain of women with twins who have good outcomes varies with prepregnancy BMI as is clearly the case for women with singleton fetuses. Inasmuch as the committee was unable to conduct the same kind of analysis for women with twins as it did for women with singletons, the committee offers the following provisional guidelines: normal weight women should gain 17-25 kg (37-54 pounds), overweight women, 14-23 kg (31-50 pounds) and obese women, 11-19 kg (25-42 pounds) at term. Insufficient information was available with which to develop even a provisional guideline for underweight women with multiple fetuses. These provisional guidelines reflect the interquartile (25th to 75th percentiles) range of cumulative weight gain among women who delivered their twins, who weighed ≥ 2,500 g on average, at 37-42 weeks of gestation.

DEVEOPMENT OF THE GUIDELINES FOR WEIGHT GAIN DURING PREGNANCY

The committee worked from the perspectives that the reproductive cycle begins before conception and continues through the first year postpartum and that maternal weight status throughout the entire cycle affects both the mother and her child. To inform its review of the literature and to guide the organization of its report, the committee reevaluated the conceptual framework that guided the development of the IOM (1990) report. To account for advances in our scientific understanding of the determinants and consequences of GWG, the committee developed a modified conceptual framework (Figure S-1). However, it retained the same scientific approach and epidemiologic conventions used previously and discussed in detail in the IOM (1990) report.

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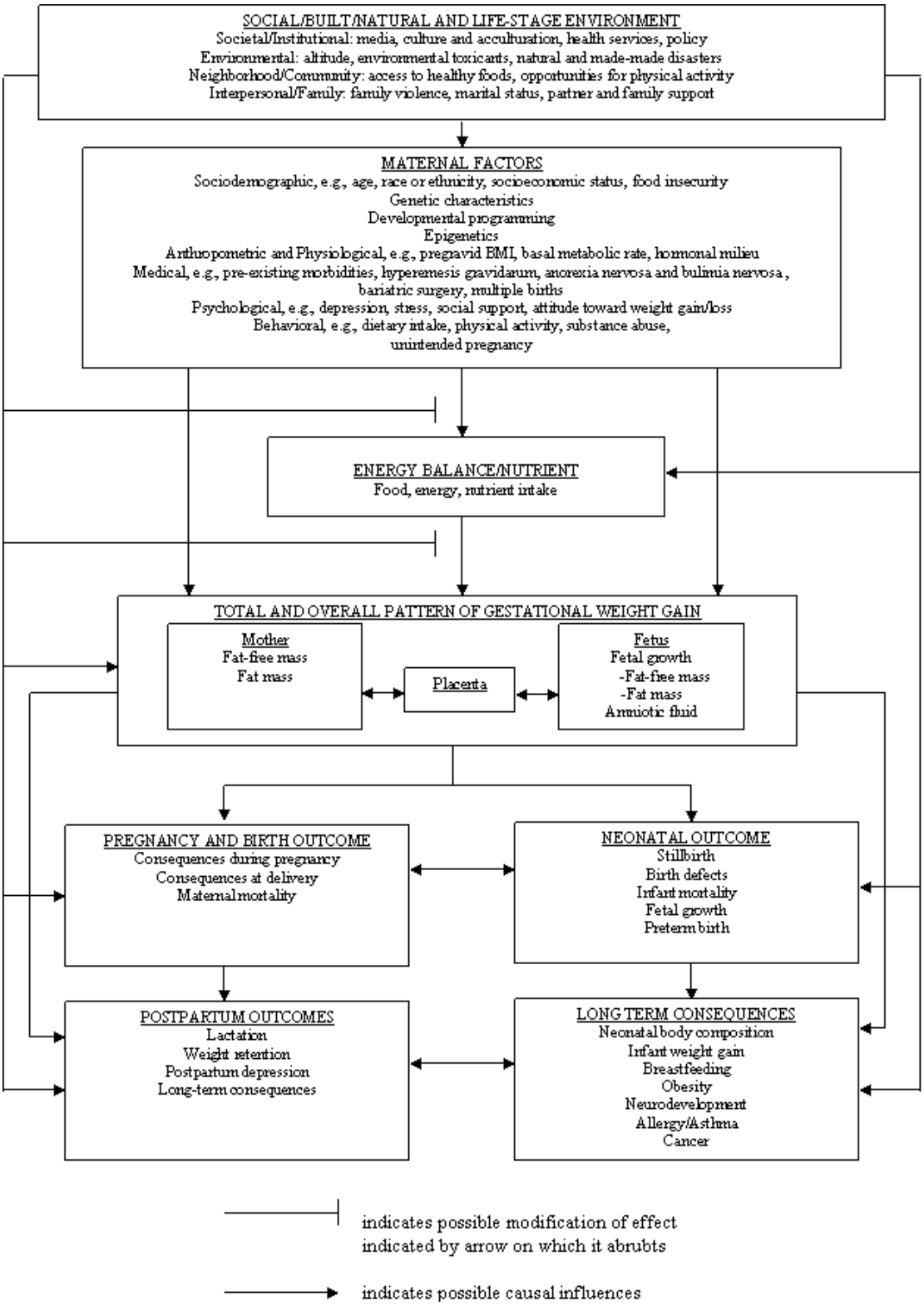

FIGURE S-1 Schematic summary of potential determinants and consequences for gestational weight gain.
SOURCE: Modified from IOM, 1990.

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The committee began its work by considering appropriate BMI cutoff points and describing trends over time in maternal prepregnancy BMI and GWG among American women. In addition, data were sought on both the determinants and consequences of GWG. The search for such data revealed major gaps in data collection and analysis.

Key Finding S-1: The WHO cutoff points for categorizing BMI have been widely adopted and should be used for categorizing prepregnancy BMI as well.

Key Finding S-2: Currently available data sources are inadequate for studying national trends in GWG, or postpartum weight, or their determinants.

Action Recommendation S-1: The committee recommends that the Department of Health and Human Services conduct routine surveillance of GWG and postpartum weight retention on a nationally representative sample of women and report the results by prepregnancy BMI (including all classes of obesity), age, racial/ethnic group, and socioeconomic status.

Action Recommendation S-2: The committee recommends that all states adopt the revised version of the birth certificate, which includes fields for maternal prepregnancy weight, height, weight at delivery, and gestational age at the last measured weight. In addition, all states should strive for 100 percent completion of these fields on birth certificates and collaborate to share data, thereby allowing a complete national picture as well as regional snapshots.

Research Recommendation S-1: The committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to conduct studies in large and diverse populations of women to understand how dietary intake, physical activity, dieting practices, food insecurity and, more broadly, the social, cultural and environmental context affect GWG.

In developing its recommendations, the committee identified a set of consequences for the short- or long-term health of the mother and the child that are potentially causally related to GWG. These consequences included those evaluated in a systematic review of outcomes of maternal weight gain prepared for the Agency for Healthcare Research and Quality (AHRQ) as well as others based on data from the literature outside the time window considered in that report. To address conflicts and gaps within the available literature, the committee commissioned four additional analyses from existing databases. The committee considered the results from these commissioned analyses in conjunction with evidence from published scientific literature.

Postpartum weight retention, cesarean delivery, gestational diabetes mellitus, and pregnancy-induced hypertension or preeclampsia emerged from this process as being the most important maternal health outcomes. The committee removed preeclampsia and gestational diabetes mellitus from consideration because of the lack of sufficient evidence that GWG was a cause of these conditions. Postpartum weight retention and, in particular, unscheduled primary cesarean delivery were retained for further consideration.

Measures of size at birth (e.g. SGA and LGA), preterm birth, and childhood obesity emerged from this process as being the most important infant health outcomes. The committee recognized that both SGA and LGA, when defined as < 10th percentile and > 90th percentile of weight-for-gestational age, respectively, represent a mix of individuals who are appropriately or inappropriately small or large. In addition, the committee recognized that being SGA was likely to be associated with deleterious outcomes for the infant but not the mother, while being LGA was likely to be associated with consequences for both the infant and the mother (e.g., cesarean delivery).

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Key Finding S-3: Evidence from the scientific literature is remarkably clear that prepregnancy BMI is an independent predictor of many adverse outcomes of pregnancy. As a result women should enter pregnancy with a BMI in the normal weight category.

Key Finding S-4: Although a record-high proportion of American women of childbearing age have BMI values in obesity classes II and III, available evidence is insufficient to develop more specific recommendations for GWG among these women.

Research Recommendation S-2: The committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to conduct studies in all classes of obese women, stratified by the severity of obesity, on the determinants and impact of GWG, pattern of weight gain, and its composition on maternal and child outcomes.

Key Finding S-5: There are only limited data available with which to link GWG to health outcomes of mothers and children that occur after the neonatal period.

Research Recommendation S-3: The committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to conduct studies on the eating behaviors, patterns of dietary intake and physical activity, and metabolic profiles of pregnant women, especially obese women, who experience low gain or weight loss during pregnancy. In addition, the committee recommends that researchers conduct studies on the effects of weight loss or low GWG, including periods of prolonged fasting and the development of ketonuria/ketonemia during gestation, on growth and on development, and long-term neurocognitive function in the offspring.

Research Recommendation S-4: The committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to conduct observational and experimental studies on the association between GWG and (a) glucose abnormalities and gestational hypertensive disorders that take into account the temporality of the diagnosis of the outcome, and (b) the development of glucose intolerance, hypertension and other cardiovascular risk factors as well as mental health and cancer later in a woman's life.

Research Recommendation S-5: The committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to conduct studies that (a) explore mechanisms, including epigenetic mechanisms, that underlie effects of GWG on maternal and child outcomes and (b) address the extent to which optimal GWG differs not only by maternal prepregnancy BMI but also by other factors such as age (especially among adolescents), parity, racial/ethnic group, socioeconomic status, co-morbidities, and maternal/paternal/fetal genotype.

Research Recommendation S-6: The committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to conduct observational and experimental studies to assess the impact of variation in GWG on a range of child outcomes, including duration of gestation, weight and body composition at birth, and neurodevelopment, obesity and related outcomes, and asthma later in childhood.

Based on the available published literature as well as the reports of its consultants, the committee ascertained the GWG value or range of values associated with lowest prevalence of the outcomes of greatest interest. When weighting the trade-off among these outcomes, the committee considered, within each category of prepregnant BMI (a) the incidence or prevalence of each of these outcomes, (b) whether the outcomes were permanent (e.g. neurocognitive deficits) or potentially modifiable (e.g. postpartum weight retention) and (c) the quality of the available data. The committee compared the resulting ranges with those developed in the

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quantitative risk analysis conducted by its consultants. Finally, the committee considered how its possible recommendations might be accepted and used by clinicians and women.

Research Recommendation S-7: To permit the development of improved recommendations for GWG in the future, the committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to (a) conduct studies to assess utilities (values) associated with short- and long-term health outcomes associated with GWG for both mother and child and (b) include these values in studies that employ decision analytic frameworks to estimate optimal GWG according to category of maternal prepregnancy BMI and other subgroups.

APPROACHES TO ACHIEVING RECOMMENDED WEIGHT GAIN DURING PREGNANCY

To meet the recommendations of this report fully, two different challenges must be met. First, a higher proportion of American women should conceive at a weight within the range of normal BMI values. Meeting this first challenge requires preconceptional counseling and, for many women some weight loss. Such counseling may need to include additional contraceptive services as well as services directed toward helping women to improve the quality of their diets and increase their physical activity. Preconception counseling is an integral part of the recommendations from the Centers for Disease Control (Johnson et al., 2006). Practical guidelines for preconceptional care are provided in *Nutrition During Pregnancy and Lactation: An Implementation Guide* (IOM, 1992). The need to meet this challenge reinforces the importance of preconceptional counseling as the cornerstone for achieving optimal outcomes of pregnancy and improved health for mothers and their children.

Action Recommendation S-3: The committee recommends that appropriate federal, state and local agencies as well as health care providers inform women of the importance of conceiving at a normal BMI and that all those who provide health care or related services to women of childbearing age include preconceptional counseling in their care.

Second, a higher proportion of American women should limit their GWG to the range specified in these guidelines for their prepregnant BMI. Meeting this second challenge requires a different set of services**.** The first step in assisting women to gain within these guidelines is letting them know that they exist, which will require educating their healthcare providers as well as the women themselves.

Action Recommendation S-4: The committee recommends that relevant federal agencies, private voluntary organizations, and medical and public health organizations adopt these new guidelines for GWG and publicize them to their members and also to women of childbearing age.

Individualized attention is called for in the IOM (1990) guidelines and was an element in all of the interventions that have been successful in helping women to gain within their target range. Guidelines on providing such care are provided in *Nutrition During Pregnancy and Lactation: An Implementation Guide* (IOM, 1992). The increase in prevalence of obesity that has occurred since this report was written suggests that this recommendation has only become more important.

In offering women individualized attention, a number of kinds of services could be considered. Health care providers should chart women's weight gain and share the results with them so that they become aware of their progress toward their weight-gain goal. To assist healthcare providers in doing this, the committee has prepared charts that could be used as a

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basis for this discussion with the pregnant woman. These charts are meant to be used as part of an assessment of the progress of pregnancy and a woman's weight gain, looking beyond the gain from one visit to the next and toward the overall pattern of weight gain. In addition, women should be provided with individualized advice about both diet and physical activity (ACOG, 2002). This may require referral to a dietitian as well as other appropriately qualified individuals, such as those who specialize in helping women to increase their physical activity. These services may need to continue into the postpartum period to give women the maximum support to return to their prepregnant weight within the first year and, thus, to have a better chance of returning to a normal BMI value at the time of a subsequent conception.

Individualized attention is likely to be necessary but not sufficient to enable most women to gain within the new guidelines. Family- and community-level factors must also be addressed if women are to succeed in gaining within these guidelines. Further research on these kinds of multilevel, ecological determinants of GWG is needed to guide the development of comprehensive and effective implementation strategies to achieve these guidelines. In addition, special attention should be given to low-income and minority women, who are at risk of being overweight or obese at the time of conception, consuming diets of lower nutritional value, and of performing less recreational physical activity.

Action Recommendation S-5: To assist women to gain within the guidelines, the committee recommends that those who provide prenatal care to women should offer them counseling, such as guidance on dietary intake and physical activity, that is tailored to their life circumstances.

Research Recommendation S-8: The committee recommends that the Department of Health and Human Services provide funding for research to aid providers and communities in assisting women to meet these guidelines, especially low-income and minority women. The committee also recommends that the Department of Health and Human Services provide funding for research to examine the cost-effectiveness (in terms of maternal and offspring outcomes) of interventions designed to assist women in meeting these guidelines.

CONCLUDING REMARKS

Although the guidelines developed as part of this committee process are not dramatically different from those published previously (IOM, 1990), fully implementing them would represent a radical change in the care provided to women of childbearing age. In particular, the committee recognizes that full implementation of these guidelines would mean:

- Offering preconceptional services, such as counseling on diet and physical activity as well as access to contraception, to all overweight or obese women to help them reach a healthy weight before conceiving. This may reduce their obstetric risk and normalize infant birth weight as well as improve their long-term health.
- Offering services, such as counseling on diet and physical activity, to all pregnant women to help them achieve the guidelines on GWG contained in this report. This may also reduce their obstetric risk, reduce postpartum weight retention, improve their long-term health, normalize infant birth weight and offer an additional tool to help reduce childhood obesity.
- Offering services, such as counseling on diet and physical activity, to all postpartum women. This may help them to eliminate postpartum weight retention and, thus, to be able to conceive again at a healthy weight as well as improve their long-term health.

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The increase in overweight and obesity among American women of childbearing age and failure of many pregnant women to gain within the IOM (1990) guidelines alone justify this radical change in care as women clearly require assistance to achieve the recommendations in this report in the current environment. However, the reduction in future health problems among both women and their children that could possibly be achieved by meeting the guidelines in this report provide additional justification for the committee's recommendations.

These new guidelines are based on observational data, which consistently show that women who gained within the IOM (1990) guidelines experienced better outcomes of pregnancy than those who did not (see Chapters 5 and 6). Nonetheless, these new guidelines require validation from experimental studies. To be useful, however, such validation through intervention studies must have adequate statistical power not only to determine if a given intervention helps women to gain within the recommended range but also to determine if doing so improves their outcomes. In the future, it will be important to reexamine the trade-offs between women and their children in pregnancy outcomes related to prepregnancy BMI as well as GWG, and also to be able to estimate the cost-effectiveness of interventions designed to help women meet these recommendations.

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Setting the Stage for Revising Pregnancy Weight Guidelines: Conceptual Framework

BACKGROUND

Improvement of maternal, fetal, and child health are key public health goals. Over the past four decades, changes in public health trends have challenged the healthcare sector to provide optimal guidance to women before, during, and after pregnancy to achieve healthy outcomes for themselves and their newborns.

The report, *Maternal Nutrition and the Course of Pregnancy* (NRC, 1970) developed from concern about high neonatal and infant mortality rates in the United States compared to other developed countries. In that report, the Committee on Maternal Nutrition recognized the positive relationship between gestational weight gain (GWG) and birth weight. The committee also noted the positive association between prepregnancy maternal weight and birth weight and the fact that higher prepregnancy maternal weight reduced the impact of GWG on birth weight. The report advised an average gestational weight gain of 24 pounds (20-25-pound range) and advised against the then-current practice of limiting GWG to 10-14 pounds.

A subsequent Institute of Medicine (IOM) report *Nutrition During Pregnancy* (IOM, 1990) offered recommendations for weight gain during pregnancy based on prepregnancy maternal body mass index (BMI). The report also made specific weight gain recommendations for population subgroups, including adolescents, members of racial and ethnic groups, women of short stature, and women carrying twins. The report also details historic trends in weight gain recommendations and guidelines. These recommendations for weight gain during pregnancy have been adopted by or have been influential in many countries. Reviews of observational studies support that women who enter pregnancy at a normal BMI and gain within the ranges recommended in the IOM (1990) guidelines are more likely to have a good birth outcome than women who gain outside the recommended ranges (Taffel et al., 1993; Abrams et al., 2000; Gross, 2006).

In the years since the release of the weight gain recommendations from the IOM (1990) report, some dramatic shifts in the demographic and epidemiologic profile of the U.S. population have occurred. Notably, prepregnancy BMI and excess GWG have increased across all population groups, particularly minority groups (Yeh and Shelton, 2005; Kim et al., 2007). These and other factors suggested a need to consider whether a revision of the IOM (1990) pregnancy weight gain guidelines is needed.

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RATIONALE FOR REVISING THE GUIDELINES

General Principles Framing the Guidelines

The IOM (1990) pregnancy weight guidelines were developed principally in response to concerns about low birth weight infants. Although adverse health outcomes for excess weight gain were considered in the IOM (1990) weight gain guidelines, these recommendations were derived largely from data collected in the 1980 National Natality Survey (Available: http://www.cdc.gov/nchs/about/major/nmihs/abnmihs.htm [accessed March 3, 2008]) and focused on preventing premature births and small-for-gestational age infants.

The IOM (1990) report and a subsequent report, *Nutrition During Pregnancy and Lactation* (IOM, 1992), identified specific actions practitioners could take to achieve the recommendations in working with patients. They also identified a series of research recommendations for epidemiologic, basic, and applied research to enable better estimates of GWG, prepregnancy weight for height, and gestational duration, which affect study design and interpretation.

INDICATORS FOR REVISING THE CONCEPTUAL FRAMEWORK OF THE GUIDELINES

In 1996 an expert work group was convened by the Maternal and Child Health Bureau of the Health Resources and Services Administration (HRSA), Department of Health and Human Services (HHS), to examine issues relating to maternal weight gain that had been published in the IOM (1990) report. The purpose of this group was to determine whether new research provided a basis for practitioners to change guidance for GWG, and to recommend future directions for research, training, and/or other programmatic initiatives. The group concluded that formal revision of the IOM (1990) weight gain recommendations was not yet warranted; however reservations were expressed that the recommendations for African-American women, young adolescents, and women of short stature were too specific (Suitor, 1997).

Since publication of the IOM reports *Nutrition During Pregnancy* (1990) and *Nutrition During Lactation* (1991) and the subsequent *Implementation Guide* (1992), the population of U.S. women of childbearing age has become more diverse. Although low birth weight remains a significant concern during pregnancy, new health concerns have emerged. These include the greater prevalence of women who are overweight or obese entering pregnancy, which puts them at high risk for pregnancy complications. For example, data from the 2003-2004 round of the National Health and Nutrition Examination Survey (NHANES) show that 28.9 percent of women of reproductive age (20-39 years old) were obese (BMI $\geq$ 30 kg/m^2) and 8.0 percent were extremely obese (BMI $\geq$ 40 kg/m^2) (Ogden et al., 2006). Women are becoming pregnant at an older age and enter pregnancy with chronic conditions such as type 2 diabetes, which also puts them at risk for pregnancy complications and may lead to increased morbidity during their post-pregnancy years (Cleary-Goldman et al., 2005; Joseph et al., 2005; Delpisheh et al., 2008).

EMERGING TRENDS FROM RESEARCH ON GESTATIONAL WEIGHT GAIN

Weight patterns (underweight and overweight) and GWG have short- and long-term consequences for the health of the mother. For example, prepregnancy BMI above normal values (19.8-26 kg/m^2) is associated with preeclampsia, gestational diabetes (GDM), cesarean delivery

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(Doherty et al., 2006; Abenhaim et al., 2007), and failure to initiate and sustain breastfeeding (Hilson et al., 1997; Li et al., 2003; Kugyelka et al., 2004). Increased maternal BMI and GWG have also been associated with higher fat mass in infants and subsequent overweight in children (Hillier et al., 2007; Oken et al., 2007).

Collectively, these trends and newer research prompted concern about the appropriateness of existing guidelines for GWG to support optimal outcomes for mother, infant, and child. Specifically there were concerns about the implications of the recommendations for (1) the health of the mother, particularly for women who are overweight, underweight, older, adolescent, or short in stature; (2) for infant and child health; and (3) for other metabolic processes that may affect the in utero environment.

Another concern that has frequently been raised by researchers and practitioners is the difference between BMI categories used in the IOM (1990) report and those used in the report *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults* from the National Heart, Lung, and Blood Institute (NHLBI, 1998) in cooperation with the National Institute of Diabetes and Digestive and Kidney Diseases, which are based on a report from the World Health Organization (1995). This is a problem for practitioners as well as for researchers. Most importantly, despite the effort made to publicize the recommendations of the IOM (1990) report, including the development of a guide to assist the medical profession to implement these guidelines (IOM, 1992), many healthcare providers have not used these guidelines and many women have not followed them (Abrams et al., 2000).

SETTING THE STAGE FOR REVISING THE GUIDELINES

In response to such concerns, the Maternal and Child Health Bureau of HHS requested that the National Research Council and the IOM convene a workshop in May 2006. The purpose of this workshop was to review trends in maternal weight; explore emerging research findings related to the complex relationship of the biological, behavioral, psychological, and social interactions that affect maternal and pregnancy weight on maternal and child health outcomes and discuss interventions. The following specific questions were addressed by the workshop were:

- What research and databases describe the distribution of maternal weight (prior to, during, and after pregnancy) among different populations of women in the United States?
- What research and databases inform understanding of the effects of different weight patterns (including underweight and overweight) during pregnancy on maternal and child health outcomes?
- What research has been conducted to describe the individual, community, and healthcare system factors that impede or foster compliance with recommended GWG guidelines?
- What opportunities exist for Title V maternal and child health programs to build on this knowledge to help childbearing women achieve and maintain recommended weight?
- What future research and data collection efforts could improve the efforts of Title V programs to support women from different racial and ethnic backgrounds in their

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efforts to comply with recommended weight guidelines and to improve their maternal health?

The workshop summary report, *Influence of Pregnancy Weight on Maternal and Child Health* (NRC-IOM, 2007), includes a review of U.S. trends in maternal weight (before, during, and after pregnancy) among different populations of women. The workshop report also includes a discussion of the determinants of GWG; the relationships among maternal weight, GWG, and the health of women and children; interventions in healthcare and community settings that help women achieve appropriate weight levels during and after pregnancy; and emerging themes that warrant further examination in future studies. Taken together, the workshop and its summary report reinforce the need to reexamine recommendations for GWG, especially in light of the current obesity epidemic, and to highlight ways to encourage their adoption.

THE COMMITTEE'S TASK

Sponsors[1] asked the IOM's Food and Nutrition Board and the Division of Behavioral and Social Sciences and Education Board on Children, Youth, and Families to review and update the IOM (1990) recommendations for weight gain during pregnancy and recommend ways to encourage their adoption through consumer education, strategies to assist practitioners, and public health strategies.

The committee was asked to address the following tasks:

1. Review evidence on the relationship between weight gain patterns before, during, and after pregnancy and maternal and child health outcomes, with particular attention to the prevalence of maternal obesity racial/ethnic and age differences, components of GWG, and implications of weight during pregnancy on postpartum weight retention and maternal and child obesity and later child health.
2. Within a life-stage framework consider factors in relation to GWG that are associated with maternal health outcomes such as lactation performance, postpartum weight retention, cardiovascular disease, metabolic processes including glucose and insulin-related issues, and risk of other chronic diseases; for infants and children, in addition to low birth weight, consider early developmental impacts and obesity related consequences (e.g., mental health, diabetes).
3. Recommend revisions to the existing guidelines, where necessary, including the need for specific pregnancy weight guidelines for underweight, normal weight, and overweight and obese women and adolescents and women carrying twins or higher-order multiples.
4. Consider a range of approaches to promote appropriate weight gain, including:

    - Individual (behavior), psychosocial, community, healthcare, and health systems;
    - Timing and components of interventions; and

[1] Sponsors include: Health Resources and Services Administration, Department of Health and Human Services; Centers for Disease Control and Prevention, Division of Nutrition, Physical Activity, and Obesity; National Institutes of Health, National Institute of Child Health and Human Development, National Institute of Diabetes and Digestive and Kidney Diseases, Division of Nutrition Research Coordination; Office on Women's Health; Office on Disease Prevention and Health Promotion; Office on Minority Health; and the March of Dimes.

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- Ways to enhance awareness and adoption of the guidelines, including interdisciplinary approaches, consumer education to men and women, strategies to assist practitioners to use the guidelines, and public health strategies; and

5. Identify gaps in knowledge and recommend research priorities.

Approach to the Task

The committee approached its task by gathering information from existing literature, which included a systematic review of the literature by the Agency for Healthcare Research and Quality (AHRQ) (Viswanathan et al., 2008) (see Appendix E for literature reviewed). The committee also gathered information from presentations by recognized experts in three workshops (see "Open Sessions"). It consulted with additional experts in relevant fields and commissioned new data analyses. Contributions made to the committee by consultants are noted throughout the report. The information-gathering activities laid the groundwork for the committee's work of deliberating on issues relevant to the task and formulating a strategy to address the scope of work. This task was not regarded by the committee as a formal systematic, evidence-based review as the full range of literature did not lend itself to this type of task. Rather, because of the wide-ranging and large literature on this subject, the committee relied on its collective expertise to determine how much weight to give to all of the sources of information at its disposal.

The committee worked from the perspective that pregnancy-related weight begins before conception and continues through the first year postpartum and affects both the mother and her child. In consideration of Task 1, given the magnitude and complexity of the task, the committee determined that it was unable to address maternal weight history before entering pregnancy other than to take prepregnant BMI into account. Whenever possible, the committee sought and presented data on outcomes associated with GWG by racial/ethnic groups. This was done in the spirit of documenting disparities across racial/ethnic groups that the committee anticipated would reflect the strong socio-economic differentials and not biological differences across these groups. This assumption is grounded in the fact that ethnicity is, by definition, a socio-cultural construct and race, the way it is defined in the U.S., has been shown to be a social and not a biological construct (Goodman, 2000).

It is noteworthy that the committee was not charged with evaluating either the safety or effectiveness of the IOM (1990) guidelines. However, observational studies clearly indicate that gaining within the 1990 guidelines is associated with better pregnancy outcomes (and, presumably, greater safety) than gaining outside of them (Taffel et al., 1993; Abrams et al, 2000; Gross, 2006). Moreover, safety and effectiveness of a set of guidelines is a function of many factors, including adoption and use of them by the health care team, acceptance and actual use of them by their target audience, any barriers the target audience might experience in achieving the guidelines and, finally, whether those who actually meet the guidelines have better outcomes.

ORGANIZATION OF THE REPORT

This report is organized into 8 chapters in which the committee describes what is known about GWG, with particular attention to demographic and other factors associated with GWG that falls above or below recommended levels, identifies data gaps, and makes recommendations based on the committee's findings.

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The report begins by introducing the reasoning for a reexamination of pregnancy weight guidelines, based on data that have been gathered since the publication of *Nutrition During Pregnancy* (IOM, 1990). To inform its review of the literature and to guide the organization of this report, the committee reevaluated the conceptual framework that guided the development of the IOM (1990) report. The committee developed a modified conceptual framework (see Figure 1-1) to account for advances in scientific understanding of the determinants and consequences of GWG. However, it retained the same scientific approach and epidemiologic conventions used previously and discussed in detail in the IOM (1990) report. Several changes in the conceptual framework are noteworthy. The committee chose to highlight the importance of numerous environmental factors as determinants of maternal factors that lead to GWG. It is recognized that some of these act through maternal factors to influence GWG and its consequences, while others may affect those consequences by other routes.

Trends in GWG are considered in Chapter 2, with particular attention to weight gain in racial or ethnic subgroups of the U.S. population. Composition and components of GWG are addressed in Chapter 3. Since the IOM (1990) report was prepared, the importance of the placenta in the dialogue between the mother and fetus has become more apparent and the physiology and metabolism of the components of weight gain, including the placenta, are also discussed in Chapter 3.

In consideration of the determinants of GWG, the committee chose to distinguish between maternal factors that are fixed at conception (e.g., age, racial or ethnic group, parity) and those that could potentially be modified during the gestation period (e.g., smoking, drug use, medical conditions that could be treated). These factors are discussed in Chapter 4.

The new conceptual framework draws attention to outcomes in the perinatal period as well as those that occur postpartum and also much later in the lives of mothers and their children. These consequences of GWG are discussed in Chapters 5 and 6. Recommendations for action and approaches to implementation are discussed in Chapters 7 and 8. Recommendations for research are presented at the end of each chapter. The data reviewed in the chapters is tabulated in accompanying appendixes.

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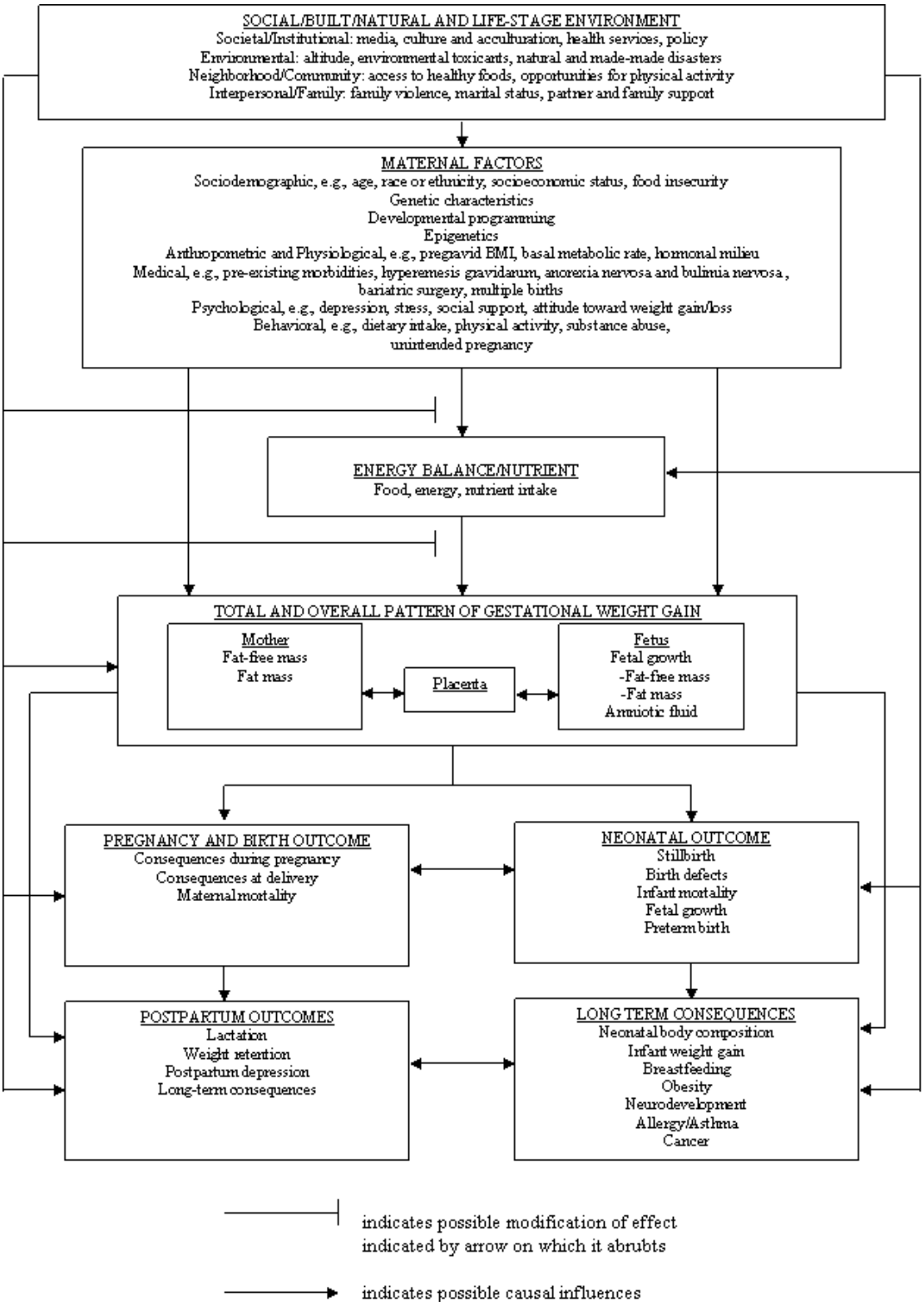

FIGURE 1-1 Schematic summary of potential determinants and consequences for gestational weight gain.
SOURCE: Modified from IOM, 1990.

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Website:
http://www.cdc.gov/nchs/about/major/nmihs/abnmihs.htm

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2

Descriptive Epidemiology and Trends

To reexamine recommendations for weight gain during pregnancy, it is important to evaluate trends since 1990 in maternal body mass index (BMI) before pregnancy as well as gestational weight gain (GWG). These are evaluated together because BMI before and after pregnancy and GWG are interrelated. It is also important to examine trends in key maternal characteristics and pregnancy outcomes that are related to maternal weight before pregnancy and also to GWG. This information provides a context for understanding the sociodemographic and behavioral environment that may influence successful promotion of healthy GWG and optimal pregnancy outcomes.

TRENDS IN MATERNAL WEIGHT AND GESTATIONAL WEIGHT GAIN

Maternal Body Mass Index

One of the most serious issues that practitioners and scientists have faced in the past 30 years is the increase in prevalence of overweight and obesity among American women of childbearing age (Flegal et al., 1998; Mokdad et al., 1999; IOM, 2005; Kim et al. 2007). The prevalence of obesity in women 12 to 44 years of age has more than doubled since 1976 (Table 2-1). Data collected by the National Center for Health Statistics (NCHS) in 1999-2004 showed that nearly two-thirds of women of childbearing age were classified as overweight (as defined by BMI $\geq$ 25 kg/m^2) and almost one-third were obese (BMI $\geq$ 30 kg/m^2) (personal communication, A. Branum, Centers for Disease Control and Prevention [CDC], December 2008). Obesity is far more common among racial or ethnic minority groups and increases in prevalence with advancing age.

Importantly, the prevalence of severe obesity, once a relatively rare condition, has increased dramatically among women of childbearing age (Table 2-1). Between 1979 and 2004, class I and II obesity doubled and class III obesity tripled. Trends are similar by age. The prevalence of all classes of obesity is lowest in white non-Hispanic women, and highest in non-Hispanic black women, where the prevalence of class I obesity approaches 25 percent, and the prevalence of class II and III obesity each exceeds 10 percent. Almost one-fifth of Hispanic women have class I obesity, with the proportions of class II and III obesity each approaching 10 percent.

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TABLE 2-1 Distribution of BMI (World Health Organization categories) from 1976 to 2004 Among U.S. Nonpregnant Women 12 to 44 Years of Age by Race or Ethnicity and Age (percentage)

	1976-1980	1988-1994	1999-2004
Total (%)			
Underweight	6.0	4.4	3.5
Normal weight	62.1	53.4	41.1
Overweight	18.8	20.8	25.3
Class I obese	7.9	12.2	15.8
Class II obese	3.5	6.0	7.7
Class III obese	1.7	3.4	6.5
By Race or Ethnicity			
Non-Hispanic white (%)			
Underweight	6.3	4.7	4.3
Normal weight	64.2	58.3	46.4
Overweight	17.9	18.4	23.3
Class I obese	7.2	10.5	13.8
Class II obese	2.9	5.3	6.9
Class III obese	1.5	2.8	5.3
Non-Hispanic black (%)			
Underweight	3.9	2.7	—[a]
Normal weight	47.8	37.3	23.4
Overweight	24.4	27.7	25.7
Class I obese	13.3	15.8	23.7
Class II obese	7.3	9.7	12.2
Class III obese	—[a]	6.8	13.3
Mexican American (%)			
Underweight	—[b]	1.9	—[a]
Normal weight	—[b]	36.0	32.0
Overweight	—[b]	32.3	32.6
Class I obese	—[b]	18.1	19.6
Class II obese		6.9	7.9
Class III obese		4.7	6.7
By Age			
Age 20-34 (%)			
Underweight	7.1	5.1	4.6
Normal weight	64.9	58.3	44.2
Overweight	16.8	18.2	23.9
Class I obese	6.9	10.6	14.8
Class II obese	3.0	5.2	7.1
Class III obese	1.4	2.6	5.4
Age 35-44 (%)			
Underweight	3.8	3.3	2.1
Normal weight	55.7	46.8	37.3
Overweight	23.2	24.2	27.1
Class I obese	10.2	14.2	17.1
Class II obese	4.8	7.0	8.6
Class III obese	—[a]	4.4	7.9

NOTE: Underweight, < 18.5 kg/m^2; normal, 18.5 to < 25.0 kg/m^2; overweight, 25.0 to < 30.0 kg/m^2; class I obese, 30.0 to < 35.0 kg/m^2; class II obese, 35.0 to < 40 kg/m^2; class III obese, ≥ 40 kg/m^2.

[a] Insufficient unweighted data to make reliable estimates.
[b] Hispanic ethnicity not available in 1976-1980 National Health and Nutrition Examination Survey (NHANES).

SOURCE: Personal communication, A. Branum, CDC, Hyattsville, Md., December 2, 2008.

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As a result, more women are already obese when they become pregnant. Based on data from the Pregnancy Risk Assessment Monitoring System (PRAMS), one-fifth of American women are obese (BMI > 29 kg/m^2) at the start of pregnancy, a figure that has risen 70 percent in the last decade (Kim et al., 2007) (Figure 2-1). Although the prevalence of overweight has increased slightly in the population as a whole and among black and white women, the prevalence of obesity doubled in white women and increased by 50 percent in black women. These statistics are based on data from only nine states; no nationally representative data are available from a modern cohort to provide trends in pregravid BMI values.

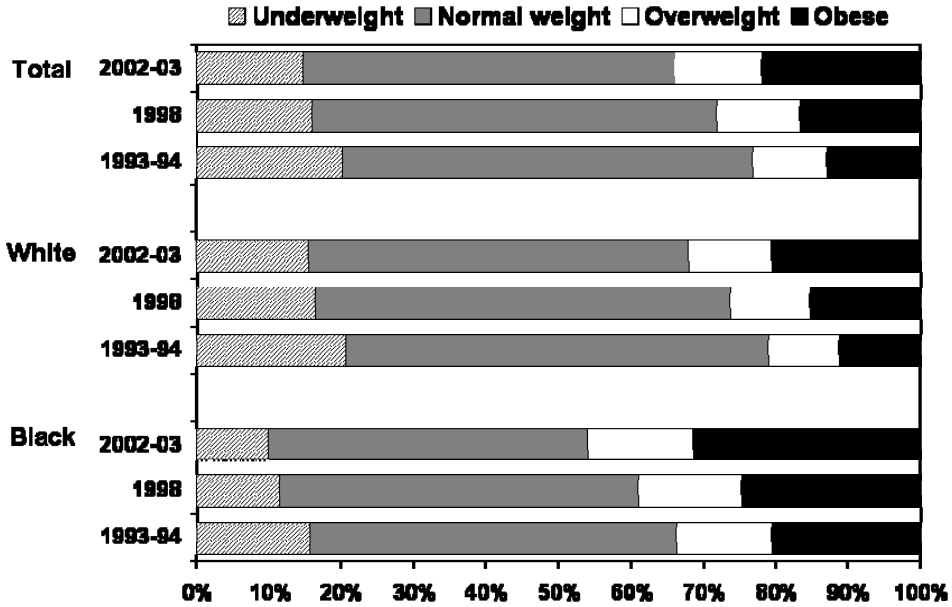

FIGURE 2-1 Trends in the distribution of BMI* from 1993 to 2003 among prepregnant U.S. women in the total population and by race or ethnicity.

*IOM BMI categories were used (underweight: < 19.8 kg/m^2; normal weight: 19.8-26.0 kg/m^2; overweight: 26.1- 29.0 kg/m^2; obese: > 29 kg/m^2).

SOURCE: Kim et al., 2007.

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Body Mass Index Classification

The report *Nutrition During Pregnancy* (IOM, 1990) recommended the use of BMI to classify maternal prepregnancy weight. The four prepregnancy BMI categories used in that report were selected to be consistent with 90 percent, 120 percent, and 135 percent of the 1959 Metropolitan Life Insurance Company's ideal weight-for-height standards—the standard most commonly used in the United States when the report was written. Since then, the World Health Organization (WHO, 1998) has developed and the National Heart, Lung, and Blood Institute (NHLBI, 1998) has adopted the use of new BMI categories. The WHO BMI categories are based on different considerations and, as a result, are defined differently than those in the Institute of Medicine (1990) report. The WHO BMI categories also include several grades or categories of obesity (see Table 2-2).

The weight gain categories identified in IOM (1990) classify more women as underweight than the more stringent WHO cutoff point, while the WHO categories classify more women as overweight and fewer women as obese, with similar differences by race or ethnicity and age. In 1999-2004, with either the IOM or WHO cutoff points, about half of women are overweight (BMI > 26 with IOM cutoff point or > 25 with WHO cutoff point) (Figure 2-2). In addition, it is impractical to expect that pediatric growth charts are available in most obstetric practices. Adolescents that follow adult BMI cutoff points will likely be categorized in a lighter group and thus advised to gain more; however, young teens often need to gain more to improve birth weight outcomes.

TABLE 2-2 Comparison of Institute of Medicine (IOM) and World Health Organization (WHO) BMI Categories

Category	IOM	WHO
Underweight	< 19.8 kg/m^2	< 18.5 kg/m^2
Normal weight	19.8-26 kg/m^2	18.5-24.9 kg/m^2
Overweight	26.1-29 kg/m^2	25.-29.9 kg/m^2
Obese Class I	> 29 kg/m^2	30-34.9 kg/m^2
Obese Class II	---	35-39.9 kg/m^2
Obese Class III	---	≥ 40 kg/m^2

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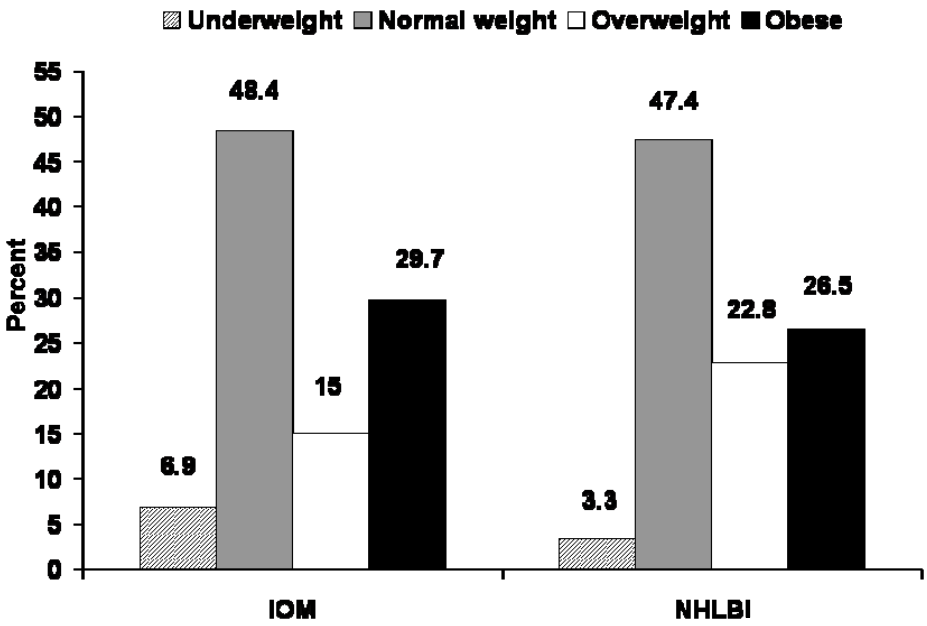

FIGURE 2-2 Distribution of BMI from 1999 to 2004 among U.S. nonpregnant women 12 to 44 years of age using the IOM* (1990) and the WHO** BMI cutoff points.

*IOM (1990) BMI categories are underweight, <19.8 kg/m^2; normal, 19.8-26.0 kg/m^2; overweight, 26.1-29.0 kg/m^2; obese, > 29 kg/m^2.

**WHO BMI categories are underweight, < 18.5 kg/m^2; normal, 18.5-24.9 kg/m^2; overweight, 25.0-29.9 kg/m^2; obese, ≥ 30 kg/m^2.

SOURCE: Personal communication, A. Branum, CDC, Hyattsville, Md., April 15, 2008.

Gestational Weight Gain

Assessment of both prepregnant BMI and GWG requires rigorous methods of data collection (see Table 2-3). Unfortunately, most of the data available to the committee were not collected with a high level of rigor, and most studies relied on recalled weight values (see Table 2-4). Although the IOM (1990) report called for collection of national data on GWG, prepregnancy height, and weight for proper surveillance, today there are still no nationally representative data with which to study trends in GWG in the United States.

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TABLE 2-3 Data Required to Assess Trends in Pregnancy-Related Maternal Weight and the Ideal and Practical Methods of Measurement and Acquisition

	Method of Measurement and Acquisition	
Required Data	**Ideal**	**Practical**
Prepregnancy weight	Measured[a] at a preconceptional visit	Recalled at the first prenatal visit using a standardized question
Prepregnancy height	Measured[a] at the first prenatal visit	
Gestational weight gain	Total gain: last measured available weight abstracted from clinical records	Total gain: maternal recall of last available weight
	Pattern of gain: requires trimester-specific or midpregnancy weight abstractions	
Gestational age at last available weight[b]	Abstracted from clinical records	
Postpartum weight	Total retention: measured maternal weight abstracted from clinical records	Total retention: recalled maternal postpartum weight
	Measured longitudinally in nonpregnant women	Cross sectionally in nonpregnant women
	Time: serial measurements 3, 6, 9, 12, and 18 months after delivery	Time: 3, 6, 9, 12, or 18 months after delivery

[a]All weight and height measurements should be performed in light clothing without shoes.
[b]The gestational age at delivery may vary substantially from the gestational age at the last prenatal visit. Thus, misclassification may result if the gestational age at delivery is used in combination with weight at the last prenatal visit to determine weight gain adequacy.

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TABLE 2-4 National Data Sources for Maternal Weight and Their Methods of Acquiring Key Variables

Data Source	Prepregnancy Weight	Prepregnancy Height	Gestational Weight Gain	Postpartum Weight	Data Coverage
Ideal	Recalled weight at first prenatal visit is abstracted from clinical records	Measured height at first prenatal visit is abstracted from clinical records	Last recorded weight is abstracted from clinical records	Measured weight at least once starting 3 months or more postpartum	50 states, little to no missing data
Standard U.S. birth certificate	Not available	Not available	Recalled at delivery	Not applicable	49 states (excludes California)
Revised 2003 U.S. birth certificate	Recalled at delivery	Recalled at delivery	Based on last recorded weight abstracted from the medical record	Not applicable	19 states in 2006
PRAMS	Recalled at 2-4 months postpartum	Recalled at 2-4 months postpartum	Obtained from birth certificates (recalled at delivery)	Not available	9 states
PNSS	Recalled at the prenatal visit or postpartum visit	Measured at the prenatal visit or postpartum visit	Recalled at the postpartum visit	Measured at WIC postpartum recertification visit	Low-income women in 26 states
IFPS II	Recalled in the postpartum period	Recalled in the postpartum period	Recalled in the postpartum period	Recalled at 3, 6, 9, and 12 months	Nationally distributed consumer opinion panel

NOTE: IFPS II = Infant Feeding Practices Study II; PNSS = Pregnancy Nutrition Surveillance System; WIC = Special Supplemental Nutrition Program for Women, Infants, and Children.

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Trends in Gestational Weight Gain

The data obtained by standard U.S. birth certificates from 49 states illustrate that from 1990 to 2005 among mothers of term, singleton pregnancies, reported weight gains of < 16 pounds and > 40 pounds both increased (Figure 2-3). Weight gain within the broad recommended range (16 to 40 pounds) (IOM, 1990) declined slowly during this 15-year period. Unfortunately, the standard birth certificate lacks data on maternal prepregnancy weight and height. Thus, data from this source cannot provide information about GWG relative to prepregnant BMI category. In addition, the data on prepregnancy weight was self-report, which are more variable than clinical measures. The loss in precision and the degree of bias due to self report must be taken into account in interpreting self-reported data.

Important differences were found in low and high gains by maternal race or ethnicity and age. The greatest increase in the proportion of women with a weight gain > 40 pounds from 1990 to 2005 was among white women (Figure 2-4). In 2005, adolescents (< 20 years old) were more likely to gain excessive weight during pregnancy than women 35 years of age and older. Between 1990 and 2005, there was a 31 percent increase in GWG of at least 40 pounds in singleton pregnancies among adolescents (NCHS, 2007a). In 2005, weight gain of < 15 pounds was more common among black and Hispanic than among white women (Figure 2-5). Within each racial or ethnic group, the proportion of women with low gains increased with advancing age.

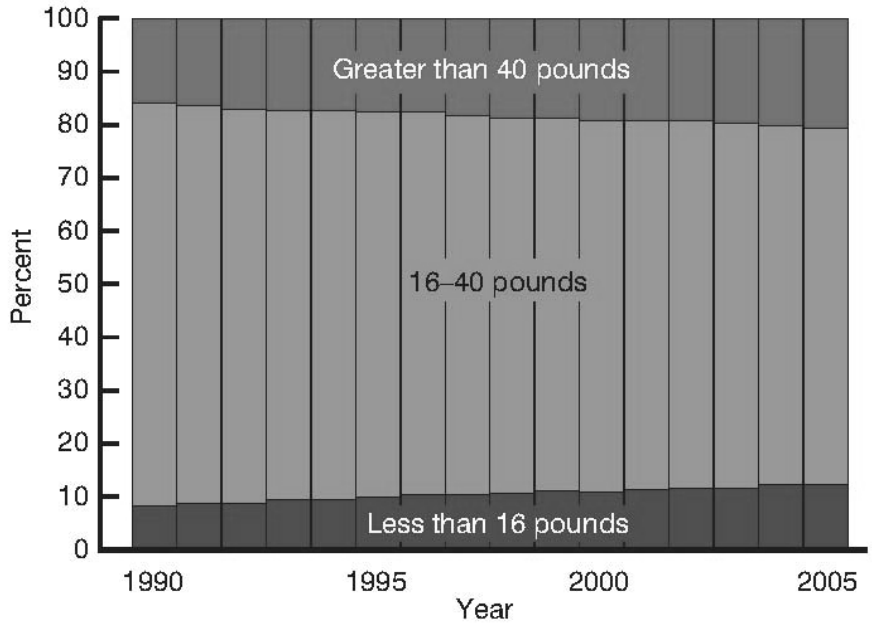

FIGURE 2-3 Weight gain during pregnancy for singleton term births in the United States, 1990-2005.
NOTES: California does not report weight gain in pregnancy. Term is ≥ 37 weeks' gestation.
SOURCE: NCHS, 2007a.

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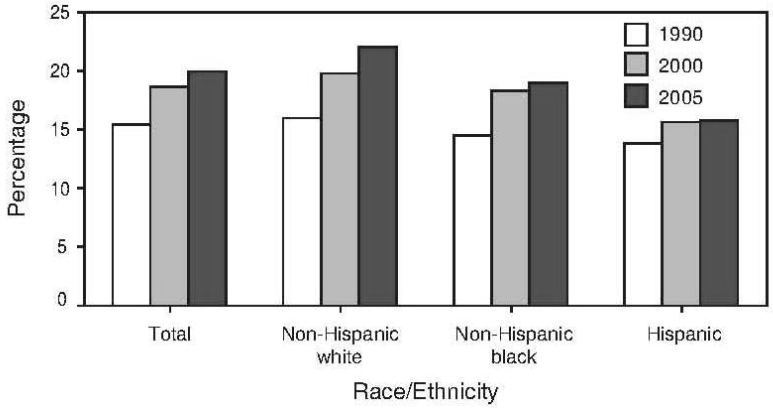

FIGURE 2-4 Percentage of women in the United States who gained more than 40 pounds during pregnancy, by race or ethnicity of the mother, 1990, 2000, and 2005.
NOTES: Includes only mothers with a singleton delivery and only non-Hispanic white, non-Hispanic black, and Hispanic mothers (who might be of any race). The total number of women who gained > 40 pounds was 456,678 in 1990, 588,253 in 2000, and 656,363 in 2005.
SOURCE: CDC, 2008a.

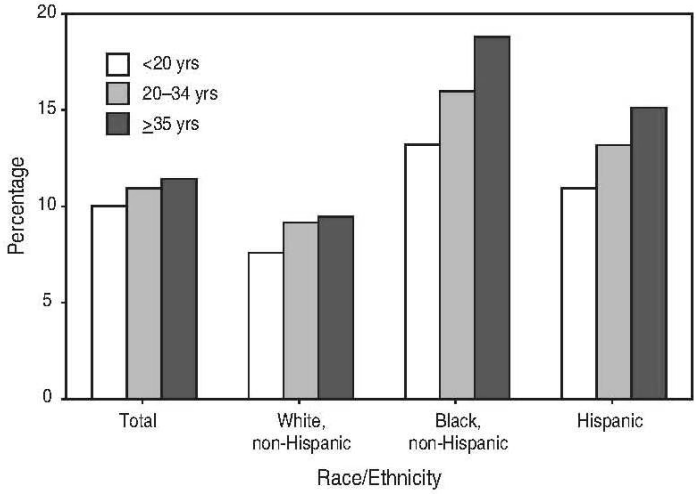

FIGURE 2-5 Percentage of women in the United States who gained less than 15 pounds during pregnancy by age and race or ethnicity of the mother, 2005.
NOTES: Includes only mothers with a term (≥ 37 weeks' gestation), singleton infant; excludes data for California.
SOURCE: CDC, 2008b.

Birth certificate data may yield more useful statistics for weight gain surveillance in the near future. After the IOM (1990) report called for collection of maternal prepregnancy weight and height, these fields were added to the 2003 revised U.S. birth certificate, and by 2006, 19 states were using the revised birth certificate.

At present, the two large surveillance systems collecting data on GWG and prepregnancy BMI in the United States, PRAMS and PNSS, (see Appendix A for descriptions) permit identification of trends in recommended weight gains, although neither system is nationally representative. For PRAMS, GWG is taken from the birth certificate and other data is either pulled from medical records or maternal recall.

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PRAMS collects GWG data from birth certificates, and maternal prepregnancy height and weight are obtained from maternal interview in the postpartum period. Currently, 37 states, New York City and the Yankton Sioux Tribe (South Dakota) participate in PRAMS (Available online: http://www.cdc.gov/prams/ [accessed February 5, 2009]). For the analysis of trends in GWG reported here, data were limited to the eight PRAMS states with at least 70 percent response rates and to women with complete data on prepregnancy BMI and singleton, term pregnancies (Alabama, Arkansas, Florida, Maine, New York [excludes New York City], Oklahoma, South Carolina, and West Virginia). Limitations in the dataset, including self-reported weight, were considered.

In 2002-2003, PRAMS data indicate that the mean GWG was highest in underweight and normal weight women and declined in overweight and obese women among all racial/ethnic groups (Figure 2-6). The mean GWG among underweight and normal weight women in all racial/ethnic groups was within the recommended range, whereas it was higher than the recommendations for overweight women. For obese women, average weight gains were well above the 15-pound recommended minimum. Similar trends were observed in 1992-1993 and 1998 (data not shown).

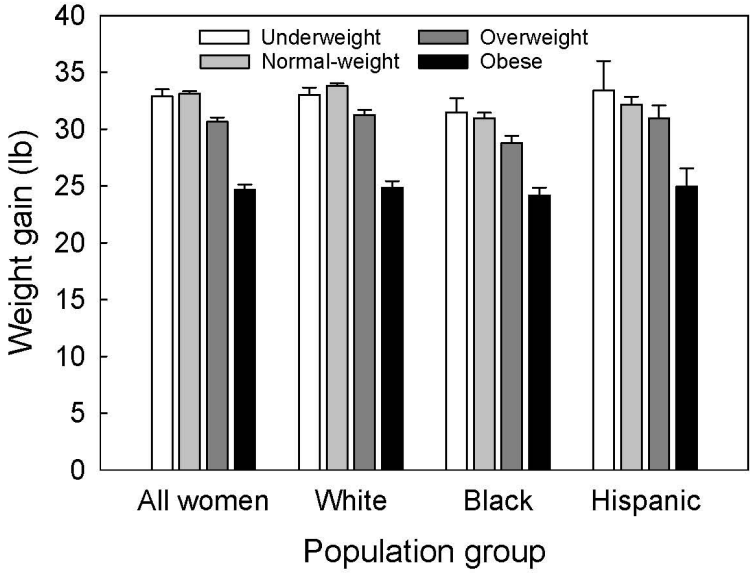

FIGURE 2-6 Mean gestational weight gain by BMI category and race or ethnicity, Pregnancy Risk Assessment Monitoring System, 2002-2003.
NOTE: WHO BMI categories were used (underweight, < 18.5 kg/m^2; normal, 18.5-24.9 kg/m^2; overweight, 25.0-29.9 kg/m^2; obese, ≥ 30 kg/m^2).
SOURCE: Information contributed to the committee in consultation with P. Dietz, CDC, Atlanta, Ga., January 2009.

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In 2002-2003, nearly half of underweight women represented in the PRAMS data gained within the range recommended by IOM (1990), while 30.6 percent and 19.5 percent gained below and above the recommendations, respectively (Figure 2-7). For normal weight women, GWG varied little over this 10-year period. There was a small decrease in the proportion of women gaining less than, while a larger proportion of women gained in excess of the IOM (1990) recommendations.

The majority of overweight women had weight gains greater than the recommended range (Figure 2-7). By 2002-2003, only about one-quarter of overweight women gained within the recommended range. For obese women, there was a modest rise in the prevalence of excessive weight gain from 1993-1994 to 2002-2003. By the end of the observation period, only one-third of obese women gained within the recommended range. Among women in all BMI categories, no more than 50 percent of women gained within the recommended range.

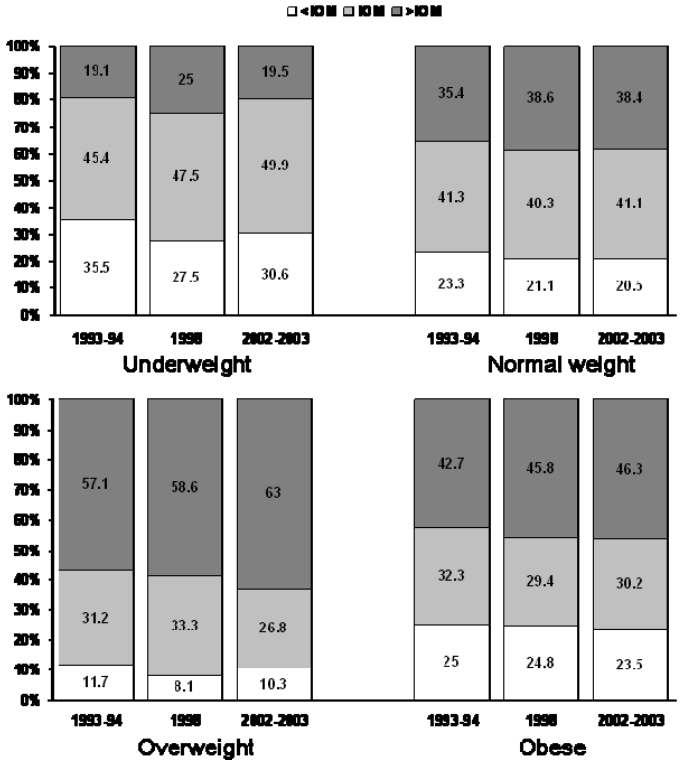

FIGURE 2-7 Distribution of gestational weight gain by prepregnancy BMI category among singleton, term deliveries from 1993 to 2003.
NOTE: IOM BMI categories were used (underweight (lean), <19.8 kg/m^2; normal, 19.8 to 26.0 kg/m^2; overweight, 26.1 to 29.0 kg/m^2; obese, > 29 kg/m^2).
SOURCE: Information contributed to the committee in consultation with P. Dietz, CDC, Atlanta, Ga., January 2009.

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The other large data source, the PNSS, collects data on low-income women participating in public health programs (predominantly Special Supplemental Nutrition Program for Women, Infants, and Children [WIC]) from 26 states, five tribal governments, and one U.S. territory. For the analyses described below, data on pregravid BMI were used to determine whether weight gains fell above, within, or below the ranges recommended by IOM (1990). In this analysis, the data also were not limited to singleton, term pregnancies. The data from the PNSS show that from 1997 to 2007 in the total population of participating women, the proportion who gained within the range recommended by IOM (1990) changed very little within BMI groups (Figure 2-8).

During the observed period, less than 30 percent of women with BMIs in the normal, overweight, and obese categories gained within the recommended ranges. The percentage of underweight women gaining within the recommended range rose slightly from nearly 36 percent in 1997 to just over 40 percent by 2007, while the percentage gaining below the recommended range declined from 41 percent to 32 percent. Furthermore, by the end of the observation period, approximately 46 percent of normal weight women, 46 percent of obese women, and 59 percent of overweight gained in excess of the recommendations (IOM, 1990).

Similar time trends were observed when the PNSS data were stratified by race or ethnicity. In all racial/ethnic groups, the rates of high weight gains increased, low weight gains decreased, and recommended weight gains varied little (Figure 2-9). Non-Hispanic black women and Hispanic women had similar rates of low weight gain and were more likely than non-Hispanic white women to gain less than the recommended levels. Non-Hispanic white women were most likely to gain weight above the recommendations (IOM, 1990).

Taken together, data from PRAMS and PNSS illustrate that less than half of the women in these populations met the IOM (1990) recommendations for GWG. Importantly, none of the data highlighted here provide information on pattern of weight gain.

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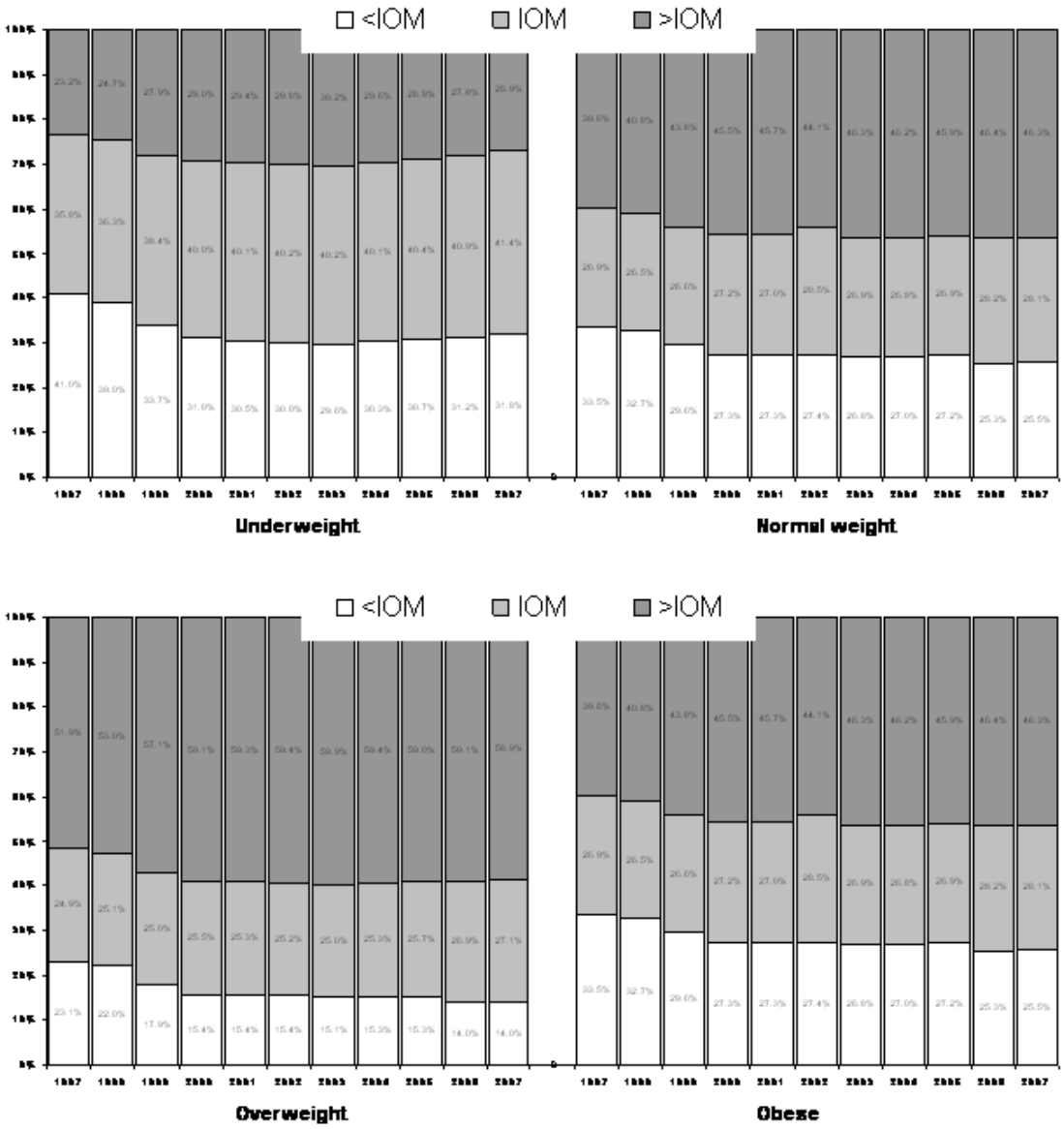

FIGURE 2-8 Distribution of gestational weight gain from 1997 to 2007 by pregravid BMI.
NOTE: BMI based on IOM categories
SOURCES: Personal communication, A. Sharma, CDC, Atlanta, Ga., December 2008; CDC, Pregnancy Nutrition Surveillance System. Available online at http://www.cdc.gov/PEDNSS/pnss_tables/pdf/national_table20.pdf [accessed February 12, 2009].

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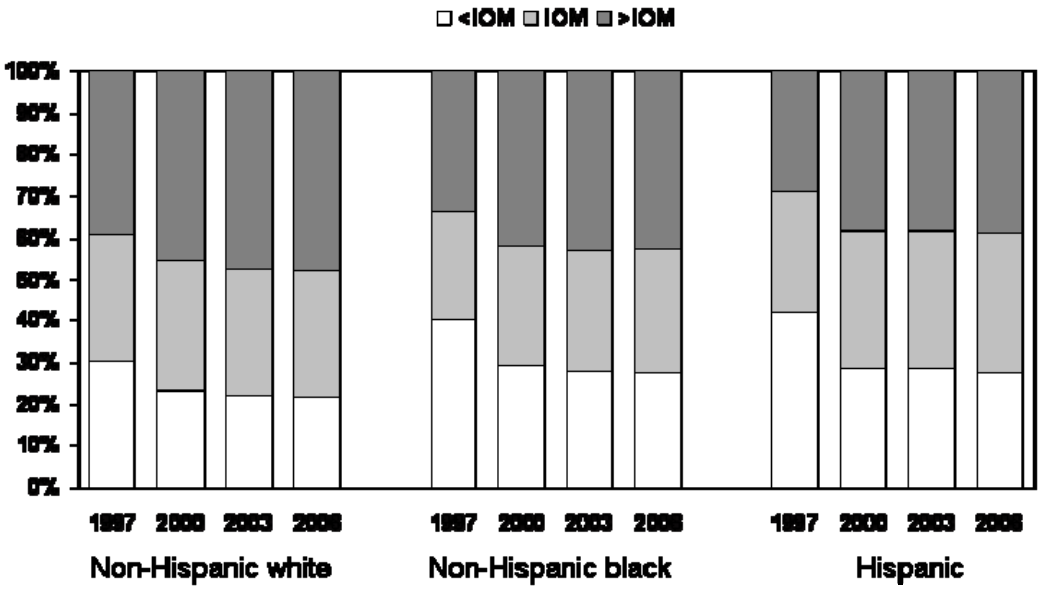

FIGURE 2-9 Distribution of gestational weight gain by race or ethnicity.
SOURCES: Personal communication, A. Sharma, CDC, Atlanta, Ga., December 2008; CDC, Pregnancy Nutrition Surveillance System. Available online at http://www.cdc.gov/PEDNSS/pnss_tables/pdf/national_table20.pdf [accessed February 12, 2009].

POSTPARTUM WEIGHT RETENTION

Postpartum weight status is usually determined by subtracting the prepregnancy weight from a weight obtained at a time after delivery. Postpartum weight status for a population can be represented in a variety of ways, including absolute weight change, percentage who retain a specific amount of weight over the prepregnancy weight (e.g., 10 or 20 pounds), or proportion of women whose BMI category changes from before to after pregnancy. Furthermore, it is important to assess postpartum weight retention according to both prepregnancy body size (e.g., BMI) and adequacy of GWG. Therefore, studies of population trends in maternal postpartum weight retention build upon and extend the data required to assess the adequacy of GWG. Unlike pregnancy, when maternal weight is monitored and routinely recorded in the clinical record, data on maternal postpartum weights are not widely available, particularly for times later in the year after birth.

In addition to the data on GWG provided by PNSS, this surveillance system also collects cross-sectional data on maternal weight at the mother's WIC recertification visit in the postpartum period. From 2004 to 2006, there were more than 1.4 million postpartum records with GWG and prepregnancy BMI in PNSS, but only about 49,000 of these occurred at 6 months postpartum or later and therefore provided useful information on postpartum weight retention in this low-income population sample (personal communication, A. Sharma, CDC, Atlanta, Ga., December 2008). Notably, PNSS data are not nationally representative and the women with postpartum records at > 24 weeks' postpartum were less likely to be non-Hispanic white and more likely to be Hispanic compared to the women with an early postpartum PNSS record.

These data suggest that at 6 months postpartum or later (median [SD], 30.6 [5.1] weeks), the mean postpartum weight retention was 11.8 (15.3) pounds. Approximately half of women retained more than 10 pounds, and one-quarter retained more than 20 pounds (personal

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communication, A. Sharma, CDC, Atlanta, Ga., December 2008). In this sample, black women retained more weight postpartum than white or Hispanic women in every BMI and weight gain category (Figure 2-10). In all BMI categories and racial/ethnic groups, mean postpartum weight retention and the percentage of women retaining > 20 and > 10 pounds increased as GWG category increased (Figure 2-11). Among all women who gained above the range recommended by IOM (1990), mean postpartum weight retention was 15 to 20 pounds (Figure 2-10). When analyses were restricted to data collected at 24 weeks' postpartum or later, more than 60 percent of women in all racial/ethnic groups who gained above the range recommended by IOM (1990) retained > 10 pounds postpartum. More than 40 percent of women who gained excessively retained > 20 pounds (Figure 2-11).

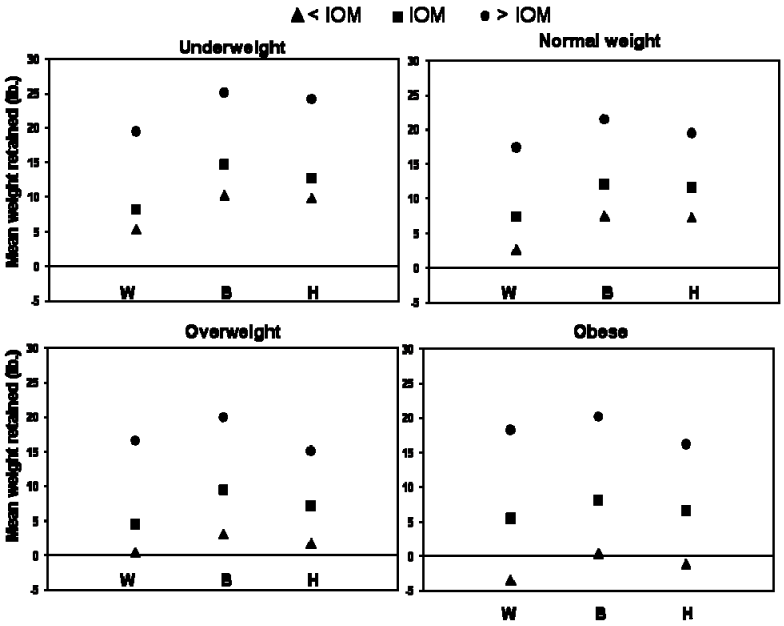

FIGURE 2-10 Mean postpartum weight retention at > 24 weeks' postpartum (mean 30.6 weeks' postpartum) by racial or ethnic group.
NOTE: W = non-Hispanic white; B = non-Hispanic black; H = Hispanic.
SOURCE: Personal communication, A. Sharma, CDC, Atlanta, Ga., December 2008.

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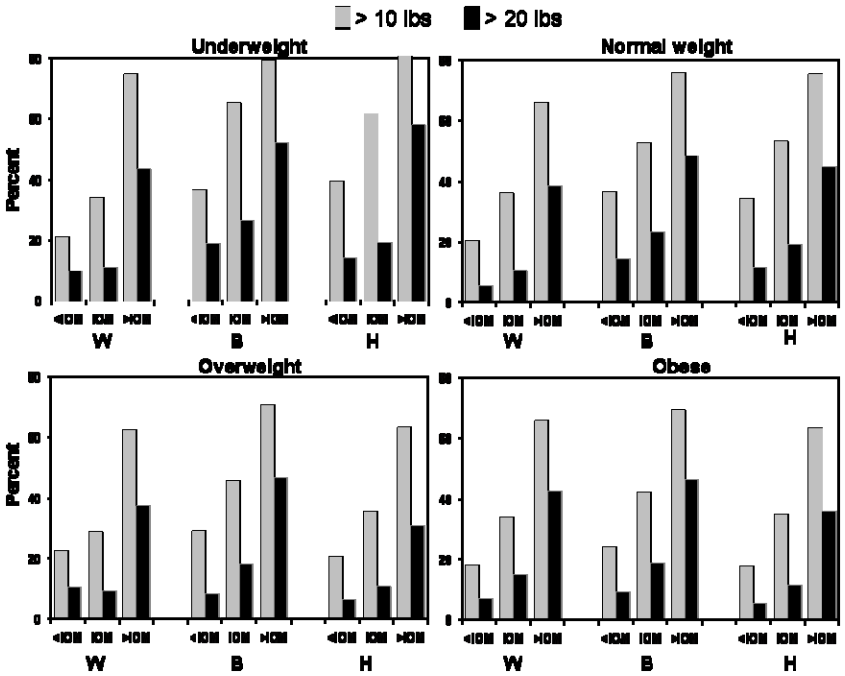

FIGURE 2-11 Percentage of women retaining more than 10 pounds and more than 20 pounds at >24 weeks' postpartum (mean 30.6 weeks' postpartum) by racial or ethnic group.
NOTE: W = non-Hispanic white; B = non-Hispanic black; H = Hispanic.
SOURCE: Personal communication, A. Sharma, CDC, Atlanta, Ga., December 2008.

The Infant Feeding Practices Study II (IFPS II) is a federally sponsored longitudinal study of approximately 4,000 mother-infant pairs that included questions about postpartum weight retention. Respondents were more likely to be non-Hispanic white and to have higher education and lower parity than the general U.S. population. At 2.0-4.9 months postpartum, one-third of women retained > 10 pounds and 12 percent retained > 20 pounds. At 11-13.9 months, only 24 percent of women retained > 10 pounds, but 12 percent still retained > 20 pounds (derived from IFPS II. Available online: http://www.cdc.gov/ifps/questionnaires.htm [accessed April 28, 2009]). In all BMI categories and at each postpartum visit, mean postpartum weight retention and the percentage of women retaining > 20 and > 10 pounds increased as GWG category increased (Figure 2-12). For normal weight and underweight women, weight retention decreased as time postpartum increased in all weight gain categories (classified according to IOM, 1990). Normal weight women who gained above the range recommended by IOM (1990), however, had a decrease in mean postpartum weight through 39 weeks' postpartum, but had an increase in their mean postpartum weight at 55 weeks (Figure 2-13). For overweight and obese women who gained above the recommended range, mean postpartum weight decreased as postpartum time increased, while obese women who gained less than the range recommended by IOM (1990) gained weight across the postpartum period. Importantly, obese women who gained within or less than the recommended range maintained a postpartum weight below their prepregnancy weight.

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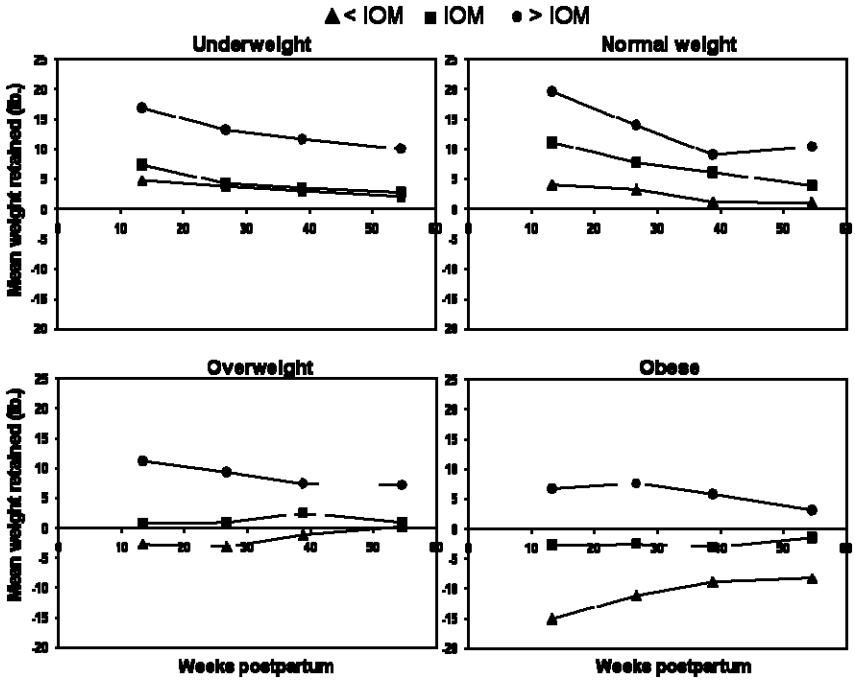

FIGURE 2-12 Mean postpartum weight retention by weight gain category (IOM, 1990) and prepregnancy BMI category across four postpartum visits in the IFPS II study. SOURCE: Derived from IFPS II. Available online: http://www.cdc.gov/ifps/questionnaires.htm [accessed April 28, 2009].

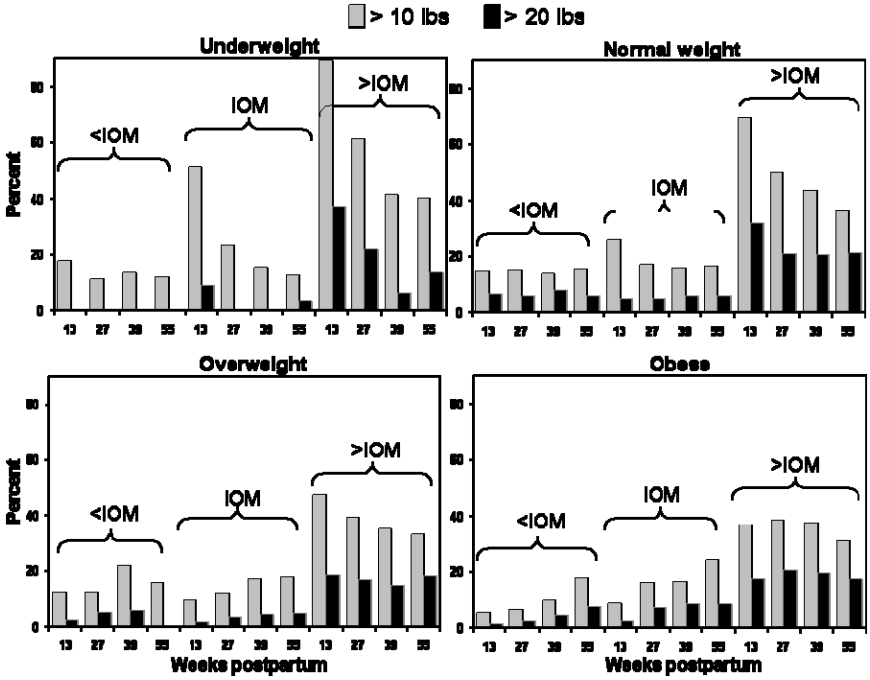

FIGURE 2-13 Percentage of women retaining greater than 10 pounds and greater than 20 pounds at 13 and 54 weeks' postpartum by weight gain category (IOM, 1990) and prepregnancy BMI category (IFPS II study).
SOURCE: Derived from IFPS II. Available online: http://www.cdc.gov/ifps/questionnaires.htm [accessed April 28, 2009].

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In summary, taken together, data from both the PNSS and the IFPS II suggest that gaining above the range recommended by IOM (1990) is associated with excess maternal weight retention postpartum, regardless of prepregnancy BMI. The data from the IFPS II highlight that for most women, weight retention declines as time postpartum increases. However, postpartum weight retention remains a problem for a large proportion of mothers, even at one year after birth. These data also show that obese women who gained within or below the recommended ranges experienced a net loss in weight from their prepregnancy weight. However, for those who gained below their recommended range, the more time that passed after the birth, the more they experienced a net increase in weight and approached their prepregnancy weight. The racially diverse PNSS suggests that among low-income women, black women retain more weight than white or Hispanic women regardless of their prepregnancy weight or GWG category. Compared with the IFPS II, which is a higher-income sample, the low-income women in PNSS retained more weight.

SOCIODEMOGRAPHIC CHARACTERISTICS OF MOTHERS

Since 1990, there has been an increase in the racial and ethnic diversity of U.S. births (Table 2-5). A greater proportion of infants in 2005 were born to nonwhite mothers, with the largest increase in births from Hispanic mothers. Childbearing by unmarried mothers sharply increased in this 15-year period to a record high of 36.9 percent. More mothers attained high levels of education; in 2005, more than one-quarter of mothers had 16 years or more of education. The proportion of births for mothers 35 years and older also increased substantially in this interval. Although the teenage birth rate had been steadily declining since 1991, preliminary data from 2006 suggest that the overall birth rate for teenagers rose 3 percent to 41.9 births per 1,000 females 15-19 years of age. Teenage mothers 10-14 years of age were the only group that did not experience an increase in birth rate during this time. Finally, the proportion of mothers who reported any smoking during pregnancy declined by about 50 percent over the rates reported prior to 1990 (CDC, 2004).

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TABLE 2-5 Distribution of Characteristics of Births in the United States, 1990 and 2005

	1990	2005
Maternal Race or Ethnicity (percentage of live births)[a]		
Non-Hispanic white	64.63	55.27
Non-Hispanic black	16.28	14.15
American Indian or Alaska Native	0.96	1.09
Asian or Pacific Islander	3.49	5.60
Hispanic	14.64	23.89
Total	100.00	100.00
Marital Status (percentage of live births)		
Married	71.98	63.10
Unmarried	28.02	36.90
Total	100.00	100.00
Education (percentage of live births)[b]		
0-8 years	6.39	6.19
9-11 years	17.44	14.74
12 years	38.37	29.80
13-15 years	20.32	21.47
16 years or more	17.48	27.80
Total	100.00	100.00
Maternal Age (percentage of live births)		
<15 years	0.28	0.16
15-17 years	4.41	3.22
18-19 years	8.14	6.80
20-24 years	26.30	25.14
25-29 years	30.71	27.34
30-34 years	21.31	22.97
35-39 years	7.64	11.68
40-44 years	1.17	2.53
45-49 years	0.04	0.15
50-54	NA	0.01
Total	100.00	100.00
Maternal Smoking (percentage of live births)	20.30	10.70

NOTE: NA = not available.

[a]Reflects percentage of total number of live births by race as presented in the table.
[b]Reflects percentage of total number of live births by education as presented in the table.

SOURCES: CDC, 2004; NCHS, 2007a; CDC/VitalStats, available online at http://www.cdc.gov/nchs/vitalstats.htm [accessed February 12, 2009].

Lifestyle Characteristics

Dietary Practices

Dietary intake No comprehensive national data is available on dietary intake practices of pregnant women. However, data from other surveys indicates that population-wide, less than 2 percent of females 14-30 years of age and less than 6 percent of females 31-50 years of age met

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the recommended number of combined fruit and vegetable servings in 1999-2000 (Guenther et al., 2006) (Figure 2-14). Additionally, approximately two-thirds of women ages 14-50 years of age did not consume at least 5 servings of fruits and vegetables per day (BRFSS, 2007; Serdula et al., 2004). See Appendix B for additional information on nutritional intake.

No other nationally representative data on dietary intake among pregnant women or women of childbearing age are available. Among the population as a whole ages 19-39 years, total energy intake increased by 18 percent (1,856 to 2,198 kilocalories [kcal] per day) from 1977-1978 to 1994-1996. This included a sharp 58 percent increase in energy from snacks (244 to 387 kcal/d) as well as the proportion of total energy from fast foods and meals eaten at restaurants, including fast-food establishments (Nielsen et al., 2002). In addition, the proportion of energy from soft drinks nearly tripled; energy from fruit drinks doubled, while energy from milk decreased (Nielsen and Popkin, 2004).

From 1994-1996 to 1999-2000, there was little change in overall diet quality as measured by the Healthy Eating Index 2005 (Guenther et al., 2006). American's diets consistently met national recommendations for total grains and meat or beans, but were far below the recommendation for whole grains, dark-green and orange vegetables, and legumes. Intakes of sodium and energy from solid fats, alcoholic beverages, and added sugars were well above national recommendations.

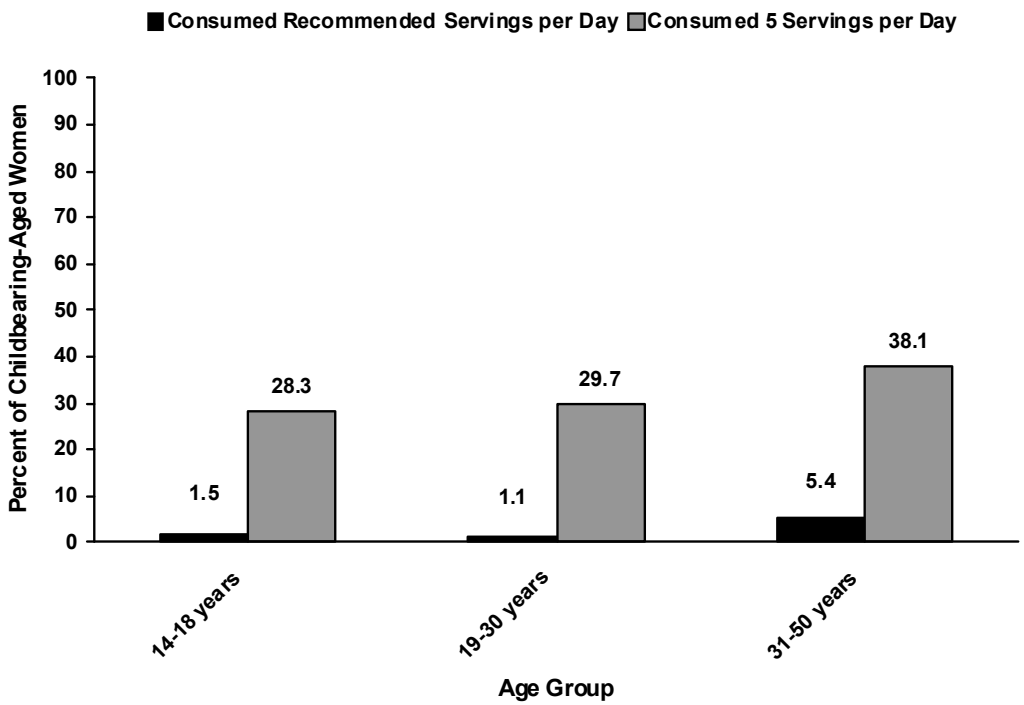

FIGURE 2-14 Percentage of U.S. childbearing-aged women who consumed the recommended number of servings of fruits and vegetables per day and five servings of fruits and vegetables per day.
NOTE: Recommended combined fruit and vegetable servings are eight servings for females age 14-18 and 31-50 and nine servings for females age 19-30.
SOURCE: Guenther et al., 2006.

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Dieting There was a steady rise in the prevalence of attempted weight loss among women of childbearing age from 1989 to 2000 (Serdula et al., 1994, 1999; Bish et al., 2005). In 2000, 60 and 70 percent of overweight and obese women, respectively, were attempting to lose weight, while 29 percent of women whose BMI was < 25 kg/m^2 also were attempting to lose weight (Bish et al., 2005). Importantly, data from the Behavioral Risk Factors Surveillance System (BRFSS) also suggest an increase in the prevalence of attempted weight loss among women who reported being pregnant. In 1989, 3.6 percent of pregnant women who participated in the BRFSS said that they were attempting to lose weight (Cogswell et al., 1996). This figure doubled to 7.5 percent in 2003 (Bish et al., 2009). Furthermore, in 2003, 34.3 percent of women were trying to maintain their weight, that is, to keep from gaining weight (Bish et al., 2009).

Food insecurity Food insecurity is defined as "whenever the availability of nutritionally adequate and safe food or the ability to acquire acceptable foods in socially acceptable ways is limited or uncertain." In 2006, 10.9 percent of U.S. households (12.6 million) had either low food security (6.9 percent) or very low food security (4.0 percent). It is difficult to obtain a nutrient-dense diet in an environment of food insecurity, and this has important implications for GWG (USDA; available online at http://www.ers.usda.gov/Publications/ERR49/ERR49.pdf [accessed April 21, 2009]).

Pregnancy and lactation require modest increases in energy but greater increases in vitamin and mineral intake. For pregnant women to gain an appropriate amount of weight and meet their nutrient requirements, dietary changes to promote high nutrient density and appropriate energy intake is required. Unfortunately, the lack of nationally representative data on pregnant and postpartum women limits understanding of dietary trends among this important population subgroup.

Physical Activity

*Healthy People 2010 (*DHHS, 2000) and the 2008 Physical Activity Guidelines (DHHS, 2008) provide recommended levels of physical activity and emphasize that inactivity has adverse health consequences. Data from the BRFSS indicate that although the proportion of women of childbearing age who reported no recreational physical activity decreased between 1994 and 2004, one in five women aged 18-29 years of age and almost a quarter of those in their thirties and forties reported no physical activity in 2004 (Figure 2-15) (CDC, 2005). Similarly, barely half of women of childbearing age met the guideline in *Healthy People 2010* for aerobic activity in 2005, although the prevalence has increased significantly since 2001 (Figure 2-16) (CDC, 2007).

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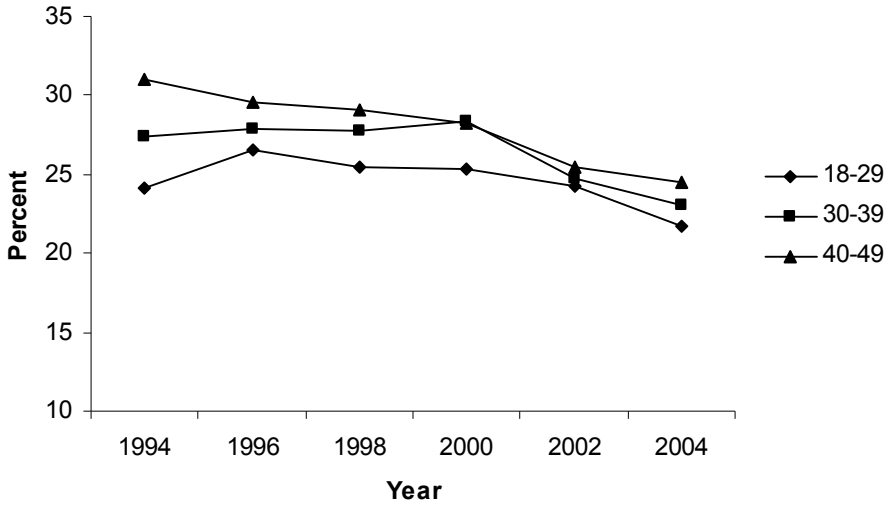

FIGURE 2-15 Trends in leisure-time physical inactivity for women of childbearing age, United States, 1994-2004.
NOTES: Leisure-time physical inactivity defined as a "no" response to the survey question, During the past month, other than your regular job, did you participate in any physical activities or exercise, such as running, calisthenics, golf, gardening, or walking for exercise? The reference time frame for the wording of this survey question was revised in 2001 to "During the past 30 days …" and was changed back to "During he past month …" in 2002. Also, in 2001, the phrase "other than your regular job" was added.
SOURCE: CDC, 2005.

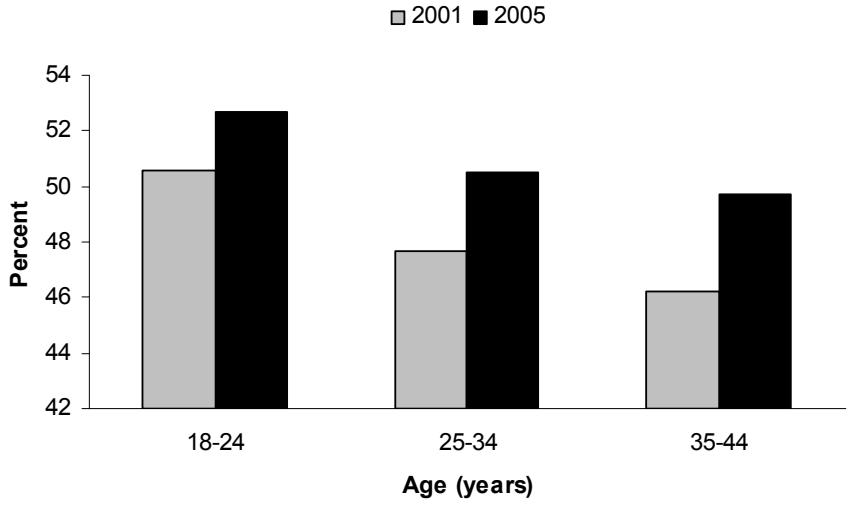

FIGURE 2-16 Trends in estimated percentage of women of childbearing age who reported meeting guidelines for regular physical activity
NOTE: Physical activity is defined as at least 30 minutes of moderate-intensity activity per day on five or more days a week, or at least 20 minutes a day of vigorous-intensity activity on three or more days a week, or both, when not working; an exercise occurrence is defined as 10+ minutes.
SOURCE: CDC, 2007.

In 2000, 15.8 percent of pregnant women met minimum physical activity recommendations (Evenson et al., 2004) and only 6 percent of pregnant women met recommendations for vigorous

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physical activity (Petersen et al., 2005). In these analyses, physical activity varied by maternal race/ethnicity, age, and education; and there was some evidence that physical activity was lower among women who worked outside the home. In 2005, almost half of white, non-Hispanic U.S. women of all ages met the *Healthy People 2010* objective for physical activity; only 36 percent of black, non-Hispanic women, 40 percent of Hispanic women, and 47 percent of other-race women did so (CDC, 2007). Physical activity increased with education, from 37 percent among women who did not graduate from high school to 53.3 percent among college graduates (CDC, 2007).

In summary, a high proportion of women of childbearing age fail to meet current guidelines for physical activity before or during pregnancy. The committee identified only limited data on physical activity or inactivity among pregnant women. The committee identified no data on postpartum mothers or physical activity according to BMI and weight change before, during, and after pregnancy.

Psychological Characteristics

Depression

Changes in appetite and weight are among the diagnostic criteria for major depression (American Psychiatric Association, 1994). In their meta-analysis, Gaynes et al. (2005) estimated that one in seven women will develop depression during pregnancy or after delivery. Although nationally representative data specific to women during and after pregnancy are not available, data for U.S. women of childbearing age illustrate striking increases in the prevalence of major depression from 1991-1992 to 2001-2002 in the total population and among white and black women (Figure 2-17) (Compton et al., 2006). Similar trends were observed among women 30 to 44 years of age, but the rates of major depression were lower than those of women age 18-29 years. Given that more than 10 percent of women of childbearing age may be depressed, screening and intervention for symptoms of depression during pregnancy may be required to achieve better GWG.

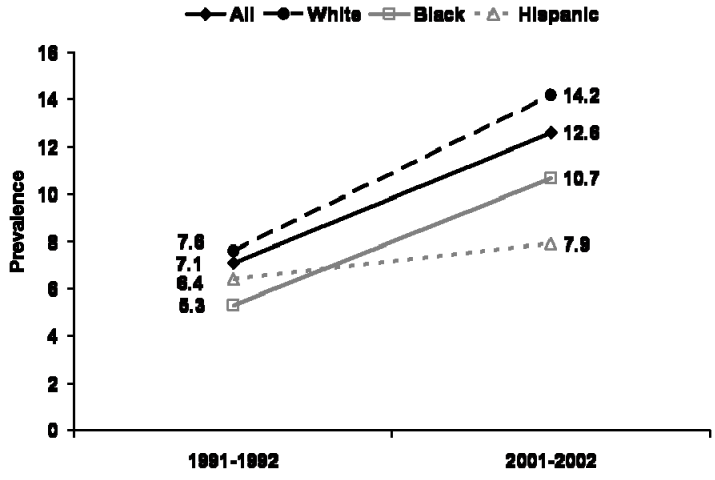

FIGURE 2-17 Prevalence of major depression among women 18-29 years of age in the United States by race or ethnicity, 1991-1992 and 2001-2002.
SOURCE: Compton et al., 2006.

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Other Psychological Characteristics

Other psychological factors that may influence GWG include stress, social support, and attitude toward weight gain (see Chapter 4). The committee did not identify any nationally representative data specific to women during and after pregnancy that were indicative of trends or prevalence of these factors related to GWG.

PREGNANCY OUTCOMES RELATED TO GESTATIONAL WEIGHT GAIN

Gestational Diabetes

Data from birth certificates collected nationally illustrate that there has been a striking increase in the prevalence of diabetes in pregnancy in each age group (Figure 2-18), with the largest increase over time among women in the oldest age group (40 or more). However, the majority of birth certificates did not distinguish between pre-gestational diabetes (diagnosis before the index pregnancy) and gestational diabetes mellitus (GDM; diagnosis during the index pregnancy).

With data from the National Hospital Discharge Survey from 1989 to 2004, Getahun et al. (2008) determined trends in the prevalence of GDM among U.S. women 14 to 45 years of age. GDM increased by 122 percent, from 1.9 percent in 1989-1990 to 4.2 percent in 2003-2004. Furthermore, among women 35 years of age and older, the rate for GDM was highest among black women.

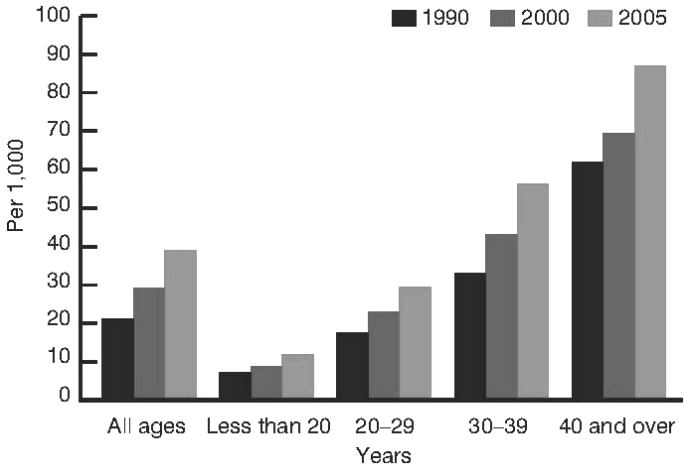

FIGURE 2-18 Diabetes rates by age of mother: United States, 1990, 2000, and 2005.
SOURCE: NCHS, 2007b.

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Preeclampsia and Gestational Hypertension

Wallis et al. (2008) investigated population trends in the incidence rates of pregnancy-induced hypertension (preeclampsia and gestational hypertension [see Appendix A for definitions]) in the United States for 1987-2004 with data from the National Hospital Discharge Survey. The age-adjusted rate of preeclampsia increased 25 percent from 1987-1988 to 2003-2004. Gestational hypertension rates nearly tripled during the same period (Figure 2-19). The authors noted that clinical diagnostic criteria, revised in the 1990s, may have simultaneously caused an exaggerated rise in the rate of gestational hypertension and an attenuated increase in the rate of preeclampsia over the study period. They concluded that the small but consistent elevation in the rate of preeclampsia is a conservative estimate of the true population-level change.

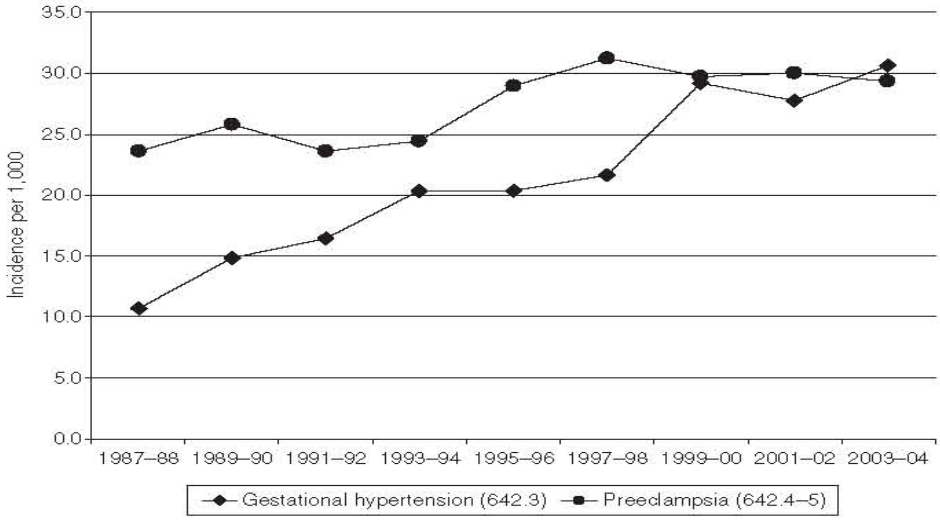

FIGURE 2-19 Age-adjusted incidence of preeclampsia and gestational hypertension per 1,000 deliveries in the United States, 1987-2004.
SOURCE: Wallis et al., 2008. Reprinted by permission from Macmillan Publishers Ltd: Wallis, A. B., A. F. Saftlas, J. Hsia, and H. K. Atrash. 2008. Secular trends in the rates of preeclampsia, eclampsia, and gestational hypertension, United States, 1987-2004. *American Journal of Hypertension* 21(5): 521-526.

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Cesarean Delivery

The rate of total cesarean deliveries in the United States increased almost fivefold between 1970 and 1988 and then declined to 20.7 percent in 1996 (Figure 2-20). Since then, the rate increased 50 percent to 31.1 percent—the highest rate ever recorded—in 2006 (Menacker et al., 2006; MacDorman et al., 2008). Primary cesareans (births to women with no previous cesarean delivery) mirror the pattern for total cesareans, while vaginal birth after a previous cesarean (VBAC) increased beginning in the mid-1980s, peaked in 1996, and then has declined since that time (MacDorman et al., 2008). An increase in primary cesarean deliveries appears to be the result of changes in obstetric practice rather than in medical risk profiles or maternal request (Menacker et al., 2006; MacDorman et al., 2008). However, a recent meta-analysis concluded that maternal obesity is associated with increased risk of cesarean delivery (Chu et al., 2007). The expanded availability of BMI data in U.S. birth certificates since 2003 will allow future researchers to more clearly understand relationships between maternal prepregnancy BMI, GWG, and cesarean deliveries in the United States.

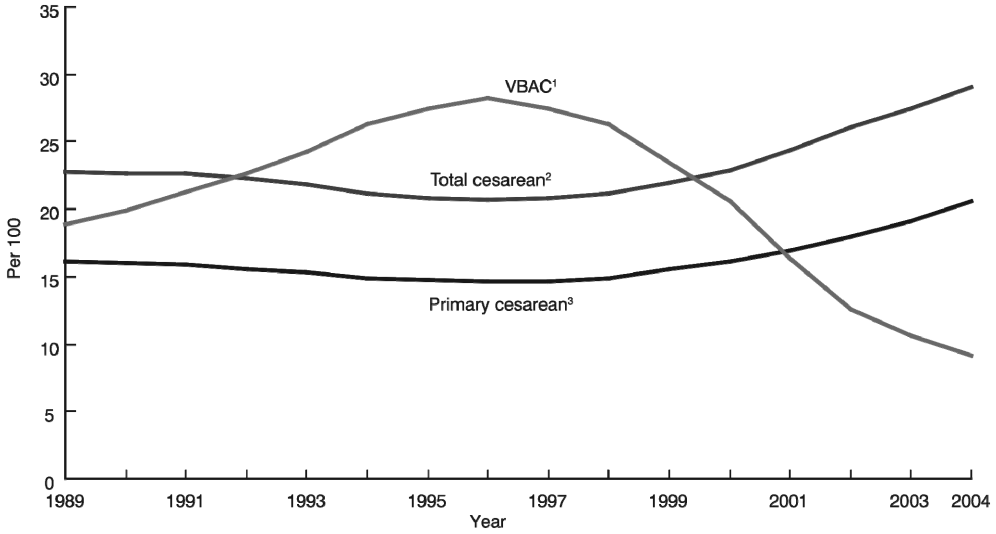

FIGURE 2-20 Total and primary cesarean rate, 1970-2004, and VBAC, 1985-2004.
SOURCE: NCHS, 2005.

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Maternal Mortality

The crude maternal mortality rate (deaths per 100,000) steadily decreased in the United States from 83.3 in 1950 to 8.2 in 1990; increased rates since 2000 are believed to be due to changes in coding and increased surveillance (Hoyert, 2007; available online at http://mchb.hrsa.gov/whusa08/hstat/mh/pages/237mm.html [accessed January 14, 2009]). Nonetheless, in 2005, the age-adjusted maternal mortality rate was 9.6 for non-Hispanic white, 8.2 for Hispanic or Latina, and 31.7 for non-Hispanic black mothers, indicating an important disparity by race. Furthermore, among women 35 years and older the mortality rate in 2005 was 28.9 for white women and 112.8 for black women (NCHS, 2007b). A recent case-control study based on a statewide Pregnancy-Associated Mortality Review in Florida reported that maternal mortality was increased three-, four-, and fivefold with class I (BMI 30-34.9), class II (BMI 35-39.9), and class III obesity (BMI ≥ 40), respectively. Given the rising rates of obesity in the population, additional studies on obesity and maternal mortality are needed (Thompson et al., 2005).

Infant Mortality

The infant mortality rate (deaths of infants less than 1 year of age per 1,000 live births) in the United States was 6.71 in 2005 (MacDorman et al., 2008). The dramatic decrease in infant mortality that occurred during the last half of the twentieth century has slowed since 2000 (Figure 2-21), and the United States has fallen behind many other developed countries in infant survival (NCHS, 2007b). Trends are similar for other measures, including early and late neonatal mortality and post-neonatal mortality, although perinatal mortality has continued to decrease steadily since 1990 (Martin et al., 2008).

Disparities in infant mortality according to maternal racial or ethnic group continue (Figure 2-22). In 2005, the infant mortality rate for non-Hispanic black mothers was three times higher than for Cuban mothers, who had the lowest rate; Puerto Rican and American Indian or Alaskan Native mothers also had rates above the national average.

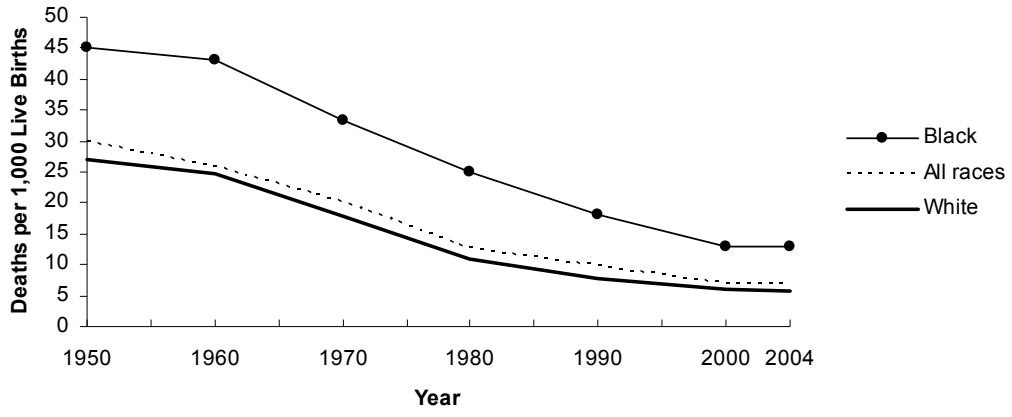

FIGURE 2-21 Infant mortality rates in the United States, 1950 through 2004, by race.
SOURCE: NCHS, 2007b.

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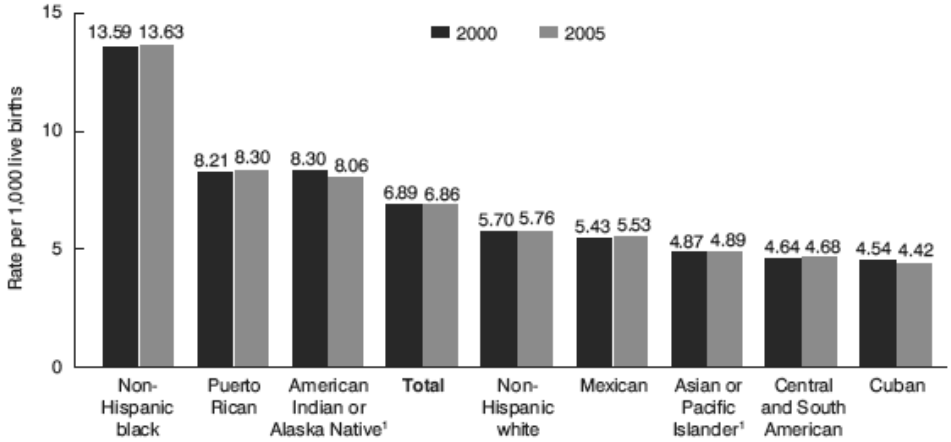

FIGURE 2-22 Infant mortality rates by race or ethnicity, 2000 and 2005.
SOURCE: NCHS, available online at http://www.cdc.gov/nchs/data/databriefs/db09.htm [accessed February 12, 2009].

Birth Weight

There is a strong association between very low birth weight (due to preterm delivery or extreme fetal growth restriction) and infant mortality that decreases as birth weight increases until it reaches about 4,500 g, when there is a slight increase in infant mortality due to problems associated with macrosomia (Mathews and MacDorman, 2007). Although rates of infant morality have decreased over time, the reverse J-shape of this relationship has not changed.

The proportion of small infants increased and large infants decreased among all reported births between 1990 and 2005 (Figure 2-23). This downward shift in the overall distribution of birth weight is attributable in part to an increase in multiple births, but the pattern is similar for singleton births. Other possible explanations for these trends in birth weight include a greater prevalence of older mothers, who tend to have more complications of pregnancy, as well as increased use of assisted reproductive technology and obstetrical procedures, including labor induction and elective cesarean deliveries.

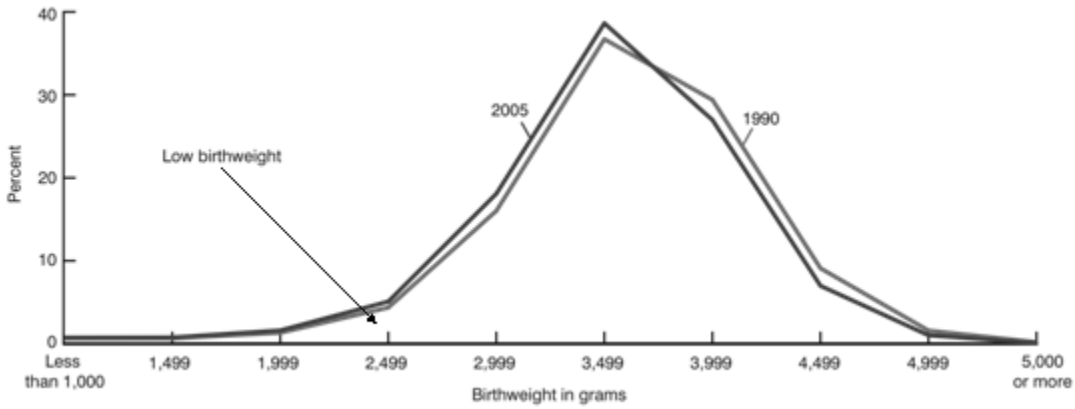

FIGURE 2-23 Percentage distribution of births by birth weight, United States, 1990 and 2005.
SOURCE: NCHS, 2007a.

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Rates for low birth weight and very low birth weight increased in the United States between 1990 and 2005, when the overall rate of low birth weight among singletons was 6.41 percent and the overall rate of very low birth weight was 1.14 percent. The lowest rates of low birth weight are among Hispanics and white infants, the highest among black infants; Native American, and Asian-Pacific Islander infants fall in between (Figure 2-24). Low birth weight also varies by maternal age, with greater prevalence among women < 20 and > 40 years of age (Martin et al., 2008).

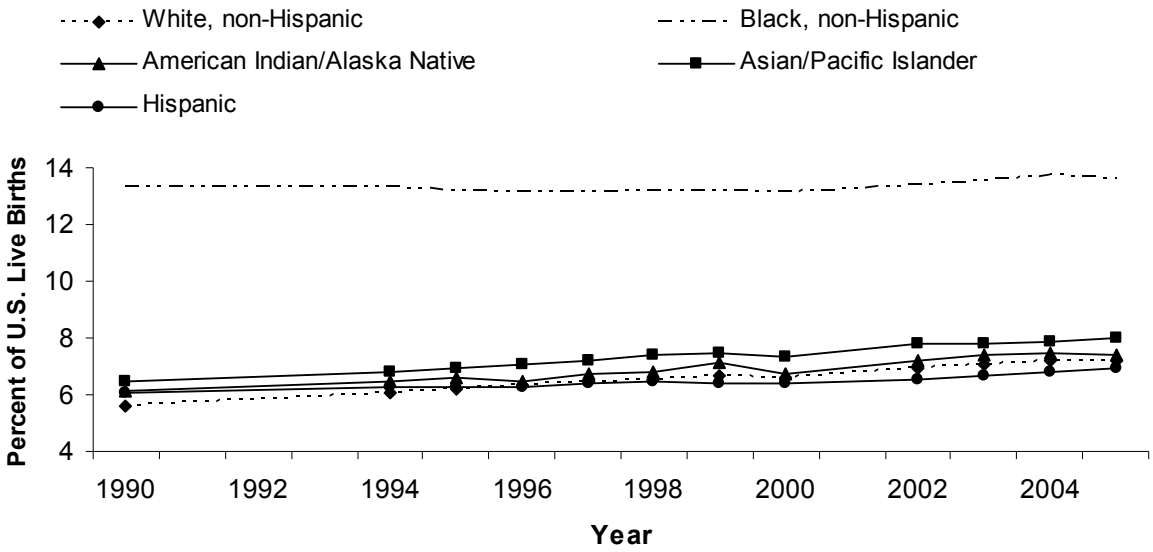

FIGURE 2-24 Trends in low birth weight of live-born singleton infants in the United States from 1990 through 2005, by race and ethnic background.
NOTE: Low birth weight is defined as less than 2,500 g.
SOURCES: NCHS, 2002; 2007a.

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Small-for-Gestational Age Births

Small-for-gestational age (SGA) is used as a proxy to examine poor fetal growth (see Chapter 4) but can also include infants who are small but healthy due to their familial genetic background (Jaquet et al., 2005; Svensson et al, 2006). SGA rates for all groups decreased between 1990 and 2000 and then increased in 2005 (Table 2-6). Rates among non-Hispanic black infants were almost twice as high as those of white infants and were not appreciably different by gender. However, Hispanic and Asian female infants had lower SGA rates than males.

TABLE 2-6 Estimates of SGA by Sex, Race or Ethnicity, and Year: United States

	1990	1995	2000	2005
Males				
Total	10.5	10.5	10.2	10.7
Non-Hispanic white	8.7	8.8	8.4	8.7
Non-Hispanic black	17.1	16.9	16.3	16.8
Hispanic	10.7	10.6	10.4	10.7
White	9.1	9.2	8.9	9.3
Black	17.0	16.8	16.2	16.5
American Indian-Alaska Native	9.9	9.7	9.4	9.8
Asian-Pacific Islander	14.0	14.4	13.9	14.5
Females				
Total	10.7	10.5	10.1	10.5
Non-Hispanic white	9.0	8.9	8.4	8.8
Non-Hispanic black	17.3	16.9	16.2	16.7
Hispanic	10.4	10.2	9.8	10.1
White	9.3	9.2	8.7	9.1
Black	17.2	16.8	16.1	16.3
American Indian-Alaska Native	9.3	9.5	9.0	9.3
Asian-Pacific Islander	13.2	13.7	13.2	13.6

NOTE: Singleton births only.
SOURCE: CDC/NCHS, National Vital Statistics System, available online at http://www.cdc.gov/nchs/VitalStats.htm [accessed February 12, 2009].

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Large-for-Gestational Age Birth

The proportion of infants born large-for-gestational age (LGA) decreased monotonically between 1990 and 2005 for males and females within all racial-ethnic groups, although American Indians-Alaska Natives had the highest rates (Table 2-7). Reasons for this decrease are not known but could include routine testing for GDM and increased cesarean deliveries performed at earlier gestational ages (Menacker et al., 2006).

TABLE 2-7 Estimates of LGA by Sex, Race or Ethnicity, and Year: United States

	1990	1995	2000	2005
Males				
Total	11.1	10.7	10.7	9.4
Non-Hispanic white	12.4	12.1	12.2	10.7
Non-Hispanic black	7.5	7.2	6.9	6.2
Hispanic	10.2	9.8	9.9	8.9
White	12.0	11.6	11.6	10.2
Black	7.5	7.2	7.0	6.4
American Indian-Alaska Native	13.8	13.6	13.2	12.0
Asian-Pacific Islander	6.5	6.2	6.1	5.4
Females				
Total	10.5	10.3	10.4	9.1
Non-Hispanic white	11.7	11.6	12.1	10.2
Non-Hispanic black	7.1	6.8	6.8	5.9
Hispanic	9.9	9.7	10.0	9.0
White	11.3	11.2	11.3	9.8
Black	7.1	6.8	6.8	6.1
American Indian-Alaska Native	14.3	13.5	13.5	12.8
Asian-Pacific Islander	6.8	6.6	6.4	5.7

NOTE: Singleton births only.
SOURCE: CDC/NCHS, National Vital Statistics System, available online at http://www.cdc.gov/nchs/VitalStats.htm [accessed February 12, 2009].

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Preterm Birth

In 2005, 12.5 percent of all births were delivered preterm. The preterm birth rate has increased 20 percent since 1990 and 9 percent since 2000 (Figure 2-25). The greatest increase has been among late preterm births, those occurring at 34-36 weeks' gestation, which have climbed 25 percent since 1990. The preterm birth rate for singleton gestations increased 13 percent from 1990 to 2005, again with late preterm births accounting for a majority of the increase. An increase in the rates of cesarean deliveries and induced births contributes to but does not completely explain this trend in late preterm births (March of Dimes, available online at http://www.marchofdimes.com/files/MP_Late_Preterm_Birth-Every_Week_Matters_3-24-06.pdf [accessed January 14, 2009]).

There is a striking racial disparity in the rate of preterm birth (Figure 2-26). In the past 15 years, non-Hispanic black women were about twice as likely as non-Hispanic white women to deliver before 37 weeks' gestation. Since 1990, the preterm birth rate increased 38 percent for non-Hispanic whites and 10 percent for Hispanic births. There was a decrease in preterm births among non-Hispanic black mothers through most of the 1990s. However, the preterm birth rate is up 12 percent since 2000.

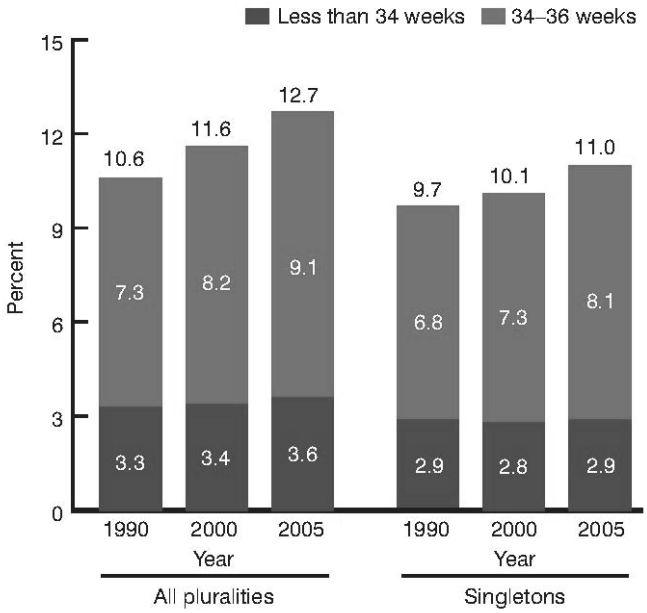

FIGURE 2-25 Preterm birth rates for all births and for singletons only: United States, 1990, 2000, and 2005
SOURCE: NCHS, 2007a.

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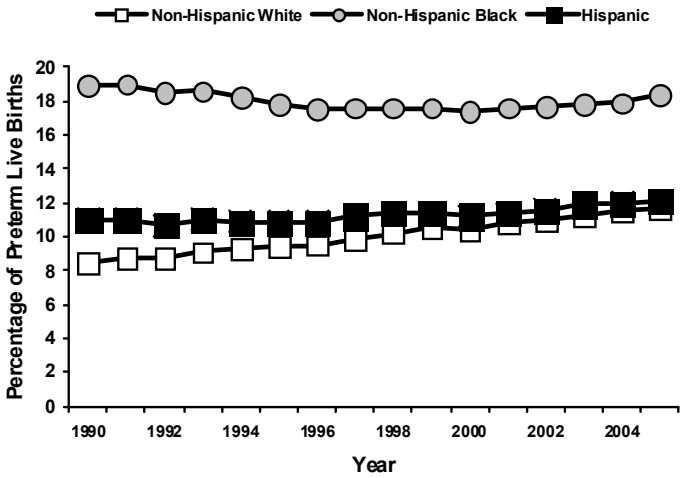

FIGURE 2-26 Trends in preterm live births in the United States by race, 1990 to 2005.
NOTE: Preterm is defined as an infant born before 37 weeks of gestation.
SOURCES: NCHS, 2007a.

Breastfeeding

Analysis of data from the Ross Laboratories Mothers Survey, a large, national survey (Ryan et al., 2002), shows that the rates of breastfeeding initiation (in-hospital) and breastfeeding at six months rose by 16 percent and 14 percent, respectively, in the 1990s. In 2001, rates were at their highest point in 40 years (Figures 2-27 and 2-28). Recent data from the National Immunization Survey, a population-based survey conducted by the CDC, shows that these rates continued to rise from 2000 to 2004.

There are remarkable disparities in rates of breastfeeding. Mothers who were white or Hispanic, older, college-educated, and not enrolled in WIC were significantly more likely to breastfeed and exclusively breastfeed in the hospital and at six months (Ryan et al., 2002).

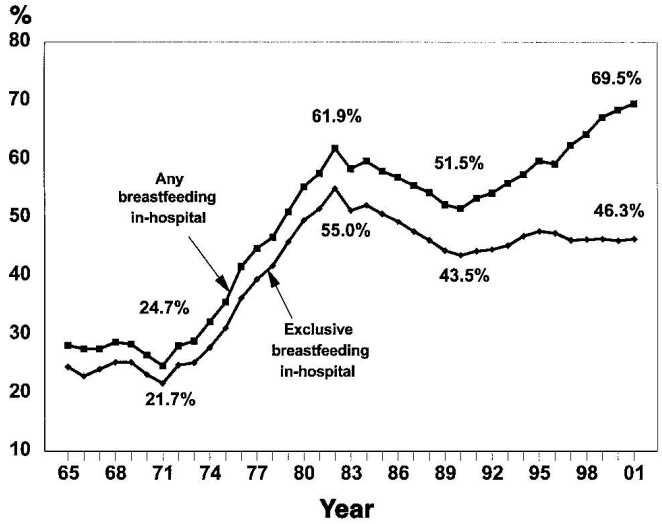

FIGURE 2-27 In-hospital breastfeeding and exclusive breastfeeding rates: 1965-2001.
SOURCE: Ryan et al., 2002. Reproduced with permission from *Pediatrics*, Vol. 110, pages 1103-1109. Copyright © 2002 by the AAP.

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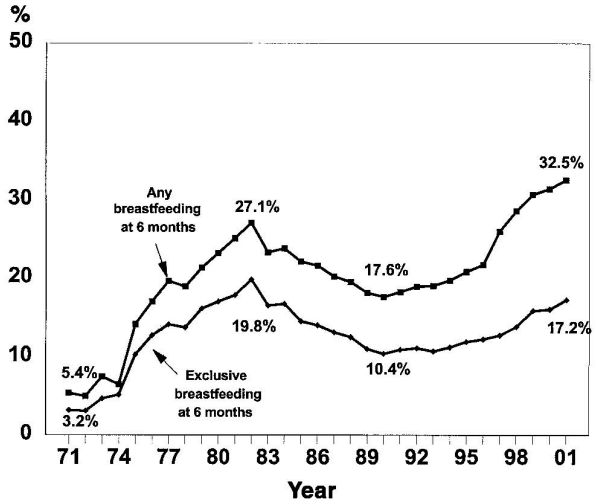

FIGURE 2-28 Breastfeeding and exclusive breastfeeding rates at 6 months of age: 1971-2001.
SOURCE: Ryan et al., 2002. Reproduced with permission from *Pediatrics*, Vol. 110, pages 1103-1109. Copyright © 2002 by the AAP.

Childhood Obesity

Nationally representative data show continuous increases in obesity (BMI ≥ 95th percentile) among American school-aged children and adolescents from 1980 to the present (http://www.cdc.gov/nccdphp/dnpa/obesity/childhood/prevalence.htm; Accessed April 15, 2009) (Figure 2-29). Recent data suggest that this trend may be slowing (Ogden et al., 2008). Population estimates from 2003 through 2006 suggest that almost a third of 2-19 year olds were at or above the 85th BMI percentile for sex and age (Ogden et al., 2008). Of these, 16 percent were above the 95th percentile, well above the *Healthy People 2010* goal of 5 percent, and 11.3 percent were above 97th percentile (rates of high BMI varied by age and race-ethnicity). Non-Hispanic black adolescents have a dramatically greater prevalence of overweight compared to non-Hispanic whites; Mexican American girls also have somewhat higher rates (Table 2-8).

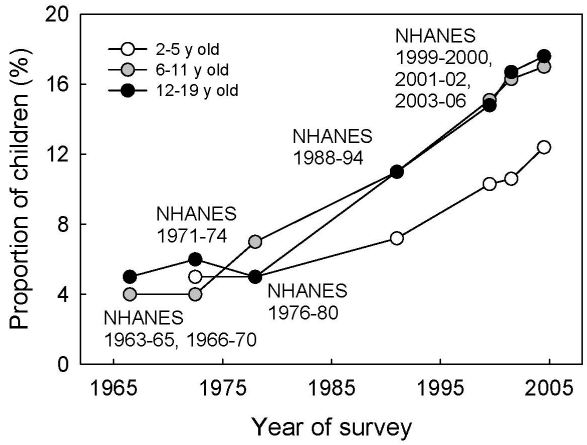

FIGURE 2-29 Prevalence of obesity (≥95th percentile) among children and adolescents, United States, 1963-2006.
SOURCES: Ogden et al., 2006; Ogden et al., 2008.

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TABLE 2-8 Prevalence of High BMI by Age Among U.S. Adolescent Girls (12-19 years of age), 2003-2006

BMI Percentile of CDC Growth Charts	Non-Hispanic Black %(SE)	Mexican American %(SE)	Non-Hispanic White %(SE)
≥ 85th	44.5 (1.5)	37.1 (1.9)	31.7 (1.9)
≥ 95th	27.7 (1.9)	19.9 (1.4)	14.5 (2.0)
≥ 97th	19.6 (1.5)	14.1 (1.3)	9.1 (1.6)

SOURCE: Odgen et al., 2008.

FINDINGS AND RECOMMENDATIONS

Findings

1. Since the release of the weight gain recommendations of IOM (1990):

    - There has been a striking increase in the prevalence of maternal overweight and obesity, particularly among black, Hispanic, and older women;
    - There has been an increase in the racial and ethnic diversity of U.S. births, as well as a rise in the proportion of older and unmarried mothers and a decrease in the proportion of teenaged mothers; and
    - Low (< 16-pound) and high (> 40-pound) GWG has become more common.

2. American women of childbearing age are far from meeting national goals for dietary intake and physical activity, yet there is a dearth of nationally representative data on dietary intake, dieting practices and food insecurity, among women of childbearing age in general and among pregnant women in particular.
3. About half of reproductive-aged American women are trying to lose weight, and another one-third of pregnant women may be attempting to maintain their weight. The prevalence of attempted weight loss during pregnancy doubled in the past 20 years.
4. Rates of preterm birth, GDM, and hypertensive disorders of pregnancy are increasing. The rise in cesarean births and the decline in LGA births appear to result from medical practice patterns and social factors.
5. In the past 10 years, improvements that were observed during the twentieth century in maternal mortality and poor infant outcomes (mortality and low birth weight) have declined or ceased.
6. There are racial and ethnic disparities in nearly all weight-related predictors and outcomes reviewed.
7. Currently available data sources are inadequate for studying national trends in GWG. Even after the IOM (1990) report called for more sophisticated analyses, major gaps in GWG surveillance remain; specifically, data on prepregnancy weight and height, reliance on self-reported weight gain, and nationally representative sources are lacking.
8. Gestational weight gain in excess of the recommended range for BMI is associated with significant postpartum weight retention.
9. Major gaps in surveillance of postpartum weight exist. Notably, most national studies lack data on postpartum weight and/or the variables needed for its proper interpretation

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(namely, prepregnancy height and weight, GWG, dietary intake, physical activity, and breastfeeding status).

Action Recommendations

Action Recommendation 2-1: The committee recommends that the Department of Health and Human Services conduct routine surveillance of GWG and postpartum weight retention on a nationally representative sample of women and report the results by prepregnancy BMI (including all classes of obesity), age, racial/ethnic group, and socioeconomic status.

Action Recommendation 2-2: The committee recommends that all states adopt the revised version of the birth certificate, which includes fields for maternal prepregnancy weight, height, weight at delivery, and gestational age at the last measured weight. In addition, all states should strive for 100 percent completion of these fields on birth certificates and collaborate to share data, thereby allowing a complete national picture as well as regional snapshots.

Supporting Actions

1. At the first prenatal visit, health care providers should record weight at last menstrual period and maternal height without shoes. Gestational weight gain should be based on measured weights (in light clothing and no shoes) abstracted from prenatal records. Gestational age at the last recorded weight should be documented, preferably through an early ultrasound, to properly evaluate adequacy of weight gain. To aid in data analysis, all data should be collected in a continuous form rather than categorically.
2. As part of maternal weight surveillance, health-care providers should document the prevalence of obesity grades I, II, and II rather than categorize women into one obesity group (BMI > 30 kg/m^2).

Areas for Additional Investigation

The committee identified the following areas for further investigation to support its research recommendations:

- The research community should conduct future monitoring of GWG; and
- Federal agencies should standardize the use of the WHO BMI cutoff points in all data collection relevant to monitoring weight gain in pregnancy.

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Websites:
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http://www.cdc.gov/PEDNSS/pnss_tables/pdf/national_table20.pdf
http://www.cdc.gov/nchs/vitalstats.htm
http://www.ers.usda.gov/Publications/ERR49/ERR49.pdf
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http://www.cdc.gov/nchs/data/databriefs/db09.htm
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3

Composition and Components of Gestational Weight Gain: Physiology and Metabolism

Gestational weight gain (GWG) is a unique and complex biological phenomenon that supports the functions of growth and development of the fetus. Gestational weight gain is influenced not only by changes in maternal physiology and metabolism, but also by placental metabolism (Figure 3-1). The placenta functions as an endocrine organ, a barrier, and a transporter of substances between maternal and fetal circulation. Changes in maternal homeostasis can modify placental structure and function and thus impact fetal growth rate. Conversely, placental function may influence maternal metabolism through alterations in insulin sensitivity and systemic inflammation and, thus influence GWG.

This chapter provides relevant background material on normal physiologic and metabolic changes that occur during pregnancy and are related to GWG. The discussion begins with a review of total and pattern of GWG in singleton, twin, and triplet pregnancies. Next, the unique chemical composition and accretion rates of maternal, placental, and fetal components of GWG are presented, followed by discussions of fundamental biology of fetal and placental growth and fetal-placental physiology underlying GWG. Lastly, pathophysiologic conditions that may adversely affect GWG are reviewed to provide a foundation for understanding changes in body weight and composition during pregnancy.

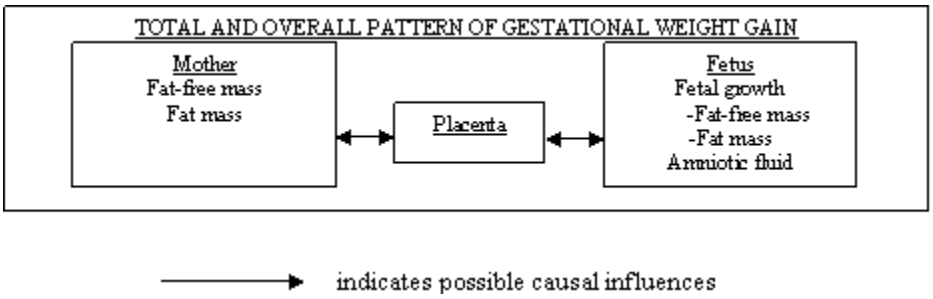

FIGURE 3-1 Schematic summary of components of gestational weight gain.

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TOTAL AND PATTERN OF GESTATIONAL WEIGHT GAIN

Total Gestational Weight Gain

The total amount of weight gained in normal-term pregnancies varies considerably among women. Nevertheless, some generalizations can be made regarding tendencies and patterns of GWG in singleton and multiple pregnancies.

Singleton Pregnancies

An examination of studies published in the United States from 1985 to the present indicate that the mean total GWG of normal weight adult women giving birth to term infants ranged from a low of 10.0 to a high of 16.7 kg (Appendix C [Tables C-1A and C-1B] contains a tabular summary of the studies examined by the committee). Among adolescents, in general, GWG tended to be higher compared with adult women (means ranged from 14.6 to 18.0 kg in the studies examined). A consistent finding across studies was an inverse relationship between GWG and pregravid body mass index (BMI). Figure 3-2 illustrates a similar relationship with data derived from Abrams et al. (1986).

Since the release of the report, *Nutrition During Pregnancy* (IOM, 1990) and its guidelines for GWG, a number of studies have examined GWG among overweight and obese women. Bianco et al. (1998) found that the mean GWG for 613 obese (BMI > 35) women averaged 9.1 ± 7.4 kg. Thirteen percent of the women, however, gained more than 16 kg, and 9 percent either lost or failed to gain weight. In a cohort study using birth certificate data from 120,251 obese women in Missouri, 18, 30, and 40 percent of the women gained < 6.8 kg in obese classes I, II, and III, respectively. The amount of total gain associated with minimal risk for preeclampsia, caesarean delivery, large for gestational age (LGA), and small for gestational age (SGA) outcomes was 4.6-11.4, and 0-4.1 for class I and II obesity, respectively; and weight loss of 0-4.1 kg for class III obesity (Kiel et al., 2007) (see Chapter 2 for definition of obesity classes).

A prospective study of a cohort of 245,526 Swedish women confirmed that GWG among obese women (BMI = 30-34.9) and very obese women (BMI ≥ 35) was lower (11.1 and 8.7 kg, respectively) than among non-obese women (Cedergren, 2006). Low GWG (< 8 kg) occurred in 30.2 and 44.6 percent of the obese and very obese women, respectively. Among the 62,167 women in the Danish National Birth Cohort with data on GWG, about 36 percent of the obese women exhibited low rates of gain (0.28 kg per week). Fifty percent gained between 0.28 and 0.68 kg per week, and 14 percent gained > 0.68 kg per week (Nohr et al., 2007).

Obese women (BMI = 30-40) participating in a prenatal intervention gained less weight (adjusted GWG = 7.52 kg) than controls (adjusted GWG = 9.78 kg), and experienced no difference in pregnancy outcome (Claesson et al., 2008). In summary, from a population perspective, obese women as a group gain less weight than non-obese women, nevertheless GWG can vary widely.

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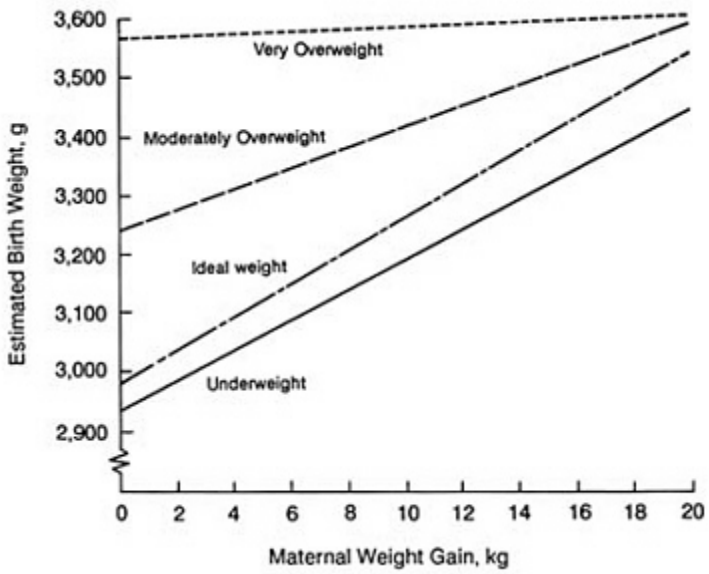

FIGURE 3-2 Birth weight as a function of maternal weight gain and prepregnancy weight for height. SOURCE: Modified from Abrams et al. (1986). This article was published in the *American Journal of Obstetrics and Gynecology*, 154(3), Prepregnancy weight, weight gain, and birth weight, pp. 503-509. Copyright Elsevier (1986).

Twin Pregnancies

Total GWG in twin pregnancies is generally higher than in singleton pregnancies; averaging from 15 to 22 kg (Appendix C, Table C-2). Outcomes associated with GWG in twin pregnancies, as with singleton pregnancies, are a function of pregravid BMI. Several studies have shown that, when stratified by pregravid BMI, increased GWG is associated with increased twin birth weight among underweight, normal weight, and overweight, but not obese, women (Brown and Schloesser, 1990; Luke et al., 1992; Lantz et al., 1996). Yeh and Shelton (2007) found that mean twin birth weights in the population studied increased incrementally from 2,237 g to 2,753 g for total GWG < 35, 35-45, 46-55, and > 55 pounds, respectively. The odds, however, of having a twin delivery at ≥ 36 weeks gestation and birth weight ≥ 2,500 g were significantly lower among women who gained < 35 pounds (AOR 0.49, 95% CI: 0.37-0.65) and significantly higher among women who gained > 55 pounds (AOR 2.24, 95% CI: 1.51-3.33) compared to those who gained 35-45 pounds. Interestingly, GWG > 55 pounds was associated with an approximate 1.5 times greater likelihood of having a maternal complication (cumulative of GDM, pregnancy-induced hypertension, preeclampsia, and anemia [AOR 1.63, 95% CI 1.02-2.60]) also of having a cesarean delivery [AOR 1.85, 95% CI 1.20-2.87]). The cumulative weight gain stratified by pregravid BMI for mothers of twins born at 37-42 weeks of gestation and with an average twin birth weight ≥ 2,500 g is shown in Table 3-1. Cumulative and rates of weight gain by trimester are presented in Appendix C, Tables C-3A and C-3B. In summary, GWG in twin gestations mirrors that in singleton pregnancies, i.e. there is an inverse relationship between maternal GWG and maternal pre-pregnancy BMI. These results suggest that a balance is needed between optimal GWG for maternal and twin outcomes.

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TABLE 3-1 Summary of Adjusted and Unadjusted* Cumulative Weight Gain, by Pregravid BMI Status for Mothers of Twins at Gestational Ages 37-42 Wk, and with Average Twin Birth weight > 2,500 g

	Cumulative Weight Gain (To 37-42 weeks)		**Interquartile 25th – 75th Percentile Ranges of Cumulative Weight Gain (To 37-42 weeks)**	
Pregravid BMI	**kg**	**lbs**	**kg**	**lbs**
Normal Weight [a] (n = 409)	20.9 ± 0.3 (21.0 ± 6.1)*	45.9 ± 0.7 (46.2 ± 13.4)*	16.8 – 24.5	37 – 54
Overweight [b] (n= 154)	18.9 ± 0.5 (18.7 ± 7.0)*	41.6 ± 1.1 (41.1 ± 15.5)*	14.1 – 22.7	31 – 50
Obese [c] (n = 143)	15.7 ± 0.5 (15.4 ± 7.2)*	34.6 ± 1.2 (34.0 ± 15.9)*	11.4 – 19.1	25 – 42

NOTES: Results are presented as least square means ± SEM from models controlling for diabetes and gestational diabetes, preeclampsia, smoking during pregnancy, primiparity, and placental membranes (monochorionicity and missing chorionicity). Total cumulative gain is also adjusted for length of gestation. Results in parentheses are the unadjusted means ± SD (also see Appendix C, Tables C-3A through C-3D)

[a] BMI = 18.5-24.9 kg/m^2
[b] BMI = 25.0-29.9 kg/m^2
[c] BMI = ≥ 30 kg/m^2

SOURCE: Historical cohort of twin births delivered at John Hopkins Hospital, Baltimore, Jackson Memorial Hospital, Miami, Medical University of South Carolina, Charleston, and University of Michigan, Ann Arbor provided by Barbara Luke, Sc.D., M.P.H., R.D., and Mary L. Hediger, Ph.D. For more details on this historical cohort, see Luke et al. (2003).

Triplet and Quadruplet Pregnancies

Fewer studies are available on triplet and quadruplet pregnancies (Appendix C, Table C-2). In a large cohort study, GWG among mothers carrying triplets was found to range from 20.5 to 23.0 kg at 32-34 weeks and for quadruplets from 20.8 to 31.0 kg at 31-32 weeks (Luke, 1998). In the same cohort, the mean total GWG in 38 triplet pregnancies was found to be 20.2 kg at 33.4 weeks (Luke et al., 1995). The rate of gain before 24 weeks gestation was 0.48 kg per week and 0.96 kg per week after 24 weeks (Luke et al., 1995). In data from a different cohort, GWG was found to be a function of BMI category. Median GWG was 15.5, 21.8, and 15 kg for low-, normal-, and high-BMI categories, respectively (Eddib et al., 2007).

Pattern of Gestational Weight Gain

The pattern of GWG is most commonly described as sigmoidal (Hytten and Chamberlain, 1991), but linear, concave, and convex patterns of weight gain have been observed as well (Villamor et al., 1998).

Singleton Pregnancies

In the report *Nutrition During Pregnancy* (IOM, 1990) the average rates of GWG for well-nourished women with uncomplicated singleton pregnancies are reported as approximately 0.45 kg per week during the second trimester and 0.40 kg per week during the third trimester. Several studies, published since that report, indicate that higher rates of GWG in the second and third

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trimesters are common among American women with normal range BMI values (Appendix C, Tables C-1A and C-1B).

The pattern of GWG by maternal BMI category was examined in a large cohort of women from University of California, San Francisco (Abrams, et al., 1995; Carmichael et al., 1997). Mean rate of gain was 0.169 kg per week in the first trimester. Mean weight gains were higher in the second (0.563 kg per week) than the third trimester (0.518 kg per week) in all groups except for obese women. The average gains in the second and third trimester were higher in underweight and normal weight women than in overweight and obese women. Birth weight was correlated most strongly with gain in the second trimester (32.8 g/kg GWG versus 18 and 17 g/kg in the first and third trimesters, respectively).

In another study, mean rates of GWG in non-obese, low-income black and white women were 2.48 kg in the first trimester and 0.49 and 0.45 kg per week in the second and third trimesters, respectively (Hickey et al., 1995). In contrast, rates of weight gain of predominantly Hispanic women (n = 7,589) participating in the Prematurity Prevention Project were similar in the second (0.52 kg per week) and third trimesters (0.53 kg per week) (Siega-Riz et al., 1996). In this study, the third-trimester gain was slightly lower in women who delivered preterm (0.50 vs. 0.53 kg per week). A similar pattern of GWG was found in adolescents, although the median gain and rate of gain were higher throughout gestation. From mid-pregnancy to term the rate of gain was 0.51 kg per week (Hediger et al., 1990). In summary, the pattern of GWG is generally higher in the second trimester and is related to maternal pregravid BMI. However, pattern of GWG can vary depending on maternal ethnicity and age.

Twin Pregnancies

A series of observational studies examined outcomes associated with the rate of GWG in women with twin pregnancies who delivered infants at 37-42 weeks gestation with mean birth weights exceeding 2,500 g. Luke et al. (1992) found that low rate of GWG, defined as < 1.0 pound/week, was associated with a significantly lower mean birth weight for twins compared to singletons (β, -0.137; p = 0.001). Significantly higher rates of GWG in the third trimester were observed among women whose mean birth weights for twins was $\geq$ 2,500 g compared to women with birth weights for twin that was < 2,500 g, regardless of BMI category. No significant differences were seen for first and second trimester GWG rates.

Among a large multiethnic population of 646 twin pregnancies at $\geq$ 28 weeks gestation, birth weight increased by 14, 20, and 17 g for each pound of weight gained between 0 and 20 weeks gestation, 20 and 28 weeks gestation, and 28 weeks to birth, respectively (Luke et al., 1997). Mean total GWG was 17.4 kg in a larger cohort of 1,564 twin births of > 28 weeks' gestation from the same population (Luke et al., 1998). In a another study from the same population database, Luke et al. (2003) found that rates of GWG associated with optimal outcomes were greater for underweight and normal weight women than for overweight and obese women. These results are similar to those of singleton pregnancies.

COMPONENTS OF GESTATIONAL WEIGHT GAIN

As pregnancy progresses, protein, fat, water, and minerals are deposited in the fetus, placenta, amniotic fluid, uterus, mammary gland, blood, and adipose tissue (Figure 3-3). The products of conception (placenta, fetus, amniotic fluid) comprise approximately 35 percent of the total GWG (Pitkin, 1976). The extent to which these changes in body composition are critical for

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normal fetal development or are incidental to pregnancy is not completely understood.

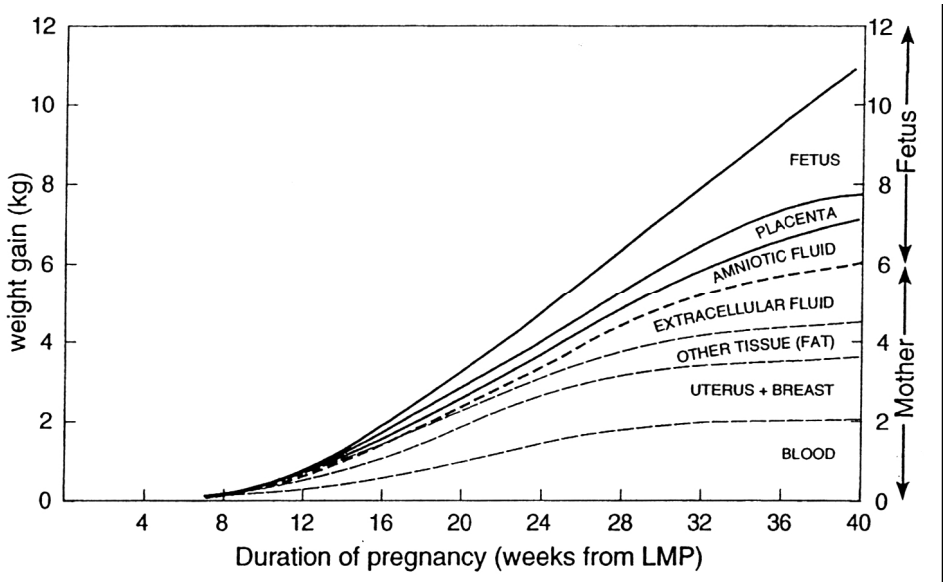

FIGURE 3-3 Components of gestational weight gain.
SOURCE: Pitkin, 1976. Nutritional support in obstetrics and gynecology. *Clinical Obstetrics and Gynecology* 19(3): 489-513. Reprinted with permission.

Maternal Components of Gestational Weight Gain

Total Body Water Accretion

Total body water (TBW) accretion is highly variable during pregnancy and largely under hormonal control. Across several studies, TBW accretion measured by deuterium or antipyrine tracers averaged about 7-8 liters (L) in healthy pregnancies (Hytten and Chamberlain, 1991). Expansion of the extracellular fluid (ECF) measured using the tracer sodium thiocyanate is estimated to be about 6-7 L. For a reference 12.5-kg GWG, total water gain at term is distributed in the fetus (2,414 g), placenta (540 g), amniotic fluid (792 g), blood-free uterus (800 g), mammary gland (304 g), blood (1,267 g), and extravascular-ECF (1,496 g) with no edema or leg edema, and ECF with generalized edema (4,697 g) (Hytten and Chamberlain, 1991). Maternal age, parity, and height did not affect the incidence of edema, but overweight women had greater generalized edema than underweight women. As pregnancy advances, plasma volume expansion measured using Evans blue dye increases up to 45 percent (Rosso, 1990). Maternal plasma volume expansion correlates positively with birth weight. Monthly bioimpedance analysis (BIA) measurements in 170 healthy pregnant women confirmed the progressive expansion of TBW, intracellular water (ICW), and ECF during pregnancy (Larciprete et al., 2003). Total body water accretion was positively correlated with birth weight, in agreement with other investigations (Langhoof-Roos et al., 1987; Lederman et al., 1997; Mardones-Santander et al., 1998; Butte et al., 2003).

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Fat-Free Mass

Protein Accretion

Protein is accrued predominantly in the fetus (42 percent), but also in the uterus (17 percent), blood (14 percent), placenta (10 percent), and breasts (8 percent) (Hytten and Chamberlain, 1991). Protein accrual occurs predominantly in late pregnancy. Protein deposition has been estimated from measurements of total body potassium (TBK) accretion measured by whole-body counting in a number of studies of pregnant women (King et al., 1973; Emerson et al., 1975; Pipe et al., 1979; Forsum et al., 1988; Butte et al., 2003). King et al. (1973) observed a rate of TBK accretion of 24 milliequivalents (meq) per week between 26 and 40 weeks' gestation. Pipe et al. (1979) found a 312 meq potassium (K) increase. Lower increments of 110 and 187 meq at 36 weeks were found over pregravid values in two other studies (Forsum et al., 1988; Butte et al., 2003). Based on a potassium-nitrogen ratio in fetal tissues of 2.15 meq potassium/g nitrogen, the total protein deposition estimated from the longitudinal studies of King et al. (1973), Pipe et al. (1979), Forsum et al. (1988), and Butte et al. (2003) is 686 g. A study of 108 black adolescents, showed a mean rate of TBK accretion of 21 meq per week between 16 and 35 weeks' gestation, consistent with adult studies (Stevens-Simon et al., 1997). In summary, these recent studies suggest that protein accretion may be less than the approximate (~1 kg) estimates of the earlier findings of Hytten and Chamberlin (1991).

Fat Mass

Fat Accretion

The distribution of fat deposited during pregnancy is distinct to pregnancy. Figure 3-4 shows that, based on serial measurements of skinfold thickness at seven sites made in 84 healthy, pregnant women, fat appears to be deposited preferentially over the hips, back, and upper thighs up to about 30 weeks gestation (Taggart et al., 1967).

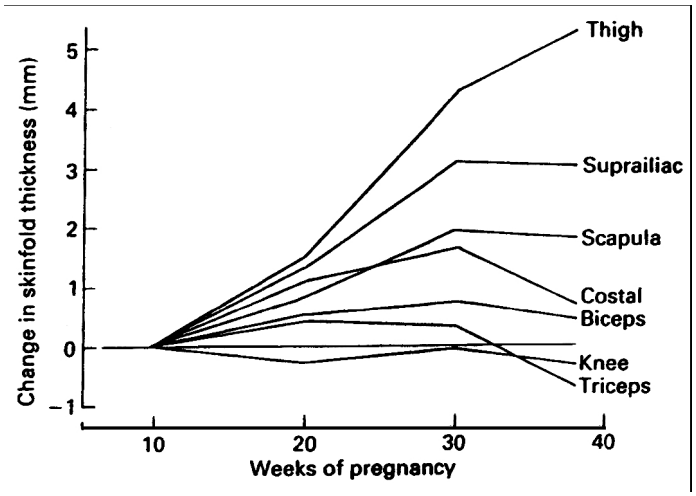

FIGURE 3-4 Longitudinal changes in skinfold thicknesses throughout pregnancy.
SOURCE: Taggart et al., 1967. Changes in skinfolds during pregnancy. *British Journal of Nutrition* 21(2): 439-451. Reprinted with the permission of Cambridge University Press.

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Evidence using computer tomography in 14 women suggests that childbearing may be associated with preferential deposition of visceral fat (Gunderson et al., 2008). However, the majority of fat deposited during pregnancy is subcutaneous. Magnetic resonance imaging to assess fat deposition and distribution in 15 healthy women before and after pregnancy (Sohlstrom and Forsum, 1995) found that of the adipose tissue gained during pregnancy, 76 percent was deposited subcutaneously, similar to the fat distribution before pregnancy. Of the total fat deposition, 46 percent was in the lower trunk, 32 percent in the upper trunk, 16 percent in the thighs, 1 percent in the calves, 4 percent in the upper arms, and 1 percent in the forearms. Postpartum, fat was mobilized more completely from the thighs than the trunk and non-subcutaneous fat in the upper trunk actually increased postpartum.

Measurement of fat mass during pregnancy is technically challenging because the usual methodologies are imprecise, invalid, or not applicable to pregnancy. Skinfold measurements lack the precision necessary to estimate changes in fat mass accurately. Two-component body composition methods based on TBW, body density, and TBK, however, are invalid during pregnancy because of the increased hydration of fat-free mass (FFM) that occurs during pregnancy. The constants for hydration, density, and K content of FFM used in two-compartment models are not applicable to pregnant women and would lead to erroneous estimations of FFM and fat mass (FM). Corrected constants for the hydration, density, and K content of FFM in pregnancy have been determined (van Raaij et al., 1988; Hopkinson et al., 1997). Two-component models that use the corrected constants are satisfactory for use with pregnant women, as are three- or four-component models (Fuller et al., 1992) in which the hydration or density of FFM is measured.

Fat accretion models estimated in pregnant women using corrected two-component models, or three- and four-component body composition are summarized in Appendix C, Table C-4. Figure 3-5 shows a four-compartment body composition model of FM, TBW, protein, bone mineral, and non-osseous mineral measured by hydrodensitometry, deuterium dilution, and densitometry (dual energy X-ray absorptometry, DXA) (Lederman et al., 1997). When applied (after pregnancy) to 200 healthy women at 14 and 37 weeks of gestation the model showed that obese women gained significantly less fat than underweight and normal weight women (8.7 vs. 12.6 and 12.2 kg, respectively). There were no differences in the amount of TBW gained among the underweight, normal weight or obese women. The majority of women studied did not conform to the recommendations of IOM (1990). Sixty-seven percent of underweight, 61 percent of normal weight, 69 percent of overweight, and 78 percent of obese women gained outside the recommended ranges. Fat accretion paralleled GWG; FM gain was positively correlated with GWG ($r = 0.81$) and inversely correlated ($r = -0.25$) with pregravid weight. For those that gained within recommendations from IOM (1990), FM gain was highest among the underweight (6.0 kg), followed by the normal weight (3.8 kg), overweight (2.8 kg), and obese (–0.6 kg). For those who gained less than the recommendations, FM gain was 0.6 kg in the underweight, 1.3 kg in the normal weight, 0.3 kg in the overweight, and –5.2 kg in the obese. For those that gained more than the recommendations, FM was highest in the underweight (6.9 kg), followed by the normal weight (6.0 kg), overweight (4.2 kg), and obese (3.1 kg).

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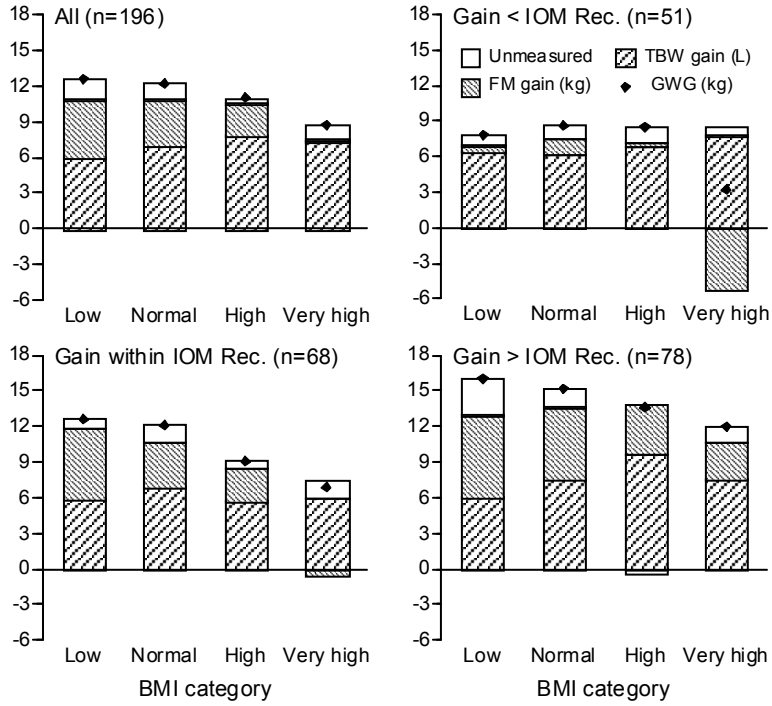

FIGURE 3-5 Body weight and composition changes in 196 women are presented by pregravid BMI category (low *n* = 21, normal *n* = 118, high *n* = 29, and very high *n* = 28). Gains in total body water and fat mass and gestational weight gain also are presented by compliance with the IOM 1990 recommendations for weight gain: women gaining less than (*n* = 51), within (*n* = 68), and more than (*n* = 78) the recommendations from IOM (1990).
SOURCE: Lederman et al., 1997.

Another four-compartment body composition model based on TBK, TBW, body volume, and bone mineral content measured by whole-body counting, deuterium dilution, hydrodensitometry, bone, and DXA (pre- and postgravid only) was used before pregnancy, at 9, 22, and 36 weeks of gestation; and at 2, 6, and 27 weeks after delivery (Butte et al., 2003) (see Figure 3-6). Protein accretion was measured by prompt-gamma activation measurements of total body nitrogen (TBN) taken before and after pregnancy. Total body K and TBN did not differ before and immediately after pregnancy, but did decline postpartum. On average, weight gain was 42 percent FM and 58 percent FFM. GWG was correlated linearly with gains in TBW (r = 0.39), TBK (r = 0.49), protein (r = 0.49), FFM (r = 0.50), and FM (r = 0.76). Gains in TBW, TBK, protein, and FFM did not differ among low-, normal- and high-BMI groups; only FM gain was higher in high-BMI group who also gained more weight. The body composition changes in those women who gained (mean 14.4 kg) within IOM (1990) recommendations were TBW (7.1 kg), TBK (5.0 g), protein (370 g), FFM (8.4 kg), and FM (4.1 kg). Postpartum weight retention positively correlated with GWG and FM gain, but not with total TBW, TBK, or FFM gain. Postpartum FM retention positively correlated with GWG and FM gain. FM retention at 27 weeks' postpartum was higher in those who gained above the recommendations (5.3 kg) than those that gained within (2.3 kg) and below (–0.5 kg) them. Birth weight was positively correlated with gains in weight, TBW, TBK, protein, and FFM, but not FM gain. Lederman et al. (1997) also found that maternal weight and FFM, but not FM, at term related to birth weight.

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In summary, much of the variance in GWG is accounted for by the increase in fat mass, because that much of an increase in FFM also represents and increase in water. Similar to what was observed in GWG, the increase in fat mass during gestation in inversely proportional to pre-gravid obesity.

The relationships between accretion of maternal fat mass as a function of pre-gravid obesity may relate to pregravid maternal metabolic function. Sixteen healthy lean women were measured for body composition, basal oxygen consumption (VO_2), and insulin sensitivity before pregnancy and at 12-14 weeks and 34-36 weeks of gestation (Catalano et al., 1998). In early pregnancy, women with abnormal glucose tolerance had smaller increases in FM (1.3 kg) and percentage FM (1.6 percent) compared to those with normal glucose tolerance (2.0 kg, 3.6 percent). Fat accretion did not differ from early to late gestation but changes in maternal insulin sensitivity were inversely correlated with changes in energy expenditure and FM accretion in early but not late pregnancy.

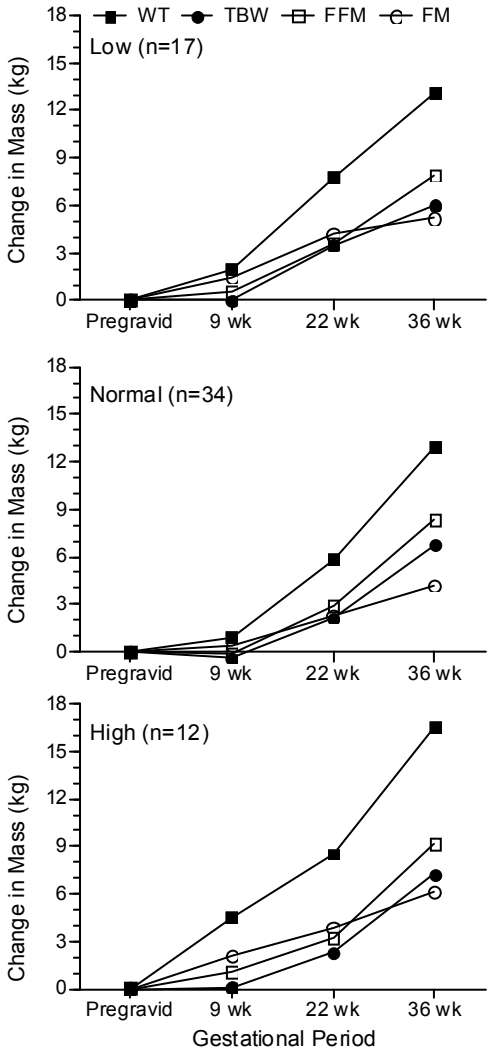

FIGURE 3-6 Changes in body weight and composition of 63 women (low pregravid BMI *n* = 17; normal pregravid BMI *n* = 34; high pregravid BMI *n* = 12) measured at 9, 22, and 36 weeks' gestation.
NOTE: WT = weight.
SOURCE: Butte et al., 2003.

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Placenta

Placental Weight

There is a linear relationship between fetal growth and placental mass, fetal weight, and placental growth in both early and late gestation (Molteni et al., 1978). There is a significant increase in the mean placental weight and the fetal-placental weight ratio with advancing gestation in pregnancies that are appropriate-for-gestational age (AGA) and LGA.

In infants that were born SGA, placental weight showed no increase after 36 weeks, but the fetal-placental weight ratio continued to increase. Therefore, although there may be further growth of the fetus, albeit not optimal, there is a lack of placental growth commonly referred to as placental insufficiency. The basis for altered placental growth and function may be related to a variety of pathologies such as nutritional, vascular (e.g., hypertension, diabetic vasculopathy), or anatomic disorders.

There are a limited number of cases of higher-order placental weights in higher multiples, but Pinar et al. (2002) published a series of reference weights from triplet pregnancies. See Table C-5 in Appendix C for normative criteria for placental weight in singleton, twin, and triplet pregnancies.

Placental Growth

Normal placental growth using human tissue is difficult to ascertain because placentas obtained from early pregnancy are often the result of an abnormal pregnancy outcome. Prior to 20 weeks, most placentas are obtained at the time of either spontaneous or elective termination. In mid-pregnancy, placentas are obtained after either a preterm delivery or placental dysfunction such as placenta previa or abruptio placenta.

Placental Development

There are structural and functional changes in placental development with advancing gestation. Teasdale (1980) described these changes in placentas delivered in healthy pregnancies between 22 and 40 weeks' gestation. The first stage of placental growth lasting through 36 weeks is characterized by increases in the parenchymal and non-parenchymal tissue. The parenchyma is composed primarily of intravillous space, the trophoblast tissue (i.e., cytotrophoblast, syncytiotrophoblast), and fetal capillaries of peripheral and stem villi. The non-parenchymal tissue is composed of the decidual and chorionic plates, intercotyledonary septa, fetal vessels, connective tissue, and fibrin deposits. The second phase of placental development lasting from 36 weeks until term is the maturation phase. The maturation phase of placental growth is characterized by an increase in fetal growth but without an increase in placental functional or parenchymal tissue. During the maturation phase there is only an increase in non-parenchymal (i.e., nonfunctional) placental tissue. These relationships are all consistent with the importance of early placental growth and development needed to support the rapid fetal growth in the last trimester of pregnancy when fetal weight increases from a mean of 1,000 g to 3,400 g in the general U.S. population.

In addition to the weight and structural changes in placental development, there also may be differences in placental function as a consequence of a women's pregravid BMI. In general, obese women are more likely to have larger placentas and neonates in comparison to average-

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weight women. As discussed previously the alterations in maternal metabolic function during pregnancy are most likely mediated through placental hormone and cytokine production, which in turn affect maternal fat accretion and nutrient availability necessary for fetal growth. Recently, Challier et al. (2008) reported that the placentas of obese women (pregravid BMI > 30 kg/m^2) had a two- to threefold increase in the number of macrophages in comparison with placentas of average-weight (pregravid BMI < 25 kg/m^2) women. There was also increased expression of the proinflammatory cytokines interleukin (IL-1), tumor necrosis factor-alpha (TNF-α), and IL-6. Hence, the chronic inflammation associated with obesity may affect placental growth and function, thereby altering maternal metabolic function and resulting in the women with pregravid obesity having decreased maternal pre-gravid maternal insulin resistance and decreased maternal fat accretion but increased placental and fetal growth.

Placental Growth

Because of the intrinsic problem of using cross-sectional data to determine normal placental growth, there developed an interest in the use of ultrasound to estimate placental growth using various volumetric measures. Bleker and Hoogland (1981) estimated placental volumes using longitudinal ultrasonographic techniques. Placental volume was 200 cm^2 at 21 weeks' gestation, 300 cm^2 at 28 weeks, and 500 cm^2 at term. The placental area was found to increase linearly until 24 weeks. There was a decreasing growth rate in the last trimester, although 15 percent of placentas showed a continuous increase through pregnancy. Abramovich (1969) was able to obtain placental weights at the time of abdominal hysterectomy with an intact amniotic sac. The average weight of the placenta at 10-12 weeks was 51 g, 12-14 weeks 66 g, 14-16 weeks 85 g, 16-18 weeks 110 g, and 18-20 weeks 141 g.

Placental Composition

The composition of the placenta varies with gestational age as well as maternal metabolic status. Approximately 88 percent of placental weight is water. In comparison, the fetus at term has approximately 80 percent water in its fat-free mass. In studies of Widdowson and Spray (1951), the composition of placentas ranging from 17 to 40 weeks' gestation was analyzed. The mean percentage of water was 88 percent, protein 11 percent, and fat 1 percent. Garrow and Hawes (1971) similarly reported that in more than 700 placentas, the blood-free placenta had approximately 10 percent protein. In a further analysis of the effect of maternal diabetes on placental composition, Diamant et al. (1982) described increased placental mass, amount of DNA, glycogen, and lipids in the placentas of women with diabetes compared to a normal glucose-tolerant control group. The relative changes in glycogen and fat exceeded the changes in weight and mg of DNA, suggesting that a true increase in glycogen and fat per placental cell may have occurred. The increase in lipids in the placenta of the women with diabetes consisted primarily of triglycerides and phospholipids but not cholesterol (See Table C-6 in Appendix C for placental lipid content).

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Fetus

Patterns of Fetal Growth for Singletons, Twins, and Triplets

Singletons With the exception of longitudinal studies using methods such as ultrasound, all measures of fetal growth are cross-sectional by definition (i.e., each fetus having been measured only once) Hytten and Chamberlain, 1991). The criteria that are commonly used are to classify fetal growth (1) SGA (i.e., birth weight less than the 10th percentile for gestational age); (2) average (AGA; i.e., birth weight between the 10th and 90th percentile for gestational age); and (3) LGA (i.e., birth weight greater than the 90th percentile for gestational age). These criteria were arbitrarily chosen to help assess the neonatal risk for both short-term and, more recently, long-term morbidity. Since that time there have been numerous other publications relative to fetal growth rates.

For the fetus that is deemed viable, fetal weight, as a measure of fetal growth, is usually determined at the time of delivery. The gestational age of viability has decreased steadily over the years and the fetus is now considered potentially viable at 23-24 weeks. Therefore, most of the fetal growth curves relating to viable fetuses rely on clinical data starting from the mid-second trimester. Although the numbers are small, there appears to be minimal variation in fetal growth through 25 weeks' gestation (Archie et al., 2006).

Recently, Thomas et al. (2000) published data comparing gestation-specific growth parameters with those developed in the late 1960s using data from 85 nurseries including 27,229 neonates. For neonates at < 30 weeks' gestation, there were smaller variances and lower average weights, lengths, and head circumferences than previously published norms. For neonates > 36 weeks' gestation, the variance was similar, but the neonates were larger and heavier. The authors concluded that using older growth curves resulted in misclassification of gender- and race-specific criteria for SGA and LGA. Since then, many investigators have observed an increase in birth weight at term (Orskou et al., 2001; Ananth and Wen, 2002; Surkan, 2004; Catalano, 2007). Hence, the use of current birth weight curves is important in the assessment of fetal growth. Oken et al. (2003) published U.S. birth weight curves based on the 1999 and 2000 United States Natality datasets from 22 through 44 weeks gestation.

Although gestational age is an important factor related to fetal growth, other factors affect not only fetal growth but the pattern of growth. These include gender (Figure 3-7)—males grow more rapidly from the mid-third trimester through term. Other factors that can affect fetal growth include maternal age, height, weight, GWG, obesity, and parity (Catalano et al., 2007). Paternal factors can also affect fetal growth, although they explain much less of the variance than maternal factors (Klebanoff et al., 1998). High altitude results in decreased fetal growth as does maternal hypoxia. Maternal medical problems, e.g. hypertensive disorders, autoimmune disease, and smoking can also result in decreased fetal growth. In contrast, maternal diabetes without evidence of vascular involvement often results in increased fetal growth (see Chapter 4 for detailed discussion).

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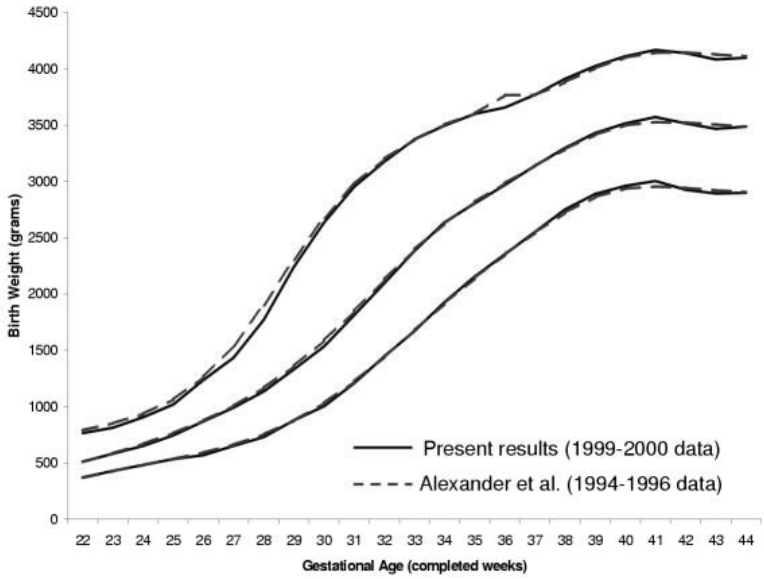

FIGURE 3-7 Select reference percentiles for birth weight at each gestational age from 22 to 44 completed weeks for all singleton infants.
SOURCE: Oken E., K. P. Kleinman, J. Rich-Edwards, and M. W. Gillman. 2003. Reprinted with permission from *BMC Pediatrics* 3: 6, by BioMed Central.

The question of ethnic differences in fetal growth and implications for neonatal health has become more relevant recently. Kierans et al. (2008) evaluated all births in British Columbia from 1981 through 2000 and examined fetal growth and perinatal mortality in Chinese, South Asian, First Nation (Native American Indian), and other (primarily Caucasian) populations. They concluded that the ethnic differences in fetal growth rates were physiologic, not pathologic.

The rate of premature delivery (i.e., before 37 weeks' gestation) in the United States is approximately 12.5 percent. As such, birth weight tables that rely on actual neonatal weights for preterm infants represent a much smaller percentage of all births. Furthermore, there is evidence that infants born prematurely are smaller than infants of the same gestational age who remain in utero (Weiner et al., 1985).

In summary, normal fetal growth is relatively uniform until mid-second trimester. At term there is much greater variation in fetal weight as a result of varying determinants of GWG and other maternal factors (see Chapter 4 for complete discussion). Lastly, there has been an increase in term birth weight in developed countries over the last two decades, most likely because of the increased prevalence of obesity.

Twins and Triplets Fetal growth in multiple gestations can be considered very similar to singleton growth until the third trimester. Although there is a tendency to consider multiple gestations as being growth restricted, Blickstein (2002) described the fetal mass of a multiple pregnancy as "growth promoted" and the smaller size of the fetus as "growth adapted". In addition to previously discussed variables that may affect fetal growth such as gender and parity, in twin gestations, chorionicity may also affect fetal growth. Ananth et al. (1998) reported that

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twins from monochorionic gestations weigh on average 66 g less than those from dichorionic gestations after correction for gestational age.

Gielen et al. (2007) reported on customized birth weight charts in more than 4,277 twin pairs in Flanders from 1964 through 2002. In their study, birth weight was affected by maternal parity and age. Zygosity, fetal gender, chorionicity, fusion of the placentas, placental weight, and site of umbilical cord insertion all influenced twin birth weights. These variables can account for as much as a 1,000 g difference in weight at term. After 40 weeks' gestation, there is a decrease in weight of twins with a monochorionic monozygotic placentation, while dichorionic dizygotic twins continue to grow. Min et al. (2000) estimated growth in 1,831 twin pregnancies using ultrasound at 2-week intervals from 20 through 40 weeks' gestation. The weight difference between twins and singleton pregnancies at their respective 50th percentiles was 147 g (10 percent) at 30 weeks' gestation, 242 g (14 percent) at 32 weeks' gestation, 347 g (17 percent) at 34 weeks' gestation, 450 g (19 percent) at 36 weeks' gestation, 579 g (22 percent) at 38 weeks' gestation, and 772 g (27 percent) at 40 weeks' gestation.

Lastly, Glinianaia et al. (2000) reported on 690 triplets born in Norway from 1967 through 1995. The birth weight by gestational age curves of the triplets were almost identical to those of singleton and twin gestations before 30 weeks. From 31 weeks of gestation onward, the median birth weight of triplets consistently diverged from that of twins. At 38 and 39 weeks' gestation the difference reached 478 and 541 g, respectively, with a weight difference between twins and triplets of 650 g in the 10th percentile at 39 weeks.

In summary, the growth rate in multiple gestations is similar to growth rate in singleton gestations up to approximately 30 weeks' gestation. In the third trimester, there is a decrease in individual fetal growth, more so in triplets than in twins and may be related to placental function.

Fetal Body Composition

The human fetus at term has a significantly different body composition than most other mammalian species. At birth the human fetus has approximately 12-16 percent body fat. In contrast, laboratory animals have 1-2 percent body fat at birth (Widdowson, 1950). Theoretically, the accrual of fetal fat has two possible sources: one is from the transfer of free fatty acids from the mother, and the second is *de novo* synthesis of fatty acids from substrates such as glucose, lactate, and acetate provided by the mother (Girard and Ferre, 1982). Regardless of the substrate source, fetal insulin is required for the fetus to increase adipose tissue stores.

The remaining tissue in the human fetus is lean body mass or FFM, which consists primarily of glycogen, protein, and water. At birth the human fetus has approximately 40 g of glycogen, primarily in muscle and liver tissue (Girard and Ferre, 1982). The protein content of the term fetus is approximately 12.8 percent of total body weight, or 15 percent of FFM (i.e., about 500 g; Fomon et al., 1982; Spady, 1989). The remainder is water. In the human fetus at term, approximately 80 percent of FFM is water (Fomon et al., 1982). Temporal changes in fetal growth and body composition have been characterized using ultrasound. The human fetus at 28 weeks weighs approximately 1 kg. Over the next 12 weeks the fetus gains approximately 2.5 kg. Fetal fat tissue begins to accrue in the mid-second trimester. Into the third trimester, however, there is a decrease in FFM as a percentage of total body weight. Bernstein et al. (1997) reported that, although the rate of fetal FFM accretion appeared linear when taken in aggregate, the compartments of FFM changed differentially. Peripheral muscle growth accelerated, and head circumference decelerated in late gestation. Fetal fat deposition accelerated as a quadratic

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function. Hence, fetal growth of FM and FFM follow unique patterns and offer an additional means to assess normal and abnormal growth.

The gold standard for estimating body composition is carcass analysis. Sparks (1984), in his review of the data on 169 carcass analyses of fetuses, concluded that the differences in FFM are less variable at each gestational age than the fat content As with GWG, the variation in fetal growth may be related to the fat content of the fetus, which may reflect the intrauterine environment, while the FFM may be more representative of genetic factors.

Koo et al. (2000) using DXA, found that among the 214 singletons studied, neonates whose birth weight was < 2,500 g had 6 to 14 percent body fat. Neonates whose birth weight was > 2,500 g had 8 to 20 percent body fat. The mean percentage of body fat for a 3,500-g infant was 16.2 percent. Using total body electrical conductivity, Catalano et al. (2003) reported that body fat was 10.4 $\pm$ 4.6 percent in 220 term healthy singleton neonates. The difference in results between the two studies primarily represents differences in methodologies.

Birth weight and neonatal body composition are related to changes in maternal body composition. Butte et al. (2003) used DXA to assess body composition in 63 term infants at 2 weeks of age and related these data to changes in maternal body composition measured using a multi-component model. Birth weight correlated positively with prepregnancy weight (r = 0.34), prepregnancy FM (r = 0.32), GWG (r = 0.35), net GWG (r = 0.26), rate of weight gain (r = 0.28), and gestational age (r = 0.49). Birth weight was correlated significantly with gestational gains in TBW (r = 0.37), TBK (r = 0.35), and FFM (r = 0.39), but not FM. Maternal FFM gains in the first, second, and third trimesters were shown by multiple regression to make independent contributions to birth weight. Maternal TBW gains during the second and third trimesters and maternal TBK gain in the third trimester were independent predictors of birth weight. Infant body composition at 2 weeks of age (FFM, FM, or percent FM) was not correlated with maternal body composition before or after pregnancy or with maternal gains in TBW, TBK, FFM, and FM during pregnancy.

As is true for birth weight, multiple factors are associated with alterations in fetal body composition. Some of these factors are genetic. For example, at birth, male fetuses have greater lean body mass than females, and as a consequence, females have a higher percentage of body fat (Catalano et al., 1995; Ibanez et al., 2008). Maternal factors that have the strongest effect on fetal growth and primarily affect fetal fat accretion include parity, GWG, and medical problems. Maternal parity is positively correlated with neonatal adiposity (Harvey et al., 2007). Birth weight is significantly greater in neonates of overweight and obese women than underweight or normal weight women because of increased FM, not FFM (Sewell et al., 2006; Hull et al., 2008). Maternal weight gain is associated with both increased fetal FFM and increased FM and is related to maternal pregravid BMI (Catalano and Ehrenberg, 2006). Maternal medical problems, such as gestational diabetes mellitus, are associated with an increase in birth weight again because of increased FM, and in the macrosomic neonate a relative decrease in FFM (Catalano et al., 2003; Durnwald et al., 2004). Environmental factors also affect fetal body composition (see Chapter 4). Maternal smoking has a negative effect on fetal growth on the order of 150 g, which primarily decreases fetal FFM (Lindsay et al., 1997). Increased altitude has been reported to be associated with a 339-g decrease in birth weight. Crown-head length was shown to be reduced by 1 cm although the sum of five skinfolds was 5 mm greater, in those born at high altitude compared to those born at sea level (Ballew and Haas, 1986). In their study of > 400 newborns using total body electrical conductivity, Catalano and Ehrenberg (2006) found that maternal

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pregravid BMI had the strongest correlation with maternal weight gain and GDM, as a factor associated with fetal adiposity.

In summary, the human fetus has a high percentage of body fat (12-16 percent) at birth compared to most mammalian species. Fetal fat mass contributes the greatest percentage of variance in birth weight, is affected by the in utero environment, and is more strongly correlated with maternal pre-gravid BMI than GWG.

Amniotic Fluid

Amniotic fluid has four major sources of volume flow into and out of the amniotic sac in late gestation (Ross and Brace, 2001). The two major inflow sources are fetal urine and lung liquid secretions. The two major outflows are fetal swallowing and intra-membranous absorption. Brace and Wolf (1989) reported on a series of 705 published amniotic fluid volumes based on either direct collection or dye dilution techniques. At 8 weeks of gestation the volume increases at a rate of 10 mL per week, and at 13 weeks the volume increases to 25 mL per week. The maximal increase in amniotic fluid of 60 mL per week occurs at 21 weeks' gestation. The weekly volume increment then decreases and reaches zero at 33 weeks' gestation (i.e., the time at which maximal volume is reached). There is wide variation in the amount of amniotic fluid in a normal pregnancy. Decreased amniotic fluid (i.e., oligohydramnios) occurs in approximately 8.2 percent of pregnancies, and increased amniotic fluid (i.e., polyhydramnios) occurs in approximately 1.6 percent of pregnancies (Ross and Brace, 2001). Oligohydramnios may occur as a consequence of fetal renal obstruction or dysplasia and may be associated with fetal growth restriction. Polyhydramnios is associated with various fetal structural anomalies such as congenital esophageal atresia, fetal anemia, congenital infections, and maternal diabetes. Given the wide range of normal amniotic fluid volume at term, this compartment may affect maternal GWG by as much as 1 kg. Therefore, amniotic fluid volume is an important component of maternal weight gain.

MATERNAL PHYSIOLOGY

The unique physiologic, metabolic, and endocrine milieu of the pregnant woman is crucial to understanding the mechanisms underlying GWG. The pregnant woman undergoes dramatic physiologic changes in anticipation and in support of fetal growth. Many of the obligatory components of GWG (for example, TBW) are directly related to the alterations in maternal physiology necessary to grow and develop a healthy fetus and placenta.

Cardiovascular Changes

In early pregnancy, cardiac output increases about 30-50 percent as a result of an increase in heart rate—primarily stroke volume—and remains elevated until term (Hytten and Chamberlain, 1991). As pregnancy progresses, blood flow increases to the uterus, kidney, skin, and probably the alimentary tract. Arterial blood pressure may decrease in mid-pregnancy. This is a result of increased peripheral vasodilatation and in order to maintain perfusion this results in an increase in cardiac output and relatively small decreases in mid-gestational blood pressure. Venous blood pressure rises in the lower limbs due to mechanical and hydrostatic pressure in the pelvis, causing edema in the lower limbs. Because of these cardiovascular changes, it is possible to have reduced exercise tolerance and dyspnea.

Physiological changes in circulation during pregnancy are marked and variable (Gabbe et al., 1991; Hytten and Chamberlain, 1991). Plasma volume increases progressively to 50 percent by

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30-34 weeks of gestation. Importantly, plasma volume expansion is correlated with clinical performance and birth weight. Poor plasma volume expansion is associated with a poorly growing fetus and poor reproductive performance. The increases in maternal plasma volume account for a significant portion of the increase in total body water during pregnancy.

Red blood cell mass also increases about 18 percent by term without iron supplementation and 30 percent with iron supplementation. Minute ventilation increases 30-40 percent by late pregnancy due to increased tidal volume. Oxygen consumption increases only 15-20 percent, resulting in an increase in alveolar and arterial P_{AO2} (partial pressure of oxygen) and a fall in P_{ACO2} (partial pressure of carbon dioxide) levels (Gabbe et al., 1991).

Renal Changes

Renal plasma flow increases 70 percent over pregravid levels by 16 weeks of gestation and is maintained until late pregnancy when it falls slightly (Gabbe et al., 1991). Glomerular filtration rate (GFR) increases early in pregnancy, up to 50 percent by term. As a result of the increased GFR, serum levels of urea and creatinine decline. Plasma osmolarity declines early in pregnancy due to a reduction in serum sodium and associated anions. There is a net accumulation of approximately 900-1,000 meq of sodium in the fetus, placenta, and intravascular and interstitial fluids. There is a large increase in tubular sodium reabsorption during pregnancy, promoted by increased aldosterone, estrogen, and deoxycorticosterone. Plasma renin activity, renin substrate, and angiotensin levels increase five- to tenfold above the pregravid values. The adaptations in maternal renal physiology during gestation are among the primary mechanisms accounting for the increase in plasma volume and hence total body water during gestation.

Endocrine Changes

The plasma concentration of corticosteroid-binding globulin (CBG) increases significantly, reflecting increased hepatic synthesis (Gabbe et al., 1991). Estrogen-induced increases in CBG lead to elevated plasma cortisol concentration; with a three-fold increase occurring by the end of the third trimester. The concentration of the metabolically active free cortisol also progressively increases through gestation due to increased production and decreased clearance. Adrenocorticotropic hormone (ACTH) level is suppressed during pregnancy due to the action of estrogen and progesterone. The plasma concentration of dehydroepiandrosterone sulfate (DHEAS) declines during pregnancy due to an increase in metabolic clearance by the placenta and maternal liver.

The renin-angiotensin system changes dramatically during pregnancy. The adrenal gland remains responsive to the trophic action of angiotensin II, even though a refractory effect of pressors to angiotensin II develops early in pregnancy. This provides a probable explanation for the expansion of plasma volume during pregnancy. The secretion of prolactin from the pituitary and uterine decidua increases steadily during pregnancy. In contrast, luteinizing hormone and follicle-stimulating hormone are suppressed to levels similar to the luteal phase of ovulation. Growth hormone secretion is inhibited presumably by placental growth hormone production.

In normal pregnancy, thyroxine-binding globulin concentration is increased and the circulating pool of extrathyroidal iodide is decreased due to increased renal clearance. These changes cause the thyroid to enlarge and to synthesize and secrete the thyroid hormones T_4 (thyroxine) and T_3 (triiodothyronine) more actively. Despite elevated total T_4 and T_3, the concentrations of active hormones (free T_4 and free T_3) are unchanged during normal pregnancy,

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with the exception of a transient increase in the first trimester in some women (Gabbe et al., 1991; Glinoer, 2004).

Adipose tissue produces an array of adipokines known to have profound effects on metabolism and fertility, but their role in reproductive performance is yet to be fully understood. In addition to adipose tissue, leptin and its receptor, TNF-α, and resistin also are expressed in the placenta (Mitchell et al., 2005). Serum adiponectin was found to be lower in the third trimester and this correlates with a decrease in insulin sensitivity (Catalano et al., 2006). Increases in maternal fat mass most likely are related to the decreases in circulating adiponectin concentrations.

Metabolic Changes

Many of the metabolic adjustments of pregnancy are well established in early pregnancy when fetal nutrient demands are still minor. Minimal nutrient balances are usually positive, reflecting the anabolic state of the fetus and the mother. In the absence of nausea or "morning sickness," most women experience an increase in appetite in the beginning of pregnancy (Gabbe et al., 1991). Several gastrointestinal changes occur during pregnancy, including decreased tone and motility of the stomach, reduced gastric acid secretion, delayed gastric emptying, and increased gastric mucous secretion as a function of increased progesterone. Motility of small intestine is also reduced during gestation; however, except for enhanced iron absorption, nutrient absorption is unchanged. These physiologic changes may affect the pattern of gestational weight gain in early gestation.

Changes in protein and nitrogen metabolism occur in early pregnancy, presumably in response to pregnancy-related hormones (Kalhan, 2000). Serum total α-amino nitrogen deceases, as does the rate of urea synthesis and the rate of transamination of branched-chain amino acids, which are aimed at conservation of nitrogen and protein accretion in pregnancy. Protein turnover on a weight basis, however, does not change (Kalhan, 2000). Serum total protein and albumin fall progressively and by term are 30 percent lower than non-pregnant values (Hytten and Chamberlain, 1991). The concentrations of binding proteins for corticosteroids, sex steroids, thyroid hormones, and vitamin D are also increased.

Changes in carbohydrate and lipid metabolism occur during pregnancy to ensure a continuous supply of nutrients to the growing fetus (Butte, 2000). In early pregnancy, glucose tolerance is normal or improved slightly, and peripheral (muscle) sensitivity to insulin and hepatic basal glucose production are normal or increase by as much as 15 percent (Catalano et al., 1991; 1992; 1993). As pregnancy advances, nutrient-stimulated insulin responses increase progressively despite only minor deterioration in glucose tolerance, which is consistent with progressive insulin resistance (Kühl, 1991). In late pregnancy, insulin action is 50-60 percent lower than in non-pregnant state (Ryan et al., 1985; Buchanan et al., 1990; Catalano et al., 1991; 1992; 1993). By the third trimester, basal and 24-hour mean insulin concentrations may double (Lesser and Carpenter, 1994). The first and second phases of insulin release are increased threefold by late pregnancy (Catalano et al., 1991). These alterations in maternal insulin sensitivity affect not only glucose metabolism but also lipid metabolism resulting in a decreased ability of insulin to suppress lipolysis (Catalano et al., 2002).

The return of normal physiologic function after delivery may occur rapidly over a matter of days—for example, an improvement in insulin sensitivity (Ryan et al., 1985)—or over a matter of weeks—for example, a return to a normal non-pregnant renal glomerular filtration rate (Sims and Krantz, 1958). The alterations in maternal physiology are mediated by placental factors as

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evidenced by the significant increase in maternal insulin sensitivity within days after delivery of the fetus and placenta. The alterations in maternal metabolism have generally been ascribed to placental hormones such as hPL, progesterone, and estrogen (Kalkhoff et al., 1979; Ryan and Enns, 1988). Recently, Kirwan et al. (2002) have reported that circulating cytokines (i.e., TNF-α concentration) were inversely correlated with insulin sensitivity.

The metabolic changes in insulin sensitivity during pregnancy are modified by inflammatory factors (Friedman et al. 1999; 2008). In women with normal glucose tolerance during pregnancy who lose significant weight postpartum, there is a return to normal metabolic function. However, in women with GDM, particularly if there is no decrease in postpartum weight or adiposity, there remains a significant inflammatory milieu that results in chronic insulin resistance, increasing the risk of diabetes and the metabolic syndrome.

Depending on the pregravid insulin sensitivity status of the woman, insulin sensitivity may increase or decrease during early pregnancy. In the very insulin-sensitive woman, insulin sensitivity most often decreases and is accompanied by an increase in adipose tissue and basal metabolic rate (Catalano et al., 1998). In contrast, in the more insulin-resistant woman (e.g., those who are obese or have GDM), insulin sensitivity often increases and is accompanied by a decrease in basal metabolic rate and potential loss of adipose tissue (Okereke et al., 2004) (Figure 3-8). These physiologic changes may help to explain in part the relative decrease in weight gain in obese insulin-resistant women compared to the greater increases in weight in lean insulin-sensitive women in early gestation. The placental factors related to these alterations in insulin sensitivity, energy expenditure, and adipose tissue are not well understood relative to metabolic alterations in late pregnancy. Although there is a significant increase in maternal leptin concentrations in early pregnancy (Hauguel-de Mouzon et al., 2006), most likely related to placental production, the increased leptin concentrations do not appear to be associated differently with energy expenditure or fat accretion between lean and obese women.

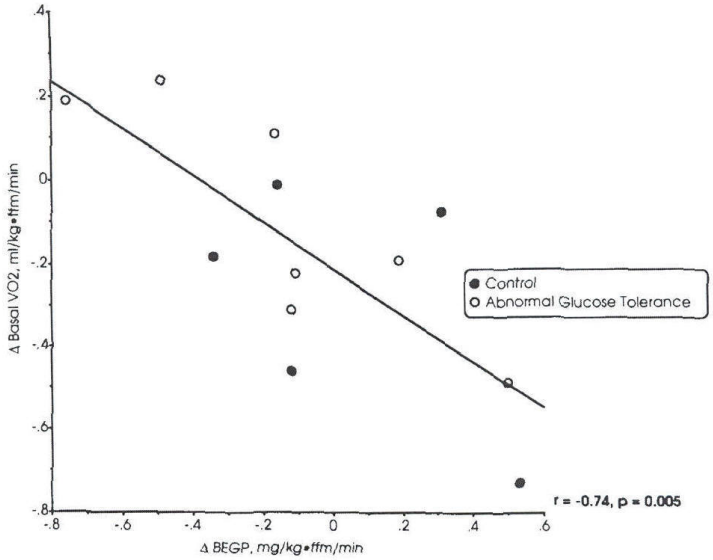

FIGURE 3-8 Alterations in basal VO_2 per kilogram of FFM per minute in relation to changes in basal endogenous glucose production
SOURCE: Catalano et al., 1998. Reprinted from *American Journal of Obstetrics and Gynecology*, Volume 179, Issue 1, Catalano P. M., N. M. Roman-Drago, S. B. Amini, and E. A. Sims, Longitudinal changes in body composition and energy balance in lean women with normal and abnormal glucose tolerance during pregnancy, pages 156-165. Copyright (2008), with permission from Elsevier.

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FETAL-PLACENTAL PHYSIOLOGY

Transport Function

Three primary functions of the placenta are to serve as a barrier or filter, to transport substances between maternal and fetal circulation, and to cover a large spectrum of endocrine activity. Changes in fetal growth rate have been shown to result from effects related to maternal homeostasis and associated changes in placental structure or function in both normal and non-normal pregnancies (Thame et al., 2004; Desoye and Kaufman, 2005; MacLaughlin et al., 2005; Swanson and Bewtra, 2008). Changes in the maternal environment have been shown to have an impact on specific steps of placental transport of the major energy substrates (i.e., glucose, lipids, amino acids; Hauguel de-Mouzon and Shafrir, 2001). For example, maternal diabetes results in increased availability of glucose, which is transported directly across the placenta for fetal utilization (Baumann et al., 2002). In contrast to glucose, which is transported along a concentration gradient, regulation of lipid transfer from maternal to fetal circulation is more complex. The placenta has the capacity to regulate the uptake, storage, and release of maternal lipids through multiple regulatory mechanisms and thus control fetal plasma lipid composition (Haggarty, 2002). Changes in the maternal environment may also modify placental endocrine function. For example, changes in maternal circulating cholesterol affect lipid metabolism in human term placenta (Marseille-Tremblay et al., 2008). Higher cholesterol uptake may subsequently impact steroidogenesis because cholesterol is the primary precursor for progesterone synthesis (Pasqualini, 2005).

Interaction of Maternal and Placental Metabolism

The question of whether or how placental function(s) may have an impact on maternal metabolism has received little attention. Besides the uterus, the feto-placental unit, intra- and extravascular fluids, and mammary gland, most of the weight gain that occurs over the course of a pregnancy lies in changes in maternal adipose tissue mass. In this context, the placental contribution to weight changes through the action of systemic factors that control the pathways of lipid synthesis and storage within the adipocyte must be taken into consideration. The placenta does not release adipogenic substrates into the maternal circulation. Hence, the most probable routes by which placental function would alter the regulation of lipogenic pathways are modulation of maternal insulin sensitivity and inflammation, as discussed previously.

Placental Hormone Production

Sex steroids and hPL, which best reflect the endocrine function of the placenta, have been primary candidates for regulation of maternal insulin sensitivity (Leturque et al., 1989). Although estrogens certainly have insulin sensitizing properties, the action of progesterone is clearly linked to diminishing insulin sensitivity and weight gain (Kalkhoff, 1982; Gonzalez et al., 2000; Xiang et al., 2007). Hence, an imbalance in placental progesterone production may be a contributing factor to maternal weight regulation. Human placental lactogen is the most abundant polypeptide hormone produced by the placenta with strong anabolic and lipolytic properties. Inasmuch as hPL enhances maternal nitrogen accrual, this process could possibly contribute to weight regulation and that possibility has been the subject of speculation (Florini et al., 1966). However, the lipolytic action of hPL on adipose tissue has received more experimental support. One consequence of the lipolytic effect of hPL is the re-orientation of maternal metabolism

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toward lipid rather than glucose utilization to favor glucose sparing for the fetus. Interestingly the ability of hPL to mediate pregnancy-induced insulin resistance, as suggested by Grumbach et al. (1968) was never fully established. Thus, the exact contribution of hPL to the regulation of maternal homeostasis remains to be established. Further, whether hPL synthesis is modified in pathologic pregnancies also has not been confirmed (Stewart et al., 1989). Just as occurs in white adipose tissue, the placenta synthesizes a large array of cytokines (Hauguel-de Mouzon and Guerre-Millo, 2006; Desoye and Hauguel-de Mouzon, 2007). All placenta-derived cytokines except leptin, which is released in large amounts in maternal circulation, likely act in either a paracrine or autocrine manner. Obesity and diabetes are associated with increased placental leptin production and maternal hyperleptinemia, but the consequences of high systemic leptin are unclear at this time (Hauguel-de Mouzon et al., 2006). Resistance to the central satiety effect of leptin during pregnancy as a possible consequence has been considered (Grattan et al., 2007).

Another potential contribution of the placenta to the regulation of maternal metabolism and subsequent alteration in maternal weight gain is systemic inflammatory priming by circulating syncytiotrophoblast microparticles (STBMs). Syncytiotrophoblast microparticles bind to monocytes and stimulate the production of inflammatory cytokines (Germain et al., 2007; Rovere-Querini et al., 2007). In addition to local placental inflammation, these microparticles are potential contributors to the altered systemic inflammatory response in pregnancy (Challier et al., 2008). Consequently, increased macrophage infiltration into maternal adipose tissue in combination with increased insulin resistance may contribute to the regulation of adipose mass during pregnancy (Xu et al., 2003).

Taken together there is little direct evidence that placental hormonal factors directly regulate maternal homeostasis and particularly quantitative changes in adipose tissue mass. The role of progesterone, hPL, and leptin in maternal insulin sensitivity and energy homeostasis remains to be established, but inflammatory mechanisms are novel potential regulatory pathways that will also have to be examined.

ABNORMAL MATERNAL METABOLISM

Weight Loss During Pregnancy

Weight loss or no GWG as a result of dietary caloric insufficiency will induce maternal hormonal and metabolic responses. Given the obligatory weight gain in the maternal tissues (uterus, breast, blood), and the fetal-placental unit, a weight gain less than ~7.5-8.5 kg would imply mobilization of maternal adipose tissue and possibly protein stores. Metabolic profile, dietary patterns, and eating behaviors of pregnant women undergoing weight loss or no weight gain have not been studied, but expected changes in fuel homeostasis can be deduced from studies conducted in pregnant women subjected to fasting.

Fasting in Pregnant Women

During 84 hours of fasting before elective termination of pregnancy at 16-20 weeks' gestation, ketonemia, increased urinary nitrogen excretion, and exaggerated reduction in gluconeogenic amino acids were detected in pregnant women (Felig, 1973). Glucose and insulin were lower, and acetoacetate and β-hydroxybutyrate were two to three times higher, in pregnant than non-pregnant women at 12-60 hours but not 84 hours (Felig and Lynch, 1970). Weight loss averaged 3.1 kg in non-pregnant women and 3.2 kg in pregnant women. Metzger et al. (1982)

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subjected lean (*n* = 11) and obese (*n* = 10) pregnant women and lean (*n* = 14) and obese (*n* = 13) non-pregnant controls to an 18-hour fast. At 12 hours, there were no significant differences between groups, but by 16 and 18 hours, substantial increases in FFA and β-hydroxybutyrate (βHA), which were inversely correlated with glucose, were seen in the pregnant women. There was a significant difference in FFA concentrations between obese and lean pregnant women only at 16 hrs of fasting. In contrast there were no significant differences in BHA levels at any time point between lean and obese women.

Ketonuria and Ketonemia in Pregnancy

As first described by Freinkel (1980), pregnancy can be considered a condition of "accelerated starvation" because of the changes in maternal metabolism. The accelerated starvation occurs because of the increase in insulin resistance, particularly related to lipid metabolism, as discussed previously. As a result, there is an increased risk of developing ketonuria and ketonemia in pregnancy even among women with normal glucose tolerance. Chez and Curcio (1987) reported that eight of nine women with clinically normal pregnancies developed ketonuria at various times during their pregnancy. Gin et al. (2006) measured capillary blood ketones and βHA using a portable capillary meter in women with normal glucose tolerance (controls) and those with GDM three times a day from 25 to 37 weeks' gestation. Fasting ketonuria was strongly correlated with ketonemia in controls but not in women with GDM. There was a chronic increase in ketonemia levels in 12 percent of the controls and 47 percent of the women with GDM.

Pregnant women develop ketonemia much earlier than non-pregnant women during prolonged fasting because of the accelerated starvation. Felig (1973) studied women between 16 and 22 weeks' gestation who elected termination of pregnancy and were willing to undergo prolonged fasting and compared them with a non-pregnant control group. After an overnight fast of at least 12 hours and for the first 36 to 60 hours of starvation, blood βHA and acetoacetate concentrations were two- to threefold higher in the pregnant group than in the non-pregnant group. The increase in lipolysis among the pregnant women was attributed to increases in hPL. The ketone concentrations in maternal blood were equivalent to those in amniotic fluid and were fortyfold above levels in fed subjects. The assumption is that amniotic fluid levels represent maternal-to-fetal transport. Felig (1973) also hypothesized that ketones become an important metabolic fuel for the fetal brain once glucose concentrations decrease, because the human fetal brain has the enzymes necessary for ketone oxidation.

Coetzee et al. (1980) reported that 19 percent of obese, insulin-dependent diabetic women on 1,000-kilocalorie (kcal) diets developed ketonuria. In contrast, in diabetic women eating higher-energy diets, only 14 percent had ketonuria, and in pregnant non-diabetic women, only 7 percent developed ketonuria. Measurement of blood ketones was never positive if the urine measure was ≤ 2 plus and acetoacetate levels were always less than 1 mmol/L. There was no difference in neonatal outcomes among the three groups.

In summary, pregnant women are more likely to develop elevated measures of blood βHA and acetoacetate during prolonged fasting (after 12-18 hours) as a result of the metabolic and hormonal changes in pregnancy. Pregnant women with diabetes are more likely to develop elevated blood ketones than women with normal glucose tolerance. Nevertheless, a substantial proportion of pregnant women with normal glucose tolerance have elevated blood ketone levels some time during gestation. Although the evidence is based on associations and does not demonstrate causality, caution should be exercised regarding weight loss during pregnancy or no

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GWG given the propensity to develop ketonemia, increased urinary nitrogen excretion, and decreased gluconeogenic amino acids and the potential to adversely impact the neurocognitive development of the offspring. There are significant consequences of caloric insufficiency, low GWG, and poorly controlled diabetes for the child and these are discussed in Chapter 6.

FINDINGS AND RECOMMENDATIONS

Findings

1. Total GWG in normal-term pregnancies displays considerable variability; nevertheless, some generalizations can be made regarding mean tendencies and patterns of GWG:

    a. A consistent inverse relationship is observed between GWG and pregravid BMI category;
    b. Mean GWG ranges from 10.0 to 16.7 kg in normal weight adults and 14.6 to 18.0 kg in adolescents giving birth to term infants;
    c. The pattern of GWG is most commonly described as sigmoidal, with mean weight gains higher in the second than the third trimester across BMI categories, except for obese women; and
    d. Lower GWGs, on the order of 11 kg and 9 kg, have been confirmed in large cohorts of obese women and very obese women, respectively.

2. The committee relied on observational GWG data of women giving birth to twins born at 37-42 weeks of gestation and with an average twin birth weight ≥ 2,500 g:

    a. Mean GWG of normal weight women with twin births ranged from 15.5 to 21.8 kg;
    b. GWG for triplets ranged from 20.5 to 23.0 kg at 32-34 weeks and for quadruplets from 20.8 to 31.0 kg at 31-32 weeks; and

3. When stratified by WHO prepregnancy BMI categories sample sizes from data on twins was insufficient to designate a value for underweight women with pregravid BMI <18.5 kg/m^2.
4. The extent to which fat mass accretion is critical rather than incidental to pregnancy is not clear, but unrestrained weight gain leads to postpartum weight retention.
5. Placental size is strongly correlated with fetal growth, averaging approximately 500 g in singleton pregnancies.
6. Amniotic fluid weight may affect maternal gestational weight gain by as much as 1 kg at term.
7. Gestational gains in weight, total body water, total body potassium, protein, and FFM, but not FM, are positively correlated with birth weight across all BMI categories.
8. Poor plasma volume expansion is associated with a poorly growing fetus and poor reproductive performance.
9. Pregnancy is a condition of systemic inflammation that also influences maternal and fetal nutrient utilization.
10. During prolonged fasting, i.e. 16-18 hours, pregnant women are more likely to develop

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elevated measures of blood βHA and acetoacetate. In women with diabetes, plasma FFA and βHA were inversely associated with intellectual development of the offspring at 3-5 years of age. Therefore, caution is warranted regarding periods of prolonged fasting and weight loss during pregnancy and the development of ketonuria.

Research Recommendations

Research Recommendation 3-1: The committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to conduct studies in all classes of obese women, stratified by the severity of obesity, on the determinants and impact of GWG, pattern of weight gain and its composition on maternal and child outcomes.

Research Recommendation 3-2: The committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to conduct studies on the eating behaviors, patterns of dietary intake and physical activity, and metabolic profiles of pregnant women, especially obese women, who experience low gain or weight loss during pregnancy. In addition, the committee recommends that researchers conduct studies on the effects of weight loss or low GWG, including periods of prolonged fasting and the development of ketonuria/ketonemia during gestation, on growth and on development and long-term neurocognitive function in the offspring.

Areas for Additional Investigation

The committee identified the following areas for further investigation to support its research recommendation. The research community should conduct studies on:

- Potential effects of maternal weight loss on components of maternal body composition for both the mother and the fetus, particularly in obese women; and
- Mechanisms by which placental hormonal factors and systemic inflammation impact the regulation of maternal metabolism during pregnancy.

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4

Determinants of Gestational Weight Gain

The total amount of weight gain during pregnancy is determined by many factors. Aside from physiological factors (discussed in Chapter 2); psychological, behavioral, family, social, cultural, and environmental factors can also have an impact on gestational weight gain (GWG). Understanding these factors as determinants of GWG is an important component of revising weight gain guidelines for women during pregnancy.

Several conceptual models guided the committee's consideration of determinants of GWG. The ecological perspective recognizes that health behavior such as GWG is influenced at multiple levels. Brofenbrenner (1979) identified multiple levels of environmental influence on health behavior in general:

- The microsystem – face-to-face interactions in specific settings, such as family, school, or a peer group;
- The mesosystem (a system of microsystems) – the interrelations among the various settings in which the individual is involved, such as that between the family and the workplace;
- The exosystem – the larger social system in which the individual is embedded, such as the extended family or community; and
- The macrosystem – cultural values and beliefs, such as cultural beliefs about GWG.

Other models which recognize the multiple determinants of health behavior or outcome include the health field model, which identifies multiple domains, including: the physical and social environments that exert influences on health behavior and outcome; and the epidemiological model which describes a triad of epidemiologic factors to model the complex and interrelated factors contributing to the increasing rate of obesity in the United States and other countries. One of the triad components describes an "obesogenic" environment as "the sum of influences that the surroundings, opportunities, or conditions of life have on promoting obesity in individuals or populations" (Swinburn and Egger, 2002). This obesogenic environment includes physical, economic, policy, and socio-cultural factors that can influence eating and physical activity behaviors.

Collectively, these models place emphasis on how the health of individuals is influenced by not only physiological functioning and genetic predisposition, but by a complex interplay of these biological determinants with social and familial relationships, environmental influences, and broader social and economic contexts over the life course. They further suggest that intervention efforts to change health behavior or outcome, such as GWG, should address not only "downstream" individual-level phenomena (e.g. physiologic pathways to disease, individual and lifestyle factors) and "mainstream" factors (e.g. population-based interventions), but also "upstream," societal-level phenomena (e.g. public policies) (IOM, 2000).

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Another model, the life-course perspective (Kuh and Ben-Shlomo, 1997), perceives life not in disconnected stages, but as an integrated continuum; it recognizes that each stage of life is influenced by the life stages that precede it, and it, in turn, influences the life stages that follow (See Chapter 6 for detailed discussion).

Some of the most significant determinants of GWG at multiple levels (social/institutional, environmental, neighborhood/community, interpersonal/family, and individual levels) occur across the life course (Figure 4-1). The following discussion begins with a review of the evidence for a direct relationship between a given determinant (identified in the Conceptual Framework) and GWG. Where data are lacking, rationale are provided for why the committee thinks that it is potentially an important determinant which merits further research. The committee's review of evidence (tabulated in Appendix D) included both epidemiologic and clinical studies. Since this research discipline is focused largely on observational studies the committee recognized the need for proof of causality for determinants and outcomes significantly associated with GWG.

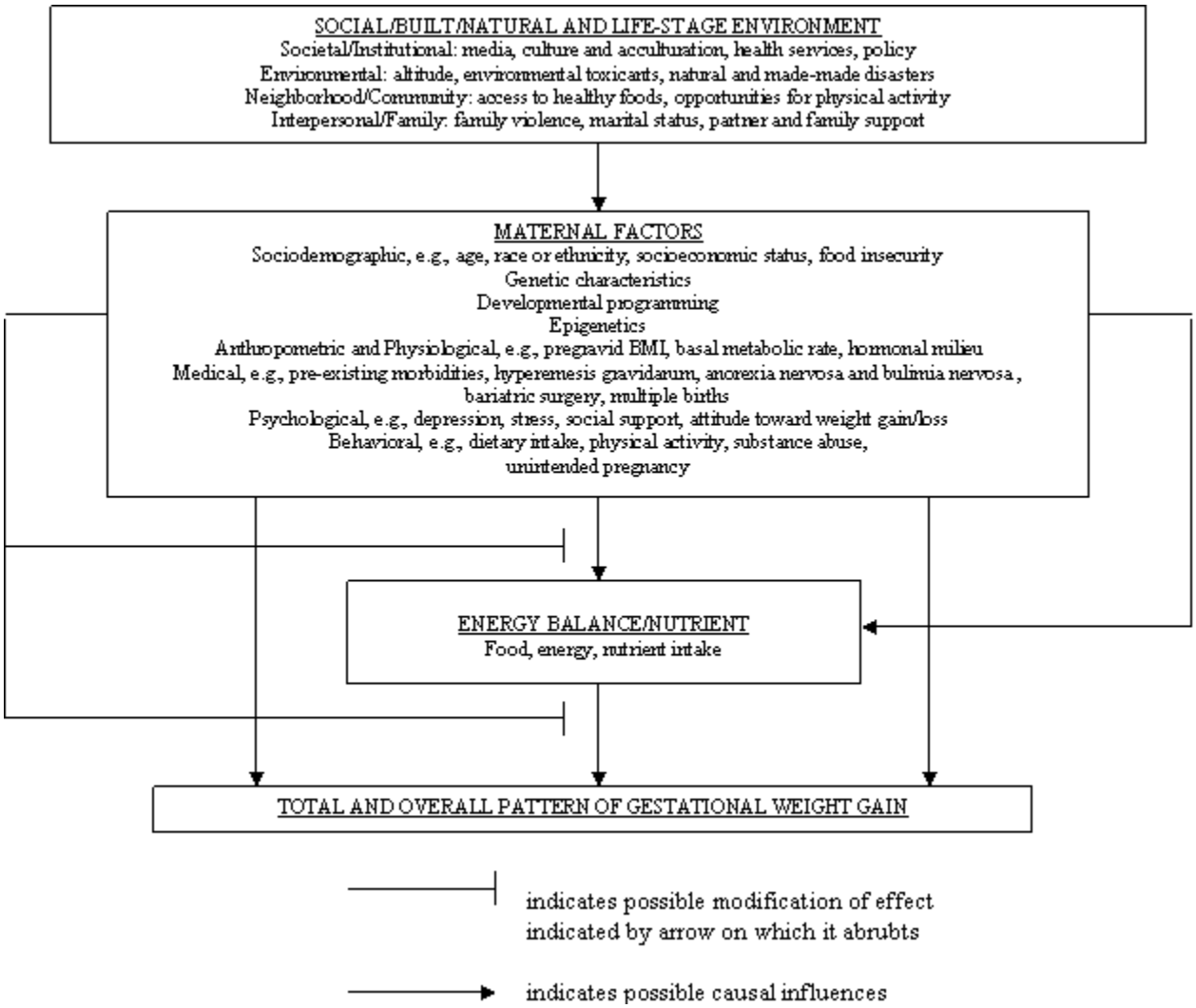

FIGURE 4-1 Schematic summary of determinants associated with GWG.

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SOCIETAL/INSTITUTIONAL DETERMINANTS

Media

The committee was unable to identify studies that examined specifically the media's influence on GWG. From a life course perspective, however, it is plausible that the media may influence GWG by having an effect on eating and exercise habits that are established well before pregnancy. Several previous reports have documented the influence of advertising and marketing on children's food, beverage, and sedentary-pursuit choices that can adversely affect energy balance (Kunkel, 2001; IOM, 2006). An extensive systematic literature review concluded that food advertisements promote food purchase requests by children to parents, have an impact on children's product and brand preferences, and affect consumption behavior (Hastings et al., 2003). Other studies have shown that the media can encourage sedentary behaviors, such as television watching, that may adversely affect energy balance (Gortmaker et al., 1996; Gortmaker et al., 1999; Robinson, 1999; IOM, 2005; Epstein et al., 2008). Such eating habits and sedentary behaviors shaped during childhood and adolescence may be carried into young adulthood and continued into pregnancy and thus indirectly affect GWG. Moreover, by influencing energy balance over the long run these habits and behaviors may also have an impact on prepregnancy body mass index (BMI) as well as other biological determinants of GWG.

Not all media influences are negative, however. Media can be used to convey consumer information and public health messages, such as those regarding youth smoking, and seat belt and child car seat use. Social marketing programs that have used the media, either as focused efforts or as part of multi-component campaigns to promote physical activity or healthy diet in adults, have produced mixed results, often because discerning their impact is a challenge. The most successful social marketing programs have received more funding, been better sustained, and were shaped by formative research (IOM, 2006).

Taken together, available evidence is inconclusive about the media's influence on GWG. However, it is plausible that the media may exert its influence indirectly by affecting prepregnancy BMI and other biological determinants, as well as influencing eating habits and sedentary behaviors that are established well before pregnancy.

Culture and Acculturation

The committee was unable to identify studies that examined specifically the effects of culture and acculturation factors on GWG. However, cultural norms and beliefs may influence dietary behavior and physical activity, thereby affecting energy balance and, indirectly, GWG. For example, there is a belief among women of all ages, ethnic groups, and income and education levels that the consumption of certain foods marks a child before birth; which may then lead to certain food preferences and avoidances (IOM, 1992; King, 2000). There is also the belief that diet can influence ease of delivery. Most women know that low GWG will produce a small infant, which will be delivered more easily than a larger one. In some cultures this knowledge may encourage women to "eat down" in late pregnancy in order to avoid a difficult birth (King, 2000). Understanding these cultural norms and beliefs is important for effectively communicating recommendations for GWG.

Acculturation, the process in which members of one cultural group adopt the beliefs and behaviors of another, is often associated with adoption of unhealthy behaviors, including food choices. Using nativity or duration of residence in the U.S. as a proxy for acculturation, several

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studies have found greater rates of overweight and obesity among children and non-pregnant adults who are more acculturated, compared to their less acculturated counterparts (Lizarzaburu and Palinkas, 2002; Hubert et al., 2005; Hernandez-Valero et al., 2007; Fuentes-Afflick and Hessol, 2008). A population-based study of 462 mothers in California found that in the three months before pregnancy, foreign-born Latinas had the lowest contribution of fat to total energy intake and the highest dietary intake of carbohydrate, cholesterol, fiber, grain products, protein foods, folate, vitamin C, iron, and zinc, compared to the dietary intake of white non-Latinas and U.S.-born Latinas (Schaffer et al., 1998). Other researchers have also documented increased risk for adverse birth outcomes, including preterm birth and low birth weight, among U.S.-born women compared to foreign-born women of the same ethnic origin (Ventura and Taffel, 1985; Scribner and Dwyer, 1989; Cabral et al., 1990; Kleinman et al., 1991; Rumbaut and Weeks, 1996; Singh and Yu, 1996; Fuentes-Afflick and Lurie, 1997; Jones and Bond, 1999; Callister and Birkhead, 2002; Baker and Hellerstedt, 2006). In these studies, however, GWG is usually not reported, and so the contribution of GWG to adverse outcomes is unclear. Taken together, there is indirect evidence that cultural and acculturation factors can influence GWG even though conclusive evidence for a direct effect is lacking.

Health Services

The U.S. Public Health Service Expert Panel on the Content of Prenatal Care recommended that pregnant women receive advice on gaining an appropriate amount of weight during pregnancy even though an influence of weight gain advice on GWG has not been conclusively demonstrated (DHHS, 1989). Several intervention studies have been conducted using nutrition advice alone (Orstead et al., 1985; Bruce and Tchabo, 1989) or such advice linked with home visits by nutritionists and supplemental food (Rush, 1981; Bruce and Tchabo, 1989), a nurse home visitation program (Olds et al., 1986), and the provision of prenatal care through multidisciplinary rather than traditional clinics (Morris et al., 1993). In three of the studies (Rush, 1981; Olds et al., 1986; Morris et al., 1993) the differences in mean GWG between intervention and control groups were not statistically significant. In two other studies (Orstead et al., 1985; Bruce and Tchabo, 1989) intervention groups gained significantly more weight than the control groups; however, the findings may be limited by gestational age bias. Additionally, only mean GWG was reported in the studies and no comparisons were made using different cutoff points based on pregravid BMI further limiting interpretation of the findings.

Brown et al. (1992) developed a prenatal weight gain intervention program based on social marketing methods. While circumstances arose that hampered full evaluation of the program, preliminary evidence suggests that GWG and birth weight of African Americans in the intervention group did not differ significantly from those of whites, whereas both weight gain and birth weight were significantly lower in African Americans than in whites in the control group.

Hickey (2000) identified several threats to the validity of previous studies on prenatal weight gain advice and actual GWG. These include, in addition to differences in pregravid nutritional status and BMI, issues such as self-selection bias, recall bias, differences in time during gestation when nutrition advice was given, variation in content and frequency of advice, the pairing of advice with other food or nonfood interventions, individual and social characteristics of the provider as contrasted with those of the pregnant woman, and racial-ethnic and socioeconomic disparities in weight gain advice given to women. Given all these threats, the evidence to evaluate the influence of prenatal weight gain advice on actual GWG is weak.

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Policy

For the purpose of this report, policy is defined broadly to include principles, guidelines, or plans adopted by an organization to guide decisions, actions, and other matters. As an example of how policy can influence GWG, the weight gain recommendations from the report, *Nutrition During Pregnancy* (IOM, 1990) have been endorsed by obstetric organizations in the United States and many other countries. A 2005 cross-sectional survey mailed to 1,806 practicing members of the American College of Obstetricians and Gynecologists (ACOG) showed that more than 85 percent of the 900 respondents report counseling their patients about GWG often or most of the time (ACOG, 2005). The survey did not, however, assess the respondents' knowledge of the IOM (1990) guidelines or the content of counseling (Power et al., 2006).

The few studies that have examined the advice given for GWG, however, have shown that women often receive inconsistent or erroneous advice. Cogswell et al. (1999) found in a survey of approximately 2,300 women that they appear to be influenced by professional advice in the weight gain they believe is appropriate and the weight gain they actually achieve. Of the 1,643 women who recalled weight gain advice, 14 percent reported being advised to gain less than the recommended levels, and 22 percent were advised to gain more. Provider advice to gain below the recommended levels was associated with actual weight gain below the recommendations (an adjusted odds ratio of 3.6), and advice above the guidelines had the same odds ratio for higher rates of gain. Moreover, 27 percent of women reported receiving no advice about GWG; thus nearly two-thirds (63 percent) of women in this study reported receiving no advice or inappropriate advice from health professionals regarding GWG.

In a more recent study, Stotland et al. (2005) found that 79 percent of the nearly 1,200 women of all BMI ranges in the study reported a target GWG, or how much weight women think they should gain during pregnancy, within the IOM (1990) guidelines, as compared to only 59 percent reported in Cogswell et al. (1999). Given that the women in the Cogswell et al. cohort delivered in 1993, the authors speculated that the IOM (1990) guidelines are now more widely applied or accepted than they were in 1993. Still, Stotland et al. (2005) found that one-third (33 percent) of women received no advice from health professionals regarding GWG, and less than half (49 percent) reported receiving advice within guidelines. In sum, the IOM (1990) guidelines appear to influence what women believe to be appropriate weight gain during pregnancy, though their influence on actual GWG may be less, in part because many health professionals are providing no or inappropriate advice about GWG.

Another example of how policy can influence GWG is the Special Supplemental Food Program for Women, Infants and Children (WIC). Rush et al. (1988) conducted a national evaluation of WIC programs and found that a reversal of low weight gain in early pregnancy and greater total weight gain during pregnancy occurred among women who enrolled in WIC compared with controls. They also found greater intake of protein, iron, calcium, vitamin C, and energy among WIC participants. However, subsequent evaluations (Joyce et al., 2008) have challenged these earlier findings and found more limited associations between WIC participation and GWG. Nonetheless, it is possible that policy that increases food access would have an impact on dietary pattern and GWG.

Policy that does not directly affect pregnant women can also have an effect on GWG. Examples include policy recommendations to restrict food and beverage advertising and marketing to young children, to develop and implement nutritional standards for all competitive foods and beverages sold or served in schools, or to promote physical activity in schools (IOM,

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2007). These policies can influence the development of children's eating and exercise habits, which will be important later in life.

ENVIRONMENTAL DETERMINANTS

Altitude

There is inconsistent evidence about the influence of altitude on GWG. Jensen and Moore (1997) examined the effect of high altitude on GWG and birth weight using Colorado birth certificates. They did not find any significant difference in GWG among women residing at 3,000 to 5,000 feet; 5,000 to 7,000 feet; 7,000 to 9,000 feet; and 9,000 to 11,000 feet. Mean birth weight, however, decreased and low birth weight rates increased with increasing altitude. The decline in birth weight associated with increase in altitude was found to be independent of and not interactive with gestational age, GWG, parity, maternal smoking, pregnancy-induced hypertension and other factors associated with birth weight (Jensen and Moore, 1997).

Environmental Toxicants

The committee was unable to identify studies that examined specifically the effects of exposures to environmental toxicants on GWG. There is some evidence linking environmental contaminants such as organophosphate and organochlorine compounds to fetal growth, but the evidence is inconsistent (Dar et al. 1992; Wolff et al., 2007). Additional research may better define the relationships among environmental exposures, GWG, and fetal growth.

Natural and Man-made Disasters

The committee was unable to identify studies that examined specifically the effects of natural or man-made disasters on GWG. However, it is plausible that disasters can affect GWG indirectly by influencing resource availability (including food supply), healthcare access, and stress levels (Callaghan et al., 2007). Several studies have documented the impact of disasters on pregnancy outcomes such as preterm birth (Weissman et al., 1989; Cordero, 1993; Glynn et al., 2001; Lederman et al., 2004) and fetal growth restriction (Eskenazi et al., 2007; Landrigan et al., 2008); however, it remains unclear whether these adverse outcomes were caused by low GWG.

NEIGHBORHOOD/COMMUNITY DETERMINANTS

Access to Healthy Foods

Evidence for a direct influence of neighborhood or community factors, such as access to healthy foods, on GWG is lacking. However, because appropriate nutrient intake and weight gain during pregnancy requires a safe and adequate food supply, it is likely women who live in areas where residents have poor accessibility to foods may be at increased risk for inadequate or inappropriate GWG and associated poor pregnancy outcomes (See Chapter 2 for trends in dietary practices and Appendix B for supplemental information). A study of urban retail food markets and birth weight outcomes in upstate New York found pregnant women who lived in proximity to urban retail corner markets without fresh produce, dairy, and other healthy foods had significantly more low birth weight infants compared to women who had access to supermarkets where healthy foods were available. These findings were independent of income level; however,

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the study did not report on GWG (Lane et al., 2008). Laraia et al. (2004) investigated associations between the distance of a supermarket from home and diet quality of pregnant women, measured by a Diet Quality Index (DQI). They found that women who lived more than four miles from a supermarket had a two-fold greater risk of falling into the lowest DQI quartile compared to women who lived ≤ 2 miles from a supermarket, but the authors also did not report on GWG.

Opportunities for Physical Activity

While a growing body of evidence has demonstrated the role of the built environment for populations at high risk for obesity (See Chapter 2 for trends in physical activity), only one study was identified that examined the relationship between neighborhood contexts and GWG. Laraia et al. (2007) conducted a study of neighborhood factors associated with physical activity and weight gain during pregnancy. They found that social spaces, defined as the presence of parks, sidewalks, and porches as well as the presence of people, including nonresidential visitors, was associated with decreased odds for inadequate or excessive GWG. The social spaces scale was also associated with decreased odds of living greater than three miles from a supermarket. These findings suggest that neighborhood environments can influence GWG by providing access to healthy foods and opportunities for physical activities.

INTERPERSONAL/FAMILY DETERMINANTS

Family Violence

Several studies examined GWG in the context of family violence (Parker et al., 1994; McFarlane et al., 1996; Siega-Riz and Hobel, 1997; Moraes et al., 2006). Siega-Riz and Hobel (1997) found in a clinic sample of 4,791 Hispanic women in Los Angeles that physical abuse was associated with a greater than threefold risk for inadequate GWG among obese and overweight women. Moraes et al. (2006) found in a study of 394 pregnant women in Brazil that those with the highest physical abuse score gained, on average, 3 to 4 kg less than women unexposed to intimate partner violence. Boy and Salihu (2004) conducted a systematic review and found that abused pregnant women had less GWG than non-abused women. These studies suggest an association between intimate partner violence and insufficient GWG.

Marital Status

Several studies have examined the relationship between marital status and GWG. Kleinman et al. (1991) and Ventura (1994), who used 1992 U.S. national data, found that unmarried mothers were more likely than married mothers to gain less than 7.3 kg during pregnancy. Olsen and Strawderman (2003) found in a cohort of 622 healthy adult women that 38 percent of married women had gained above IOM (1990) guidelines, compared to 42 percent for women who were separated or divorced, and 48 percent for single women. They also found that 21 percent of married women had gained below IOM (1990) guidelines, compared to 23 percent for single women and 29 percent for women who were separated or divorced. Thus married women were more likely to gain within the IOM (1990) recommended weight gain range than single or separated/divorced women.

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Partner and Family Support

Evidence to support a relationship between partner support and GWG is lacking at this time. One study examined the influences of partner support on attitudes or behaviors toward GWG. Dipietro et al. (2003) found in a cross-sectional study of 130 women with low-risk pregnancies that partner support was associated with negative pregnancy body image, but not with attitudes or behaviors toward GWG. Siega-Riz and Hobel (1997) found that receiving financial support from the infant's father was significantly associated with decreased risk of poor GWG for overweight and obese women, but not for underweight or normal weight women.

Several studies have examined the influence of family support on GWG. Stevens-Simon et al. (1993) found in a sample of 99 pregnant adolescents that attitudes toward GWG were directly related to their perceived family support. Negative weight gain attitudes were most common among heavier adolescents, depressed adolescents, and adolescents who did not perceive their families as supportive. Gutierrez (1999) reported from a study of 46 pregnant Mexican American adolescents that the most powerful factors contributing to good food practices during pregnancy were maternal concern about the well-being of the infant, role of motherhood, and family support system; the contribution of family support to GWG attitudes or actual GWG was not reported in this study.

MATERNAL FACTORS

Sociodemographic Factors

Gestational Weight Gain in Adolescents

Adolescent pregnancy has been associated with increased risk of preterm delivery, low birth weight, SGA births, and increased risk of neonatal mortality, although reported risk associations vary (Chen et al., 2007). To reduce these risks, the IOM (1990) report recommended that pregnant adolescents gain weight within the ranges for adult women unless they were under 16 years of age or less than two years post-menarche. In either of these cases, adolescents were encouraged to gain at the upper limits of the GWG guidelines for their prepregnancy BMI category.

The youngest adolescents as well as somewhat older adolescents who conceive soon after menarche may still be growing themselves (Scholl and Hediger, 1993). Even girls who become pregnant for a second time during adolescence may still be growing. Scholl et al. (1990) showed that adolescents who were still growing during a first pregnancy delivered infants whose birth weight did not differ from those who were not growing. This was not true among adolescents who were still growing during a second pregnancy; their infants were significantly lighter at birth than those who were not growing themselves. The possibility of a competition for nutrients between the still-growing adolescent gravida and her fetus has been advanced as an argument for recommending relatively higher gains for at least some pregnant adolescents. What has been found instead is that still-growing adolescents are not mobilizing their fat gain during pregnancy to enhance fetal growth but, rather, are supporting the continued development of their own fat stores (Scholl et al., 1994).

In a retrospective review of natality data from 2000, Howie et al. (2003) reported an increased likelihood for excessive GWG among adolescents compared to older women. Other

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authors have corroborated that younger adolescents have a higher GWG compared to older adolescents and adults, but whether the infant benefits from this greater weight-gain is not yet clear (Hediger et al. 1990; Scholl et al., 1990; Stevens-Simon et al., 1993). This is in part because—as is also the case for adult women—increases in GWG not only reduce the risk of delivering a low birth weight infant but also increases the risk of delivering a macrosomic infant (Scholl et al., 1988). Nielsen et al. (2006) showed that birth weight outcomes improved in all prepregnancy BMI groups when GWG increased from below to within the lower half of the weight gain recommended by IOM (1990) in a cohort of 815 pregnant African-American adolescents. Further gains were not beneficial, particularly for infants of adolescents with a high prepregnancy BMI.

The possibility that adolescents who gained at the upper end of the range for their BMI category might have an excess risk of postpartum weight retention or the later development of obesity was not considered in formulating the 1990 guidelines, but has long been recognized as a possible downside of recommending relatively high weight gains for them (McAnarney and Stevens-Simon, 1993). Adolescents who have given birth are heavier (Gigante et al., 2005) with more adipose tissue (Gunderson et al., 2009) than adolescents who have not. Gestational weight gain was a significant predictor of increase in BMI 6 and 9 years post delivery in all prepregnancy BMI categories among the 330 primiparous black adolescents studied by Groth (2008). In addition, those who gained above the IOM (1990) guidelines were more likely to have become obese by 9 years post delivery than those who gained within the guidelines.

In summary, the relationship of GWG to fetal and birth outcomes, postpartum weight retention, and risk for future overweight/obesity appears to be generally similar to that for adult women. However, information on these subjects is more limited for pregnancy among adolescents, particularly younger adolescents, than it is for adult women. Data generated since the IOM (1990) report, particularly related to the risk of developing postpartum weight retention and obesity in adult women who had been pregnant as young adolescents, support the recommendation that "…until more is known, adolescents less than two years post-menarche should be advised to stay within the IOM-recommended BMI-specific weight range without either restricting weight or encouraging weight gain at the upper end of the range" (Suitor, 1997).

Gestational Weight Gain in Older Women

Increased maternal age is significantly associated with risk for adverse pregnancy outcomes, including stillbirth (Fretts, 2005; Reddy et al., 2006), low birth weight, preterm birth, and SGA (Cnattingius et al., 1992; Delpisheh et al., 2008). In addition to poor outcomes, pregnancy in older women is also associated with increased risk for pregnancy complications, e.g. hypertension, diabetes, placenta previa, and placental abruption (Joseph et al., 2005).

Women who become pregnant after age 35 differ from their younger counterparts in several factors that can influence pregnancy outcome, including prepregnancy weight (or BMI) and GWG. In a study of obese and non-obese women who were pregnant, Gross et al. (1980) found that a greater proportion of obese subjects were older and of higher parity than non-obese subjects. The obese subjects also had higher rates of chronic hypertension, diabetes, and inadequate GWG. Prysak et al. (1995), in a retrospective comparison of pregnancy characteristics between older (≥ 35 years old) and younger (25-29 years old) nulliparous women, found that the older women had significantly lower mean GWG than the younger women. In addition, obesity was significantly greater in the older compared to the younger women.

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Pregnant women over 35 years of age who were enrolled in the WIC program were evaluated by Endres et al. (1987) for nutrient intake, prepregnancy weight, and GWG compared to adolescents aged 15-18 years. Prepregnancy BMI was calculated for both groups and more than 50 percent of the older women were identified as obese prior to pregnancy. The study found no significant difference in total nutrient intake between the groups (neither met the RDAs), but the younger women had higher mean energy intakes ($P = 0.006$) and greater cumulative GWG in the third trimester (9.5 kg versus 7.6 kg) than the older women. In sum, several studies reported higher prepregnancy BMI and lower GWG among older women, compared to their younger counterparts. The contributions of GWG to birth outcomes, postpartum weight retention, and subsequent overweight/obesity among older women remain unclear.

Table 4-1 summarizes reports from the last three decades on GWG by age and racial/ethnic group.

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TABLE 4-1 Effect of Chronological Maternal Age on GWG

Reference	Age (yrs)	Racial/Ethnic Group	Number in Sample	Weight Gain (kg)	Coefficient of Variation, %
Ancri et al.	12-17	Caucasian	26	13.4	26
(1977)	18-19	(one black woman)	22	12.4	31
	20-24		24	11.1	17
	25-32		26	10.7	18
Frisancho et al.	12-13	Latin-American	28	9.0	18
(1983)	14		104	9.8	22
	15		296	9.9	26
	16		565	9.7	25
	17		229	10.0	26
	18-25		46	9.7	16
Horon et al.	< 16	Black, White	422	12.5	NR [a]
(1983)	20-24		422	12.5	NR
Loris et al.	13-15.9	Mixed group	18	17.2	23
(1985)	16-17.9		84	17.1	40
	18-19.9		25	17.3	54
Meserole et al.	13-15	Mixed group	24	14.5	32
(1984)	16-17		25	17.9	35
Endres et al.	15-18	Mixed group	46	12.0	NR
(1985)	19-30		198	11.0	NR
Muscati et al.	14-17	NR	90	16.5	36
(1988)	18-19		135	15.1	36
	20-35		461	13.8	39
Scholl et al. (1988)	16.9 ± 1.3 [b]	Black, White, Hispanic	696	14.7	39
Haiek and Lederman	< 16	Black Americans, Black	90	14.6	NR
(1989)	19-30	Latin Americans, White Non-Latin Americans, White Latin Americans	90	16.9	NR
Hediger et al.	≤ 18	Puerto Rican	304	13.7	±5.6 [b]
(1990)		Black	501	13.8	±5.7 [b]
		White	514	15.9	±5.7 [b]
Stevens-Simon et al.	< 16		52	14.9	±5.9 [b]
(1993)		N/A			
	16-19		89	13.9	±6.0 [b]
Prysak et al.	25-29	White or other	1,054	15.0	±4.9 [b]
(1995)	≥ 35		890	14.2	±5.4 [b]
Gutierrez (1999)	13-18	Mexican-American	46	14.5	±4.5
Nielsen et al. (2006)	< 17	African-American	776	14.5	±6.9

[a] NR = Not reported.
[b] Standard deviation.

SOURCE: Modified from IOM, 1990.

Race or Ethnicity

Few studies have examined racial-ethnic differences in GWG, and even fewer studies have considered the influence of the many possible determinants of GWG among different racial/ethnic groups or alternatively, adjusted for race/ethnicity in their analyses. Caulfield et al.

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(1996), for example, found that among 2617 black and 1253 white women delivering at a university hospital during 1987-1989 only 28.2 and 32.5 percent of black and white women, respectively gained within the ranges recommended by IOM (1990).

Black women are at increased risk for gaining less weight than recommended, when controlled for maternal pre-pregnancy BMI, height, parity, education, smoking, hypertension, duration of pregnancy, and fetal sex. Chu et al. (2009) assessed the amount of GWG among 52,988 underweight, normal weight, overweight, and obese U.S. women who delivered a singleton, full-term infant in 2004-2005 using PRAMS data (2004-2005). They found that black women were significantly more likely than white women to gain less than 15 pounds, but less likely than white women to gain more than 34 pounds. A review of birth records of 913,320 singleton births in New York City from 1995 to 2003 found that Asian and non-Hispanic black women were more likely to gain 0 to 9 kg, whereas Hispanic and non-Hispanic white women were more likely to gain 20+ kg during pregnancy (information contributed to the committee in consultation with Stein [see Appendix G, Part III). Table 4-2 presents GWG among women of different race and ethnicity in this study population.

Taken together, the limited data on the influence of race/ethnicity on GWG is suggestive of inadequate GWG among some racial/ethnic groups. However, the paucity of data on a national level and the lack of observational studies based on pre-pregnancy BMI preclude drawing any conclusions about the influence of race/ethnicity on GWG (see Chapter 2 and Figure 2-6 for trends in GWG for racial/ethnic groups by prepregnancy BMI).

TABLE 4-2 Bivariate Association between Gestational Weight Gain and Race or Ethnicity among Singleton Births, New York City, 1995–2003, *N* = 913,320

	Gestational Weight Gain			
	0 – 9 kg	**10 – 14 kg**	**15 – 19 kg**	**20+ kg**
	***N* = 234,764**	***N* = 333,968**	***N* = 223,366**	***N* = 121,192**
	***N* (percent)**	***N* (percent)**	***N* (percent)**	***N* (percent)**
Maternal race or ethnicity				
Non-Hispanic white	56,817 (20.3)	112,814 (40.4)	75,274 (26.9)	34,517 (12.3)
Non-Hispanic black	69,294 (29.2)	77,868 (32.8)	54,412 (22.9)	35,899 (15.1)
Hispanic	78,528 (26.9)	99,705 (34.1)	70,694 (24.2)	43,513 (14.9)
Asian	29,086 (29.0)	42,137 (41.9)	22,251 (22.1)	6,964 (6.9)
Other	1069 (30.1)	1,444 (40.7)	735 (20.7)	299 (8.4)

SOURCE: Information contributed to the committee in consultation with C. Stein (see Appendix G, Part III).

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Socioeconomic Status

The committee found limited evidence for associations between GWG and socioeconomic status (SES), and few studies that considered the influence of the many possible determinants of GWG among different SES groups; or alternatively, adjusted for SES in their analyses (see Appendix D). Using 2004-2005 PRAMS data, Chu et al. (2009) found that women with less than 12 years of education were more likely to gain less than 15 pounds, and less likely to gain more than 34 pounds, compared to women with more than 12 years of education (Table 4-3). This analysis, however, did not control for pre-pregnancy BMI or other factors that could potentially influenced GWG.

TABLE 4-3 Gestational Weight Gain (pounds) by Selected Characteristics among Women Delivering Full-term, Singleton Births (Underweight Women Excluded), PRAMS, 2004-2005

	≤ 14 (n = 8091)[a]		**15-24 (n = 9970)[a]**		**25-34 (n = 14,545)[a]**		**35-44 (n = 10,311)[a]**		**≥ 45 (n = 7112)[a]**	
Characteristic	**Percent[b]**	**SE[b]**	**Percent[b]**	**SE[b]**	**Percent[b]**	**SE[b]**	**Percent[b]**	**SE[b]**	**Percent[b]**	**SE[b]**
Age, y (n)[a]										
14-19 (5249)	15.4	0.8	16.9	0.8	25.7	0.9	20.4	0.9	21.7	0.9
20-24 (12,477)	15.3	0.5	19.3	0.5	26.7	0.6	20.3	0.5	18.4	0.5
25-29 (13,483)	15.8	0.5	18.6	0.5	28.5	0.6	22.2	0.5	15.0	0.5
30-34 (11,169)	15.1	0.5	18.6	0.5	30.8	0.6	22.1	0.6	13.4	0.5
≥ 35 (7651)	15.9	0.6	19.8	0.7	32.2	0.8	20.8	0.7	11.2	0.6
Race/ethnicity										
White (27,393)	13.3	0.3	17.4	0.3	30.0	0.4	22.7	0.4	16.6	0.3
Black (7790)	21.7	0.7	21.1	0.6	23.9	0.7	18.2	0.6	15.1	0.6
Hispanic (7428)	17.3	0.7	21.2	0.7	29.3	0.8	20.1	0.7	12.1	0.6
Other (7221)	16.4	0.8	19.9	0.9	30.6	1.1	19.8	0.9	13.5	0.8
Education, y (n)										
< 12 (8154)	19.6	0.7	21.1	0.7	25.7	0.8	18.0	0.7	15.7	0.7
12 (15,550)	17.3	0.5	19.4	0.5	26.0	0.5	19.9	0.5	17.4	0.5
> 12 (25,667)	12.7	0.3	17.8	0.3	31.7	0.4	23.3	0.4	14.5	0.3
Parity (n)										
0 (20,782)	11.5	0.3	15.9	0.4	28.3	0.5	24.3	0.4	20.1	0.4
1-2 (23,911)	16.8	0.4	20.5	0.4	29.8	0.4	20.3	0.4	12.7	0.3
≥ 3 (5100)	23.2	0.9	22.9	0.9	28.3	0.9	14.8	0.7	10.8	0.7
Total (50,029)	15.5	0.2	18.8	0.3	28.9	0.3	21.4	0.3	15.5	0.2

x^2 test used for difference in gestational weight gain by maternal age, race/ethnicity, educational level, and parity were all statistically significant ($P < .001$)

[a] Based on unweighted data.

[b] Based on weighted data

SOURCE: Reprinted from Chu S. Y., W. M. Callaghan, C. L. Bish and D. D'Angelo. Gestational weight gain by body mass index among US women delivering live births, 2004-2005: fueling future obesity. *American Journal of Obstetrics and Gynecology*. Copyright (2009), with permission from Elsevier.

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Food Insecurity

Food insecurity is closely tied to socioeconomic status and thus, it is included in the discussion even though it is arguably a modifiable factor. Several studies have identified a relationship between food insecurity, defined as "whenever the availability of nutritionally adequate and safe food or the ability to acquire acceptable foods in socially acceptable ways is limited or uncertain" and overweight/obesity (Anderson, 1990). These studies have shown a higher prevalence of overweight and obesity among women living in food insecure households compared to women living in food secure households (Frongillo et al., 1997; Olson, 1999; Townsend et al., 2001; Adams et al., 2003; Basiotis and Lino, 2003; CDC, 2003; Crawford et al., 2004). The mechanisms mediating this association are not well understood, but reports in the literature addressing eating patterns support the idea that food deprivation can result in overeating (Olson and Strawderman, 2008). Polivy (1996) found that food restriction or deprivation, whether voluntary or involuntary, results in a variety of changes including the preoccupation with food and eating. It has also been suggested that food-insecure households tend to purchase calorie-dense foods that are often high in fats and added sugars as an adaptative response to food insecurity (Drewnowski and Darmon, 2005).

More recently, Jones and Frongillo (2007) found food insecurity without hunger to be associated with risk for overweight/obesity, but not with subsequent weight gain in women of all racial/ethnic groups. Wilde and Peterman (2006) examined the relationship between food insecurity and change in self-reported weight over 12 months in a national sample of non-pregnant women. These investigators found that women in households that were marginally food secure were significantly more likely to gain 4.54 kg (10 pounds) or more in a year compared to women in food secure households.

While food insecurity and obesity have been shown to be positively associated in women, little is known about the direction of causality between food insecurity and obesity. Olson and Strawderman (2008) found in a cohort of 622 healthy adult women from rural areas followed from early pregnancy until two years postpartum that food insecurity in early pregnancy was not associated with increased risk of obesity at two years postpartum. However, obesity in early pregnancy was significantly associated with increased risk of food insecurity at two years postpartum, suggesting that the causal direction of the relationship between food insecurity and obesity likely goes from obesity to food insecurity. Moreover, they found that women who were both obese and food insecure in early pregnancy were at greatest risk of major gestational and postpartum weight gain, suggesting that food insecurity may play a role in GWG (trends in food insecurity are shown in Chapter 2).

Genetic Characteristics

The role of DNA sequence variation in the regulation of body weight is being investigated in many laboratories around the world. However, the role of genetic factors in the modulation of weight gain during pregnancy has not received much attention to date. The committee was unable to identify studies dealing with familial aggregation or heritability of GWG. The only evidence comes from a small number of reports focusing on the contribution of single nucleotide polymorphisms (SNPs) in specific genes on GWG. At present no study has considered the important issue of nutrition or physical activity interactions with genes on GWG.

Several studies have considered the effect of the Trp64Arg allelic substitution in the beta 3 adrenergic receptor gene (ADRβ3) on weight gain during pregnancy (Festa et al., 1999; Yanagisawa et al., 1999; Alevizaki et al., 2000; Tsai et al., 2004; Fallucca et al., 2006). The

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homozygotes for the 64Arg allele were found to have gained more weight from baseline to gestational weeks 20 to 31 in Austrian mothers (Festa et al., 1999). In pregnant mothers with type 2 diabetes, a gain in BMI of more than 5 units was found to have a prevalence of 12.2 percent in homozygotes for the Trp allele, 19.2 percent for the heterozygotes and 28.6 percent for the Arg allele homozygotes (Yanagisawa et al., 1999). However, in the latter study from Japan, there were no differences among the three genotypes for GWG. In a study from Greece, no differences among the ADRB3 genotypes were found for the rate of weight gain (g/day) calculated from the difference between the prepregnancy reported body weight and the weight measured between weeks 28 and 36 of gestation (Alevizaki et al., 2000). Similarly, no differences were observed among genotypes in a Taiwanese population for weight gain at 24 to 31 weeks of gestation (Tsai et al., 2004). In the largest study to date, 627 pregnant women from Italy were studied and no effect of the ADRB3 polymorphism on GWG was found (Fallucca et al., 2006). In the same study, a marker in the insulin receptor substrate 1 (IRS-1) gene was also not associated with GWG.

The Pro12Ala polymorphism in the peroxisome proliferator-activated receptor gamma 2 (PPARδ2) was typed in pregnant Turkish women (Tok et al., 2006). Among 62 women who had gestational diabetes (GDM), those with the Ala allele gained more weight during pregnancy but this was not observed in 100 non-diabetic pregnant women. The 825 C/T base substitutions, a common polymorphism of the G-protein beta-3 subunit gene, was studied in 294 women with uncomplicated, singleton pregnancies with term deliveries ranging from 37 to 40 weeks (Dishy et al., 2003). Pregnant women homozygous for the T allele (17.4 ± 0.9 kg) gained significantly more weight than the C allele carriers (15.1 ± 0.4 kg). However, the sample was composed of women from various ethnic ancestries which may have affected the results in an undetermined manner.

From this small body of data, it is not possible to conclude whether there is a role for specific genes and alleles in GWG. None of the studies reported to date were based on sufficiently large sample sizes to ensure that adequate statistical power was available to identify the effects of alleles or genotypes with a small effect size. Studies conducted on large samples of ethnically homogeneous pregnant women will be needed to be able to understand the contribution of specific genes and sequence variants to GWG.

Genetics and Birth Weight

Gestational weight gain is associated with the weight of the infant at birth even though there may not always be a cause and effect relationship and despite the fact that reverse causation often cannot be excluded. In this context, it is useful to consider the role that genetic factors may play in the variation of birth weight. In particular, it is important to understand the potential role of risk alleles at specific genes on risk for SGA and LGA.

The topic of the heritability of birth weight has been addressed for more than 50 years in the scientific literature. The evidence up to the late 1970s was reviewed (Robson, 1978) in a three volume treatise on Human Growth. The conclusion was that the fetal genotype played a small role on birth weight, probably of the order of 10 percent, while the maternal genotype accounted for about 24 percent of the total variance. These estimates were derived from data on full siblings, half-siblings, first cousins, mother-child, father-child, and monozygotic and dizygotic twins.

Recent twin studies have consistently generated slightly higher significant genetic components for birth weight in the range of 20 percent to 40 percent (Vlietinck et al., 1989;

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Whitfield et al., 2001; Dubois et al., 2007). A recent report from Norway on birth weight was obtained in the mother, father, and up to three singleton offspring (included data from 101,748 families) (Lunde et al., 2007). It concluded that the fetal genetic component of birth weight adjusted for birth order, sex and generation reached 31 percent. The heritability estimates reached 31 percent for birth length and 11 percent for variation in gestational age. Given the ample statistical power of the latter study, 31 percent represents the most valid and reliable heritability estimate to date of the contribution of the fetal genes to birth weight (Beaty, 2007). The latter is concordant with the 25 percent value reported in another large Norwegian study of trios composed of mother-father-firstborn child (Magnus et al., 2001).

Importantly, variation in birth weight is influenced by a number of other factors besides the genetic makeup of the newborn. Several studies have found a role for the maternal genotype on the weight of the newborn. In the large Norwegian study cited above, maternal genetic factors accounted for 22 percent of the variation in birth weight (Lunde et al., 2007). Of particular interest is whether there is a paternal genetic component to birth weight.

In a study of 6,811 white singletons and their natural parents, the effect of parental height and weight on the length and weight at birth of an offspring was evaluated (Griffiths et al., 2007). It was observed that the effects of parental height on birth weight are similar for the two parents. However, the influence of the mother's weight on the infant's birth weight was stronger than that of the father. In a report on parental role in the familial aggregation of SGA based on 256 cases, it was found that both parents contributed almost equally to the risk (Jaquet et al., 2005). The risk of SGA for an infant at birth was 4.7 times greater for mothers and 3.5 times for fathers who were themselves small for gestational age compared to those who were of average weight for gestational age. The risk of a SGA infant was however 16 times higher when both parents had been SGA (Jaquet et al., 2005). The most compelling data for a role of paternal birth weight on weight of the offspring at birth comes once again from a Norwegian study. A total of 67,795 trios of father-mother-firstborn child were used to plot the birth weight of infants against paternal birth weight by classes of maternal birth weight (Magnus et al., 2001). The regression of a child's birth weight on the father's birth weight was 0.137 while that on the mother's birth weight reached 0.252. The effect of paternal birth weight was about the same within each category of maternal birth weight, with no significant interaction effects between parental birth weight levels.

Evidence for a role of specific genes with a focus on their implications for diabetes on birth weight is limited (McCarthy and Hattersley, 2008). Glucokinase encoded by the *GCK* gene is an enzyme that phosphorylates glucose to glucose-6-phosphate in the pancreas, where it serves as a glucose sensor and is the rate limiting step in glucose metabolism. A defect in the pancreatic glucose sensing mechanisms of the fetus could potentially reduce weight at birth and have profound effects on the regulation of glucose and insulin later in life. Mutations altering highly conserved amino acids in *GCK* were genotyped in 58 offspring and their mothers from the UK (Hattersley et al., 1998). When a mutation was present in the fetus but not carried by the mother, weight at birth was diminished by more than 500 g. A concordant observation was that in 19 pairs of siblings discordant for a *GCK* mutation, the infant with the mutation weighed about 500 g less at birth than the other sibling (See Figure 4-2). When a mutation was absent in the fetus but present in the mother, mean birth weight was higher by about 600 g. When the mutation was present in both mother and fetus, body weight at birth was normal. The low and high birth weights associated with a number of *GCK* missense mutations are thought to reflect variation in fetal insulin secretion resulting from the *GCK* fetal genotype and indirectly from the fetal

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response to maternal hyperglycemia (Hattersley et al., 1998). This may represent an explanation for some of the fetal programming cases in which there is an association between low birth weight and later insulin resistance and type 2 diabetes.

In a short report of four cases from Italy exhibiting different *GCK* mutations, three had substantially lower than average birth weight (Prisco et al., 2000). One recent study focused on the effect of the A allele (SNP at position -30) at the *GCK* gene on birth weight (Weedon et al., 2005). Using data from 2,689 mother-child pairs, the A allele in the mother was associated with a 64 g increase in the offspring birth weight. There was no effect of the offspring *GCK* genotype at this particular mutation on birth weight.

Hepatocyte nuclear factor 1 beta (HNF1β) is a transcription factor, encoded by the *HNF1β* gene, critical for the development of the pancreas. Birth weight was studied in 21 patients with *HNF1β* mutations (Edghill et al., 2006). Weight at birth was low in all cases, with a median weight of 2.7 kg. In 13 of these patients born to unaffected mothers, 69 percent were SGA at birth, with a median percentile weight of 3 (Figure 4-2).

Another transcription factor, hepatocyte nuclear factor 4 alpha (HNF4α) is involved in the regulation of pancreatic insulin secretion. The *HNF4α* gene is responsible for MODY-1 and accounts for about 4 percent of all maturity-onset diabetes in the young (MODY) cases (McCarthy and Hattersley, 2008). Mutations in *HNF4α* also associate with type 2 diabetes. Weight at birth was studied in 108 infants from families with *HNF4α* mutations (Pearson et al., 2007). Birth weight was increased by 790 g in *HNF4α* mutation carriers compared to non-mutated family members (Figure 4-2). Fifty-four percent of mutation carriers were macrosomic compared with 13 percent for non-mutation family members.

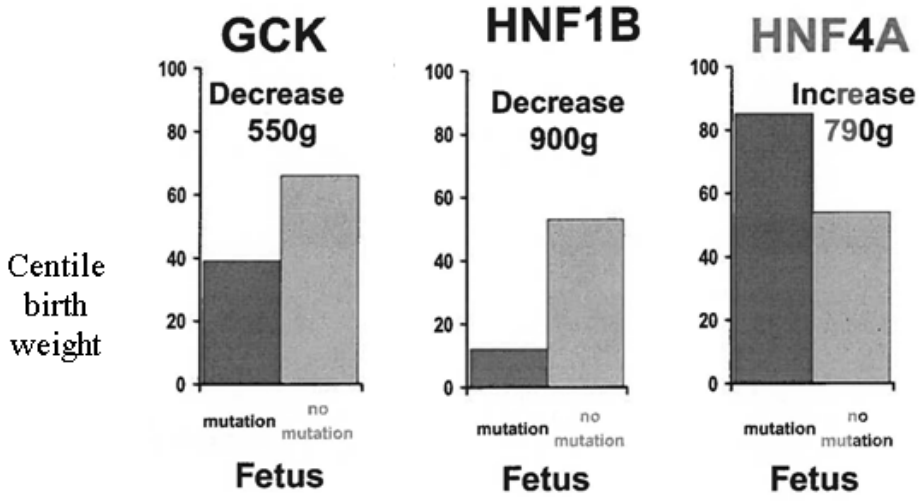

FIGURE 4-2 The impact on birth weight of a fetus inheriting three common maturity-onset diabetes in the young (MODY) gene mutations. Birth weight is presented in centile birth weight with the fetus inheriting the mutation in black and in comparison a fetus without the mutation in gray.
SOURCE: Modified from McCarthy and Hattersley, 2008. Copyright © 2008 American Diabetes Association From Diabetes®, Vol. 57, 2008; 2889-2898. Modified with permission from *The American Diabetes Association.*

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In another candidate gene study, a common SNP in the fat mass and obesity associated gene (*FTO)* was investigated for its relationship to weight at birth in 234 full-term, healthy newborns (Lopez-Bermejo et al., 2008). An allelic variant known to influence body weight and fat mass in children and adults was not associated with birth weight but an association became evident after about two weeks postnatally.

Another line of evidence for a role of genes on birth weight and thus perhaps on GWG comes from scanning the whole genome. Three studies have dealt with genome-wide linkages using panels of highly polymorphic markers and birth weight. The first was based on 269 Pima Indians from 92 families and 503 autosomal microsatellite markers (Lindsay et al., 2002). A quantitative trait locus (QTL) was identified on chromosome 11 (logarithmic odds [LOD] for an imprinted locus = 3.4), suggesting that a paternally imprinted gene at map position 88 cM was influencing birth weight in this population (Lindsay et al., 2002). Subsequently, a QTL on chromosome 6q was shown to be linked to birth weight in Mexican-Americans from the San Antonio Family Birth Weight Study (LOD = 3.7) and partially replicated in a European-American population (LOD = 2.3) (Arya et al., 2006). The latest study using this approach was also on Hispanic newborns from Texas (Cai et al., 2007). Birth weight was available from birth certificates for 629 children from 319 families. Birth weight was highly heritable in this population and a QTL was identified on 10q22 with a LOD score of 2.6.

From this body of data, one can draw the preliminary conclusion that: (a) there is a fetal genotype effect on weight at birth (about 30 percent of the adjusted variance), (b) both parents' genes influence birth weight with a stronger effect for maternal genes, (c) specific allelic variants have been associated with weight at birth, (d) mutations in *GCK* and *HNF1*β are associated with low birth weight, (e) mutations in *HNF4*α are associated with high birth weight, and (f) a few quantitative trait loci on chromosomes 6, 10, and 11 have been uncovered from genome-wide linkage scans. None of the high risk alleles identified thus far have been studied for their potential contributions to GWG with or without control for the weight of the infant. The issue of the contribution of specific genes and variants to human variation in birth weight would greatly benefit from a number of genome-wide association studies with comprehensive panels of markers, particularly in cohorts with large sample size and substantial numbers of small- and large-for-gestational age infants. It will also be critical in the future to design studies that will make it possible to define the maternal and fetal alleles at key genes which associate with increased risk for GWG outside recommended ranges in the context of maternal dietary and physical activity habits.

Developmental Programming

Among the multitude of factors influencing GWG, early developmental programming may increase risk for GWG above recommended ranges. Developmental programming (physiological, metabolic or behavioral adaptation resulting from exposure or lack thereof to hormones, nutrients, stress and other agents at critical periods during embryonic or fetal development) suggests that exposures and experiences during sensitive developmental periods in utero, and possibly early postnatal life may encode the functions of organs or systems that become manifest as risk factors for disease later in life (Barker, 1998; Seckl, 1998).

One example to illustrate how developmental programming may influence maternal GWG is the suggestion that developmental programming could influence the ability to respond to and cope with repeated exposure to stress. This could in turn, provide a mechanism by which some women may be at greater risk for excessive GWG. Animals and humans subjected to chronic and

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repeated stress exhibit elevated basal glucocorticoid levels and exaggerated hypothalamic-pituitary-adrenal (HPA) response to natural or experimental stressors (Sapolsky, 1995). Epidemiologic evidence suggests there may be a relationship between elevated glucocorticoid levels and physiologic changes consistent with metabolic syndrome, including increased adiposity (Pasquali et al, 2006; Barat et al., 2007). Hyperactivity of the HPA axis has been hypothesized to play a role in development of abdominal obesity and insulin resistance (Bjorntorp, 1993, 1996; Bjorntorp and Rosmond, 2000). A potential mechanism for HPA hyperactivity is through diminished feedback inhibition of pituitary activity resulting from down-regulation of glucocorticoid receptors in the brain (Vicennati and Pasquali, 2000). Over time HPA hyperactivity and excess glucocorticoid secretion can lead to both hyperinsulinemia and insulin resistance with subsequent increased risk of type II diabetes (Vicennati and Pasquali, 2000). These observations suggest that GWG could be influenced by not only factors that arise in pregnancy, but also by in utero developmental events that may predispose the mother to HPA dysregulation.

Even though the evidence for a role of developmental programming during fetal life on the risk of obesity and late-onset metabolic diseases is growing, the committee was unable to identify studies that directly examined the influences of programming on GWG in the mother. Consequences of high GWG to the child that may be related to developmental programming are discussed in Chapter 6.

Epigenetics

In addition to developmental programming, another line of evidence suggests that modifications in DNA and histone proteins could translate into phenotypic differences that often mimic those associated with DNA sequence variants. Such DNA and nucleoprotein alterations have been collectively referred to as "epigenetic events". Epigenetic events begin to occur early after fertilization, are typically stable, and influence gene expression. There is already compelling evidence to suggest that nutritional factors can entrain DNA methylation and modifications in histone proteins (Waterland and Jirtle, 2003); some of which occur at the embryonic stage in key tissues (Sinclair et al., 2007; Waterland et al., 2008). Such events are known to lead to the silencing (or switching off) of genes particularly when they occur in their promoter regions. Cytosine residue (in CpG islands) and histone (H3 and H4) methylation, acetylation or other chemical modifications occurring during early fetal life provide a mechanism, although it is not the only one, by which programming of the developing organism beyond the blueprint specified in the genomic DNA may occur.

However, it is important to recognize that epigenetic events can occur throughout life and may thus account for some of the phenotypic variation observed among adults. In this regard, the observation that the pattern of DNA methylation in monozygotic twins diverges more as they become older is of great interest (Fraga et al., 2005). It reinforces the view of those who believe that considerable phenotypic differences can arise among individuals with the same genotype. Such phenotypic variations in physiology and behavior have been observed before in inbred rodent strains but no satisfactory explanations have been provided thus far for them.

Future progress in understanding the role of programming and epigenetic factors on GWG will require increased attention not only to the role of DNA sequence variation but also to the potential influence of early programming and epigenetic events and their lasting impact on pregnant women.

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Anthropometric and Physiological Factors

Pregravid BMI in concert with physiological changes in a woman's basal energy expenditure and hormonal milieu that occur during pregnancy can influence her GWG and may predict both maternal and fetal outcomes.

Pregravid BMI

Epidemiological studies, based largely on self-reported data, suggest that GWG is influenced by maternal BMI and is an important determinant of both maternal and neonatal health. In the United States, Chu et al. (2009) assessed PRAMS data for GWG by prepregnancy BMI among 52,988 women delivering full-term singleton infants from 2004-2005 (Table 4-4). This analysis found that GWG decreased as BMI increased, however, even though obese women gained less than normal or overweight women; about one-fourth of them still gained 35 pounds or more. Using a multivariable regression model, they showed that maternal prepregnancy obesity was the strongest predictor of low GWG, followed by higher parity, African American or Hispanic racial identity, and higher maternal age. In Germany, Voigt et al. (2007) analyzed perinatal statistics from over 2.3 million singleton deliveries from 1995-2000. This analysis concluded that overall, relatively short and heavy women had lower GWGs than tall and thin women.

Although pregravid BMI can predict GWG there are also metabolic changes in pregnancy, i.e. basal metabolic rate (BMR), total energy expenditure (TEE), and hormonal changes that are independent of BMI which can influence GWG.

TABLE 4-4 Gestational weight gain (pounds) by prepregnancy BMI among mothers delivering full-term, singleton births, PRAMS, 2004-2005

	≤ 14 (n = 8442)[a]		15-24 (n = 10,583)[a]		25-34 (n = 15,477)[a]		35-44 (n = 10,942)[a]		≥ 45 (n = 7544)[a]	
BMI Group	**Percent[b]**	**SE[b]**	**Percent[b]**	**SE[b]**	**Percent[b]**	**SE[b]**	**Percent[b]**	**SE[b]**	**Percent[b]**	**SE[b]**
Underweight (BMI, < 18.5 kg/m2)	10.5	0.9	17.7	1.1	34.4	1.5	23.2	1.3	14.2	1.0
Normal (BMI, 18.5-24.9 kg/m2)	10.4	0.3	16.1	0.3	31.8	0.4	24.7	0.4	17.1	0.3
Overweight (BMI, 25.0-29.9 kg/m2)	15.7	0.5	20.3	0.5	27.5	0.6	20.5	0.5	16.1	0.5
Obese (BMI, ≥ 30.0 kg/m2)	29.8	0.7	24.4	0.6	22.1	0.6	13.1	0.5	10.7	0.5
Total	15.3	0.2	18.7	0.3	29.1	0.3	21.4	0.3	15.5	0.2

χ^2 test for the difference in gestational weight gain by body mass index (BMI) group was statistically significant ($P < .001$).

[a] Based on unweighted data.

[b] Based on weighted data; percentages were age adjusted.

SOURCE: Reprinted from Chu S. Y., W. M. Callaghan, C. L. Bish and D. D'Angelo. Gestational weight gain by body mass index among US women delivering live births, 2004-2005: fueling future obesity. *American Journal of Obstetrics and Gynecology*. Copyright (2009), with permission from Elsevier.

Insulin, Leptin, and Hormonal Milieu

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Basal Metabolic Rate

The metabolic response to pregnancy varies widely among women. Prentice et al. (1989) reported on longitudinal changes from pre-conception through 36 weeks gestation in eight healthy well nourished women. The mean GWG at 38 weeks gestation was 14.4 ± 4.1 kg. Lean body mass increased linearly to a mean of 6.7 ± 1.6 kg by 36 weeks gestation. Measured BMR varied from 8.6 to 35.4 percent above pregravid BMR, although some obese subjects showed significant decreases in BMR up to 24 weeks gestation (r = 0.84). In pregnant women, the relative cost of exercise for 120 minutes was approximately 10 percent of total energy expenditure. The authors concluded from finding a small range for energy savings from either minor physical activity or thermogenesis along with high variability in BMR during pregnancy, that offering prescriptive energy intake recommendations would be impractical because it is impossible to know how an individual woman's metabolism will respond.

Durnin (1991) reported on longitudinal changes in energy expenditure during pregnancy among Scottish and Dutch women. Among this cohort, an increase in BMR was not seen until 16 weeks gestation and was followed by a mean increase of 400 kcal/day over pregravid BMI. The total energy cost of pregnancy was estimated at 69,000 kcal. Adjusting for dietary energy intake (~22,000 kcal) the authors estimated that decreased physical activity or increased efficiency of work accounted for an additional savings of ~47,000 kcal. Similarly Forsum et al. (1985) found an increase in BMR throughout gestation in a study of Swedish women.

Lawrence et al. (1985), studied how women in a developing country responded to increasing food intake during pregnancy. Pregnant women in the Gambia who followed their normal dietary pattern experienced energy sparing of 11,000 kcal with no increase in BMR above pregravid BMI until 30 weeks gestation. Further, the women showed a mean GWG of 6 kg with no increase in adipose tissue mass. When their baseline diet was supplemented with 723 kcal/day in additional food, BMR increased by approximately 1,000 kcal over pregravid BMI. Women whose diets were supplemented with additional food had a mean 8 kg increase in GWG and a 2 kg increase in fat mass. Food supplementation had no effect, however, on the energy cost of activity and did not result in increased birth weight when physical work was decreased.

Goldberg et al. (1993) used the doubly labeled water method (International Dietary Energy Consulting Group, 1990) to assess BMR, energy intake, and body composition in 12 affluent women at pre-conception and at 6 week intervals from 6 through 36 weeks gestation. Estimated changes in BMR, total energy expenditure (TEE) and fat deposition were 112 ± 104 MJ, 243 ± 279 MJ, and 132 ± 127 MJ, respectively. The mean total energy cost of pregnancy calculated from BMR, TEE, and energy deposited as fat was 418 ± 348 MJ. The women's self-reported energy intake however was only 208 ± 272 MJ, a significant underestimate of the calculated additional energy cost of pregnancy. Again, the variability in the individual biological response shown in this study supports the impracticality of prescriptive recommendations for energy intake during pregnancy.

A similar prospective study by Butte et al. (2004) of measured energy expenditure in women by prepregnant BMI showed that women in the highest BMI group accumulated greater fat mass (8.4 kg) compared to those in the low BMI group (5.3 kg). The increase in fat mass accounted for most of the variance in total weight gain among BMI groups. In both the low and high BMI groups mean TEE decreased in the second trimester but increased in the third trimester. When adjusted for fat free mass (FFM), TEE decreased in all BMI groups toward the end of gestation. Using multiple regression analysis, the change in TEE throughout the course of gestation was

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related to prepregnancy BMI and percent body fat as well as weight gain and increase in FFM. These variables accounted for 33 percent of the variance in 24 hour TEE, primarily from change in BMR. Physical activity accounted for very little net increase in TEE and actually decreased in all groups with advancing gestation.

Hormonal Milieu

Maternal pregravid insulin sensitivity may vary up to 2- to 3-fold, depending on factors such as obesity, level of fitness and genetic make-up. Over the course of pregnancy a 40-60 percent decrease in insulin sensitivity occurs, depending on pregravid metabolic status (Catalano et al., 1993; Catalano et al., 1999). For example, a 50 percent decrease in insulin sensitivity in both a thin athletic woman and an obese sedentary woman with type 2 diabetes may represent a 2-fold or greater quantitative change in insulin sensitivity between them by the end of gestation. In the last 12 weeks of pregnancy, when fetal weight increases on the average from 1.0 kg to 3.5 kg, decreased insulin sensitivity increases the availability of energy to support fetal growth (Hytten and Chamberlain, 1991).

These changes in insulin sensitivity occur over a matter of months whereas in a non-pregnant individual they may occur over many years. Swinburn et al. (1991) in a longitudinal study (3.5 years) showed that, in Pima Indians, those that were insulin resistant gained less weight over time than insulin sensitive subjects (3.1 versus 7.6 kg, $p = 0.0001$). The percent change in weight per year was correlated with glucose utilization ($r = 0.34$, $p = 0.0001$). Whether similar physiologic relationships also apply to decreased GWG in obese women is not known. Swinburn et al. (1991) may not have had sufficient power to account for the greater inter-individual variability that was observed. There is, however, preliminary data showing that at least in early pregnancy changes in maternal BMR and fat accretion are inversely related to the changes in insulin sensitivity in a small number of subjects (Catalano et al., 1998). Whether increased energy intake in obese insulin-resistant women during pregnancy has a greater effect on maternal and fetal fat accretion than in non-obese women remains to be determined.

Cytokines

Both leptin and adiponectin are two adipocytokines related to fat accretion that have been examined in pregnancy. Leptin is produced in relatively large quantities by the placenta and is transferred primarily into the maternal circulation (Hauguel-deMouzon et al., 2006). Maternal leptin concentrations increase by 12 weeks gestation and have a significant positive correlation with both maternal body fat and basal metabolic rate in both early and late gestation (Highman et al., 1998). Using a stepwise regression analysis, Kirwan et al. (2002) showed that leptin made a significant contribution to changes in insulin sensitivity during gestation. A possible role for leptin in maternal metabolic adaptations to pregnancy may be found in the relationship between circulating leptin and increased maternal fat oxidation (Okereke et al., 2004).

Adiponectin is a unique circulating cytokine that is positively correlated with insulin sensitivity and negatively correlated with adiposity (Cnop et al., 2003). In contrast to leptin and other cytokines, adiponectin is made exclusively in the maternal and fetal compartments, and not in the placenta (Pinar et al., 2008). There is no transfer of leptin from mother to fetus or vice versa. Lower adiponectin concentrations have been reported in women with previous GDM (Winzer et al., 2004) and leptin was shown to decrease over the course of pregnancy in women

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with GDM compared to women with normal glucose tolerance (Retnarkaran et al., 2004; Williams et al., 2004).

In summary both leptin and adiponectin are correlated with various components of maternal metabolism such as energy expenditure and adiposity. However, there are no direct mechanistic effects relating to the changes in maternal weight gain described in human pregnancy. Indirectly these cytokines through their effects on maternal insulin sensitivity may represent markers of other mechanisms effecting gestational weight changes.

Medical Factors

Pre-Existing Morbidities

The committee was unable to identify studies that directly examined pre-existing morbidities as determinants of GWG. However, in general the pre-conceptional health status of a woman is important for optimal pregnancy outcome. This is particularly true for chronic diseases such as inflammatory bowel disease and systemic lupus erythematosus. In women with inflammatory bowel disease, and in particular Crohn's disease, the level of disease activity during pregnancy is related to disease activity at conception. Fonager et al. (1998) reported a decrease in birth weight and increased preterm delivery in women with active Crohn's disease at conception. Similarly in women with lupus complicating pregnancy, pregnancy outcomes are improved if lupus has been quiescent for at least six months before conception (Cunningham et al., 2005).

Hyperemesis Gravidarum

Although as many as 70-85 percent of pregnant women will have nausea and occasional vomiting in pregnancy (Jewell and Young, 2003), this often resolves by the second trimester. There are usually no long-term sequelae and treatment is mostly symptomatic including avoidance of certain foods and small frequent meals. However, approximately 0.5-2.0 percent of pregnant women will develop hyperemesis gravidarum (ACOG, 2004). The most commonly cited criteria for hyperemesis gravidarum include: persistent vomiting unrelated to other medical conditions, ketonuria, and weight loss of 5 percent or greater of prepregnancy weight at < 16 weeks' gestation (Goodwin et al., 1992). Other associated findings include dehydration, ketonuria, and electrolyte imbalance. The underlying etiology of this disorder is not known with certainty but rapid increases in circulating human chorionic gonadotropin (HCG) and estrogen in early pregnancy have been associated with the condition (Furneaux et al., 2001; Goodwin, 2002).

In mild cases of nausea and vomiting there appears to be no adverse effect on maternal weight gain or pregnancy outcome. However, among women with hyperemesis gravidarum there is evidence of decreased GWG and a higher risk of low birth weight. Gross et al. (1989) reported on 64 women with a diagnosis of hyperemesis gravidarum. In the women who lost > 5 percent of their prepregnancy body weight, total GWG was lower (9.6 ± 2.4 versus 13.7 ± 3.2 kg, $p < 0.05$), and fetal growth was compromised, i.e. smaller percent weight for gestational age (38 percentile versus 72 percentile, $p < 0.025$) and increased growth restriction (30 percent versus 6 percent, $p < 0.01$) compared to women with a similar diagnosis that lost < 5 percent of their prepregnancy weight. Vilming and Nesheim (2000) and Bailit (2005) also reported that women with hyperemesis gravidarum had overall lower GWG and birth weight in comparison with a control group. Evidence for long-term outcomes on infant growth was not found.

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Anorexia Nervosa and Bulimia Nervosa

Anorexia nervosa and bulimia nervosa are frequently encountered in young women of reproductive age. Anorexia is defined as body weight less than 85 percent of expected for age and height. The prevalence in women of reproductive age is between 0.5-1.0 percent. Whereas bulimia is defined as weight at the minimally normal range but where the individual employs binge eating and subsequent compensatory methods such as self-induced vomiting, laxative or diuretic medications to avoid appropriate weight gain. Bulimia occurs in 1-3 percent of young women. A dysfunctional perception of body weight and shape is common to both disorders (Wisner et al., 2007).

Sollid et al. (2004) in a Danish register-based follow-up study reported the results of 302 women with eating disorders before pregnancy and who were delivered 504 children and were compared with 900 control subjects who were delivered 1,552 children. There was almost a two-fold increased risk of preterm delivery and SGA. (Unfortunately the investigators were not able to obtain any information on prepregnancy BMI or GWG. In a smaller study from Sweden, Kouba et al. (2005) reported that among 49 women with previously diagnosed eating disorders 22 percent had a relapse of their eating disorder in pregnancy. The women with either a past or current eating disorder were at significantly increased risk of hyperemesis, and delivered children with significantly lower birth weight and head circumference as compared with a control group. Although there were no significant differences in GWG between the groups, the anorectic women (n = 24) gained less weight than women with previous history of eating disorders (10.4 ± 3.9 versus 12.1 ± 2.6 kg, $p < 0.05$). The authors speculated that potential causes for the decreased fetal growth in the women with a history of eating disorders include their inability to achieve the recommended weight gain of 11.5-16.0 kg during pregnancy. There was no significant difference in intake of folate, protein or total caloric intake between the two groups. Bulik et al. (2008) found among a cohort of 35,929 pregnant Norwegian women, that 35 reported broad anorexia nervosa, 304 bulimia nervosa, 1,812 binge eating disorder, and 36 eating disorder not otherwise specified (EDNOS)-purging type in the six months before or during pregnancy. Prepregnancy BMI was lower in anorexia, and higher in binge eating disorder than the referent group, and anorexia, bulimia, and binge eating disordered mothers reported greater GWG.

Bariatric Surgery

Parallel to the trend of increasing prevalence of obesity in the U.S., is an increase in the number of bariatric surgeries performed as treatment. The reported total number of bariatric surgical procedures performed in the U.S. increased from approximately 13,365 in 1998 to approximately 72,177 in 2002 (Santry et al., 2005; Davis et al., 2006). Furthermore, most of the procedures were performed on women; 81 percent in 1998 and 84 percent in 2002.

The American College of Obstetricians and Gynecologists (ACOG) published a Committee Opinion on Obesity and Pregnancy addressing the issue of bariatric surgery and pregnancy (ACOG, 2005). ACOG recommends that obese women who have undergone bariatric surgery receive the following counseling before and during pregnancy:

- Patients with adjustable gastric banding should be advised that they are at risk of becoming pregnant unexpectedly after weight loss following surgery.

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- All patients are advised to delay pregnancy for 12-18 months after surgery to avoid pregnancy during the rapid weight loss phase.
- Women with gastric banding should be monitored by their general surgeons during pregnancy because adjustments of the band may be necessary.
- Patients should be evaluated for nutritional deficiencies, including iron, B12, folate, vitamin D and calcium, and supplemented with vitamins as necessary.

With respect to GWG, three studies reported a decrease in weight gain during a subsequent pregnancy in women who had bariatric surgery (Skull et al., 2004; Dixon et al., 2005; Ducarme et al., 2007). Nutritional complications during pregnancy, such as folate and B_{12} deficiencies, are also associated with bariatric surgery (Gurewitsch et al., 1996).

No prospective randomized trials of pregnancy outcome in obese women treated by bariatric surgery were identified. However, there have been reports using the patient as her own control i.e. a pregnancy outcome before bariatric surgery and a subsequent pregnancy outcome after having a bariatric procedure (Marceau et al., 2004; Skull et al., 2004; Dixon et al., 2005) or retrospective case controlled studies (Ducarme et al., 2007). Incidence of GDM and hypertensive disorders were found to be decreased in the studies of Skull et al. (2004), Dixon et al. (2005), and Ducarme et al. (2007). The effect of bariatric surgery on the risk of fetal macrosomia and birth weight are inconclusive. Marceau et al. (2004) and Ducarme et al. (2007) reported a decreased risk of macrosomia in women following bariatric surgery. In contrast, neither Dixon et al. (2005) nor Skull et al. (2004) reported a decrease in macrosomia. Care must be taken in the interpretation of these studies because of their retrospective nature and use of various definitions of outcome measures.

Twins and Higher Order Pregnancy

The presence of multiple fetuses in a pregnancy has an influence on total GWG. In comparison to singleton birth the additional components of the products of a twin gestation (fetus, placenta and amniotic fluid) account for up to two additional kilograms in GWG (See discussion in Chapter 3). The effects of GWG on maternal and child health outcomes for multiple births are discussed in Chapters 5 and 6, respectively.

Psychological Factors

Depression

Several investigators have reported positive associations between GWG and depressive symptoms. Bodnar et al. (in press) followed a sample of 242 mostly well-educated white women through pregnancy and assessed clinical depression through structured interviews at 20, 30, and 36 weeks gestation. The study found that all women with GWG below the ranges recommended by IOM (1990) had an elevated prevalence of major depression, regardless of their pregravid BMI (Bodnar et al., in press). Hickey et al. (1995) conducted a prospective study of depressive symptoms at 24-26 weeks and inadequate GWG in a large cohort of low-income, non-obese black and white women. The authors reported that white women in the highest quartile of depressive symptom score were three times as likely as women in the lowest quartile to have weight gain below the ranges recommended by IOM (1990). No relationship was found,

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however, between depression and low weight gain in black women (Hickey et al., 1995). In a cohort of over 4,000 Hispanic women, self-reported feelings of depression during the pregnancy were found to be negatively associated with GWG (Siega-Riz and Hobel, 1997).

In a recent study, pregnant women who gained in excess of the ranges recommended by IOM (1990) were more likely to have high depressive symptoms than women who met the weight gain recommendations (Webb et al., 2009). Casanueva et al. (2000) used a case-control study to test for associations between maternal depressive symptoms and fat deposition among pregnant adolescents in Mexico City. Body weight and anthropometric measures of skinfold thickness were used to determine fat deposition beginning at 20 weeks gestation through four weeks postpartum. The results of this study indicated an association between depressive symptoms and excessive fat deposition in Mexican adolescents. In cross-sectional studies, high depressive symptoms have been linked with negative attitudes about GWG (Stevens-Simon et al., 1993; Dipietro et al., 2003). Women who are concerned before and during pregnancy about their weight gain have higher depressive scores in the week following delivery (Abraham et al., 2001).

Cameron et al. (1996) studied a biracial sample of 132 women in mid-gestation and found that GWG had no association with depressive symptoms in the second trimester. Third-trimester weight was negatively correlated with depression score, but only among white women with low self-esteem. A positive association between GWG and depression score was found for white women with high self-esteem, whereas GWG had no effect on depression in black women (Cameron et al., 1996). Walker and Kim (2002) used data from a longitudinal study of postpartum weight patterns in low income women to test for relationships between depressive symptoms and GWG and birth weight. Regression analyses found that depressive symptoms were not significantly associated with GWG. Collectively, the majority of studies indicate that low and high GWG may be a marker of depression during pregnancy. Trends in depression among women of child-bearing age are shown in Chapter 2.

Stress

Among studies that evaluate stress, social support, or depression and its relationship to postpartum weight retention there is no consistent evidence in support of a relationship between stress and GWG or increased postpartum weight retention. The impact, however, of psychosocial factors such as stress on GWG and postpartum weight retention may be underestimated as a result of the limitations in measurement and data analysis in observational studies. An additional confounding factor is that stress can have different kinds of effects depending on how an individual responds.

The influence of psychological stress as a factor in GWG and pregnancy outcome was examined in a controlled prospective study of a group of 60 women utilizing an urban prenatal clinic (Picone et al., 1982). Psychological stress was assessed using a social readjustment rating scale from the Holmes-Rahe life events questionnaire. This study found a correlation between higher stress scores and lower GWG, independent of nutrient or caloric intake. This finding suggests that stress did not affect food intake in these subjects, rather the utilization of calories and nutrients from the foods consumed to support pregnancy was impacted.

A robust association between the appraisals of stress and sufficiency of coping resources and adequacy of GWG in crude or adjusted models was not identified. However, when evaluating the risk ratio differences observed between women who gained inadequate or excessive weight (relative to women who gained adequate weight), the former tended to have a stronger, albeit modest, link to perceived stress than the latter. This distinction is comparable to a pattern also

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cited previously in another investigation (Brawarsky et al., 2005). In that study, women who reported high stress during pregnancy tended to gain weight below clinical guidelines in contrast to those who did not report significant prenatal stress. This relationship failed to emerge among those who gained weight in excess of clinical advice. This finding is also consistent with earlier work that typically reported higher stress in relation to insufficient GWG (Orr et al., 1996).

Social Support

There is inconclusive evidence for a role of social support as a determinant of GWG. In a prospective study of 806 low-income, non-obese pregnant women, Hickey et al. (1995) found that the levels of social support did not predict low GWG for either black or white women. Casanueva et al. (1994) reported on the impact of psychological support, given to a group of adolescents during pregnancy, on GWG. Adolescents who received additional psychological support by a psychotherapy team gained, on average, 2.8 kg more than adolescents who did not receive support. More recently, Olson and Strawderman (2003) found the effect of social support on GWG varied significantly by BMI group. Low social support among low, normal, and obese women was associated with significantly more weight gain than that of their counterparts with average or high social support. However, obese women who had low social support gained significantly less weight relative to those with average or high social support.

Attitudes Toward Weight Gain or Weight Loss

Several studies have examined the relationship between maternal attitude toward weight gain during pregnancy and actual GWG. Palmer et al. (1985) developed an 18-item scale measuring pregnant women's attitude toward their own weight gain and found among 29 white, middle-class women that positive attitude was significantly associated with higher actual weight gain. Steven-Simon et al. (1993) found, in a study of 99 pregnant adolescents, that weight gain was significantly related to 4 of 18 scale items but not the total attitude scale score. However, Copper et al. (1995) found in a sample of 1,000 black and white low-income women that the attitude score was not significantly related to GWG. Maternal attitude toward weight gain was found to be influenced by prepregnancy BMI; thin women tended to have positive attitudes and obese women tended to have negative attitudes about GWG (Copper et al., 1995). Taken together, the evidence is inconclusive regarding the influence of maternal attitude on actual GWG.

For the majority of women, weight loss during pregnancy is discouraged. However, a small percentage (8.1) of women reported in the Behavioral Risk Factor Surveillance Survey (BRFSS) that they attempted to lose weight during pregnancy (CDC, 1989; 1991). Another survey of women who reported being pregnant and also trying to lose weight indicated that prevalence of weight loss behavior during pregnancy occurred among those who reported drinking and smoking (12.7 percent), women in the first trimester of pregnancy (9.4 percent), those who were diabetic (9 percent), and those with very high BMIs (6.9 percent) (Cogswell et al., 1996). Cohen and Kim (2009) reviewed aggregated multiple year data between 1996 and 2003 from the BRFSS (1989) and found weight loss attempts during pregnancy were more frequent among women over 34 years of age (6.2 percent) and Hispanic women (13.1 percent). Carmichael et al. (2003) reported in a population-based case control study of 538 cased and 539 control infants that restricted food intake or fad dieting by the mother during the first trimester of pregnancy was associated with significant risk for neural tube defect among both food restrictors (OR = 2.1 [95% CI: 1.1-4.1]) and dieters (OR = 5.8 [95% CI: 1.7-10]) compared to controls. Interestingly,

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no significant increased risk for neural tube defect was detected for dieting behaviors during the three months prior to conception.

Behavioral Factors

The issue of energy balance is central relative to the evaluation of GWG, regardless of whether it is in gravid or pregravid women.

Dietary Intake

Dietary factors may influence GWG however, a significant difference in GWG from balanced energy/protein supplementation in normal weight women was not supported in a systematic review of 10 trials (Kramer and Kakuma, 2003). Two trials reviewed in Kramer and Kakuma (2003) among women who were obese (Campbell, 1983) or had high GWG (Campbell and MacGillivray, 1975) showed energy/protein restriction was associated with a significant reduction in weekly maternal weight gain (weighted mean difference of 255 [95% CI= -436.56 to -73.0] g/week).

Several observational studies have also examined the relationship between prepregnancy BMI, caloric intake, and GWG. Bergmann et al. (1997) reported on data in 156 healthy German women. The authors defined maternal weight gain as "net weight gain", i.e. the weight gain of the mother from the end of the third trimester minus the measured weight in the first trimester, excluding the weight of the fetus and placenta. The authors defined high BMI as > 24. Net weight gain was related to both maternal pregravid BMI and energy intake. There was a decrease in net weight gain in the high BMI group (4.2 kg) as compared to the medium BMI group (6.2 kg) and low BMI group (5.9 kg). In the high BMI group, parity was a significant co-variable. The lower weight gain was confined to the multigravid women, whereas the primigravid high BMI group actually had greater net weight gain. These associations did not appreciably change when adjusted for energy intake, which did not vary during the course of pregnancy. Neither maternal BMI nor energy intake was related to birth weight.

Olson and Strawderman (2003) used a proxy measure for energy intake by questioning women about changes in the amount of food eaten prior to and during pregnancy. They found in a clinic sample of 622 healthy pregnant women that consuming either "much more" or "much less" food during rather than prior to pregnancy was associated with greater (3.67 pounds; $P < 0.001$) and less (-3.16 pounds; $P < 0.05$) GWG, respectively, compared with maintaining similar levels of food intake. Their multivariable model found that women who ate "much more" during than before their pregnancy had an adjusted odds ratio of 2.35 for excessive GWG. Lagiou et al. (2004) found in a clinic sample of 224 pregnant women that increased GWG by the end of the second trimester of pregnancy was associated with higher total energy intake as well as a higher proportion of protein and lipids of animal origin and lower proportion of carbohydrates.

More recently, Olafsdottir et al. (2006) reported on the relationship of dietary factors relating to GWG in 495 healthy Icelandic women using food frequency questionnaires. Optimal weight gain in normal weight women was defined as between 12-18 kg and for overweight women between 7-12 kg. Eleven percent of overweight women had inadequate weight gain (≤ 7 kg). Additionally, 55 percent of overweight women gained excessive weight (>12 kg) and 20 percent of normal weight women gained excessive weight (>18 kg). A "suboptimal" weight gain resulted in a mean birth weight of 3,591 ± 447 g while excessive weight gain resulted in a mean weight of 3,872 ± 471 g.

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Analyses from the Pregnancy, Infection, and Nutrition Study (Deierlein et al., 2008) showed that compared to women consuming diets within the lowest quartile for energy density (defined as the number of calories/g of food consumed) during the second trimester, women consuming diets with energy density values in the third and highest quartiles gained a significant excess of over 1 kg in total GWG.

Beyond general food intake, several studies have also examined consumption of different types of food as well as macronutrient and micronutrient intake. Steven-Simon and McAnarney (1992) showed in a study of adolescents that those who consumed fewer than three snacks a day had slower weight gains during pregnancy. Olson and Strawderman (2003) found women who consumed three or more servings of fruits and vegetables per day gained 1.81 pounds less than those who consumed fewer servings during pregnancy. Olafsdottir et al. (2006) found that among women in Iceland, the percentage of energy intake from various macronutrients is an important predictor of weight gain only among overweight women and late in pregnancy. Compared with women gaining suboptimal weight, the diet of overweight women gaining excessive weight had higher energy percentage from fat and lower energy percentage from carbohydrates. They also found that consumption of dairy products and sweets in late pregnancy was associated with a decreased risk of inadequate gain and an increased risk of excessive gain during pregnancy.

In a small randomized clinical trial of a low-glycemic versus a high-glycemic diet, Clapp (2002) found that the women on the low-glycemic diet gained less weight during pregnancy (22.9 compared with 40.9 pounds). The mechanisms involved were thought to include changes in: daily digestible energy requirements (i.e. metabolic efficiency), substrate utilization (glucose oxidation versus lipid oxidation), and insulin resistance and sensitivity (Clapp, 2002). However, Deierlein et al. (2008) reported no statistical effect of glycemic load alone on total GWG or weight gain ratio. Their findings suggest that race/ethnicity may interact with glycemic processes, such that white women with glycemic load increases were more sensitive to increased weight gain during pregnancy; this was not true for black women

Altogether several studies have demonstrated a relationship between energy intake and GWG. Additional evidence suggests that dietary intake may also influence GWG however the evidence is insufficient to draw a conclusion.

Physical Activity

The American College of Obstetricians and Gynecologists (ACOG) took the position in 2002 that, in the absence of either medical or obstetric complications, 30 minutes or more of moderate exercise a day on most, if not all, days was recommended for pregnant women (ACOG, 2002). The ACOG report emphasized that participation in a wide range of recreational activities appears to be safe for pregnant women. Participation in activities with a high potential for trauma to the woman or fetus, however, should be avoided.

Published reviews on exercise and pregnancy concluded that the balance of evidence suggests a benefit of exercise during pregnancy, especially for maternal outcomes (Morris and Johnson, 2005; Gavard and Artal, 2008). Moderate exercise during a low risk pregnancy was found to be safe for both the mother and fetus and to improve overall maternal fitness and well-being as well as maternal and fetal outcomes (Morris and Johnson, 2005).

The report of the Physical Activity Guidelines Advisory Committee (DHHS, 2008) concluded that:

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a) Moderate-intensity leisure time physical activity is not associated with an increased risk of low birth weight, preterm delivery, or early pregnancy loss; and
b) Participation in vigorous activities has been associated with small reductions in birth weight compared to less active women (Hegaard et al., 2007; Leet and Flick, 2003) but not with gestational age at birth or birth weight (Evenson et al., 2002; Duncombe et al., 2006).

Gavard and Artal (2008) concurred with the latter findings however a Cochrane Review (Kramer and McDonald, 2006) concluded that the evidence was insufficient to evaluate the risks or benefits of exercise in pregnant women for infant outcomes.

Several studies have examined the effects of regular physical activity on GWG (Abrams et al., 2000; Siega-Riz et al., 2004). Based on theoretical energy calculation alone, it appears that regular physical activity has the potential to prevent excessive GWG. The main issue then becomes whether it can be shown to work in practice. A number of observational studies but few randomized controlled trials have been reported on this topic. A small number of reports have addressed the issue of the prevalence of physical activity behavior in pregnant women. A cross sectional survey of pregnant women found that about 48 percent reported some exercise participation during pregnancy (Hinton and Olson, 2001). The most common activities were walking, swimming and aerobics. In general, the proportion of exercising pregnant women declines across trimesters of pregnancy. In one study of 388 pregnant women, 41 percent were active before pregnancy (Ohlin and Rossner, 1994). By the third trimester, only 14 percent of the women continued to participate in aerobic exercise.

Two meta-analyses and several reviews have concluded that the level of physical activity in pregnant women did not have an influence on GWG (Lokey et al., 1991; Sternfeld et al., 1995; Stevenson, 1997; Kramer and Kakuma, 2003; Morris and Johnson, 2005). However, the meta-analyses did not take into account a number of key factors, including the most critical one: the level of physical activity-related energy expenditure. If the energy cost of the exercise program is very low, it should not be surprising that its influences on GWG cannot be shown.

Some observational studies suggest that maintaining an active lifestyle or adding physical activity to the normal daily schedule of the pregnant woman may attenuate GWG. Clapp and Little (1995) compared exercising women who became pregnant and who continued to exercise at least three times per week to a group of women who stopped exercising once they became pregnant. The rate of GWG and of subcutaneous fat accretion (determined by skinfold thickness) was similar between the two groups during the first and second trimesters but the exercising women gained significantly less body weight and skinfold thickness during the third trimester. On average, the pregnant women who continued to exercise gained about 3 kg less. These observations were from a Norwegian study of 467 pregnant women who answered a questionnaire on physical activity level in week 36 of their pregnancy (Haakstad et al., 2007). Women who exercised regularly had significantly lower weight gain than inactive women in the third trimester only.

In a study of 96 obese women with GDM self-enrolled in either a diet (n = 57) or an exercise plus diet (n = 39) program during the last two months of pregnancy, the mean weight gain per week was less in the exercise plus diet group (0.1 ± 0.4 kg versus 0.3 ± 0.4 kg) (Artal et al., 2007). The exercise session consisted of walking on the treadmill or cycling in a semi-recumbent position once a week followed by unsupervised exercise at home for the remaining six days. The

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exercise plus diet group exercised for 153 ± 91 min per week. Complications, infant birth weight, and the proportion of cesarean deliveries were comparable between the two groups.

Based on the limited available evidence, the Physical Activity Guidelines Advisory Committee concluded that "unless there are medical reasons to the contrary, a pregnant woman can begin or continue a regular physical activity program throughout gestation, adjusting the frequency, intensity, and time as her condition warrants" (DHHS, 2008). The committee added that "in the absence of data, it is reasonable for women during pregnancy and the postpartum period to follow the moderate-intensity recommendations set for adults unless specific medical concerns warrant a reduction in activity". It is commonly recognized however that adequately powered randomized, controlled intervention studies on the potential benefits and risks of regular physical activity at various dose levels in pregnant women are needed.

Physical activity, such as work, spontaneous activity, fidgeting, and personal chores as well as exercise account for a widely variable fraction of total energy expenditure. In some, this may reach only about 15 percent of daily energy expenditure while in others it may be as high as 50 percent (Hill et al., 2004). Most recently, Lof et al. (2008) assessed the effects of maternal physical activity level (PAL) and BMI on GWG in 223 healthy Swedish women. Pregravid PAL was related to decreased weight gain in the third trimester, about 0.10 kg/week less in the high PAL than in the low or medium PAL groups. Maternal BMI was inversely associated with weight gain in the second trimester but there was a positive association between maternal BMI and GWG in the third trimester. However, maternal smoking, parity, education, age, pre-gravid PAL explained only four percent of the variance in maternal weight gain and PAL was not related to birth weight.

In sum, several studies have demonstrated an inverse relationship between the level of physical activity and GWG. Based on energetic fundamentals alone, maintaining a reasonable level of exercise-related energy expenditure during pregnancy should moderate GWG. Energy requirements based on PAL are provided in Appendix B.

Substance Abuse

Cigarette smoking Taken together, early studies examining associations between decreasing GWG and amount of reported smoking show inconclusive results. Rush (1974) found a strong relationship between amount of smoking and decreasing GWG ($p < 0.01$) while Garn et al. (1979) found no association between smoking and non-smoking mothers and GWG. Several investigators examined whether smoking had a negative effect on caloric intake as a causative factor for higher incidence of SGA in smoking mothers. Haworth et al. (1980) found that women who smoked during pregnancy actually had higher mean caloric intakes with no difference in GWG; but a greater number of low birth weight infants than non-smokers. Similarly, Papoz et al. (1982) found higher mean caloric intake and lower birth weight in women who smoked during pregnancy. More recently, Furuno et al. (2004) found no significant difference in mean GWG between smoking and non-smoking mothers but did find a slightly increased (1.3-fold) risk for low GWG among smokers.

Although there is limited evidence that cigarette smoking may be inversely associated with GWG there is a preponderance of evidence that supports an independent effect of smoking on birth weight (Muscati et al., 1988; Wolff et al., 1993; Adriananse et al., 1996). Seckler-Walker and Vacek (2003) examined the effect of smoking on birth weight independent of GWG and found that gains in infant birth weight among mothers who stopped smoking during pregnancy were not related to GWG, but rather to the independent effect of smoking on birth weight.

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Alcohol use Little information is available about effects of alcohol consumption on GWG. Wells et al. (2006) assessed biological, psychological and behavioral characteristics to determine associations with inadequate or excessive GWG. This analysis found no significant association between smoking and drinking and GWG outside the IOM (1990) guidelines. Little et al., (1986) found no difference in GWG between infrequent (< 7.5 g/day), occasional (7.5-15 g/day), and regular (≥ 15g/day) alcohol consumers during pregnancy. In a study of determinants of GWG in poor black adolescent mothers, Stevens-Simon and McAnarney (1992) found that alcohol use was more frequent among mothers who experienced rapid GWG. Alcohol, however, is a potent teratogen and its effects on pregnancy outcome are independent of GWG (Hanson et al., 1978; Little et al., 1986; Jacobsen et al., 1994; Bagheri et al., 1998). Thus, any impact of alcohol consumption on GWG is of little relevance compared to its teratogenic effects.

Drug use Amphetamines are anorectic drugs and their use during pregnancy would be expected to result in low GWG. Smith et al. (2006) assessed a cohort of 1,618 pregnant women that included 84 methamphetamine users. Analysis of GWG in the methamphetamine exposed group showed that those who used the drug in the first two trimesters but ceased use by the third trimester gained significantly more weight than either women who used throughout pregnancy or non-exposed women, suggesting the anorexic effects of methamphetamine are limited to continuous use, and there may be a rebound in weight gain if the mother stops use. Nevertheless, this study found exposure to methamphetamines increased the incidence of SGA births 3.5 times over the non-exposed group. Graham et al. (1992) conducted a prospective study with 30 women who were social users of cocaine during the first trimester of pregnancy. No significant differences were found between the drug users and non-users for GWG, delivery complications, birth weight, and other adverse outcomes. Chronic use of cocaine, however, has been shown to be associated with adverse maternal and fetal consequences (Wagner et al., 1998; Ogunyemi and Hernandez-Loera, 2004).

Unintended Pregnancy

Evidence for an effect of unintended pregnancy on GWG appears to be conflicting. Hickey et al. (1997) found that mistimed or unplanned pregnancy was associated with an increased risk for insufficient GWG among black but not among white women. In a study by Siega-Riz and Hobel (1997), planned pregnancy was associated with a marginally statistically significant decreased risk for insufficient GWG, but only among the low and normal weight subjects in a Hispanic cohort. Using data from the National Longitudinal Survey of Labor Market Experiences of Youth, Marsiglio and Mott (1988) found in a cohort of 6,015 primiparous women that not desiring a pregnancy was not a significant predictor of very low prenatal weight gain. Several large population-based surveys have not found an association between GWG and planned pregnancy (Kost et al., 1998; Wells et al., 2006).

VULNERABLE POPULATIONS

Seasonal Migrant Workers

The actual number of migrant farm workers currently in the U.S. in not known, but estimates are that at least 3-5 million migrant and seasonal workers come to the U.S. each year (CDC, 1997). Further, approximately 16 percent of migrant workers are women. Data about GWG among migrant women in four states was obtained through the Pregnancy Nutrition Surveillance

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System (PNSS). Analysis of the data collected showed that about 52 percent of migrant women gained less than the range recommended by IOM (1990) compared to 32 percent of non-migrant women. Mean weight gain was also lower for migrant women (22.9 pounds) compared to non-migrant women (29.7 pounds). However, even though migrant women had lower GWG than non-migrant women, the prevalence for adverse birth outcomes (low birth weight, very low birth weight, preterm birth, and small for gestational age) was similar for both groups (CDC, 1997). Similarly, Reed et al. (2005) found that migrant women had higher rates of pregnancy-related risk factors but lower rates of adverse birth outcomes compared to non-migrant women.

Military

The committee was unable to identify studies that specifically examined GWG among women in military service. Several studies found women in active-duty experience greater stress but less job control, as well as higher rates of depression, compared to a parity-matched control group of dependent military wives (Magann and Nolan, 1991; O'Boyle et al., 2005), but it is unclear how these factors might influence GWG. One study surveyed pregnant women with deployed partners (Haas and Pazdernik, 2006). Women whose partners were deployed showed a greater tendency to deliver a large infant and reported more stress and changed eating habits, compared to women whose partners were not deployed; however, the results were not statistically significant. No difference was seen in the gestational age at delivery, percentage with vaginal delivery, average number of children at home, self-reported stress, or reported GWG.

Women Incarcerated During Pregnancy

The U.S. Department of Justice estimates that women offenders account for about 16 percent of the total corrections population (Bureau of Justice Statistics, 1999). Recent estimates show that the number of women under the jurisdiction of State or Federal prison authorities increased 1.2 percent from year-end 2007, reaching 115,779 (Available: http://www.ojp.usdoj.gov/bjs/prisons.htm. Accessed April 13, 2009). Of women who are incarcerated, most are of child-bearing age and approximately 6 percent are pregnant (Bureau of Justice Statistics, 1994; Safyer and Richmond, 1995).

While there are no studies that have examined the direct effect of incarceration on GWG *per se*, several studies (Martin et al., 1997a, 1997b; Bell et al., 2004) have examined its effect on birth weight. Martin et al. (1997a) found that a higher number of pregnancy days spent incarcerated was found to be associated with higher infant birth weight. Furthermore, Martin et al. (1997b) also found that infant birth weights among women incarcerated during pregnancy were not significantly different from women never incarcerated; however, infant birth weights were significantly worse among women incarcerated at a time other than during pregnancy than among never-incarcerated women and women incarcerated during pregnancy, suggesting certain aspects of the prison environment, such as shelters and regular meals, may be protective particularly for high-risk pregnant women.

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FINDINGS AND RECOMMENDATIONS

Findings

1. There is a lack of evidence on societal/institutional (media, culture/acculturation, health services, policy), environmental (altitude, exposures to environmental toxicants, disasters), and neighborhood determinants (access to healthy foods, opportunities for physical activities) of GWG.

    a. Few of the studies reviewed considered the influence of the many possible determinants of GWG among women of different racial/ethnic and socioeconomic groups, or alternatively, adjusted for race/ethnicity or SES in their analyses.
    b. There is insufficient evidence to evaluate the influences of psychological factors such as depression, stress, social support, or attitude toward GWG on actual GWG.
    c. There remains a lack of information to relate dietary intake or physical activity to GWG even though they are primary determinants of weight gain in non-pregnant individuals.

2. Married women are more likely to have appropriate GWG than unmarried women. Intimate partner violence is associated with insufficient GWG. There is a paucity of studies examining the influence of partner/family support on GWG.
3. GWG is generally higher among adolescents and lower among women > 35 years of age, although the relationship of GWG among these groups to birth outcomes, post-partum weight retention, and subsequent risk for overweight/obesity remains unclear.
4. There is a lack of evidence on GWG among vulnerable populations, specifically, seasonal migrant workers, women in military service, and women incarcerated during pregnancy.
5. The IOM (1990) GWG guidelines appear to influence what women believe to be appropriate weight gain during pregnancy, though their influence on actual GWG is less clear in part because many health professionals are providing no or inappropriate advice about weight gain during pregnancy.
6. There is growing evidence suggesting that specific fetal and maternal genes and alleles can influence GWG, though both parental genotypes appear to affect birth weight. The effect of developmental programming and epigenetic events on GWG is strongly suspected but direct evidence is still lacking. Leptin and adiponectin may represent markers of insulin sensitivity or other mechanisms affecting gestational weight changes.

Research Recommendation

Research Recommendation 4-1: The committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to conduct studies in large and diverse populations of women to understand how dietary intake, physical activity, dieting practices, food insecurity and, more broadly, the social, cultural and environmental context affect GWG.

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Areas for Additional Investigation

The committee identified the following areas for further investigation to support its research recommendations. The research community should conduct studies on:

- social, cultural, and environmental contexts of GWG. Findings from these studies should help guide the development of implementation strategies for GWG recommendations;
- healthcare providers' knowledge, attitude, and behavior with respect to GWG recommendations. These studies should identify facilitators and barriers to adoption of GWG recommendations by healthcare providers in their clinical practice;
- partner and family influences on GWG;
- influences of genetic factors, epigenetic events, and developmental programming on GWG;
- how GWG affect birth outcomes, postpartum weight retention, and overweight and obesity in later life among adolescents and older women. Findings from these studies should be used to reevaluate the appropriateness of GWG recommendations for these women.
- determining whether maternal biomarkers such as leptin, adiponectin, and other markers of insulin sensitivity can be used to enhance clinical prediction of adverse birth outcomes and guide further interventions for women with GWG outside the recommended ranges. Data on relevant biomarkers should be made available through databases such as the Federal Human Nutrition Research and Information Management (HNRIM) System Database; and
- influences of psychological factors, such as depression, stress, social support, and attitude toward GWG on actual GWG.

The Department of Health and Human Services (HHS) or other appropriate federal agencies should:

- track racial-ethnic and socioeconomic disparities in GWG and that the research community conduct studies on how GWG affect birth outcomes, postpartum weight retention, and overweight and obesity in later life among women of different racial-ethnic and socioeconomic groups;
- collect nationally representative data on dietary intake, physical activity, and food insecurity among prepregnant, pregnant and postpartum women**,** and report these data by prepregnancy body mass index (including all classes of obesity), age, racial/ethnic group, and socioeconomic status; and
- collect data on GWG among vulnerable populations.

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5

Consequences of Gestational Weight Gain for the Mother

Women whose weight gain during pregnancy is outside of the recommended ranges may experience various adverse maternal outcomes, which may include increased risk for pregnancy-associated hypertension, gestational diabetes (GDM), complications during labor and delivery, and postpartum weight retention and subsequent maternal obesity as well as an increased risk for unsuccessful breastfeeding. As noted in Chapter 1 and discussed in detail in Chapter 2, there is an increased prevalence in the United States of women who are overweight or obese entering pregnancy, also putting them at greater risk for several of these same adverse pregnancy outcomes. Additionally, more women are becoming pregnant at an older age and are thus entering pregnancy with chronic conditions, such as type 2 diabetes that could contribute to increased morbidity during both the prenatal and postpartum periods.

To review the evidence on outcomes related to gestational weight gain (GWG) within or outside the 1990 Institute of Medicine (IOM) guidelines, the Agency for Healthcare Research and Quality (AHRQ) commissioned a comprehensive, systematic evidence-based review of the literature since the release of the IOM (1990) report *Nutrition During Pregnancy*. This review included evidence on the consequences of GWG for both the mother and infant (Viswananthan et al., 2008). The committee used this review as a foundation for discussion of the state of the science for GWG and maternal outcomes in this chapter as well as for infant outcomes in Chapter 6. It is important to note that research since 1990 has focused on the consequences of gains *above* the IOM (1990) recommendations—to the exclusion of gains *below* those recommendations—on maternal and infant outcomes. The reader is referred to IOM (1990) for a discussion of the consequences of gaining too little weight gain during pregnancy.

The following briefly reviews the state of the science before the IOM (1990) report then summarizes findings from the recently published AHRQ evidence-based review on outcomes of gestational weight gain (GWG) (Viswanathan et al., 2008). These discussions also include articles published since the AHRQ report in which associations between GWG and maternal outcomes are examined (see Appendix F for summary data tables).

To interpret the rating scales from the AHRQ report, the committee indicates how the articles were rated and how the strength of the evidence was determined. The methodological approach and system of rating articles used in the AHRQ review is provided in Appendix E.

CONCEPTUAL FRAMEWORK: CONSEQUENCES OF GESTATIONAL WEIGHT GAIN FOR THE MOTHER

The committee's conceptual framework (see Chapter 1) illustrates a model for maternal and child outcomes consequent to GWG outside the ranges recommended by the IOM (1990) report (Figure 5-1). There are numerous potential causal factors, including environmental factors, that can influence the determinants of GWG and its consequences and others that may affect those

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consequences by other routes. These consequences, i.e. adverse health outcomes to the mother, can arise in the prenatal and/or postpartum periods. Among the well-studied prenatal maternal outcomes that result from excessive GWG are pregnancy-associated hypertension (including preeclampsia and eclampsia) and risk of complications in labor and delivery. In the postpartum period, weight retention can lead to higher weight status in subsequent pregnancies as well as weight retention and other long-term maternal health consequences such as increased risk for type 2 diabetes and cardiovascular disease. Unfortunately the literature in this area does not allow inference of causality because it is based solely on observational studies.

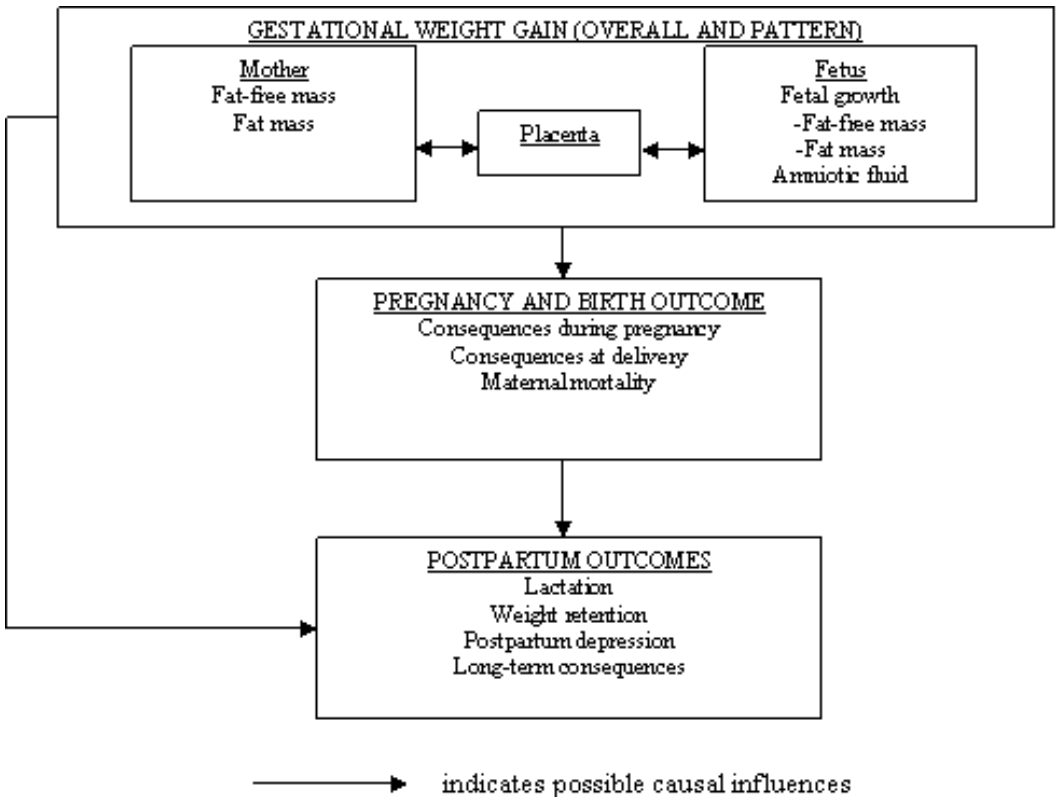

FIGURE 5-1 Schematic summary of maternal consequences associated with gestational weight gain.

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CONSEQUENCES DURING PREGNANCY

Gestational Diabetes and Impaired Glucose Tolerance

Pregnancy is frequently accompanied by a pronounced physiological decrease in peripheral insulin sensitivity (reviewed in Chapter 3). Gestational diabetes, the development of abnormal glucose tolerance only during pregnancy, generally arises from the combination of decreased peripheral insulin sensitivity and beta-cell dysfunction. It is well established that women who are obese when they enter pregnancy tend to develop a more pronounced insulin resistance and are at greater risk for GDM than are non-obese women (Dahlgren, 2006; Chu et al., 2007). The incidence of GDM has increased dramatically in recent decades (See Chapter 2). From 1989 to 2004 there was a relative increase in prevalence of GDM of 122 percent for the U.S. population as a whole; and 260 percent among African American women (Getahun et al., 2008).

Most women with normal glucose tolerance develop elevated blood ketones with ketonuria at various times during pregnancy (Chez and Curcio, 1987). Pregnant women with diabetes, on the other hand, are more likely to develop sustained elevated blood ketones and ketonuria than women with normal glucose tolerance in pregnancy. Gin et al. (2006), who measured capillary blood ketones and beta-hydroxybutyrate in women with normal glucose tolerance (controls) and those with GDM three times a day between 25 and 37 weeks gestation, found that fasting ketonuria was strongly correlated with ketonemia in controls but not women with GDM. Maternal ketonuria or acetonuria during pregnancy is a concern because it can result in neonatal or childhood neurocognitive dysfunction (see discussion in Chapters 3 and 6).

As described in the AHRQ review (Viswanathan et al., 2008), the literature since 1990 includes 11 published articles that together provide weak evidence in support of an association between GWG and development of abnormal glucose metabolism (either GDM or impaired glucose tolerance). Four of the studies reported that GWG above the range recommended in the IOM (1990) report was positively associated with abnormal glucose tolerance (Edwards et al., 1996; Kieffer et al., 2001; Kabiru and Raynor, 2004; Saldana et al., 2006). Three studies reported that women whose GWG was below the recommended range had a higher likelihood of GDM (Thorsdottir et al., 2002; Brennand et al., 2005; Kieffer et al., 2006) and four studies found no significant association between GWG and glucose tolerance (Bianco et al., 1998; Murakami et al., 2005; Seghieri et al., 2005; Hackmon et al., 2007). All but one study (Saldana et al., 2006) were limited by the use of total GWG as the exposure variable rather than weight gain until the time of diagnosis. This is problematic because management of GDM includes dietary counseling and efforts to control weight gain.

Catalano et al. (1993) reported that weight gain in women who developed GDM was less than in women with normal glucose tolerance primarily as a result of their greater pregravid weight. When GWG was assessed separately for early, mid- and late gestation in women with GDM, there was a significantly decreased rate of weight gain in overweight women with GDM, although only from 30 weeks gestation until delivery. There is biologic plausibility for an effect of GWG on the development of glucose tolerance: higher GWG could result in greater fat deposition, which could then influence insulin sensitivity. The body of evidence to date, however, is weak in support of such an association.

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Hypertensive Disorders

Hypertensive disorders during pregnancy include pregnancy-induced hypertension, pre-eclampsia, and eclampsia. The risk for pregnancy-induced hypertension is greater among women who enter pregnancy overweight or obese. Thadhani et al (1999) examined the relationship between pre-gravid BMI, elevated cholesterol, and the development of hypertensive disorders of pregnancy among 15,262 women. The age-adjusted relative risk for developing gestational hypertension was 1.7 and 2.2 for women with BMI values of 25-29.9 and $\geq$ 30 kg/m^2, respectively, compared to women with BMI values < 21 kg/m^2.

Preeclampsia is about twice as prevalent among overweight and about three times as prevalent among obese women as it is among normal weight women (Sibai et al., 1997; Catalano et al., 2007). Furthermore, the severity of the disease increases as BMI increases (Bodnar et al., 2007). The IOM (1990) report described the relationship between GWG and hypertensive conditions as being unclear because of limited and inclusive data. Since that report was published, 12 studies were examined in the AHRQ review (Viswanathan et al., 2008). Out of five studies (two rated fair and the rest rated poor) that examined the outcome of pregnancy-induced hypertension (Edwards et al., 1996; Bianco et al., 1998; Thorsdottir et al., 2002; Brennand et al., 2005; Jensen et al., 2005), an association between higher GWG and pregnancy-induced hypertension was found in only two of them (Thorsdottir et al., 2002; Jensen et al., 2005). The studies, however, differed in control for confounding. Thorsdottir et al. (2002) adjusted for age, parity, height, and gestational age. Jensen et al (2005) adjusted for 2-hour oral glucose tolerance test (OGTT) result, maternal age, prepregnancy BMI, gestational age (continuous variables), parity, smoking, and ethnic background. As a result, the relationship between increased GWG and onset of hypertension continues to remain unclear.

The outcome of preeclampsia has been examined in a total of 10 studies (Edwards et al., 1996; Ogunyemi et al., 1998; Thorsdottir et al., 2002; Kabiru and Raynor, 2004; Brennand et al., 2005; Murakami et al., 2005; Cedergen, 2006; Wataba et al., 2006; DeVader et al., 2007; Kiel et al., 2007), of which seven were rated fair and the rest were rated of poor quality. Overall, an association between higher total GWG and higher risk of pre-eclampsia was found in six of these studies (Edwards et al., 1996; Ogunyemi et al., 1998; Brennand et al., 2005; Cedergen, 2006; DeVader et al., 2007; Kiel et al., 2007). Lower total weight gains were found to be protective in four studies (Brennand et al., 2005; Cedergren, 2006; DeVader et al., 2007; Kiel et al., 2007). Those studies that did not find an association for high total GWG (Thorsdottir et al., 2002; Kabiru and Raynor, 2004; Murakami et al., 2005; Wataba et al., 2006) were primarily conducted in women who were not overweight or obese (two were conducted in Japan). Two studies not included in the AHRQ review using birth certificate data from the state of Missouri (Kiel et al., 2007 and DeVader et al., 2007), showed similar results, namely that GWG above the recommended range leads to higher risk of preeclampsia among overweight women (Langford et al., 2008). These studies were also limited by methodological problems associated with the use of total weight gain as the exposure as opposed to a weight gain before the diagnosis of preeclampsia. There was also a lack of a consistent definition for preeclampsia across these studies, which makes it difficult to compare them.

Preeclampsia is a condition noted for a decrease in the normal (50-60 percent) expansion in maternal intravascular (plasma) volume. The condition may also affect weight gain in early gestation. In addition, increased vascular permeability and decreased plasma oncotic pressure, caused by preeclampsia, can lead to increased edema and excessive weight gain in late gestation. Hence placental dysfunction in early gestation may effect both early and late weight gain—albeit

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in opposite directions. These physiologic parameters preclude the use of total weight as a measure of GWG in preeclampsia.

Other Quality of Life Measures

As noted in Chapter 4, the influence of psychosocial status on GWG has been examined in several studies. However, there was no evidence available about possible effects of GWG on a woman's mental health during pregnancy and there was no information in the IOM (1990) report related to other measures of quality of life.

There are eight studies covered in AHRQ review (Viswanathan et al., 2008) on other antepartum outcomes. Topics include a composite outcome for discomfort in general (Rodriguez et al., 2001), physical energy and fatigue (Tulman et al., 1998), stretch marks (Madlon-Kay, 1993; Atwal et al., 2006), heartburn (Marrero et al., 1992), gallstones (Lindseth and Bird-Baker, 2004; Ko, 2006), and hyperemesis (Dodds et al., 2006). Three of these studies were rated as fair (Tulman et al., 1998; Rodriguez et al., 2001; Ko, 2006) and five as poor-quality (Marrero et al., 1992; Madlon-Kay, 1993; Lindseth and Bird-Baker, 2004; Atwal et al., 2006; Dodds et al., 2006). Overall, there was no association between higher GWG and the outcomes of interest except for the two studies in which stretch marks were examined (Madlon-Kay, 1993; Atwal et al., 2006). This association was weak because of the small sample size, study design (one was a cross-sectional study), and the lack of adjustment for confounding factors. In the one study in which hyperemesis was examined, women who gained a total of < 7 kg had an increased likelihood of more antenatal admissions for this outcome (Dodds et al., 2006). For this outcome in particular, GWG was not a causal factor but was more likely the result of having had hyperemesis during the pregnancy.

In summary, evidence for an association between GWG and pregnancy complications such as GDM and gestational hypertensive disorders is inconclusive because of inconsistent results and methodological flaws. The evidence for the outcome of mental health during pregnancy is understudied.

CONSEQUENCES AT DELIVERY

In IOM (1990), the link between GWG and complications during labor and delivery was viewed as a consequence of delivering a large-for-gestational age (LGA) infant. This report concluded that the contribution of GWG to these outcomes was quite small. Since then, the literature has grown and the outcomes related to delivery have been subdivided so as to understand the process of labor more fully.

Induction of Labor

In the AHRQ review (Viswanathan et al., 2008) five studies were reviewed related to an association between GWG and induction of labor (Ekblad and Grenman 1992; Kabiru and Raynor 2004; Jensen et al., 2005; Graves et al., 2006; DeVader et al., 2007.) The strength of the evidence from these studies was rated weak for an association between high GWG and labor induction or failure of labor induction. A statistically significant increase in the outcomes associated with high GWG was found in all of the studies. Comparisons across studies however were not meaningful because of differences in the definition of high GWG and a lack of control for confounding factors.

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Length of Labor

Three studies in the AHRQ review (Viswanathan et al., 2008) examined associations between GWG and length of labor (Ekblad and Grenman, 1992; Johnson et al., 1992; Purfield and Morin, 1995). The evidence was rated weak for an association between higher GWG and longer duration of labor. Two of the three studies found a significant increase in the length of labor with higher weight gains, but both lacked control for confounding factors (Ekblad and Grenman, 1992; Purfield and Morin, 1995).

Mode of Delivery

The association between GWG and mode of delivery has been examined in many studies. A total of 21 studies reviewed in the AHRQ review (Viswanathan et al., 2008) examined this association using GWG as a continuous or categorical variable unrelated to the IOM (1990) guidelines (Ekbald and Grenman, 1992; Johnson et al., 1992; Purfield and Morin, 1995; Witter et al., 1995; Bianco et al., 1998; Shepard et al., 1998; Young and Woodmansee, 2002; Joseph et al., 2003; Chen et al., 2004; Kabiru and Raynor, 2004; Brennand et al., 2005; Jensen et al., 2005; Murakami et al., 2005; Rosenberg et al., 2005; Cedergren, 2006; Graves et al., 2006; Wataba et al., 2006; DeVader et al., 2007; Jain et al., 2007; Kiel et al., 2007; Sherrard et al., 2007). Overall, these studies provided moderate evidence of an association between high GWG and cesarean delivery; only four studies failed to find an association (Bianco et al., 1998; Brennand et al., 2005; Murakami et al., 2005; Graves et al., 2006). An important factor to consider in this literature is the route of previous delivery for multiparous women. Only half of the studies reviewed adjusted for this and, among those that did, five also adjusted for co-morbidities that could also have contributed to the route of delivery (e.g. GDM and preeclampsia) (Witter et al., 1995; Shepard et al., 1998; Joseph et al., 2003; Rosenberg et al., 2005; Sherrard et al., 2007). Higher weight gains were associated with instrumental deliveries in three (Purfield and Morin, 1995; Kabiru and Raynor, 2004; Cedergren, 2006) studies but not in two others (Ekblad and Grenman, 1992; DeVader et al., 2007).

When GWG was categorized according to the ranges recommended in the IOM (1990) report, the body of research provided moderate evidence that weight gain above the recommended ranges was associated with cesarean delivery among normal- and underweight women. In contrast, the evidence among obese and morbidly obese women was rated as weak (Parker and Abrams, 1992; Edwards et al., 1996; Bianco et al., 1998; Thorsdottir et al., 2002; Stotland et al., 2004; Hilakivi-Clarke et al., 2005; DeVader et al., 2007; Kiel et al., 2007).

There was a consistent observation (noted in 10 studies) that women who were overweight or obese before pregnancy were at higher risk of cesarean delivery compared to women who entered pregnancy at a lower BMI (Johnson et al., 1992; Witter et al., 1995; Shepard et al., 1998; Joseph et al., 2003; Chen et al., 2004; Murakami et al., 2005; Rosenberg et al., 2005; Graves et al., 2006; Jain et al., 2007; Sherrard et al., 2007).

Maternal Mortality

Both the IOM (1990) report and the AHRQ review (Viswanathan et al., 2008) concluded that there was no information on the relationship between GWG and maternal mortality. From a theoretical perspective, if GWG above recommended ranges is associated with LGA infants and shoulder dystocia in settings that do not allow for immediate cesarean delivery or attendance by a trained clinician, the mother could die during childbirth. In such an event, the immediate cause

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of death would be attributed to the size of the infant and associated labor and delivery complications. This impedes the study of consequences of GWG on maternal mortality. In summary, current evidence supports a strong association between GWG above recommended ranges and increased risk of cesarean delivery. There is no evidence, however, to support an association of GWG with maternal mortality in countries where women have ready access to obstetric care.

POSTPARTUM CONSEQUENCES

Lactation

IOM (1990) reported only one study that examined the relationship between GWG and milk quality and quantity (Butte et al., 1984). There was no relationship between GWG and these outcomes in this study.

The AHRQ review (Viswanathan et al., 2008) includes four studies on the association of GWG, categorized according to the recommendations of IOM (1990), with lactation performance (Rasmussen et al., 2002; Li et al., 2003; Hilson et al., 2006; Baker et al., 2007). These studies provide evidence rated moderate that low weight gain is associated with decreased initiation of breastfeeding, and weak for any association between GWG and duration of exclusive or any breastfeeding. Three of the studies reviewed showed that obese women had a shorter duration of breastfeeding (both exclusive and any breastfeeding) regardless of GWG (Rasmussen et al., 2002; Hilson et al., 2006; Baker et al., 2007). Subsequently, Manios et al. (2008), in a cross-sectional study done in Greece, found results similar to those in the AHRQ review (Viswanathan et al., 2008). This study found that women with higher prepregnancy BMI were less likely to initiate breastfeeding and that GWG had no effect on either initiation or duration of breastfeeding.

Postpartum Weight Retention

A woman's weight immediately after delivery of the fetus, placenta, and amniotic fluid is termed her postpartum weight. In the subsequent days to weeks, the increase in the woman's extracellular and extravascular water that occurred during pregnancy is lost and her plasma volume returns to prepregnancy values. The amount of weight that remains then minus her pregravid weight is termed postpartum weight retention. It reflects the increased breast tissue being used for lactation as well as any remaining fat mass that was gained during pregnancy.

IOM (1990) stated that women with GWG well beyond the recommended ranges are more likely to retain weight postpartum and are at increased risk for subsequent obesity. The emphasis of the IOM (1990) guidelines, however, was on infant outcomes rather than maternal postpartum weight retention in an effort to optimize birth weight.

The AHRQ review only included studies that directly examined associations between GWG and postpartum weight retention and did not include those that used parity or childbearing as a proxy for GWG. The report only found two studies that examined differences in the amount of fat retained in the postpartum period for GWG according to IOM (1990) categories (Butte et al., 2003; Lederman et al., 1997). Butte et al. (2003) examined a convenience sample of non-smoking women aged 18-40 from Houston (17 underweight, 34 normal weight, 12 overweigh/obese). Body composition was measured using dual-energy x-ray absorptiometry (DXA) before and after pregnancy and weight was obtained before pregnancy, during pregnancy,

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and after pregnancy. Results showed that maternal fat retention was significantly higher among women who gained above (5.3 kg) compared to those who gained within (2.3 kg) or below (-0.5 kg) the IOM (1990) guidelines.

Lederman et al. (1997) studied 196 non-smoking women aged 18-36 years, recruited from 3 prenatal clinics in New York City. Women who gained below the IOM (1990) recommendations had the lowest fat gain from 14 to 37 weeks of gestation compared to those with an intermediate and those with the highest fat gain. In addition the study found that, among obese women who gained within the IOM (1990) guidelines, the amount of body fat change (-0.6 kg) was significantly lower than among women in the other BMI groups who also gained within the recommendations (6.0 for underweight, 3.8 for normal, and 2.8 kg for overweight women). Unfortunately no test of significance was conducted. These data suggest, however, that higher GWG results in higher maternal fat gains, although the evidence for this is weak because of the limited number of studies and small sample sizes.

The AHRQ review (Viswanathan et al., 2008) separated the studies on postpartum weight retention into three categories according to the time postpartum when weight retention was assessed: short-term, less than 11 weeks; intermediate, 3 months to 3 years, and long-term, greater than 3 years. Within the short-term (≤ 11 weeks) studies, there was weak evidence for a relationship between GWG as a continuous variable and postpartum weight retention (Muscati et al., 1996). However, when GWG was categorized according to IOM (1990), there was a moderate, consistent relationship. Four studies showed that GWG exceeding the IOM (1990) guidelines was associated with higher postpartum weight retention (Stevens-Simon and McAnarney, 1992; Scholl et al., 1995; Luke et al., 1996; Walker et al., 2004). This observation was consistent for women irrespective of age.

In the intermediate-term (3 months to 3 years), one study rated good (Harris et al., 1999), three studies rated fair (Ohlin and Rossner, 1990; Soltani and Fraser, 2000; Walker et al., 2004), and one study rated poor (Parham et al., 1990) showed moderate evidence for a relationship between GWG above recommended ranges and greater postpartum weight retention. The strength of the evidence was the same for subjects who gained above the IOM (1990) prepregnancy guidelines as for those who stayed within the guidelines, based on five studies rated fair (Scholl et al., 1995; Walker, 1996; Rooney and Schauberger, 2002; Olson et al., 2003; Amorim et al., 2007) and one study rated poor (Keppel and Taffel, 1993). Thus, overall, higher GWG is associated with greater postpartum weight retention measured at 3 to 36 months postpartum. The authors noted, however, that the data should be interpreted cautiously because of a lack of consistent adjustment for covariates such as nutrition and exercise. In interpreting these data, it is important to note that the relationship between GWG and postpartum weight retention depends not only on dietary intake and physical exercise, but also on breastfeeding behavior. In the only available study that considered prepregnancy BMI, GWG, and breastfeeding simultaneously, Baker et al. (2008) showed that women from the Danish National Birth Cohort with reasonable weight gains (e.g. ~12 kg) and exclusively breastfed for 6 months as currently recommended would have no weight retention at 6 months postpartum. For racial/ethnic groups, only one study was available. Keppel and Taffel (1993) used a nationally representative database to show that black women retained more weight than white women regardless of GWG.

In the long-term (> 3 years), the evidence is less conclusive for a relationship between GWG and postpartum weight retention. One study rated good, Callaway et al. (2007) found a weak association between GWG and weight of the mother 21 years after the pregnancy, while in

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another study rated fair, Linne et al. (2003) found that women who became overweight after 15 years had higher GWG in the index pregnancy compared to women who remained within a normal weight range (although no adjustment was made for confounding). In the latter study, the authors also concluded that women who began pregnancy at a higher BMI tended to stay on the same weight trajectory later in life (Linne et al., 2004). There was moderate evidence in support of a relationship between gaining above the IOM (1990) guidelines and greater postpartum weight retention based on findings from three studies rated as fair (Rooney and Schauberger, 2002; Rooney et al., 2005; Amorim et al., 2007); however the amount of weight retained was small.

Studies by Gunderson (2004) and Rosenberg et al. (2003), although not included in the AHRQ review, provide information that is consistent with its conclusions. The work of Nohr et al. (2008) also largely corroborates these earlier findings and strengthens the evidence for an association between GWG and postpartum weight retention in the intermediate period. Nohr et al. (2008) gathered data from 60,892 women with term pregnancies in the Danish National Birth Cohort. They linked these data to birth and hospital-discharge registers. After adjustment for multiple confounding factors, they reported that women who gained 16-19 kg or ≥ 20 kg were at 2.3- and 6.2-fold higher odds of retaining ≥ 5 kg at 6 months postpartum than women who gained only 10-15 kg. The study results were attenuated for the measurements obtained at 18 months postpartum, which was only based on approximately one-half of the original study cohort.

A major concern with postpartum weight retention is that a woman may move into a higher BMI category than she was in before pregnancy. Being in this higher category is then associated with a greater risk of pregnancy complications and adverse birth outcomes in a subsequent pregnancy. Scholl et al. (1995) calculated that adolescent women (12-29 years old) had a 2.8-fold higher risk of becoming overweight at 6 months postpartum if their rate of weight gain during pregnancy was > 0.68 kg per week than women with lower gains. Gunderson et al. (2000) observed a similar magnitude of effect in adults when she calculated the risk of becoming overweight at the start of the second pregnancy with weight gains above the IOM (1990) recommendations in the first. Nohr et al. (2008) also showed that with GWG between 16-19 kg, 12 to 14 percent of women with pregravid BMIs > 18.5 kg/m^2 move up one category of weight status at 6 months postpartum and that this increases to 25 percent with weight gains > 20 kg.

Overall the evidence suggests that low GWG is moderately associated with initiation of breastfeeding and that there is a strong association between higher GWG and postpartum weight retention (3 months to 3 years). The outcome of mental health is understudied and worthy of exploration.

Postpartum Depression

There were no data on the relationship of GWG and postpartum depression in the IOM (1990) report. The AHRQ review (Viswanathan et al., 2008) does not include data on this relationship. The committee was unable to identify new data on this possible relationship.

LONG-TERM CONSEQUENCES

The IOM (1990) report did not address long-term maternal outcomes of GWG. Excess postpartum weight retention could exacerbate these problems (see discussion above) and contribute to the development of chronic conditions that include diabetes, hypertension, and other cardiovascular risk factors (Arendas et al., 2008).

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Type 2 Diabetes/Metabolic Disorders

The committee was unable to identify published studies that examined the possible association between GWG and the development of metabolic disorders later in a woman's life. Such an association is biologically plausible because of the link between GWG and postpartum weight retention. Gunderson et al. (2008) showed that childbearing was associated with increased visceral fat postpartum, but their study did not include data on GWG. Berg and Scherer (2005) reviewed evidence on the role of adipose tissue in systemic inflammation and determined that the distribution of fat is important as well as the amount. Visceral fat in obese subjects was shown to be more strongly associated with insulin resistance than visceral fat in lean subjects.. Lim et al. (2007) identified a relationship between abnormal glucose tolerance at one year post-partum and increased visceral fat in women who had GDM that was independent of maternal age and BMI.

Cardiovascular Disorders

The committee was also unable to identify any published studies that examined a direct association between GWG and the development of cardiovascular disorders later on in life. However, obesity, preeclampsia, or toxemia of pregnancy is linked to long-term sequelae that include cardiovascular disease (Bellamy et al., 2007; Zhang et al., 2008).

Other Adverse Health Outcomes

Mental Health

The topic of mental health of the mother is not addressed in the AHRQ review (Viswanathan et al., 2008). Two small studies (Jenkin and Tiggemann, 1997; Walker, 1997) provide weak evidence regarding the connection between post-partum weight retention up to one year post-delivery and self-esteem/depression. These studies did not control for prepregnancy BMI.

Cancer

There is weak evidence for an association of GWG with risk of breast cancer. One retrospective cohort study of 2,089 Finnish women showed a positive relationship between weight gain in the upper tertile (> 15 kg) and post-menopausal breast cancer risk, after adjustment for prepregnancy BMI (RR = 1.62, 95% CI: 1.03-2.53) (Kinnunen et al., 2004). In a nested case-control study of 65 cases of breast cancer in this cohort, the BMI at the time of diagnosis did not change the findings. Although there was a relationship between increased weight gain and an increased risk of post-menopausal breast cancer in this cohort, in another population of Finnish women weight gain of > 16 kg during pregnancy and increased BMI during adult life was associated with a reduced risk of pre-menopausal breast cancer (Hilakivi-Clarke et al., 2005).

Overall there is insufficient evidence to link GWG to long-term health consequences of the mother as a result of the lack of studies in this area.

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CONCLUDING REMARKS

There is a general lack of research that relates GWG to maternal outcomes beyond the first year postpartum other than for postpartum weight retention and subsequent obesity. This is understandable because most of the outcomes that are of the greatest interest, such as cardiovascular disease, cancer, and depression take longer to study because they occur later in the woman's life. It is well established however that obesity is associated with increased morbidity and mortality (hypertension, dyslipidemia, diabetes mellitus, cholelithiasis, coronary heart disease, osteoarthritis, sleep apnea, stroke, and certain cancers) (Must et al., 1992; Troiano et al., 1996; Allison et al., 1999; Calle et al., 2003; Gregg et al., 2005). Furthermore, for subsequent pregnancies, maternal overweight and obesity are associated with higher rates of cesarean delivery, GDM, preeclampsia and pregnancy-induced hypertension as well as postpartum anemia (Bodnar et al., 2004).

Overall, the consequences for the mother of GWG above recommended ranges appear to be well-substantiated for outcomes such as cesarean delivery and postpartum weight retention. The studies that have examined glucose abnormalities and hypertensive disorders of pregnancy have been methodologically flawed and thus do not provide sufficient evidence to support or refute a possible association. For GWG below recommended ranges, the only outcome for which there is any substantial evidence is initiation of breastfeeding. There are no available studies of a relationship between low GWG and increased maternal mortality among American women.

FINDINGS AND RECOMMENDATIONS

Findings

1. The literature related to GWG and maternal outcomes does not allow inference of causality since it is based solely on observational studies.
2. Evidence for an association between GWG and pregnancy complications such as glucose abnormalities and gestational hypertension disorders is inconclusive and problematic due to methodological flaws and that the outcome of mental health during pregnancy is understudied.
3. There is a strong association between higher GWG and increased risk of cesarean delivery.
4. There is no research on the effect of GWG on maternal mortality from which they could make any conclusions.
5. Low GWG is moderately associated with failure to initiate breastfeeding.
6. There is a strong association between higher GWG and postpartum weight retention in the immediate postpartum period (three months to three years).
7. The outcome of mental health is understudied.
8. There is insufficient evidence to link GWG to long-term health consequences of the mother due to the lack of studies in this area.
9. Maternal prepregnancy weight status is an important independent predictor of maternal short and long term outcomes.

Recommendations for Action and Research

Action Recommendation 5-1: The committee recommends that appropriate federal, state and local agencies as well as health care providers inform women of the importance of

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conceiving at a normal BMI and that all those who provide health care or related services to women of childbearing age include preconceptional counseling in their care.

Research Recommendation 5-1: The committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to researchers to conduct observational and experimental studies on the association between GWG and (a) glucose abnormalities and gestational hypertensive disorders that take into account the temporality of the diagnosis of the outcome, and (b) the development of glucose intolerance, hypertension and other CVD risk factors as well as mental health and cancer later in a woman's life.

Research Recommendation 5-2: The committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to conduct studies that (a) explore mechanisms, including epigenetic mechanisms, that underlie effects of GWG on maternal and child outcomes and (b) address the extent to which optimal GWG differs not only by maternal prepregnancy BMI but also by other factors such as age (especially among adolescents), parity, racial/ethnic group, socioeconomic status, co-morbidities, and maternal/paternal/fetal genotype.

Areas for Additional Investigation

The committee identified the following areas for further investigation to aid in future revisions of GWG recommendations. The research community should conduct studies on:

- Associations between gestational weight gain and maternal mortality;
- Effects of GWG on maternal mental status during pregnancy, in the postpartum period and in long-term.
- The causal nature of how gestational weight gain leads to short and long term maternal outcomes.

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6

Consequences of Gestational Weight Gain for the Child

The authors of the report, *Nutrition During Pregnancy* (IOM, 1990) emphasized short-term outcomes in developing guidelines for recommended gestational weight gain (GWG) ranges. This resulted from a lack of data on long-term outcomes and the fact that the research community was only beginning to understand the importance of the intrauterine environment for long-term health. In the interim, the literature on the topic has expanded, and more information is now available on neonatal as well as long-term consequences of both inadequate and excessive GWG during pregnancy.

Only by knowing the magnitude of causal relationships can one say with confidence that recommending a certain amount of GWG will result in altered frequency of adverse child health outcomes. The limited experimental data from randomized controlled trials in humans, however, impedes efforts to determine how much of any observed association is causal. It is possible that associations of GWG with outcomes do not result from GWG itself, but rather to underlying factors that influence both weight gain and the outcomes (e.g. maternal diet composition or physical activity level). In particular, it is important to determine whether these relationships are independent of prepregnancy body mass index (BMI) or if they differ by prepregnancy BMI. This is because observational studies are often limited by mixing effects of confounding factors with the predictor of real interest.

Although reverse causality is less of a problem in cohort than in cross-sectional studies, confounding is not readily controlled in any observational study. Only with large, well-designed and carefully controlled randomized studies can causal relationships be inferred with a high degree of confidence. Otherwise, inferences must be made using the best data available in consideration of plausible biologic mechanisms, confounding and other aspects of the study methodology, and the patterns of results.

The following reviews the current evidence and strives to quantify, wherever possible, causal relationships between GWG and childhood outcomes.

GENERAL CONCEPTS

Causal Concepts

The committee generated a general conceptual causal model of determinants (see Chapter 1) for maternal and child outcomes that may result from excessive or inadequate GWG (Figure 6-1). This model fits well with two paradigms that offer useful conceptual frameworks for considering long-term effects on the offspring. One, termed the life course approach to chronic disease, invokes two axes (Kuh and Ben-Shlomo, 2004). The first is time. Factors may act in the pre-conceptional through the prenatal period, into infancy, childhood, and beyond to determine

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risk of chronic disease. The second is hierarchical: these factors can range from the social/built/natural environment (macro) through behavior, physiology, and genetics (micro) (See Chapter 4). Factors interact with each other over the life course, with different determinants being more or less important at different life stages.

The other paradigm, called developmental origins of health and disease, focuses primarily on the prenatal and early postnatal periods, because they are the periods of most rapid somatic growth and organ development (Gillman, 2005; Sinclair et al., 2007; Hanson and Gluckman, 2008). Both of these frameworks invoke the concept of programming, which refers to perturbations or events that occur at early, plastic, and perhaps critical phases of development that can have long-lasting, sometimes irreversible, health consequences. The period of plasticity may vary for different organs and systems (Gluckman and Hanson, 2006). The model predicts that adult risk factors can only partially modify the trajectories of health and disease patterns established in earlier life (Barker et al., 2002; Ben-Shlomo and Kuh, 2002; McMillen and Robinson, 2005; Sullivan et al., 2008).

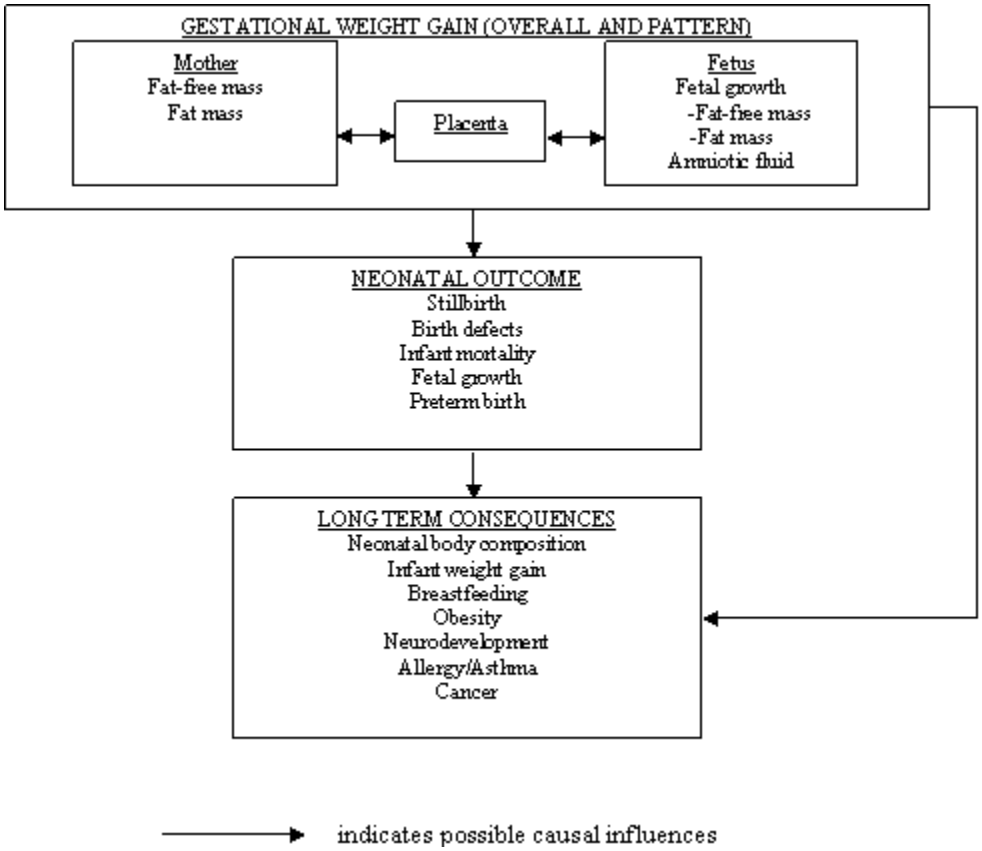

FIGURE 6-1 Schematic summary of neonatal, infant, and child consequences of GWG.

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Developmental programming, including the possible role of epigenetics as potential determinants of GWG, is discussed in Chapter 4. In this chapter, the role of developmental programming as a mechanism for some of the effects of GWG on postnatal outcomes is discussed. Many animal models have demonstrated that altering the environment in utero can have lifelong consequences. Perturbations of the maternal diet during pregnancy (typically by severe energy or protein restriction; administering hormones such as glucocorticoids; mechanical means, such as ligation of the uterine artery; or inducing anemia or hypoxia) have postnatal consequences on a number of metabolic and behavioral traits. Effects are inducible in rodents and in mammals, including non-human primates. Inasmuch as humans differ from other animal species in duration of pregnancy, placentation, and other important factors, the importance of the findings from animal studies lies not in the specific interventions but rather in the general principle that altering the supply of nutrients, hormones, and oxygen to the growing embryo and fetus or exposing them to stressors and toxicants can have long-term effects. Much of this animal research has focused on obesity-related outcomes such as adiposity, fat distribution, sarcopenia, insulin sensitivity, glucose intolerance, and blood pressure. These are leading causes of morbidity—and ultimately, mortality—in the U.S. and, as discussed below, GWG appears to be related to offspring obesity. The ways in which GWG could influence a number of child health outcomes through developmental programming is also discussed below.

Until recently, most of the research in animal models has concentrated on the long-term effects of interventions that cause offspring to be born small, typically small-for-gestational age (SGA) rather than early. Such work has been a good companion to a series of epidemiologic observations made within the last two decades that lower birth weight, apparently resulting from both reduced fetal growth and reduced length of gestation, is associated with higher risks of central obesity, insulin resistance, the metabolic syndrome, type 2 diabetes, hypertension, and coronary heart disease. These associations are potentiated by rapid weight gain in childhood (Bhargava et al., 2004; Barker et al., 2005).

It is important to note, however, that in recent years researchers have recognized that higher birth weight is also associated with later obesity and its consequences. Given that greater GWG is associated with increased weight at birth (reviewed below) and that total GWG and excessive gain—which are based on total GWG and prepregnancy BMI—appear to be rising over time (see Chapter 2), these observations regarding higher birth weights raise questions about the long-term adverse effects of higher weight gains in pregnancy. Accompanying these observations are newer animal experiments that involve "overnutrition" of the mother during pregnancy, which are also discussed briefly below.

In addition, it is critical to recognize that effects of GWG, or indeed any factor that alters the in utero environment, may have long-term effects on the offspring without any alterations of fetal growth or length of gestation. Thus the most important epidemiologic evidence for long-term effects of GWG does not depend on birth weight, gestational age or birth weight-for-gestational-age as exposures or outcomes, but rather provides data on the direct associations of GWG with various health outcomes in the offspring.

With these distinctions in mind, the committee considered "fetal growth" outcomes, including small-for-gestational age (SGA) and large-for-gestational age (LGA), and preterm birth as short-term outcomes. These measures have demonstrable and substantial associations with neonatal morbidity and mortality. Other short-term outcomes include stillbirth and birth defects.

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In contrast, neonatal body composition is included in the discussion of long-term outcomes because of the hypothesis (still unproven, however) that relative amounts of adiposity and lean mass—and their physiologic consequences—in fetal and neonatal life are important in setting long-term cardio-metabolic trajectories.

It also bears noting that this report focuses primarily on GWG, rather than prepregnancy BMI. Nevertheless, because the two factors are closely linked, one must account for confounding and effect modification by BMI in addressing offspring effects of GWG. Also it is possible that factors in infancy or childhood (e.g., growth in stature, adiposity, and infant feeding) could mediate effects of GWG on long-term child health.

Potential Mechanisms Linking Gestational Weight Gain to Long-Term Offspring Health

The existence of plausible biological mechanisms is one criterion for establishing causal relationships between GWG and child health outcomes. The following discussion focuses primarily on potential mechanisms linking GWG to offspring obesity and its consequences. Gestational weight gain is clearly about weight, so it is appropriate to address weight-related outcomes. Also most of the emerging evidence on long-term outcomes is based on these endpoints. The epidemiologic evidence for effects of GWG on other important child health outcomes are addressed later this chapter.

Although the following discussion focuses primarily on long-term child outcomes, similar mechanisms may also underlie associations of GWG with fetal growth. One issue that hampers inferences regarding fetal growth is that it is usually characterized by (gestational-age-specific) weight at birth, with less consideration of trajectory of weight from conception, body length or body composition (see Chapters 3 and 4 for a review of existing studies that address these issues). In contrast to the prenatal period, serial measurement of length/height and weight is common, even though data on body composition are relatively scarce.

Insulin resistance and glucose intolerance during pregnancy may mediate effects of GWG on long-term child outcomes. Weight gain in pregnancy is partly a gain in adiposity, which is accompanied by a state of relative insulin resistance starting in mid-pregnancy, among other metabolic alterations (Reece et al., 1994; Williams, 2003; Catalano et al., 2006; King, 2006; Hwang et al., 2007) (also see Chapter 3). This is an adaptive response, as it allows more efficient transfer of fuels across the placenta to the growing fetus (King, 2006). In overweight and obese pregnant women, these changes are magnified. For example, insulin resistance is more severe than in normal weight women, substantially raising the risk of impaired glucose tolerance and frank gestational diabetes (GDM).

In pregnant women who have hyperglycemia, the fetus also experiences hyperglycemia, as glucose freely crosses the placenta. In a sequence that Freinkel et al. (1986) termed "fuel-mediated teratogenesis" nearly 30 years ago, fetal hyperglycemia causes fetal hyperinsulinemia, leading to increased adiposity in the fetus. This increase is reflected in larger size at birth, which translates to higher rates of LGA and lower rates of SGA newborns (see discussion below and Chapter 3). Presumably through programming mechanisms, the increased fetal adiposity also results in increased adiposity in the growing child.

Other fuels besides glucose may also be involved. Increased fetal production of anabolic hormones and growth factors in combination with the increased levels of glucose, lipids, and amino acids that occurs in GDM result in fetal macrosomia and increase the risk for neonatal complications (Catalano et al., 2003). Crowther et al. (2005) and Pirc et al. (2007) showed that

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diet and insulin therapy along with blood glucose monitoring in pregnant women with mild GDM could lower plasma insulin and leptin (but not glucose) concentrations in cord blood as well as major adverse birth outcomes. This intervention more than halved the risk of macrosomia (birth weight > 4,500 g) (Crowther et al., 2005).

This physiologic milieu may also increase the risk for long-term complications, particularly obesity. But long-term follow-up of children from this and similar randomized trials is necessary to determine if treatment of GDM or impaired glucose intolerance during pregnancy can reduce adiposity and related physiology. Results from observational studies, however, suggest this possibility. Among 5-7 year-old children in two American health plans, Hillier et al. (2007) showed that risk of high weight-for-age was lower among those whose mothers had been treated for GDM than those who had not been treated. The weight status of the "treated" offspring was similar to those whose mothers had normal glucose tolerance.

The Freinkel hypothesis is supported by animal experiments, such as those of van Assche and colleagues (1979) and more recently Plagemann and colleagues (1998), who have pharmacologically induced GDM in rats. They observe fetal hyperglycemia and hyperinsulinemia, as hypothesized, as well as changes in the hypothalamus that give rise to hyperphagia, overweight, and impaired glucose tolerance in the offspring as they mature.

Another way to induce offspring metabolic derangement in rats is through over-feeding the pregnant dam. Samuelsson et al. (2008) found that maternal diet-induced obesity resulted in increased adult adiposity and evidence of cardiovascular and metabolic dysfunction compared to the offspring of lean dams. Earlier work by Dorner et al. (1988) and Diaz and Taylor (1998) showed that a period of overfeeding or GDM in the pregnant dam during a developmentally sensitive period in gestation could change the metabolic phenotype of the immediate offspring that would then persist over two succeeding generations. In their review of animal studies, Aerts and Van Assche (2003) demonstrate that these intergenerational physiologic effects are maternally transmitted, most likely through epigenetic processes.

Seemingly paradoxically, in animal experiments it is also possible to produce offspring insulin resistance, features of the metabolic syndrome, and diabetes, including GDM, by reducing energy or macronutrient intake of the mother during pregnancy. This situation can also result in intergenerational amplification of obesity and its consequences. For example, glucose metabolism is altered in the grand-offspring of female rats who had been protein restricted during pregnancy and lactation (Benyshek et al., 2006).

Overall, animal experiments show that offspring obesity and related metabolic sequelae can result from maternal over- or underfeeding during pregnancy, from experimental manipulation like pharmacologic induction of GDM, or from mechanical means like uterine artery ligation. Epigenetic modifications likely explain many of these phenomena (Simmons, 2007). A human counterpart to the animal experimental work is epidemiologic studies showing that higher birth weight is related to later obesity and type 2 diabetes, while lower birth weight is associated with central obesity, the metabolic syndrome, and indeed, type 2 diabetes as well (Gillman, 2005). In other words, a U-shape relationship exists between birth weight and obesity-related health outcomes.

The extent to which these observations have relevance for GWG guidelines is still unclear. Few animal studies directly assess the influence of GWG on short- or long-term offspring outcomes. Animal experimentalists typically do not measure weight gain during pregnancy, and it is not clear whether appropriate animal models exist to study GWG and obesity-related outcomes in the offspring. Neither is it clear that models of either diet-induced obesity or GDM

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are instructive for assessing effects of GWG. Similarly, human population studies that rely on birth weight or its components, duration of gestation, and size at birth as predictors of later outcomes (e.g., Hofman et al., 2004; Hovi et al., 2007) do not directly assess GWG.

Further, intervention studies to treat GDM do not in themselves provide evidence for making recommendations for appropriate GWG. Only randomized trials that alter weight gain during pregnancy can address that goal directly. Recently Wolff and colleagues (2008) analyzed results from 50 participants in a randomized controlled trial of reducing weight gain among obese pregnant women. The intervention was successful in restricting GWG: mean weight gain in the intervention group was 6.6 kg (± 5.5 kg) v. 13.3 kg (± 7.5 kg) in the control group a mean difference of 6.7 kg (95% CI: 2.6-10.8 [p = 0.002]). Insulin and leptin concentrations were also reduced, although glucose values were hardly altered.

Overall this small trial, along with other data from experimental animals and human subjects, raises the possibility that moderating GWG among obese pregnant women may reduce the risk of insulin resistance and glucose intolerance during gestation and, in turn, childhood obesity, but larger and longer-term studies are needed to address this question directly.

EFFECTS ON NEONATAL MORBIDITY AND MORTALITY

Although there is a substantial literature on prepregnancy BMI and neonatal morbidity and mortality, the literature on GWG in relation to these outcomes remains more limited, with the exception of its influence on fetal growth (Cedergren, 2006; Kiel et al., 2007). Infant mortality is strongly associated with maternal prepregnancy BMI as are a number of other clinically important outcomes, including stillbirth and preterm birth (Figure 6-2). Given that GWG, which is lower on average for heavier women, differs in relation to prepregnancy BMI, studies that examine GWG without stratifying by prepregnancy BMI are subject to confounding. These component relationships (prepregnancy BMI and GWG, and prepregnancy BMI and health outcome), as seen in Figure 6-2, are sufficiently strong that studies of GWG and neonatal outcomes that fail to account for prepregnancy BMI are of limited value in addressing the independent effects of GWG.

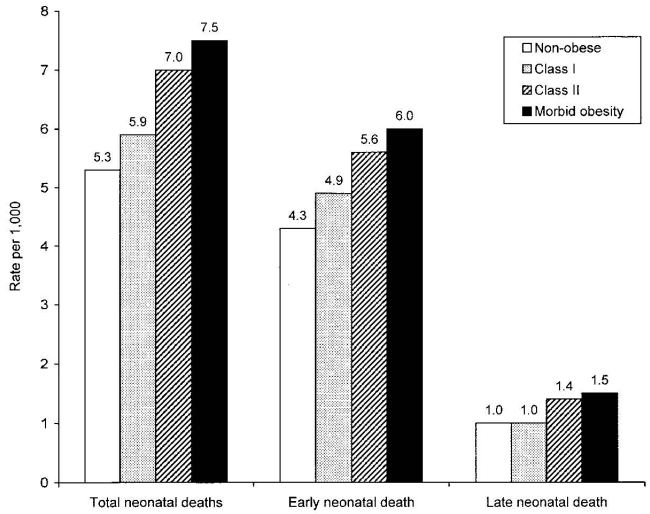

FIGURE 6-2 Rate of neonatal, early, and late neonatal death by obesity subclass.

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SOURCE: Salihu et al., 2008. Obesity and extreme obesity: new insights into the black-white disparity in neonatal mortality. *Obstetrics and Gynecology* 111(6): 1410-1416. Reprinted with permission.

Stillbirth

Inadequate or excessive GWG has the potential to affect fetal viability in later pregnancy, specifically risk of stillbirth (defined as pregnancy loss after 20 weeks' gestation). Both Naeye (1979) and NCHS (1986) showed that women with low prepregnancy BMI tended to have elevated risk of fetal or perinatal mortality (a combination of stillbirth and neonatal mortality) when combined with low GWG, but women with elevated prepregnancy BMI experienced increased risk of adverse outcomes when combined with excessive GWG. In an analysis from the California Child Health and Development Studies of the School of Public Health, University of California, Berkeley, Tavris and Read (1982) found a strong inverse association between total GWG and fetal death but it was eliminated by restriction to births of greater then 35 weeks' gestation. Thus, the association was an artifact of using cumulative weight gain as the predictor, strongly related to duration of gestation with stillbirths of notably shorter gestational duration than live births. A case-control study of stillbirths in Sweden reported a strong positive association between prepregnancy BMI and stillbirth, with odds ratios approaching 3.0 for obese women, but the authors reported no effect of GWG measured in either early or late pregnancy among term births (Stephansson et al., 2001). The large size of the study (649 cases and 690 controls) is notable as well as the ability to consider an array of covariates, but the results for total GWG were not presented in the publication.

In summary, the research on GWG and stillbirth remains quite limited in quantity and quality. In addition to considering prepregnancy BMI, there is a need to avoid the error of comparing total GWG in pregnancies resulting in stillbirths with those resulting in live births because of the time in pregnancy when stillbirth is likely to occur. Although early studies suggested adverse effects of low GWG among women with low prepregnancy BMI and also of high GWG among women with elevated prepregnancy BMI, more detailed studies have not been done to corroborate or refute this pattern. Recent, better studies largely do not support an association between GWG and stillbirth.

Birth Defects

The authors of the IOM (1990) report found no studies of GWG associations with birth defects and recognized that the etiologic period for congenital defects is so early in pregnancy that the issue of weight gain was not likely to be relevant. Although there is a growing literature on prepregnancy BMI and congenital defects that suggests that there is an increased risk of birth defects with increasing BMI (Watkins et al., 2003; Anderson et al., 2005; Villamor et al., 2008), only one recent study addressed GWG in relation to birth defects directly. Infants born to mothers who gained less than 5 kg or less than 10 kg during pregnancy were at increased risk of neural tube defects (Shaw, 2001). In addition, one report indicated that dieting to lose weight during pregnancy was associated with an increased risk of neural tube defects (Carmichael et al., 2003). Although there are several pathways by which GWG and birth defects may be related, including a shared influence of diet on both high and low weight gain related to an abnormally developing fetus, a direct causal effect of GWG on risk of birth defects is precluded by the timing of these events during gestation.

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Infant Mortality

Infant mortality is obviously of great clinical and public health importance and is often used as a summary indicator of a population's reproductive health status. Concern with fetal growth and preterm birth as health outcomes is based largely on their known relationships with mortality (as well as morbidity). As a result, studies that directly address mortality as the outcome are of particular relevance and can help to interpret the patterns seen for the studies of intermediate outcomes such as preterm birth or growth restriction.

Only limited research exists in which an association between GWG and infant mortality is assessed. Perinatal mortality was examined in only one study (NCHS, 1986) noted in the IOM (1990) report. Recently, Chen et al. (2009) examined maternal prepregnancy BMI and GWG in the National Maternal and Infant Health Survey (NMIHS) and considered 4,265 infant deaths and 7,293 controls, a large study that was analyzed appropriately. Among underweight and normal-weight women, low GWG was associated with a marked increase in infant mortality, with relative risks on the order of 3-4 compared to those with the highest GWG. Among overweight and obese women, there were more modest effects of GWG, but both lower and higher GWG were associated with around 2-fold increases in risk of infant mortality. In all cases, the patterns were stronger for neonatal deaths (in the first 30 days of life) than for postneonatal deaths (those occurring after one month but before the completion of one year). As seen in Table 6-1 within BMI strata, the relative risk for neonatal death was 3.6 for the lowest weight gain group among underweight women, 3.1 among normal weight women, 2.0 among overweight women, and 1.2 among obese women, showing a diminishing effect of low GWG with increasing BMI. At high GWG, the relative risks for neonatal mortality for underweight, normal weight, overweight, and obese women were 1.0, 1.2, 1.4, and 1.8, respectively, which showed the exact opposite tendency. Here excessive GWG was more strongly associated with neonatal death with increasing BMI. Maternal age at delivery did not affect neonatal mortality. After adjusting for gestational age at delivery, no association was found between teenage pregnancy and neonatal mortality. The same general pattern was seen for post neonatal deaths; but was less pronounced (Table 6-1).

More studies of infant mortality are needed, but the evidence from Chen et al. (2009) warrants serious consideration not only because of the importance of the outcome but also because of the implications for the more voluminous literature on fetal growth and preterm birth. Although this study did not link GWG to those intermediate outcomes and the intermediate outcomes to mortality, the strength of the patterns and their parallels with studies of fetal growth add credibility to the presumption that causal chain from GWG to adverse birth outcomes to death is operative. Based on limited volume of research, but one well-done study, the evidence for a link to infant mortality can be considered moderate.

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TABLE 6-1 Maternal Prepregnancy BMI and Gestational Weight Gain of Infant Deaths and Controls (1988 National Maternal and Infant Health Survey [NMIHS])

Maternal Prepregnancy BMI (kg/m^2)	Total Weight Gain During Pregnancy [a] (kg)	Neonatal Death OR[b] (95% CI)	Postneonatal Death OR[b] (95% CI)	Infant Death OR[b] (95% CI)
< 18.5	< 6.0	3.55 (1.92-6.54)	2.96 (1.42-6.15)	3.26 (1.86-5.72)
	6.0 – 11.6	1.35 (0.88-2.06)	1.34 (0.83-2.14)	1.34 (0.93-1.92)
	12.0 – 17.6 [c]	1.00	1.00	1.00
	≥ 18.0	0.99 (0.63-1.54)	0.55 (0.32-0.95)	0.79 (0.53-1.17)
18.5 – 24.9	< 6.0	3.07 (2.45-3.85)	1.96 (1.51-2.55)	2.58 (2.12-3.14)
	6.0 – 11.6	1.41(1.19-1.68)	1.12 (0.92-1.36)	1.29 (1.11-1.49)
	12.0 – 17.6 [c]	1.00	1.00	1.00
	≥ 18.0	1.15 (0.96-1.37)	0.94 (0.77-1.15)	1.06 (0.91-1.23)
25 – 29.9	< 6.0	1.98 (1.34-2.92)	0.81 (0.51-1.29)	1.42 (1.02-1.99)
	6.0 – 11.6	1.20 (0.85-1.68)	0.64 (0.43-0.95)	0.94 (0.71-1.25)
	12.0 – 17.6 [c]	1.00	1.00	1.00
	≥ 18.0	1.41(1.00-2.00)	0.87(0.58-1.31)	1.16 (0.87-1.56)
≥ 30	< 6.0	1.19 (0.69-2.06)	0.81 (0.40-1.62)	1.04 (0.64-1.70)
	6.0 – 11.6	0.67 (0.39-1.17)	0.91 (0.47-1.78)	0.78 (0.48-1.26)
	12.0 – 17.6 [c]	1.00	1.00	1.00
	≥ 18.0	1.78 (0.96-3.33)	1.29 (0.58-2.84)	1.61 (0.92-2.81)

NOTE: Midpoint and range values for outcomes (neonatal death, postnatal death, infant death) are derived using a separate reference group for each BMI category.

[a] Weight gain during pregnancy projected to 40 weeks' gestation.
[b] Adjusted for race, maternal age at pregnancy, maternal education, maternal smoking during pregnancy, child's sex, live birth order, and plurality
[c] Referent group for comparisons within BMI stratum

SOURCE: Modified from Chen et al. (2009).

Fetal Growth

The relationship of GWG to fetal growth was considered in some detail in the IOM (1990) report. Much of the literature on consequences of fetal size fails to separate preterm delivery from fetal growth restriction, which is problematic for the purposes of this report. As noted in IOM (1990) and by others, smaller size at birth is associated with increased fetal and infant mortality, cerebral palsy, hypoglycemia, hypocalcemia, polycythemia and birth asphyxia, persistent deficits in size, and persistent deficits in neurocognitive performance (Pryor et al., 1995; Goldenberg et al., 1998). Health consequences of small size at birth tend to follow a dose-response relationship with elevated relative risks at the lowest weights. Large size causes delivery complications, including shoulder dystocia and other forms of birth injury, as well as cesarean delivery, maternal death, and fistulae (IOM, 1990).

Birth weight combines duration of gestation with rate of fetal growth. Infants on a given growth trajectory who are born earlier will weigh less than infants on the same growth trajectory who are born later. To isolate fetal growth, studies often use SGA and LGA, which compare the infant's weight to the distribution of birth weight of all infants in the same week of gestation.

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Most commonly, those infants in the lowest and highest 10th percentiles are classified as SGA and LGA, respectively, but some use the more extreme values of the 5th percentile or two standard deviations or more below or above the mean. The cutoff points for those percentiles may be specific to gender, race/ethnicity, and/or parity in addition to week of gestation. There is some controversy about the use of racial/ethnic-specific norms, in particular because their biological meaning is in doubt. Black infants in the U.S. have a markedly different weight distribution than non-black infants (of varying race/ethnicity). Identification of deviations from group-specific norms is a useful means of predicting mortality; although separate group-specific norms could be interpreted as acceptance of differences in birth outcome by race/ethnicity as absolute. Such differences are not absolute, however, because health disparities are strongly influenced by social and behavioral factors. The term intrauterine growth restriction (IUGR) is generally applied to births that are designated as a lower weight than would have been attained had the pregnancy been a "normal" one. Obviously the definition of "normal" or "expected" is problematic because it is not known what would have happened had conditions been different—only what did happen. Thus results may not be comparable across studies when different indices are used.

At the time of the IOM (1990) report, the evidence for an effect of GWG on fetal growth was viewed as "quite convincing." Increased GWG was related to increased birth weight, and the report noted that the strength of that relationship varied as a function of prepregnancy BMI. The lower the prepregnancy BMI, the stronger the association between increased GWG and increased birth weight. Among obese women, the association between increased GWG and increased birth weight was questionable. The patterns of influence of GWG on fetal growth were evident both for mean birth weight and for the tails of the distribution, usually described as IUGR or macrosomia (variably defined as > 90th percentile of birth weight for gestational age or > 4,000 grams).

In addition to the consistent observational data that linked inadequate GWG, especially in underweight and normal weight women, with increased risk for SGA, and the evidence that linked excessive weight gain, especially in overweight and obese women, with increased risk of LGA and its sequelae, there is some evidence from randomized trials in women that is important. A series of early randomized trials of dietary supplements, carefully reviewed by Susser (1991), provides very little support for the argument that increased energy or protein intake during pregnancy enhances fetal growth in general. Only for women who were near starvation was the evidence linking improved nutrition to GWG to fetal growth supportive of a causal relationship. For other groups of pregnant women, there was no benefit and some indication of possible harm from ingesting supplements with high protein concentrations. In contrast, results from a Cochrane systematic review suggested a consistent benefit of supplementation in reducing risk of SGA, though this does not necessarily mean that such benefits were mediated by GWG (Kramer and Kakuma, 2003).

Recent randomized trials have focused directly on the impact of *limiting* GWG to determine whether this results in short-term metabolic effects or improved clinical outcomes. Polley et al. (2002) randomized normal weight and overweight women (~ 30 in each class and arm of the trial) to assess the impact of a multifaceted program designed to maintain GWG within recommended guidelines. The intervention yielded benefits in preventing excessive GWG only among normal weight women. Women whose GWG was moderated had infants that weighed 93 g less on average than controls. Fewer of the treated number developed GDM or had cesarean deliveries.

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In summary, the issue of whether the association between GWG and fetal growth is causal cannot be answered with certainty based on the available evidence. Observational data provide replicated indications of a strong association between GWG and increased risk of SGA, especially in underweight and normal-weight women, and between higher GWG and increased risk of LGA, particularly among overweight and obese women. Randomized trials are either only indirectly applicable (because they are from less-relevant populations and time periods) or are too small to provide strong evidence. Biological considerations also need to be taken into account in judging the likelihood that the associations from observational studies are causal.

There are several possible explanations for the associations between GWG and fetal growth that have been reported: GWG is causally related to fetal growth, both GWG and fetal growth are independently affected by maternal diet and/or physical activity, and both GWG and fetal growth have shared genetic or other intrinsic biological determinants. If the non-causal explanations are correct, then manipulating GWG will not affect fetal growth directly. However, if the same behavioral changes that produce a more optimal GWG also happen to result in a more optimal fetal growth, then there would be a benefit realized. If there is a shared, intrinsic biological basis for a link between GWG and fetal growth, genetic or otherwise, modification of GWG would not affect on fetal growth. In the absence of clear evidence on the causal pathway, the committee presumes that the relationship between GWG and fetal growth was causal in an effort to ensure that the guidelines are protective of the health of the fetus and infant.

The Agency for Healthcare Research and Quality (AHRQ) evidence-based review on outcomes of GWG (Viswanathan et al., 2008) provides a thorough summary of the literature on GWG and birth outcomes. That report included studies published between 1990 and 2007, so that it supplements the IOM (1990) report; and the committee expanded on the AHRQ review by considering studies published subsequently (see Chapter 5 and Appendix F). The committee did not exhaustively review the studies from before 1990 given the thorough work of the IOM (1990) committee. Earlier studies are mentioned only when the information they provide affects the overall conclusions.

The AHRQ review (Viswanathan et al., 2008) considered 25 studies of variable quality that addressed GWG and birth weight as a continuous measure. Every one of those studies demonstrated an association between higher GWG and higher infant birth weight. Although there was substantial variability in magnitude of effect across studies, in general differences were on the order of 300 grams in birth weight from lowest to highest GWG categories. Among the stronger studies, the AHRQ review found that for each 1 kg increase in GWG, birth weight rose 16.7-22.6 grams. Fewer studies considered weight gain by trimester, and they tended to show a lower increase in birth weight per unit increase in GWG in the third than in the first or second trimester.

A smaller but still sizable number of studies (13) examined the relationship of GWG to risk of low birth weight (LBW, defined as < 2,500 g). These studies showed that risk of LBW diminishes as GWG increases, particularly as total gain exceeds 25-30 pounds. Although the magnitude of association varied substantially across studies, in general the highest GWG category had roughly half the risk of a LBW infant compared to the lowest GWG category. At the other end of the birth weight spectrum, 12 studies considered infant macrosomia, defined as birth weight > 4,000 or > 4,500 grams. Recognizing the variability in definitions of macrosomia and GWG categories, the committee found that the studies showed a consistent trend for increased risk of macrosomia with increasing GWG. Relative risks were 2-3 for macrosomia in the highest compared to the lowest GWG category. These results consistently indicate that the

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relationship of GWG to birth weight applies across the full range of weights and is not limited to the low or high end of the distribution. However, because birth weight is a combination of fetal growth and length of gestation, studies that separate these two components are more informative.

The AHRQ review (Viswanathan et al., 2008) also considered studies of SGA that did not stratify by prepregnancy BMI and identified 15 of them that showed a consistent pattern of diminishing risk of SGA with increasing GWG. It is difficult to provide quantitative estimates of the magnitude of this effect given variable study methods and results, but as for LBW, relative risks on the order of 2-3 across extreme GWG categories are typical. The six studies that stratified by prepregnancy BMI similarly found lower GWG was associated with increased risk of SGA births. While methods and results were again variable, the studies did not strongly suggest that prepregnancy BMI modified the relationship between GWG and SGA, in contrast to the interpretation in the IOM (1990) report.

In the 10 studies in which GWG and LGA were considered, there was reasonably consistent support for a positive association. For each 1 kg increment in GWG, the relative risk of LGA increased by approximately a factor of 1.1, and comparing the highest to lowest categories of GWG yielded relative risks on the order of 2. The studies that stratified by prepregnancy BMI did not show notable differences in the GWG–LGA association across BMI categories, with only a modest tendency towards a stronger association among women with lower prepregnancy BMI.

Three additional studies that addressed GWG and birth weight appeared after the period covered by the AHRQ review (Viswanathan et al., 2008). Lof et al. (2008), whose focus was on the role of physical activity in relation to GWG and pregnancy outcome, noted that GWG during weeks 12-33 (unadjusted for prepregnancy BMI) was modestly correlated with increased birth weight, $r = 0.13$ ($P = 0.05$), stronger than the relationship for GWG during weeks 12-25 or 25-33 alone. Segal et al. (2008) found similar results in a study of obesity and family history of diabetes in relation to pregnancy outcome, with an adjusted correlation coefficient of 0.19 ($P = 0.09$) between weight gain before the oral glucose tolerance test and birth weight, accounting for prepregnancy BMI in the analysis.

Nohr et al. (2008) conducted the most informative analysis of the independent effects of prepregnancy BMI and GWG among over 60,000 births from the Danish National Birth Cohort. The authors considered the relationship of GWG with SGA and LGA as well as the interaction of prepregnancy BMI and GWG in relation to birth weight. They report statistically significant but generally modest indications of an interaction between prepregnancy BMI and GWG with the exception of a stronger association of low GWG with SGA among underweight women.

Subsequent analyses of this data (information contributed to the committee in consultation with Nohr) revealed that the relative risk of SGA associated with lower (< 10 kg) versus medium GWG (10-15 kg) among underweight women was 2.1, while it was 1.7 for normal weight women, 1.6 for overweight women, and 1.3 for obese women. The increased risk of LGA associated with very high GWG (≥ 20 kg) versus medium GWG (10-15 kg) was 3.7 for underweight women, 2.6 for normal weight women, 2.0 for overweight women, and 1.8 for obese women, again suggesting that the effect of GWG is dampened with increasing prepregnancy BMI. This analysis is an important contribution to quantifying the magnitude of effect of GWG on birth weight, consistent with the large body of previous studies and demonstrating an overall shift of less SGA and more LGA (and higher mean birth weight) with increasing GWG (see Appendix G, Part I).

Dietz et al. (in press) provided analyses from the Pregnancy Risk Assessment Monitoring System (PRAMS) in which they considered estimated associations between GWG and delivery

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of a SGA infant using three definitions of SGA: > two standard deviations below the mean birth weight for gestational age, a customized measure, < 10th percentile of expected birth weight for gestational age; and < 10th percentile of birth weight for gestational age using a population-based reference. Population-based information about 104,980 singleton term births in 2000-2005 from 29 states participating in PRAMS provided the basis of the analysis. Risk of LGA births or births > 4,500 g yielded clear and similar findings; with increasing weight gain, there was a markedly increased risk of LGA births, present among all BMI groups, but most pronounced on a relative scale among the women with the lowest BMI. The magnitudes of association were striking, with more than a 10-fold gradient in risk from lowest to highest weight gain categories for underweight women, and a 3- to 4-fold gradient in risk for women in the other BMI categories (Table 6-2).

In summary, the evidence that GWG is related to birth weight-for-gestational age is quite strong and the magnitude of that association is large, with relative risks of SGA with low GWG on the order of 2-3. It appears that the entire birth weight distribution is shifted upward with increased GWG, reducing the risk of SGA and increasing the risk of LGA as the mean birth weight rises. The evidence that this pattern is enhanced among women with low prepregnancy BMI is moderately strong as well. The IOM (1990) report suggested consideration of a different relationship between GWG and fetal growth among young mothers. As they noted, however, it was unclear whether this pattern was reflective of a different causal process, and the subsequent literature has not strengthened the support for differential effects by maternal age group. Only limited research is available on the potential for different effects of GWG on fetal growth by ethnicity, smoking status, or other maternal attributes, and the results that are summarized in the AHRQ review are inconsistent. There is moderate support for a stronger effect of GWG that occurs during the first or second trimester on birth weight-for-gain than there is for third trimester GWG (Viswanathan et al., 2008).

TABLE 6-2 Adjusted* Odds Ratios for Association of Total GWG with SGA Stratified by Prepregnancy BMI

	Prepregnancy BMI			
	Lean	**Normal**	**Overweight**	**Obese**
Total GWG (kg)	**AOR (95% CI)**	**AOR (95% CI)**	**AOR (95% CI)**	**AOR (95% CI)**
0.4-6.7	1.0 (0.7, 1.3)	1.3 (1.1, 1.5)	1.0 (0.7, 1.4)	1.5 (1.2, 1.8)
6.8-11.7	Ref	Ref	Ref	Ref
11.8-16.3	0.6 (0.5, 0.7)	0.6 (0.5, 0.7)	0.7 (0.5, 0.7)	0.6 (0.5, 0.7)
16.4-20.8	0.4 (0.3, 0.5)	0.5 (0.4, 0.5)	0.5 (0.3, 0.6)	0.4 (0.3, 0.5)
≥20.9	0.3 (0.2, 0.3)	0.2 (0.2, 0.3)	0.3 (0.2, 0.5)	0.3 (0.2, 0.5)

NOTES: Adjusted for infant gender and gestational age; and maternal race/ethnicity, age, marital status, education, Medicaid recipient, parity, and smoking during pregnancy. Lean BMI = < 19.8 kg/m^2; Normal BMI = 19.8-26 kg/m^2; Overweight BMI = 26.1-28.9 kg/m^2; Obese BMI = > 29 kg/m^2
SOURCE: Dietz et al. (in press). This article will be published in the *American Journal of Obstetrics and Gynecology*, Copyright Elsevier (2009).

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Preterm Birth

Preterm birth (< 37 weeks' completed gestation) is a critical indicator of maturity. Although defined dichotomously, risks of death and morbidity are a direct function of the degree of prematurity, with births at the margins, 33-36 weeks' gestation, most common and at modestly increased risk of health problems. In contrast, earlier births, < 33 weeks' gestation, are rarer events but at much greater risk. The spectrum of health consequences of preterm birth includes acute respiratory, central nervous system, and gastrointestinal disorders, and long-term deficits in neurobehavioral development (IOM, 2007) and possibly adverse cardiometabolic outcomes (Hofman et al., 2004; Hovi et al., 2007). An early delivery may well reflect the better alternative as compared to an intrauterine death, whether resulting from clinical intervention (an increasingly common "cause" of preterm birth) or natural processes. Nonetheless, the high and growing frequency of preterm birth in the U.S. makes this a critical endpoint to consider in relation to GWG.

At the time of the IOM (1990) report, the volume and quality of literature on preterm birth was quite limited. Several studies suggested that low GWG was associated with increased risk of preterm birth, but as they demonstrated, much of that may have been resulted from the simple error of failing to recognize that the shortened period of pregnancy (i.e., preterm birth) limits the duration of time over which weight can be gained. Comparing term and preterm births on total weight gain is meaningless for that reason. Data generated on behalf of this committee (information contributed to the committee in consultation with: Herring [see Appendix G, Part II] and Stein [see Appendix G, Part III]) provided some of the first information on GWG and preterm birth to consider prepregnancy BMI, which is predictive of both preterm birth (higher risk with lower BMI) and GWG (higher GWG with lower BMI). The results of that effort suggested little effect of rate of net weight gain (the only proper measure to compare pregnancies of varying duration) on risk of preterm birth.

The AHRQ review (Viswanathan et al., 2008) included a detailed summary of the findings of 12 more recent studies that considered the relationship of rate of GWG to preterm birth, generally defined as delivery before the completion of 37 weeks' gestation. Using rate of weight gain rather than total amount gained takes into account the truncated period of pregnancy—and thus truncated opportunity to gain weight—for women who give birth before term. There was a consistently increased risk of preterm birth in both the lowest and highest GWG categories. It is difficult to summarize the quantitative impact because the studies used varying definitions of high and low rates of weight gain and different analytic methods to characterize the relationship with preterm birth. In studies that provided relative risks comparing higher and lower GWG to the middle range of GWG, the relative risks were on the order of 1.5-2.5 for both the higher and lower GWG groups, with greater consistency for the influence of lower GWG on preterm birth.

A number of these studies examined effect modification by prepregnancy BMI (Siega-Riz et al., 1996; Spinillo et al., 1998; Schieve et al., 1999; Dietz et al., 2006; Nohr et al., 2007), and those reports were consistent in finding a stronger effect of a lower rate of GWG on preterm delivery among underweight women. As prepregnancy BMI increased, the magnitude of increased risk associated with a lower rate of GWG diminished. There was some evidence that the increased risk of preterm birth associated with a higher rate of GWG was greater with increasing BMI, so that the optimal GWG shifted downward with higher prepregnancy BMI. Four studies applied the IOM (1990) guidelines to define adequacy of GWG, and all of these

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reported increased risk of preterm birth associated with inadequate GWG among underweight and normal-weight women.

Some studies considered the clinical presentation of preterm birth (Siega-Riz et al., 1996; Spinillo et al., 1998; Nohr et al., 2007), and some considered severity of prematurity (Dietz et al., 2006; Stotland et al., 2006) in their analyses. Though limited in quantity, the results of these studies do not provide a clear suggestion that the association between GWG and preterm birth differs by clinical presentation or severity. More recently, Rudra et al. (2008) considered preterm birth subtypes in relation to prepregnancy BMI and GWG. They reported that greater GWG during gestational weeks 18-22 was weakly associated with lower risk of spontaneous preterm birth and higher risk of medically indicated preterm birth, with some variation in these patterns in relation to prepregnancy BMI.

Biological Plausibility

The pathogenesis of spontaneous preterm delivery has not been clearly elucidated but may involve five or more primary pathogenic mechanisms: activation of the maternal or fetal hypothalamic-pituitary-adrenal (HPA) axis, amniochorionic-decidual or systemic inflammation, uteroplacental thrombosis and intrauterine vascular lesions, pathologic distention of the myometrium, and cervical insufficiency (IOM, 2007). The committee found no studies that directly linked GWG to activation of the maternal or fetal HPA axis; however, several animal studies have linked periconceptional undernutrition to accelerated maturation of fetal HPA axis resulting in preterm delivery (Bloomfield et al., 2003; Bloomfield et al., 2004; Kumarasamy et al., 2005).

With respect to amniochorionic-decidual or systemic inflammation, the committee also found no direct link to GWG. However, it is plausible that maternal undernutrition may increase the risk of preterm delivery by suppressing immune functions or increasing oxidative stress. Macro- or micronutrient deficiencies can adversely affect maternal immune functions. For example, iron-deficiency anemia can alter the proliferation of T- and B-cells, reduce the killing activity of phagocytes and neutrophils, and lower bactericidal and natural killer cell activity, thereby increasing maternal susceptibility to infections (Allen, 2001). Furthermore, protein and/or micronutrient deficiencies may impair cellular antioxidant capacities because proteins provide amino acids needed for synthesis of antioxidant defense enzymes, such as glutathione and albumin (reactive oxygen species scavengers), and many micronutrients themselves are antioxidants. Increase in reactive oxygen species, such as oxidized low-density lipoprotein and F2-isoprostanes (lipid peroxidation products), may contribute to cellular toxicity, inflammation, vasoconstriction, platelet aggregation, vascular apoptosis and endothelial cell dysfunction (Luo et al., 2006), which may also activate the pathway to preterm delivery involving uteroplacental thrombosis and intrauterine vascular lesions.

The committee cannot establish a causal relationship between GWG and preterm delivery based on available evidence. Although there are intriguing data linking macro- and/or micronutrient deficiencies to accelerated maturation of fetal HPA axis and altered immune functions and/or increased oxidative stress that suggest biological plausibility, important questions regarding timing, threshold, content, and interactions remain unanswered. These uncertainties about a direct causal relationship between GWG and preterm delivery guided the committee's approach to decision analysis in Chapter 7, which weighed the trade-offs of GWG with and without taking into account preterm delivery as an outcome.

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In summary, there is strong evidence for a U-shape association between lower GWG and preterm birth among normal weight and underweight women, and moderate evidence for an association of higher GWG and preterm birth. The magnitude of the association is fairly strong, with relative risks on the order of two, but difficult to summarize because of variability in the definitions of higher and lower rates of weight gain. In addition, as noted in the chapter introduction, the lack of strong biological plausibility between GWG and preterm calls into question the causal relation between the two. There is no empirical basis for suggesting modifiers of this relationship other than prepregnancy BMI, for which the data are clear in showing that associations of low GWG with preterm are stronger among underweight women.

LONG-TERM CONSEQUENCES

The IOM (1990) report recommendations for GWG were focused largely on avoiding inadequate weight gain and thus the short-term consequences of low fetal growth and prematurity (see Chapter 1). Since its publication, the emergence of epidemic obesity in the American population has raised the possibility that excessive GWG may also be harmful. A small number of studies now address the associations of GWG with adiposity at birth and with markers of childhood obesity and its cardiometabolic sequelae.

Neonatal Body Composition

As reviewed above, GWG is directly associated with fetal growth as measured by birth weight for gestational age. For outcomes related to long-term adiposity, however, it is important to measure not only weight (and length) at birth, but also body composition. Catalano and colleagues performed a series of studies relating maternal characteristics to neonatal body composition measured by total body electrical conductivity, a method no longer in use. One set of studies compared infants who were born at term to overweight/obese women (pregravid BMI > 25 kg/m^2; n = 76) with those born to lean/average weight women (n = 144) (Sewell et al., 2006). As expected, GWG was higher among lean/average (mean 15.2 kg) than overweight/obese (13.8 kg) women. Among the overweight/obese sample, stepwise regression analyses that included pregravid weight as a covariate revealed that the higher the GWG, the higher the newborn fat mass. The authors did not report the correlation among the lean women, presumably because the associated p-value exceeded 0.05. In another study, however, which combined diabetic and non-diabetic pregnant women (total n = 415), Catalano and Ehrenberg (2006) found that GWG was directly associated with birth weight, including both lean and fat mass. These results are consistent with those of Udal et al. (1978), who found a direct association between GWG and sum of eight neonatal skinfold measurements among 109 non-diabetic mothers who delivered term infants, an association that was independent of prepregnancy weight, gestational age, smoking and family history of diabetes.

The findings of these studies raise the possibility that higher GWG may lead to long-lasting adiposity in the offspring. Studies that link GWG with body composition from birth onwards in populations from developed countries would be quite informative but are presently unavailable.

Infant Weight Gain

Rapid weight gain during infancy is associated with obesity later in life (Baird et al., 2005; Monteiro and Victoria, 2005; Gillman, 2008). It is unclear whether this pattern is particularly an issue among infants who are born SGA (Ong and Loos, 2006; Taveras et al., 2009). Therefore,

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infant weight gain may serve as a surrogate, or intermediate marker, of later adiposity. Although it may be more feasible to obtain intermediate markers than ultimate health outcomes, they are rarely perfect surrogates and are sometimes misleading. Therefore, one should view any associations of GWG with surrogate outcomes—even in randomized trials—with caution, and only as suggestive of effects on actual health outcomes. Also, this line of evidence would be strengthened by studies with serial measures of body composition, not just weight (with or without length) from birth onwards.

Only one study was identified that addressed GWG and infant weight gain, and it was not the primary goal of the study. Ong et al. (2000) conducted a prospective study of 848 term infants born in the UK who had weight measured at birth and at two and five years of age. The 30.7 percent of children who gained more than 0.67 weight standard deviations in the first two years of life had more adiposity at age five than the other children, but they also had been lighter, shorter, and thinner at birth. The mothers of these children were not more likely to have had a higher prepregnancy BMI or to have gained more weight during pregnancy.

Breastfeeding Initiation and Maintenance

Breastfeeding Outcomes

Breastfeeding is an important outcome to study not only because it may be associated with reduced obesity, and is therefore an intermediate like infant weight gain, but also because it predicts other health outcomes such as reduced otitis media and gastrointestinal illness and better cognition. Observational studies (see Chapter 5) have documented a relationship between excessive weight gain during pregnancy and poor breastfeeding outcomes.

Long-term Effects on Obesity

Despite the importance of this issue, high-quality studies that link GWG with obesity and related health outcomes in childhood are only beginning to be published. The AHRQ review (Viswanathan et al., 2008) identified only one cohort study that examined childhood obesity in relation to weight gain according to the IOM (1990) guidelines. Oken et al. (2007) analyzed data from Project Viva, a prospective study of predominantly non-low-income pregnant women and their children in Massachusetts (Table 6-3). Among the sample of 1,044 mothers included in this analysis, 51 percent gained excessive, 35 percent adequate and 14 percent inadequate weight during pregnancy. Compared with inadequate GWG, adequate and excessive gain were associated with odds ratios of 3.77 (95% CI: 1.38, 10.27) and 4.35 (1.69, 11.24), respectively, for obesity at 3 years of age (BMI > 95th percentile versus < 50th percentile) in analyses adjusted for key covariates. In addition, the authors found higher BMI z-score, sum of triceps and subscapular skinfold thicknesses, and systolic blood pressure for each 5-kg increment in total GWG.

The AHRQ review (Viswanathan et al., 2008) found three other studies that assessed total GWG and childhood adiposity. One of these studies (Ong et al., 2000) examined weight gain from birth to two years as an outcome, as was discussed earlier. Sowan and Stember (2000) examined outcomes through 14 months of age. In their fully adjusted model, each 5-pound increment in total weight gain was associated with an odds ratio of 0.8 for obesity defined as BMI > 84th percentile within the study population (n = 630). Inferences from this study are uncertain for a number of reasons. In a study, Li et al. (2007) empirically derived three weight-

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gain trajectories through childhood. Gestational weight gain was a predictor of the "early-onset" trajectory (which was defined as "children with an early-onset of overweight that persisted throughout childhood") total weight gain of at least 45 pounds (versus 25-35 pounds) was associated with an increased risk of 1.7 for being in the early-onset rather than the normal trajectory class in an analysis adjusted for maternal BMI and other factors.

Since the publication of the AHRQ review (Viswanathan et al., 2008), Wrotniak et al. (2008) studied approximately 10,000 seven-year-old term-born offspring of participants in the 1950s-1960s Collaborative Perinatal Project (see Table 6-3). Not surprisingly, mean maternal BMI (21.9 kg/m^2), total weight gain (9.5 kg), and birth weight (3.23 kg), and the proportions of women with excessive gain (11 percent) and children with obesity (defined as BMI > 95th percentile—5.7 percent) were lower than in current cohorts. Both total weight gain and excessive weight gain were associated with child obesity. For example, compared with adequate gain, excessive gain was associated with an adjusted odds ratio of 1.48 (95% CI: 1.06, 2.06) for BMI ≥ 95th versus < 95th percentile). The association appeared stronger for women who entered pregnancy underweight (BMI < 19.8 kg/m^2) than for heavier mothers.

In another recent study, Moreira et al. (2007) found that total weight gain was directly associated with childhood overweight defined by the International Obesity Task Force standards (Cole et al., 2000) (see Table 6-3). Compared with weight gains < 9 kg, gains ≥ 16 kg were associated with an adjusted odds ratio for overweight of 1.27 (95% CI: 1.01–1.61).

Among nearly 12,000 participants in the Growing Up Today Study, Oken et al. (2008) found a strong, nearly linear association between total GWG and obesity (BMI >95th versus < 85th percentile) at the age of 9-14 years after adjusting for maternal BMI and other covariates (see Figure 6-3 and Table 6-3). Overall, each 5-lb increment in GWG was associated with an odds ratio of 1.09 (95% CI: 1.06-1.13) for obesity. Expressing GWG in terms of IOM (1990) recommended weight gain ranges, the authors found that, compared to adequate weight gain, the odds ratio for excessive gain was 1.42 (95% CI: 1.19-1.70). Inadequate gain was not clearly associated with lower risk of obesity. The authors did not find that maternal BMI modified associations of GWG with adolescent obesity, although—if anything—the association was weaker among underweight mothers, in contrast to the findings of Wrotniak et al. (2008).

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TABLE 6-3 Published Studies ($N > 1{,}000$) Relating Total GWG to Child Obesity

	Moreira et al., 2007	**Oken et al., 2007**	**Oken et al., 2008**	**Wrotniak et al., 2008**
Age at Outcome (y)	6-12	3	9-14	7
N	4,845	1,044	11,994	10,226
Birth Years	1990-1997	1999-2002	1982-1987	1959-1966
GWG Exposure	<9 kg 9-13.5 13.6-15.9 16+	per 5 kg; A/E v. I	per 5 lb; I/E v A	per 1 kg; I/E v A
Child BMI Outcome	OR for overweight (IOTF)	OR for $\geq 95^{th}$ v. $< 50^{th}$	OR for $\geq 95^{th}$ v. $< 85^{th}$	OR for $\geq 95^{th}$ v. $< 95^{th}$
Outcome Prevalence	~19.5%	9.0%	6.5%	5.7%
Overall results	1.0 (ref) 1.12 (0.91-1.37) 1.20 (0.90-1.60) 1.27 (1.01-1.67)	1.66 (1.31-2.12) [1.44 (1.17- 1.79) for BMI 95th v. $< 85^{th}$][a] A 3.77 (1.38-10.3) E 4.35 (1.69, 11.2)	1.09 (1.06-1.13) I 0.91 (0.74-1.13) E 1.42 (1.19-1.70)	1.03 (1.02, 1.05) I 0.88 (0.68, 1.14) E 1.48 (1.06, 2.06)
Approximate OR per 5 kg (11 lb)	1.14 (ninth root of 1.27 raised to the fifth power)[b]	1.44	1.21 (1.09 raised to the power 2.2)	1.16 (1.03 raised to the fifth power)
Maternal BMI Category			WHO Categories	IOM Categories
Underweight	NA	NA	0.94 (0.71-1.23)	1.09 (1.01, 1.16)
Normal weight	NA	NA	1.09 (1.04-1.14)	1.02 (1.14, 2.23)
Overweight	NA	NA	1.11 (1.04-1.19)	1.02 (1.14, 2.23)
Obese	NA	NA	1.04 (0.95-1.15)	1.02 (1.14, 2.23)

[a] Not in published paper but subsequently calculated by authors (personal communication, E. Oken, Harvard Medical School and Harvard Pilgrim Health Care, Boston, Ma., December 2008)

[b] Assumes OR of 1.27 is for 9-kg difference between top and bottom categories

NOTES:

A = adequate GWG per 1990 IOM recommendations; E = excessive; I = inadequate
BMI = body mass index
OR = odds ratio; values in parentheses are 95% confidence intervals
NA = data not available
WHO BMI categories = < 18.5, 18.5-24.9, 25-29.9, 30+ kg/m^2
IOM BMI categories = < 19.8, 19.8-26.0, 26.0-29.0, > 29.0 kg/m^2
IOTF = International Obesity Task Force (Cole, 2000)

SOURCES: Moreira et al., 2007; Oken et al., 2007; Oken et al., 2008; Wrotniak et al., 2008.

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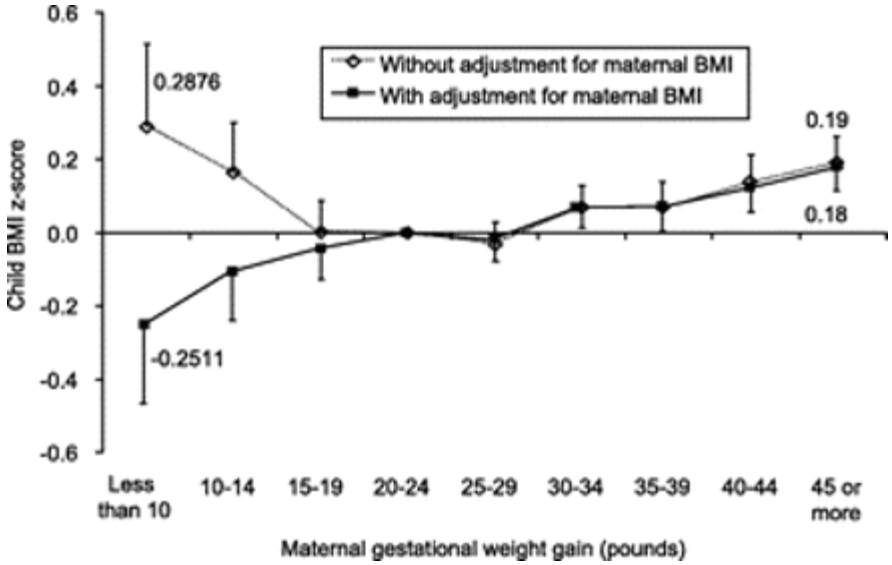

FIGURE 6-3 Associations of maternal gestational weight gain with child BMI *z* score at age 9-14 years, with and without adjustment for maternal prepregnancy BMI. All estimates are adjusted for maternal age, race/ethnicity, marital status, household income, paternal education, child sex, gestation length, age, and Tanner stage at outcome assessment.
SOURCE: Oken et al., 2008. Maternal gestational weight gain and offspring weight in adolescence. *Obstetrics and Gynecology* 112(5): 999-1006. Reprinted with permission.

Other studies have not demonstrated associations between GWG and adiposity-related measures in the offspring. Some of these were suggestive but small (Gale et al., 2007), while others were sufficiently large but did not focus on GWG as a main study exposure or adequately control for confounding factors (Fisch et al., 1975; Maffeis, 1994; Whitaker, 2004).

Overall, evidence to date is suggestive but not conclusive that GWG outside the ranges recommended by IOM (1990) is associated with higher BMI in the offspring. Evidence for effect modification by maternal BMI is scant. No data are available to address the timing of weight gain during pregnancy. Most of the studies rely on child's BMI as the only outcome, but direct measures of adiposity and cardio-metabolic status would strengthen evidence. Only one study to date has reported on blood pressure as an outcome (Oken et al., 2007), although a recent report suggests that higher amounts of weight gain are associated with increase in left ventricular mass from birth to six months (Geelhoed et al., 2008).

The National Children's Study will offer an opportunity to address many of these limitations. Now in early recruitment, this is a cohort study of parents and children from the prenatal (or, in a subset, preconceptional) period through 20 years of age. The sample, to include 100,000 children, is meant to be representative of the US population. This sample will be large enough to assess effect modification by race/ethnicity and other characteristics as well as maternal BMI, and to address timing of weight gain during pregnancy. Child outcomes will include direct measures of adiposity and cardio-metabolic risk factors from birth onwards in addition to length/height and weight. Statistical modeling will allow adjustment for many potential confounding variables.

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Nevertheless, as discussed in the chapter opening, even strong observational studies that have valid exposure and outcome measures, large sample sizes, and appropriate control for confounding cannot fully address the question of causality. Randomized controlled trials that are designed to modify GWG and include follow-up of the children would provide the most compelling evidence for or against intensive clinical or public health efforts to curb excessive weight gain.

Other Outcomes

Neurodevelopment

The effect of alterations in fuel metabolism during pregnancy resulting from intended or unintended weight loss, fasting or poorly controlled diabetes include ketonemia and/or ketonuria, and consequences from those conditions for the neurocognitive development of the infant (see Chapter 3). The committee reviewed the evidence for long-term neurodevelopmental consequences of ketonemia/ketonuria in pregnancy (See Appendix G). As a result of the association between lower GWG and SGA (see discussion above) one indirect way to evaluate the impact of GWG on neurodevelopment is by assessing associations of term and preterm SGA with neurodevelopmental outcomes.

Long term neurodevelopment in term SGA Only observational prospective studies and review articles were identified that evaluate long-term neurodevelopmental outcomes in term SGA infants. With the exception of one study conducted in China, all were conducted in industrialized nations including the U.S. (Goldenberg et al., 1996; Nelson et al., 1997). Of 18 studies identified, six examined neurodevelopmental outcomes during infancy/childhood (Watt and Strongman, 1985; Nelson et al., 1997; Sommerfelt et al., 2000; Hollo et al., 2002; Geva et al., 2006; Wiles et al., 2006), nine during adolescence (Westwood et al., 1983; Paz et al. 1995; Pryor et al., 1995; Goldenberg et al., 1996; Strauss, 2000; Paz et al., 2001; O'Keefe et al., 2003; Indredavik et al., 2004; Peng et al., 2005; Kulseng et al., 2006) and two during adulthood (Viggedal et al., 2004; Wiles et al., 2005).

A study from a Finnish cohort found that SGA children performed worse at school than gestational age-matched controls at 10 years of age (Hollo et al., 2002). Another cohort of children in Norway found a slightly lower mean intelligence quotient (IQ) at five years of age associated with SGA. However, in this study parental factors were more strongly related with IQ than SGA (Sommerfelt et al., 2000).

In studies of development related to SGA results have been mixed. Nelson et al. (1997) found that SGA was not associated with the Bayley Mental Development Index (MDI) and Fagan Test of Infant Intelligence score at one year of age. In contrast, they found that SGA was associated with a lower Bayley Psychomotor Development Index (PDI) among black males but not among females or white infants. Watt and Strongman (1985) documented that SGA was inversely associated with MDI developmental scores at 4 months, whereas Goldenberg et al. (1996) found an inverse relationship between SGA and IQ at 5.5 years of age. Wiles et al. (2006) did not find a relationship between low birth weight and behavioral problems at 6.8 years of age.

An effect size analysis was conducted based on cognitive measures. Of the 18 studies reviewed, 12 reported cognitive scores by SGA status. Of these, two reported lower Bayley scores (Watt and Strongman, 1985; Nelson et al., 1997) and 10 reported lower IQ measures (Westwood et al., 1983; Paz et al. 1995; Pryor et al., 1995; Goldenberg et al., 1996; Sommerfelt

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et al., 2000; Paz et al., 2001; Hollo et al., 2002; Viggedal et al., 2004; Peng et al., 2005; Kulseng et al., 2006) associated with term-SGA status, although these differences were not always statistically significant. Among infants, the Bayley score difference associated with SGA ranged from 4-7 points (Watt and Strongman, 1985; Nelson et al., 1997). Among children (Goldenberg et al., 1996; Sommerfelt et al., 2000; Hollo et al., 2002), IQ differentials were 4-5 points. Among adolescents (Westwood et al., 1983; Paz et al. 1995; Pryor et al., 1995; Paz et al., 2001; Peng et al., 2005; Kulseng et al., 2006) the corresponding range was 2-12 points.

The upper range limit was derived from a study conducted in China that did not control for socioeconomic and other potential confounding factors (Peng et al., 2005). The only study among adults that reported IQ documented a relatively large 19-point IQ differential (based on scores' median instead of average) associated with term-SGA infants. However, it also failed to account for confounding factors (Viggedal et al., 2004). Overall, studies consistently reported small cognitive differentials associate with being born at term and SGA. The meaning of these small differentials is unclear as in all studies average scores among individuals born SGA fell within the normal IQ range.

Although there was a large body of evidence, associations between SGA and longer term neurodevelopmental outcomes among term newborns were inconsistent, especially among adolescents. Among studies that supported this association, major methodological shortcomings (which included substantial attrition rates, lack of standard definitions of SGA across studies, not properly accounting for key confounders [such as socioeconomic status and parental cognitive functioning as well as asphyxia at birth], and lack of testing for effect modification by environmental factors) limit their interpretation. The committee's evaluation of the evidence concurs with previous reviews (Grantham-McGregor, 1995; Goldenberg et al., 1998) that SGA is associated with minimal neurologic dysfunction (e.g., poor school performance) and is not associated with major handicaps, such as cerebral palsy, unless accompanied by asphyxia at birth and that, as a whole, the studies were not designed to identify the influence of SGA, independent of socioeconomic factors, on lower IQs associated with SGA.

Long-term neurodevelopment in preterm SGA In preterm SGA infants, the majority of longitudinal studies reviewed focused on extremely premature (Feldman and Eidelman, 2006; Kono et al., 2007; Paavonen et al., 2007; Leonard et al., 2008) or very low birth weight (VLBW) (Litt et al., 1995; Hack et al., 1998; Brandt et al., 2003; Kilbride et al., 2004; Litt et al., 2005; Feldman and Eidelman, 2006; Hille et al., 2007; Paavonen et al., 2007; Strang-Karlsson et al., 2008a, 2008b) infants. Among 14 studies in children, 11 found that SGA was associated with cognitive and/or neurodevelopment impairments, although this relationship may be modified by degree of postnatal catch-up growth and maternal-child interactions (Casey et al., 2006; Feldman and Eidelman, 2006). In general, the effect size was proportional to the severity of prematurity (Calame et al., 1983; Feldman and Eidelman, 2006; Kono et al., 2007). The two studies conducted among adolescents found an association of VLBW with IQ (Hille et al., 2007) and breathing-related seep disorders (Paavonen et al., 2007). Among adults, VLBW was associated with emotional instability (Strang-Karlsson et al., 2008b) and SGA with lower head circumference among individuals who did not fully catch-up in their head circumference growth during their first 12 months of life.

An effect size analysis was conducted based on cognitive measures. Of 19 studies reviewed, 13 reported cognitive scores by SGA status; and of these one reported a lower Bayley score (Feldman and Eidelman, 2006), and 12 reported lower IQ measures (Escalona, 1982; Calame et al., 1983; Silva et al., 1984; Holwerda-Kuipers, 1987; Litt et al., 1995; McCarton et al., 1996;

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Hutton et al., 1997; Kilbride et al., 2004; Litt et al., 2005; Casey et al., 2006; Hille et al., 2007; Kono et al., 2007) associated with preterm SGA status, although these differences were not always statistically significant. Among 2-year old children, one study found an 8-point difference in the Bayley Mental Development Index score (Feldman and Eidelman, 2006). In contrast, a study conducted among 3.5-year-old children found no differences in IQ scores associated with preterm SGA (Escalona, 1982). Among the rest of studies with children (Calame et al., 1983; Silva et al., 1984; Holwerda-Kuipers, 1987; Litt et al., 1995; McCarton et al., 1996; Hutton et al., 1997; Kilbride et al., 2004; Litt et al., 2005; Casey et al., 2006; Kono et al., 2007), IQ differentials were 2-11 points. The only study among adults that reported IQ, documented a 2-point differential associated with VLBW. Overall, the cognitive differentials appear to be relatively stronger among individuals born SGA preterm (mean ± std. dev: 6.5 ± 3.8 IQ points, n = 11 studies) than among those born SGA term (5.3 ± 3.0, n = 9 studies IQ points). However, as with term SGA, the meaning of these still relatively small differentials is unclear because in the vast majority of studies, the average scores for individuals born preterm SGA fell within the normal IQ range.

The overwhelming majority of studies reviewed support an association between preterm SGA and lower neurodevelopment in the longer term. Consistent with the studies on term SGA, many of the studies on preterm SGA did not properly control for key perinatal (e.g., asphyxia), socio-economic, parental, and home environment confounders (e.g., maternal-child interactions). In addition, although some studies included term births as reference groups (Calame et al., 1983; Silva et al., 1984; Holwerda-Kuipers, 1987; Litt et al., 1995; Hack et al., 1998; Brandt et al., 2003; Kilbride et al., 2004; Litt et al., 2005; Paavonen et al., 2007; Leonard et al., 2008; Strang-Karlsson et al., 2008a, 2008b), others used preterm subgroups as comparison groups (McCarton et al., 1996; Hutton et al., 1997; Casey et al., 2006; Kono et al., 2007). Thus, the effect size or the proportion of the variance in neurodevelopmental outcomes that can be attributed to being born premature per se or to the combination of prematurity and SGA still needs to be determined taking into account these study design limitations.

In summary, as was the case with infant mortality, one must link GWG to being born preterm or small- or large-for-gestational age and, from there, to neurodevelopmental outcomes. This sequence is biologically plausible and it is possible that it is causal, but the evidence to establish causality is not available.

Apgar score Apgar score (see Glossary in Appendix A) assessments are usually conducted one and five minutes after birth and scores can range from 0 to 10. They are not adequate predictors of longer term morbidity and mortality although very low scores (0-3) associated with low birth weight do predict neonatal mortality. Apgar scores in term infants, even at five minutes do not correlate well with neurological outcomes (ACOG, 2006). The AHRQ review (Viswanthan et al., 2008) identified five studies in which the influence of GWG on newborn's Apgar score was examined (Stevens-Simon and McAnarney, 1992; Nixon et al., 1998; Cedergren et al., 2006, Stotland et al., 2006, Wataba et al., 2006). These studies provide only modest evidence that excessive GWG is associated with low Apgar score, and one study suggested that low GWG in nulliparous women also predicts low Apgar score.

Childhood cognition No published studies directly examine the link between GWG and neurocognitive development in infants and children. As discussed in Chapter 3, however, weight loss or failure to gain during pregnancy due to dietary caloric insufficiency may possibly induce maternal hormonal and metabolic responses that may have subsequent consequences for the intellectual development of the child. Because of the obligatory weight gain in maternal tissues

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(uterus, breast, blood) and the fetal-placental unit, a weight gain less than ~7.5-8.5 kg would likely result in mobilization of maternal adipose tissue and possibly lean body mass.

No studies have addressed the gestational metabolic milieu or offspring outcomes of the pregnant woman who experiences weight loss. Studies do exist, however, on the associations of ketonemia or ketonuria, which can occur among pregnant women subjected to short-term fasting (see Chapter 3), on cognition in offspring. Some, but not all, of these studies have found an association between biomarkers of maternal metabolic fuel alterations and child intellectual development (Stehbens et al., 1977; Rizzo et al. 1991; Silverman et al. 1991). In contrast, Persson et al., (1984) and Naeye and Chez (1981) did not find any association of maternal acetonuria, weight loss or low GWG with psychomotor development and IQ in children (see Chapter 3). In summary, although no studies specifically address the impact of very low GWG or weight loss on child intellectual development, some evidence suggests that biomarkers of short-term negative energy negative balance during pregnancy may be related to the child's intellectual development. These associations may be limited to women with diabetes during pregnancy.

Allergy/Asthma

Preterm birth is a risk factor for childhood asthma, often as a result of suboptimal lung function and resulting neonatal respiratory morbidity (Dombkowski et al., 2008). Inasmuch as GWG has, in turn, been associated with risk of preterm birth, the committee considered the plausibility that GWG may also be a risk factor for childhood asthma. In a case-control study of 262 African-American 4-9-year-old children receiving care at a hospital-based clinic, Oliveti et al. (1996) used maternal self-report and child medical records to examine pre- and perinatal risk factors for asthma, defined by physician diagnosis in addition to wheezing or coughing that required asthma medication. Among children with asthma, 24.6 percent were born preterm compared to 13.7 percent of controls. Multivariate logistic regression analyses showed that odds of prevalent asthma were 3.42 (95% CI: 1.72-6.79) times higher among women who gained less (versus more) than 20 total pounds during pregnancy. However, the authors neither adjusted for prepregnancy BMI nor examined the BMI-GWG interaction.

Gestational weight gain may be associated with an increased risk for asthma in offspring through alteration of the developing fetal immune system. Willwerth et al. (2006) found that both inadequate and excessive GWG were associated with increased cord blood mononuclear cell proliferative responses to stimulation (OR = 2.3 and 2.6, respectively), compared to controls with adequate GWG. In this study, however, maternal smoking (OR = 18) was the major determinant of the response.

Cancer

Whether associations exist between GWG, birth weight, and risk for childhood cancers is not clear, however, there are a few studies that have examined the possibility.

Childhood leukemia There may be an indirect relationship between GWG and childhood leukemia because of the established relationship between higher GWG and macrosomia (see discussion above). A recent meta-analysis (Hjalgrim et al., 2003) estimated that the odds for acute lymphoblastic childhood leukemia (ALL) were higher (OR = 1.26 [95% CI: 1.17-1.37]) for infants with birth weight over 4,000 g compared to those under 4,000 g. Although not statistically significant, results were of similar magnitude for acute myelogenous leukemia (AML). McLaughlin et al. (2006) examined the association between pregnancy outcomes and

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leukemia diagnosed before 10 years of age registered in the state of New York. Information on prepregnancy weight (BMI not generally available) and GWG was obtained from birth certificates. Multivariate regression analyses, adjusted for prepregnancy weight, showed that total GWG greater than 14 kg conferred an increased risk for ALL (OR = .31 [95% CI: 1.07, 1.60]). No interaction of GWG with maternal weight was found and there was no association of GWG with AML. The authors speculated that higher GWG could result in higher fetal exposure to insulin-like growth factor I (IGF-I) which in turn may increase the risk of childhood ALL.

Breast cancer Almost two decades ago, Trichopoulos (1990) hypothesized that breast cancer originates from alterations in the prenatal endocrine milieu, in particular higher estrogen levels. Although longitudinal studies are unavailable, observational studies showing direct associations between birth weight and breast cancer provide some support for an association (Michels et al., 1996; Vatten et al., 2002; Ahlgren et al., 2007). Therefore, it is of interest to examine determinants of hormone levels in the maternal-placental-fetal unit. Lagiou et al. (2006) examined the association between GWG and maternal sex hormones at 16 and 27 weeks of gestation. After adjusting for prepregnancy BMI and other covariates no associations were found between GWG and maternal estradiol, estriol or prolactin levels. However, among women with high GWG, there was an association between lower levels of maternal progesterone and of sex hormone binding globulin (-0.7 percent [95% CI -1.5, 0.0] at 16 weeks and -1.2 percent [95% CI -2.0, -0.4] at 27 weeks, respectively, for every 1 kg increment in GWG.

In addition, one study directly addressed the association of GWG with incident breast cancer. Analyzing data from the Finnish Cancer Registry, Kinnunen et al. (2004) found that mothers in the upper tertile of GWG (> 15 kg) had a 1.62-fold higher breast cancer risk than mothers who gained within the recommended range (11–15 kg), after adjusting for parity, mother's age at menarche, at first birth, and at index pregnancy, and prepregnancy BMI. Together these findings provide some support for the hypothesis that excessive weight gain in pregnancy could lead to elevated breast cancer risk in the offspring.

Attention Deficit Hyperactivity Disorder It is possible that maternal body fat reserves and GWG can influence fetal central nervous pathways that ultimately result in behavioral disorders such as attention deficit hyperactivity disorder (ADHD) because the human brain rapidly develops during gestation and the early postnatal period. Only one study (Rodriguez et al., 2008) was identified that examined the associations of early pregnancy BMI and GWG with ADHD. Within three cohorts of 7-12-year-old Scandinavian children, teachers rated the children's inattention and hyperactivity symptoms with standard questionnaires. About 8.5 percent of children were classified as having ADHD symptoms. A large majority of women (86.4 percent) had a normal prepregnancy BMI and adequate GWG (mean gestational age = 39.6 ± 1.6 weeks; and mean birth weight = 3.6 ± 0.5 kg). Multivariate linear regression analyses showed that among women with a high prepregnancy BMI, GWG (weekly gain in 100-g increments) was associated with increased odds of child ADHD (OR = 1.24, 95% CI 1.07-1.44). Among lean women, those who experienced weight loss during pregnancy also had higher odds for child ADHD than their counterparts who did not lose weight during gestation (OR: 1.52, 95% CI: 1.07-2.15). The mechanisms for these effects are unknown, although the authors speculated on the possibility of neurotoxin transfer from maternal adipose tissue to the developing fetal brain.

CONCLUDING REMARKS

Assessing the impact of GWG on child outcomes requires both a short- and long-term outlook. Strong observational evidence links GWG directly with fetal growth, so that higher

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weight gain predicts LGA and lower weight gain predicts SGA, both themselves markers of neonatal morbidity. The literature on preterm birth is more ambiguous because of a less-extensive body of epidemiologic evidence, a nonlinear relationship between GWG and preterm birth, and uncertainty about biologic mechanisms. Even the proper measurement of GWG to take into account the shortened time period of pregnancy is subject to some uncertainty. The U-shaped association of GWG with preterm birth is harder to interpret than the monotonic dose-response gradient with birth weight for gestational age, post-partum weight retention, and childhood obesity. It may reflect distinct causal processes on the low and the high end of GWG.

Among the most important long-term child outcomes are obesity and its sequelae, chiefly cardio-metabolic consequences and neurodevelopmental disorders. Observational evidence is growing that GWG predicts childhood adiposity after adjusting for key factors including prepregnancy BMI, although some older studies do not show this association. The evidence for neurodevelopmental outcomes is dependent on inferences from intermediate endpoints of fetal size and duration of gestation.

Randomized trials are especially important because to date there is no appropriate animal experimental model for GWG, thus reducing one of the criteria—biological plausibility—that epidemiologists often use to support causal inference. Nevertheless, as reviewed in this chapter, pathways involving insulin resistance and fetal hyperglycemia may underlie associations of GWG with subsequent obesity in the offspring. At the other end of the spectrum, reduced GWG is associated with lower fetal growth and preterm birth, themselves associated with later central obesity, insulin resistance, and metabolic syndrome. Very little current evidence, however, suggests that inadequate or low GWG predicts obesity-related outcomes in children.

FINDINGS AND RECOMMENDATIONS

Findings

1. Causal inferences relating GWG to childhood outcomes are tenuous as a result of the paucity of experimental studies.
2. Epidemiologic support for an association between gestational weight gain and stillbirth is weak; there are few methodologically sound studies.
3. Many epidemiologic studies are consistent in showing a linear, direct relationship between GWG and birth weight for gestational age. Thus, lower GWG predicts SGA, and higher GWG predicts LGA. Despite a limited number of randomized controlled trials, biological plausibility from animal models is strong. Relative risks appear higher among women with lower prepregnancy BMI.
4. The evidence for a relationship between GWG and preterm birth, or continuous gestational age, weaker, and biological plausibility is weak. Most studies show associations between lower GWG and preterm birth among underweight, and to a lesser extent, normal weight women. Higher GWG among all BMI categories may also be associated with preterm birth. Evidence is insufficient on associations with spontaneous versus induced preterm birth.
5. A small number of studies show that GWG is directly associated with fat mass in the newborn period. Insufficient evidence is available on associations between GWG and adiposity in infancy.

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6. A small number of relatively large and recent epidemiologic studies show that higher GWG is associated with childhood obesity as measured by BMI. Although biological plausibility is strong, evidence is insufficient to address effect modification by maternal BMI. Only one study has examined blood pressure as an outcome (finding associations in the same direction as BMI) and none has evaluated fat mass or other cardiometabolic consequences of adiposity.
7. Lower GWG may be associated with risk of childhood asthma, chiefly through complications of preterm birth although evidence is limited.
8. Higher GWG may be associated with ALL, breast cancer, and ADHD. Data are limited.
9. Concern exists that metabolic consequences of weight loss during pregnancy may be associated with poorer childhood neurodevelopmental outcomes. Data are limited but raise the possibility that ketonemia among diabetic women could lead to suboptimal neurologic development.

Recommendation for Research

Research Recommendation 6-1: The committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to conduct observational and experimental studies to assess the impact of variation in GWG on a range of child outcomes, including duration of gestation, weight and body composition at birth, and neurodevelopment, obesity and related outcomes, and asthma later in childhood.

Areas for Additional Investigation

The committee identified the following areas for further investigation to support its research recommendations. The research community should conduct studies on:

- Child outcomes related to GWG to provide support for causal inference. Randomized trials and a combination of observational epidemiology and animal models may be a more attainable benchmark to enhance certainty regarding causal links between GWG and infant outcomes;
- Statistical models that follow sound theoretical frameworks and clearly distinguish among confounding, mediating, and moderating (effect modifying) variables. Statistical models based on path analysis such as structural equation modeling may be able to improve interpretation of complex data; and
- Preventing excessive weight gain with all of the attributes listed above for observational studies. Even relatively small studies that can evaluate intermediate endpoints, if not the clinically important outcomes, would make a significant contribution.

Furthermore, future research on GWG and child outcomes should:

- Not assume linear relationships between GWG and offspring obesity, but should look for U- or J-shaped associations as well;
- Determine whether the pattern of maternal weight gain matters for short- or long-term child outcomes, e.g., whether weight gain earlier in pregnancy is more harmful than later gain; and

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- Determine whether critical or sensitive periods of adiposity accretion exist in pregnant women, and if so when weight gain is an adequate measure to capture those periods.

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7

Determining Optimal Weight Gain

INTRODUCTION

In this chapter, the approach used by the committee for arriving at its recommendations for revision of the current guidelines for weight gain during pregnancy is discussed. First, a brief discussion of the principles used by the committee to develop a strategy for making its recommendations is presented. The strategy is then described in some detail, along with the results of applying this approach. Finally, the committee's recommendations are detailed and discussed.

As was the case for the report, *Nutrition During Pregnancy* (IOM, 1990), the committee used a conceptual framework to organize the evidence for a causal relationship between gestational weight gain (GWG) and both short- and long-term outcomes. The frequency and severity of these outcomes were considered. In particular, although a possible trade-off between maternal and child health was recognized in the Institute of Medicine report (IOM, 1990) as being a consequence of changing the weight gain guidelines, evaluation of that trade-off was not possible with the data then available. The committee made evaluating this trade-off a central element of its process to develop new guidelines, while recognizing that although the available data have increased, they are still less than fully adequate for this purpose. In making its recommendations, the committee sought to recognize unintended consequences and to develop guidelines that are both feasible and potentially achievable. It is important to note that these guidelines are intended for use among women in the United States. They may be applicable to women in other developed countries, however, they are not intended for use in areas of the world where women are substantially shorter or thinner than American women or where adequate obstetric services are unavailable.

STRATEGIC APPROACH FOR DEVELOPING RECOMMENDATIONS

The committee identified a set of consequences for the short- or long-term health of the mother and the child that are potentially causally related to GWG. These consequences included those evaluated in a systematic review of outcomes of maternal weight gain prepared for the Agency for Healthcare Research and Quality (AHRQ) (Viswanathan et al., 2008) as well as others based on data from the literature outside the time window considered in that report. To develop estimates of both the risk of these outcomes and their frequency in the population, the committee used data available in the published literature and also commissioned additional analyses (see below).

The committee considered several of potential outcomes associated with GWG and compared their incidence, long-term sequelae, and baseline risk (additional information about these outcomes appears in Appendix G). Postpartum weight retention, cesarean delivery, gestational diabetes mellitus (GDM), and pregnancy-induced hypertension or preeclampsia emerged from this process as being the most important maternal health outcomes. The committee

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removed preeclampsia from consideration because of the lack of sufficient evidence that GWG was a cause of preeclampsia and not just a reflection of the disease process in this condition. The committee also removed GDM from consideration because of the lack of sufficient evidence that GWG was a cause of this condition. Postpartum weight retention and, in particular, unscheduled primary cesarean delivery were retained for further consideration.

Measures of size at birth (e.g. small-for-gestational age [SGA] and large-for-gestational age [LGA]), preterm birth and childhood obesity emerged from this process as being the most important infant health outcomes. The committee recognized that both SGA and LGA, when defined as < 10th percentile and > 90th percentile of weight-for-gestational age, respectively, represent a mix of individuals who are appropriately or inappropriately small or large. In addition, the committee recognized that being SGA was likely to be associated with deleterious outcomes for the infant but not the mother, while being LGA was likely to be associated with consequences for both the infant and the mother (e.g., cesarean delivery). The committee addressed this mix of outcomes in the approach used to develop its recommendations (see below).

Previous Approaches for Developing Weight Gain Recommendations

Many approaches have been and are currently being used for making recommendations for how much weight women should gain during pregnancy. At one extreme is the advice from the National Center for Clinical Excellence in the United Kingdom that women should not be weighed at all during pregnancy "as it may produce unnecessary anxiety with no added benefit" with the exception being "pregnant women in whom nutrition is of concern" (National Collaborating Centre for Women's and Children's Health, 2008). In the United States, the 1970 report *Maternal Nutrition and the Course of Pregnancy* (NRC, 1970) recommended a single target, and average gain of 10.9 kg (24 pounds), with a range of 9.1-11.3 kg (20-25 pounds). This target was based on the amount of weight that healthy women gain when meeting the physiologic needs of pregnancy (e.g., the products of conception, expansion of plasma volume, red cell mass and maternal fat stores).

Still another approach has been used in Chile. Since 1987, maternal weight gain recommendations for the Chilean population have been based on a single target, but instead of an absolute amount of weight, a proportion (120 percent) of the woman's "standard weight" for her height is used (Rosso, 1985; Mardones and Rosso, 2005). This results in a recommendation for a higher gain in underweight women and a lower gain in heavier women, with an upper limit of 7 kg for women with prepregnant weights over 120 percent of the standard (Figure 7-1). The objective of this recommendation is to increase birth weight among underweight women, and it is considered successful in having done so (Mardones and Rosso, 2005). The IOM (1990) report also recommended higher gains for underweight women and lower gains (but at least 6.8 kg) for heavier women. The desired outcome was expressed as specific target ranges for each of 3 prepregnant body mass index (BMI) groups. The rationale for this approach was to achieve the birth weight (3-4 kg) associated with "a favorable pregnancy outcome" in all prepregnant BMI groups while avoiding the birth of infants with weight > 4 kg because of "the possible risks to the mother and infant of complicated labor and delivery" (IOM, 1990).

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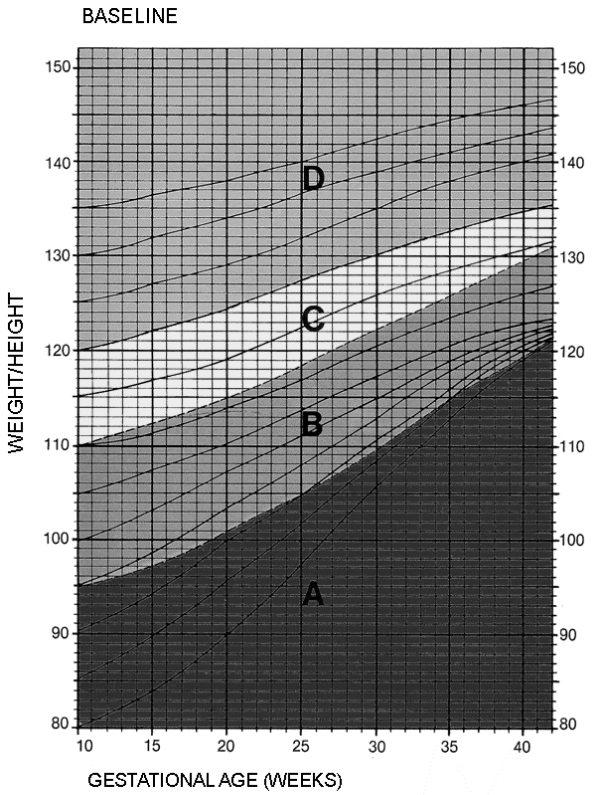

FIGURE 7-1 Graphic showing weight increase for pregnant women
NOTE: A = underweight; B = normal weight; C = overweight; D = obese
SOURCE: A weight gain chart for pregnant women designed in Chile, Mardones F. and P. Rosso. Copyright © 2005, Maternal and Child Nutrition. Reproduced with permission of Blackwell Publishing Ltd.

In constructing their recommendations, both the Chilean investigators (Mardones and Rosso, 2005) and the IOM (1990) committee explicitly recognized the trade-off between raising the birth weight of infants born to underweight women and increasing the risk of high birth weight in some infants as well as obesity and other undesirable outcomes in their mothers. In fact, the IOM (1990) committee recommended that a formal decision analysis be undertaken "in which probabilities and utilities (values) are assigned to each potential outcome" so as to assist in balancing the risks and benefits of any recommendation.

Since the publication of the IOM (1990) report, several groups of investigators have offered their own approaches for determining the optimal GWG. All of the studies of this type identified by the committee are discussed below. It is noteworthy that, with one exception (Nohr et al., 2008), maternal and infant outcomes beyond the immediate neonatal period were not included in these investigations. In each of these investigations, the researchers studied GWG as a categorical, not a continuous, variable, and each group defined the categories differently. Bracero and Byrne (1998) used data from 20,971 pregnant women and their singleton infants who were delivered at a single hospital in New York City (1987-1993). They identified the GWG at which the proportion of women who had infants with any one of 11 adverse perinatal outcomes was minimal. This list included outcomes not generally associated with GWG. Adverse maternal outcomes were not considered. In general, they found that this point was at a higher GWG than recommended in the IOM (1990) report. Therefore, they recommended gains of 16.3-18.1 kg

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(36-40 pounds), 14.1-18.2 kg (31-40 pounds) and 11.8-13.6 kg (26-30 pounds) for underweight, normal weight and overweight or obese women categorized by the cutoff points in the IOM (1990) report, respectively.

Cedergren (2007) conducted a population-based cohort study (1994-2004) that included the data from 298,648 Swedish women obtained from the Swedish Medical Birth Registry. She calculated the risk of a variety of pregnancy outcomes by maternal prepregnant BMI category. She did this "to estimate weight gain limits that were associated with a significantly decreased risk of the most clinically dangerous situations for the mother and the infant." It is important to note that her selection of adverse outcomes "was not based primarily on possible correlations with weight gain or maternal BMI" (Cedergren, 2007). In addition to SGA and LGA, her analysis included six maternal and seven fetal outcomes that were unweighted for either their frequency or severity. Preeclampsia was included, but not GDM. With this approach, Cedergren (2007) found that the optimal GWG was lower than that recommended in the IOM (1990) report in all categories, especially for overweight or obese women.

In three studies that used population-based cohorts from Missouri, DeVader et al. (2007) studied 94,696 normal weight women (1999-2001), Langford et al. (2008) studied 34,143 overweight women (1990-2004), and Kiel et al. (2007) studied 120,251 obese women (1990-2001) who delivered full-term, singleton infants. These three groups of investigators calculated the risk of pregnancy outcomes that are routinely collected on all birth certificates according to reported GWG. DeVader et al. (2007) and Langford et al. (2008) assessed the risk of these outcomes according to whether the women had gained < 11.4 kg (25 pounds), 11.4-15.9 kg (25-35 pounds) or > 15.9 kg (> 35 pounds). Both groups found that the primary hazard of gaining less than the IOM (1990) report recommendation was delivering an SGA or low birth weight (< 2500 g) infant (Langford et al., 2008 only); gaining in excess of the recommendation was associated with an increased risk of several adverse outcomes, including preeclampsia, cesarean delivery and delivery of an LGA or macrosomic infant (Langford et al., 2008 only). After balancing these risks, DeVader and her colleagues (2007) concluded that the "ideal" gestational weight gain for their population of normal weight women was 11.4-15.5 kg (25-34 pounds). Langford and her colleagues (2008) found that overweight women "should gain within the current recommendations (15-25 lbs)" and that "there may be additional benefit of gaining below the recommendations, specifically in the 6-14 lbs range."

The number of obese women in their sample was large enough so that Kiel et al. (2007) were able to distinguish among obesity classes I, II, and III. They found that the risk of delivering an SGA infant continued to decrease with increasing degrees of maternal obesity and was minimal among women who gained < 6.8 kg (15 pounds) during pregnancy. In addition, although the pattern of increasing risk of preeclampsia, cesarean delivery and LGA birth with increasing GWG was the same across the obesity classes, Kiel et al. (2007) found that the point at which the risk of these outcomes considered as a group was minimal differed for each obesity class. This minimal risk corresponded to GWG of 4.5-15.5 kg (10-25 pounds), and 0-4.1 kg (0-9 pounds) for obesity class I and obesity classes II and III, respectively. In all of these studies of women from Missouri, the authors chose to consider outcomes that have been related to GWG (although the validity of using preeclampsia is open to question, see Chapter 5). As was the case for Cedergren's analysis (2007), these investigators did not consider the frequency or severity of these events and the outcomes of pregnancy were restricted to those at delivery.

In the most recent of the research reports in which authors have tried to identify optimal GWG, Nohr and colleagues (2008) used data from the Danish National Birth Cohort (1996-

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2002). This study included 60,892 women with term pregnancies. Data on weight before pregnancy, weight gain during pregnancy and postpartum weight were obtained during telephone interviews of the mother and outcome data were obtained from birth and hospital discharge registries. Nohr et al. (2008) calculated the risks of a variety of maternal and neonatal outcomes associated with prepregnant BMI and GWG and their interaction. For those with a strong independent association with GWG and little possibility of reverse causality (unscheduled primary cesarean delivery, SGA, LGA, and postpartum weight retention ≥ 5 kg), they calculated the absolute risk of these outcomes for women in each of the four major categories of prepregnant BMI. Although the trade-off between reducing the risk of SGA and increasing the risk of cesarean delivery was evident in these data as it was in those from Sweden (Cedergren, 2007) and Missouri (Devader et al., 2007; Kiel et al., 2007; Langford et al., 2008), what is unique in this presentation is the inclusion of postpartum weight retention. Nohr et al. (2008) showed a dramatic increase in postpartum weight retention ≥ 5 kg with increasing GWG in all categories of prepregnant BMI. Nohr and her colleagues (2008) calculated the proportion of women who had changed from one BMI category to another at six months postpartum according to their GWG. In this analysis, only 0.4 percent of underweight women had become overweight at the highest GWG (≥ 20 kg) studied. Thus, they concluded that high GWG was "probably not disadvantageous for either underweight women or their infants" (Nohr et al., 2008). For normal weight, overweight and obese women, however, the tradeoff between SGA and these other outcomes particularly postpartum weight retention, occurred at lower GWG values: 16-19 kg, 10-15 kg and <10 kg, respectively (Nohr et al., 2008). As was the case for the other studies, Nohr et al. did not weight their outcomes by their frequency or severity; however, it is clear that the authors sought the point of minimum risk of SGA and postpartum weight retention ≥ 5 kg in their decision making.

Although the analytic approaches used by these research groups have many similarities, their conclusions about optimal weight gain varied widely (Table 7-1). This was particularly striking for underweight and normal weight women, but was also present for overweight women. The differences in conclusions may have resulted from the different mix of outcomes that were considered. The report of Nohr and coworkers (2008) was the only one to exclude preeclampsia and include postpartum weight retention. Cedergren (2007) included a number of outcomes of pregnancy that lack a clear association with GWG. None of these reports included the development of obesity during childhood as an outcome or provided information about the consequences of variation in GWG among women in the racial and ethnic subgroups common in the American population or among women who are young or short—groups that were explicitly considered in the IOM (1990) report. As noted above, none of these analyses was weighted (at least explicitly) by the severity or frequency of the adverse outcomes considered and the categories of GWG were constructed separately by each group of investigators.

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TABLE 7-1 Summary of Research Published since the IOM (1990) Report in which Recommendations for Optimal Weight Gain During Pregnancy are Developed

		Proposed Optimal Weight Gain During Pregnancy (kg)					
Maternal Prepregnant BMI (kg/m^2)	**1990 IOM Guidelines (kg)**	**Bracero and Byrne, 1998**	**Cedergren, 2007**	**DeVader et al., 2007**	**Kiel et al., 2007**	**Langford et al., 2008**	**Nohr et al., 2008**
IOM BMI Categories							
Underweight (< 19.8)	12.5-18	16.4-18.2	—	—	—	—	—
Normal weight (19.8-26.0)	11.5-16	14.1-18.2	—	11.4-15.5	—	—	—
Overweight (26.0-29.0)	7-11.5	11.8-13.6	—	—	—	6.8-10.9 (or 2.7-6.4)	—
Obese (> 29)	≥ 6	11.8-13.6	—	—	—	—	—
WHO BMI Categories							
Underweight (< 18.5)	—	—	4-10*	—	—	—	> 20
Normal weight (18.5-24.9)	—	—	2-10*	—	—	—	16-19
Overweight (25-29.9)	—	—	< 9	—	—	—	10-15
Obese (≥ 30)	—	—	< 6	—	—	—	< 10
Obese Class I (30-34.9)	—	—	—	—	4.5-11.4	—	
Obese Class II (35-39.9)	—	—	—	—	0-4.1	—	—
Obese Class III (≥ 40)	—	—	—	—	loss of 0-4.1	—	—

*BMI cutoff of 20 kg/m^2.

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APPROACH USED BY THE COMMITTEE IN DEVELOPING ITS RECOMMENDATIONS

To address these conflicts and gaps within the available literature, the committee commissioned several additional analyses that informed its decision making (Table 7-2) (see Appendix G). Dr. Ellen Nohr provided two sets of analyses from the Danish National Birth Cohort. She expanded her published analyses (Nohr et al., 2008) to provide information on an additional lower and an additional higher category of GWG and replicated her published analyses for obese class I women separately from obese class II and III women. She conducted analogous new analyses several important subgroups of the population of pregnant women, namely primiparous, short, and young women as well as smokers (information contributed to the committee in consultation with Nohr [see Appendix G, Part I]). Dr. Amy Herring analyzed the 1988 National Maternal and Infant Health Survey (NMIHS) to explore the association between GWG and outcomes important to the committee separately for white and black women. She also linked it to its 1991 follow-up to examine the association between GWG and postpartum weight retention in this sample. She was unable to examine the long-term weight status of infants born LGA because access to the data could not be obtained in a timely manner (information contributed to the committee in consultation with Herring [see Appendix G, Part II]). Dr. Cheryl Stein analyzed adverse outcomes associated with GWG stratified by racial/ethnic group in the subsample of births during 1995-2003 in New York City for which prepregnant BMI was available (information contributed to the committee in consultation with Stein [see Appendix G, Part III]). In addition, the committee commissioned a quantitative analysis of risk trade-offs between maternal and child health outcomes associated with GWG by Dr. James Hammitt (information contributed to the committee in consultation with Hammitt [see Appendix G, Part IV]).

The committee relied on both standard criteria for evaluating the quality of research studies (such as those provided by the American Academy of Pediatrics, 2004) as well as its expert judgment when evaluating the evidence. It used evidence from the published scientific literature as well as the analyses it commissioned. In the development of its recommendations, the committee evaluated the overall quality of the evidence as well as the balance between benefits and risks. The committee relied on the highest level of evidence (randomized controlled trials, and experimental studies in women and animal models). However, few such experimental studies were available in the literature relevant to the committee's task. In addition, the committee used data from the general population in those instances in which data on minority populations were unavailable.

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TABLE 7-2 Research Commissioned by the Committee: Characteristics of the Datasets Used

	Consultant		
Characteristic	**Nohr**	**Herring**	**Stein**
Population Studied	• Danish National Birth Cohort • National sample • 1996-2002 • *n* = 60,892 • Singleton term births	• National Maternal and Infant Health Survey (NMIHS) • Nationally representative sample • 1988 linked to 1991 follow-up	• New York City births • 1995-2003 • Subset of 34,307 births (those with maternal height among 913,320 singleton births)
Subgroups Available	• Primiparous versus multiparous • < 20 years old versus older • Smokers versus non-smokers • Short versus non-short stature • Obesity classes II and III • GWG < 5 kg and = 25 kg	• White versus black	• White versus black
Outcomes Included	• SGA/LGA • Emergency cesarean delivery • PPWR (≥ 5 kg at 6 months)	• Primary cesarean delivery (*n* = 5,433) • Preterm birth (*n* = 7,728) • SGA and LGA (*n*= 7,748) • PPWR, 6-12 months (*n* = 1,089) • Breastfeeding initiation and duration • Infant mortality	• Spontaneous preterm birth • Primary cesarean delivery • SGA/LGA

NOTE: GWG = gestational weight gain; SGA = small-for-gestational age; LGA = large-for-gestational age; PPWR = postpartum weight retention.

CONSTRUCTION OF GUIDELINES FOR GESTATIONAL WEIGHT GAIN

Prepregnant BMI Category

After the publication of the IOM (1990) report, the World Health Organization (WHO) held a consultation that developed a categorization of BMI values for adults based on different cutoff

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points (WHO, 1995). The WHO cutoff points were subsequently endorsed by the National Institutes of Health (NHLBI, 1998). These categories have been widely adopted in the United States and internationally and, if used in formulating recommendations for GWG, would provide opportunities for a consistent message to women and health care providers about weight status for all groups of adults, including women of childbearing age. For these reasons, the committee adopted the WHO BMI categories for its recommendations.

Evidence from the scientific literature is remarkably clear that prepregnant BMI is an independent predictor of many adverse outcomes of pregnancy (see Chapter 5). These data provide ample justification for the choice made in the IOM (1990) report to construct weight gain guidelines according to prepregnant BMI. That approach has been retained in the current document.

Special Populations

Women of Short Stature

The IOM (1990) report guidelines recommended that women of short stature (< 157 cm) should gain at the lower end of the range for their prepregnant BMI. The committee was unable to identify evidence sufficient to continue to support a modification of GWG guidelines for women of short stature (Vishwanathan et al., 2008). The limited data available to the committee indicated that women of short stature had an increased risk of emergency cesarean delivery but that this risk was not modified by GWG; they did not have an increased risk of having an SGA or LGA infant or of excessive postpartum weight retention compared to taller women (Appendix G). No information was available with which to evaluate whether a modification of guidelines might be necessary for very short (<150 cm) women.

Adolescents

Evidence to continue to support a modification of the GWG guidelines for adolescents (females < 20 years old) was also insufficient (Vishwanathan et al., 2008) (see Chapter 4). For adolescents < 18 years old, the WHO BMI cutoff points for overweight and obesity often do not correspond to the 85th and 95th percentiles, respectively, of the Centers for Disease Control (CDC) pediatric growth charts that used to assess growth in these girls [Available: http://www.cdc.gov/nchs/data/nhanes/growthcharts/set2/chart%2016.pdf (accessed December 3, 2008)]. The younger the girl, the more likely it is that she will reach the 85th or 95th percentile of the growth charts at a lower BMI value than the corresponding WHO cutoff points. Thus, if the adult cutoff points are used to determine the prepregnant BMI category of younger adolescents, some of them will be categorized as being in a lighter group. As a result, the GWG recommendation would be higher than would be the case if the pediatric growth charts were used to categorize them. The committee determined that this was a tolerable risk for two reasons. First, research has shown that young teens often need to gain more than adult women to have an infant of the same size (Scholl, 2008). Second, it would be difficult to implement a recommendation in obstetric practices to use pediatric growth charts to categorize the prepregnant BMI of these girls.

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Women with Multiple Fetuses

The evidence base for women carrying multiple fetuses remains, as it was in 1990, limited. In that report (IOM, 1990), women carrying twins were encouraged to gain 16-20.5 kg (35-45 pounds) without respect to their prepregnancy BMI category. However, recent data suggest that the weight gain of women with twins who have good outcomes varies with prepregnancy BMI (see Chapter 3) as is clearly the case for women with singleton fetuses. Unfortunately, the committee was unable to conduct the same kind of analysis for women with twins as it did for women with singletons because the necessary data are unavailable. Therefore, the committee offers provisional guidelines, which are based specifically on the work of Luke and Hediger (Appendix C) and are corroborated by the work of others (Chapter 4). Unfortunately, these data sources do not provide sufficient information to develop provisional guidelines for underweight women. The provisional guidelines are: normal weight women should gain 17-25 kg (37-54 pounds), overweight women, 14-23 kg (31-50 pounds) and obese women, 11-19 kg (25-42 pounds) at term. These provisional guidelines reflect the interquartile (25th – 75th percentiles) range among women who delivered their twins, who weighted ≥ 2,500 g on average, at 37-42 weeks of gestation.

Racial/Ethnic Group

The descriptive observational data cited in Chapter 4 suggested that inadequate GWG was more common in some racial/ethnic groups. However, only Dr. Stein's analysis of data from New York City in 1995-2003 and Dr. Herring's analysis of the nationally representative data from the NMIHS in 1988-1991 provided insight into whether a woman's racial or ethnic group modified the relationship between GWG and the various outcomes of interest. The predominant finding from these analyses was that racial/ethnic group did not modify the association between GWG and these outcomes. As a result the committee concluded that, although confirmatory research is needed, its recommendations should be generally applicable to the various racial or ethnic groups that make up the U.S. population.

Obesity Classes II and III

Although a record-high number of American women of childbearing age have BMI values in obesity classes II and III, the evidence identified and reviewed by the committee was insufficient to develop more specific recommendations for GWG among these women.

Parity

It has long been known that primiparous women have smaller infants than multiparous women (as reviewed in Chapter 4) and they also gain more weight. The analyses by Nohr (information contributed to the committee in consultation with Nohr [see Appendix G, Part I]). show that primiparous women must gain more weight during pregnancy than multiparous women to have equally low risk of having an SGA birth, but primiparous women are similar to multiparous women in their likelihood of retaining ≥ 5 kg at 6 months postpartum in every category of prepregnant BMI. This means that the tradeoff between lowering the risk having an SGA infant and increasing the risk of retaining an excessive amount of weight postpartum occurs at a different GWG value for primiparous and multiparous women. This is a novel finding that warrants additional study.

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Smokers

It has also long been known that smokers have smaller infants than non-smokers. Analyses prepared by Nohr (information contributed to the committee in consultation with Nohr [see Appendix G]) show that smokers who gain more weight, as expected, have larger infants, but they also retain more weight postpartum. For example, among normal weight multiparous women, smokers would have to gain at least 16-19 kg instead of 5-9 kg to have a 10 percent risk of having an SGA infant. If they were to gain in this higher range, their risk of retaining ≥ 5 kg at 6 months postpartum would become over 20 percent instead of being about 5 percent. Thus, the weight gain trade-off to prevent an SGA birth is particularly unfavorable for smokers, which is perhaps because (as reviewed in Chapter 4) at least some of the effect of smoking on birth weight is independent of GWG. As a result, additional GWG may fail to increase birth weight but, nonetheless, still increase postpartum weight retention. This unfavorable trade-off is best resolved by smoking cessation.

DEVELOPMENT OF RECOMMENDED WEIGHT-GAIN RANGES

Guidelines for Gestational Weight Gain

As was the case for the current guidelines for GWG, the committee chose to formulate the new guidelines with a range for each category of prepregnant BMI. This range reflects the imprecision of the estimates on which these recommendations are based, the reality that good outcomes are achieved with a range of weight gains and the many additional factors that may need to be considered when making a recommendation for an individual woman.

To develop these ranges (Table 7-3), the committee proceeded as follows. Based on the available published literature (Appendix E and F) as well as the reports of its consultants (Appendix G), committee ascertained the GWG value or range of GWG values associated with lowest prevalence of the outcomes of greatest interest (namely, cesarean delivery, postpartum weight retention, preterm birth, small- or large-for gestational age birth and childhood obesity). When weighting the trade-off among these outcomes, the committee considered, within each category of prepregnant BMI (a) the incidence or prevalence of each of these outcomes, (b) whether the outcomes were permanent (e.g. neurocognitive deficits) or potentially modifiable (e.g. postpartum weight retention) and (c) the quality of the available data. The committee compared the resulting ranges with those developed in the quantitative risk analysis conducted by its consultant, Dr. Hammitt. Finally, the committee considered how its recommendations might be accepted and used by clinicians and women.The committee intends these guidelines be used in concert with good clinical judgment as well as a discussion between the woman and her prenatal care provider about diet and exercise. If a woman's GWG is not within the proposed guidelines, prenatal care providers should consider other relevant clinical evidence as well as both the adequacy and consistency of fetal growth and any available information on the nature of excess (e.g. fat or edema) or inadequate GWG before suggesting that the woman modify her pattern of weight gain. The safety of intentional weight loss during pregnancy among obese women has not been determined. Thus, priority should be given to addressing weight loss issues preconceptionally or between pregnancies, not during pregnancy.

In constructing these guidelines, the committee recognized that they fall within the category of personalized medicine. Use of these guidelines will require standardized assessment procedures to inform clinical judgment as well as support of ancillary services (e.g. counseling

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on nutrition and physical activity) or other interventions that might be deemed necessary to achieve them. Thus, the committee recognizes that full implementation of these guidelines may entail additional medical expenses. The committee did not attempt to estimate the magnitude of these potential additional medical expenses.

TABLE 7-3 New Recommendations for Total and Rate of Weight Gain during Pregnancy, by Prepregnancy BMI

	Total Weight Gain		**Rates of Weight Gain* 2nd and 3rd Trimester**	
Prepregnancy BMI	**Range in kg**	**Range in lbs**	**Mean (range) in kg/week**	**Mean (range) in lbs/week**
Underweight (< 18.5 kg/m^2)	12.5–18	28–40	0.51 (0.44–0.58)	1 (1–1.3)
Normal weight (18.5–24.9 kg/m^2)	11.5–16	25–35	0.42 (0.35–0.50)	1 (0.8–1)
Overweight (25.0–29.9 kg/m^2)	7–11.5	15–25	0.28 (0.23–0.33)	0.6 (0.5–0.7)
Obese (≥ 30.0 kg/m^2)	5–9	11–20	0.22 (0.17–0.27)	0.5 (0.4–0.6)

* Calculations assume a 0.5–2 kg (1.1– 4.4 lbs) weight gain in the first trimester (based on Siega et al., 1994; Abrams et al., 1995; Carmichael et al., 1997)

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Rate of Weight Gain

Pregnant women typically gain ~1-2 kg in the first trimester. According to the new recommended GWG values, normal weight women should gain ~0.4 kg per week in the second and third trimesters of pregnancy. Underweight women should gain slightly more (~0.5 kg per week) and overweight women slightly less (~0.3 kg per week) than this amount (Table 7-3). Obese women should gain about ~0.2 kg per week (Table 7-3). These guidelines were constructed based on the assumption that GWG is linear during the second and third trimesters of pregnancy.

The IOM (1990) report made a series of recommendations about how to implement its guidelines in the context of caring for an individual patient. As they remain appropriate, the committee endorses the key elements of these recommendations. These key elements are:

1. Before conception, use consistent and reliable procedures to measure and record the woman's weight and height without shoes in the medical record.
2. Determine the woman's prepregnancy BMI.
3. Measure the woman's height without shoes and weight in light clothing at the first prenatal visit carefully by procedures that have been rigorously standardized at the site of prenatal care and use consistent, reliable procedures to measure weight at each subsequent visit.
4. Estimate the woman's gestational age from the onset of her last menstruation or from an early ultrasound examination.
5. At the initial comprehensive prenatal examination, set a weight gain goal together with the pregnant woman that is based on her prepregnant BMI and other relevant considerations, and explain to her why weight gain is important.
6. Monitor the woman's prenatal course to identify any abnormal pattern of gain that may indicate a need to intervene, displaying the results graphically for the woman (see Chapter 8, Figures 8-1 through 8-4). When abnormal gain appears to be real, rather than a result of an error in measurement or recording, try to determine the cause and then develop and implement corrective actions jointly with the woman.

DISCUSSION OF THE NEW GUIDELINES

These new guidelines differ from those issued in 1990 in two important ways. First, they are based on a different set of cutoff points for prepregnant BMI. Compared to the new cutoff points used in the 1990 guidelines, using the WHO guidelines reduces the proportion of the population in the underweight and obese groups, as these groups are based on more extreme BMI values, and raises the proportion of the population in the normal weight and overweight groups, as these groups are based on wider ranges of BMI values.

Second, these new guidelines include a specific, relatively narrow range of recommend gain for obese women. This recommendation reflects the data available to the committee, the preponderance of which cover women in obesity class I (BMI 30.0-34.9 kg/m^2) rather than obesity classes II and III. As noted in Chapter 2, in the last two decades more American women of childbearing age have prepregnant BMI values in obesity classes II and III. Unfortunately, only two studies provide data on women in these obesity classes (Kiel et al. 2007; information contributed to the committee in consultation with Nohr [see Appendix G]) and few of the women

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studied gained < 5 kg. It is possible, based on the data collected in these investigations, that weight gains < 5 kg may be associated with a more favorable trade-off among outcomes than higher gains. However, the committee's review showed insufficient evidence to recommend gains this low and thus it was concerned about the potential for doing harm that is associated with fetal growth restriction and ketonemia (see Chapters 3 and 6). Ketonemia can occur with the accelerated starvation that is characteristic of pregnancy and may be more frequent with low weight gains. The committee recognized that women in obesity classes II and III may, without intervention, gain little during pregnancy and manage their pattern of dietary intake so as to avoid ketonemia and other problems. However, there is no evidence to determine whether a guideline for very low weight gain during pregnancy among women in obesity classes II and III would be managed well enough by these women and their care providers to avoid ketonemia.

Although there is ample justification for continuing to structure the new guidelines according to maternal prepregnancy BMI, this approach is not without limitations. Maternal height, for example, has long been known to be a determinant of birth weight among women with a narrower range of prepregnancy weight (40-80 kg) than commonly observed today (Tanner and Thomson, 1970). In addition, height appears to be a stronger predictor of GWG than prepregnancy BMI (Straube et al., 2008). However, the research necessary to show that height or another attribute might be a superior alternative to prepregnancy BMI for constructing guidelines for subgroups of pregnant women has not been conducted.

The committee based its guidelines, in part, on the presumption that the extensive, consistent observational data that link GWG to fetal growth, as measure by SGA and LGA, as well as those that link GWG to postpartum weight retention are causal. The committee recognizes, however, that the simple model in which increased caloric intake increases maternal weight and maternal weight, in turn, increases fetal weight, is likely to be more complex—and may even be incorrect. The limited results from randomized trials among undernourished women provide indications of this pathway in some cases (Susser, 1991). The results from more recent but very small randomized trials designed to control excess weight gain (see Chapter 8) provide suggestive support for this pathway. Although there are possible non-causal explanations linking GWG to fetal growth, including diet composition, affecting both GWG and fetal growth independently, or shared genetic determinants of GWG and fetal growth, none of these alternatives has been proven valid. Therefore, the committee determined that it would be prudent to consider the evidence linking inadequate GWG, especially in underweight and normal weight women, with increased risk of SGA; and the evidence linking excessive GWG, especially in overweight and obese women, with increased risk of LGA and its consequences in developing these guidelines.

As additional experimental data are generated to confirm or refute a causal interpretation of the evidence linking GWG and fetal growth, this reasoning may need to be revised. In contrast, the likelihood that the link from increased caloric intake to increased GWG and, in turn, from increased GWG to increased postpartum weight retention is causal seems more certain. However, postpartum weight retention reflects not only GWG, but also maternal actions postpartum, including but not limited to changes in dietary intake and physical activity associated with new motherhood as well as breastfeeding behavior (Baker et al., 2008).

It is noteworthy that these guidelines are structured as the ranges associated with good outcomes for both mother and infant. For example, women who are more concerned with postpartum weight retention than with the birth of a small baby, can choose to gain at the lower instead of the higher end of the range for their prepregnancy BMI category.

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As American women of childbearing age have become heavier, the trade-off between maternal and child health created by variation in GWG has become more difficult to reconcile than it was when prevention of SGA births was paramount and there was relatively low risk of excessive weight retention postpartum and childhood obesity with additional GWG. The effort made by the committee to project the short- and long-term consequences of GWG for both mothers and their children so as to reconcile the trade-offs between them is a unique feature of the process used to develop these new guidelines. For this purpose, the committee used data from the NMIHS (information contributed to the committee in consultation with Herring [see Appendix G, Part II]) to provide estimates for the probability infant mortality and data from the Danish National Birth Cohort (Nohr et al., 2008) to provide estimates for the probability of postpartum weight retention related to GWG within each category of prepregnant BMI. Dr. Hammitt linked the data on postpartum weight retention to estimates of morbidity and mortality associated with additional maternal weight. Similarly, data from the Growing Up Today Study (Oken et al., 2008) and supporting studies (see Chapter 6) were used to provide estimates of the risk of childhood obesity at ages 9-14 years related to additional GWG. The committee chose these three outcomes because they are quantitatively important and their consequences could be estimated with available data. Dr. Hammitt used the literature currently available to calculate quality adjustments for each outcome, which resulted in quality-adjusted life-years (QALY) for comparison across outcomes (information contributed to the committee in consultation with Hammitt [see Appendix G, Part IV]).

In broad terms, the results of this quantitative risk analysis by Dr. Hammitt provided support for the GWG guidelines that the committee developed from published and commissioned research. Although it was possible to develop this quantitative analysis of risk trade-offs, the data needed to support a more complete and persuasive analysis were unavailable. In particular, information is needed on associations between GWG and longer-term maternal outcomes, such as postpartum weight retention and later reproductive function and health as well as child health outcomes, such as fetal growth restriction, child neurocognitive outcomes and obesity. Such data would include not only the frequencies of outcomes but also the utilities associated with each to calculate appropriate quality adjustments.

Overall, these guidelines are remarkably similar to those included in the IOM (1990) report. The research that has appeared since their publication as well as the committee's commissioned analyses support the robustness of the prior recommendations. It remains true that, within a given prepregnancy BMI category, healthy women can deliver healthy infants at a relatively wide range of weight gain values. Unfortunately, an already large and increasing proportion of the population is gaining outside of the prior recommendations (see Chapter 2) and this is also likely to be case with these new guidelines. As a result, it is time to focus attention on helping women to adhere to these guidelines. If research on adherence is conducted with experimental designs of adequate statistical power, such studies could finally provide causal evidence of gaining within these new guidelines results in superior outcomes of pregnancy for both mother and infant.

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FINDINGS AND RECOMMENDATIONS FROM THE COMMITTEE'S ANALYSES

Findings

The committee found that:

1. The WHO cutoff points have been widely adopted for categorizing BMI among non-pregnant adults and should be used for categorizing prepregnancy BMI as well; the committee also found that these categories are also acceptable to use for categorizing the prepregnancy BMI of adolescents.
2. Evidence from the scientific literature is remarkably clear that prepregnant BMI is an independent predictor of many adverse outcomes of pregnancy. As a result, women should enter pregnancy with a BMI in the normal weight category.
3. Although a record-high number of American women of childbearing age have BMI values in obesity classes II and III, available evidence is insufficient to develop more specific recommendations for GWG among these women.
4. There are only limited data available to link GWG to health outcomes of mothers and children that occur after the neonatal period.
5. There is insufficient evidence to continue to support a modification of GWG guidelines for African-American women, women of short stature or adolescents younger than 16 years of age.
6. There is insufficient data with which to establish how much more weight women carrying multiple fetuses should gain beyond that recommended for women carrying singleton fetuses.
7. The committee reaffirms the clinical recommendations in IOM (1990) for implementation of these guidelines.
8. There is insufficient evidence to reject the possibility that racial/ethnic group modifies the association between GWG and important maternal and child health outcomes.

Recommendation for Action

Action Recommendation 7-1: The committee recommends that federal agencies, private voluntary organizations, and medical and public health organizations adopt these new guidelines for GWG and publicize them to their members and also to women of childbearing age.

Recommendation for Research

Research Recommendation 7-1: To permit the development of improved recommendations for GWG in the future, the committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to (a) conduct studies to assess utilities (values) associated with short- and long-term health outcomes associated with GWG for both mother and child and (b) include these values in studies that employ decision analytic frameworks to estimate optimal GWG according to category of maternal prepregnancy BMI and other subgroups.

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Additional Recommendation for Research

Additional Research Recommendation 7-1: The committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to conduct studies among women carrying multiple fetuses that link GWG to relevant health outcomes among both mothers and their infants.

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8

Approaches to Achieving Recommended Gestational Weight Gain

To understand the challenges that may arise in implementing the proposed guidelines on gestational weight gain (GWG) presented in Chapter 7, the committee reviewed the present environment for childbearing (see Chapter 2 for details). The committee also reviewed the interventions that had been conducted to improve GWG in response to the Institute of Medicine (IOM, 1990) guidelines and considered the guidance that these interventions might provide for implementation of these revised guidelines. Although proposing a complete implementation and evaluation plan is beyond the scope of this committee, this chapter provides a framework from which such a plan can be developed.

Current Context for Childbearing and Gestational Weight Gain

Women who are having children today are substantially heavier than at any time in the past (see Chapter 2). Over half of their pregnancies are unwanted or mistimed (IOM, 1995). These facts highlight the difficulties that women face in achieving one of our primary recommendations, namely that they should conceive at a weight within the normal range of BMI values. It is beyond the committee's scope of work to consider how to achieve this objective. Nonetheless, it is important for women to do so and for the government as well as private voluntary organizations to assist them.

The same factors that have caused women of childbearing age to be heavier than in the past challenge them to meet the previous (IOM, 1990) as well and these new guidelines for GWG. Some of the trends that are of concern include an increase in consumption of foods with low nutrient density. This has special implications for pregnancy and lactation, which require modest increases in energy but greater increases in vitamin and mineral intake. In addition, national data (see Chapter 2) indicates that a high proportion of women of childbearing age fail to meet current guidelines for physical activity. Improvement in these statistics could contribute toward helping women enter pregnancy at a healthy weight as well as to meet the proposed guidelines for GWG.

These new guidelines should also be considered in the context of data on women's reported GWG. Compared to data assembled from the studies reviewed by the committee, in which information was available for relatively large samples of women, the mean gains of underweight women are within the new guidelines (Table 8-1). This is less often the case for normal weight women, where the mean gain in some samples is at or above the upper limit of the new guidelines. This indicates that a substantial proportion of normal weight women would exceed desired GWG ranges according to the new guidelines. The mean GWG values for overweight and obese women exceed the upper end of the new guidelines by several kilograms.

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TABLE 8-1 Gestational Weight Gain (kg) by Prepregnant BMI Categories among Large Studies Compared to New Guidelines

		Study				
Prepregnant BMI Category	**New GWG Guidelines**	**Sweden, National (1994-2002)[a]**	**Danish National Birth Cohort (1996-2002)[b]**	**Pregnancy Risk Assessment Monitoring System (2002-03)[c]**	**New York City Vital Statistics Birth Data (1995 to 2003)[d]**	**Pregnancy, Infection, and Nutrition Cohort Study (2001-2005)[e]**
Underweight (< 18.5 kg/m^2)	12.5 - 18.0	13.5 ± 0.03 (SEM) (*n* = 72,361)	15.3 ± 5.1 (SD) (*n* = 2,648)	14.8 ± 0.27 (SEM) (*n* = 1,628)	15.1 ± 5.01 (SD) (*n* = 1,632)	15.4 ± 4.4 (SD) (*n* = 176)
Normal weight (18.5-24.9 kg/m^2)	11.5 - 16.0	13.8 ± 0.01 (SEM) (*n* = 368,063)	15.8 ± 5.2 (SD) (*n* = 41,569)	15.0 ± 0.10 (SEM) (*n* = 11,513)	15.1 ± 5.25 (SD) (*n* = 19,892)	16.6 ± 5.3 (SD) (*n* = 652)
Overweight (25.0-29.9 kg/m^2)	7.0 - 11.5	13.2 ± 0.02 (SEM) (*n* = 153,769)	14.7 ± 6.4 (SD) (*n* = 11,861)	13.9 ± 0.16 (SEM) (*n* = 5,027)	14.1 ± 6.07 (SD) (*n* = 7,893)	15.5 ± 6.2 (SD) (*n* = 126)
Obese (≥ 30 kg/m^2)	5.0 – 9.0	---	10.5 ± 8.3 (SD) (*n* = 4,814)	11.2 ± 0.20 (SEM) (*n* = 4,588)	11.9 ± 6.84 (SD) (*n* = 4,890)	12.0 ± 7.1 (SD) (*n* = 277)
Obese, class I (30-35 kg/m^2)	Not specified	11.1 ± 0.05 (SEM) (*n* = 43,128)	11.4 ± 7.5 (SD) (*n* = 3,541)	---	12.7 ± 6.53 (SD) (*n* = 3,077)	---
Obese, class II (35-40 kg/m^2)	Not specified	8.7 ± 0.11 (SEM) (*n* = 14,713)	7.7 ± 9.4 (SD) (*n* = 1,273)	---	11.1 ± 7.17 (SD) (*n* = 1,166)	---
Obese, class III (≥ 40 kg/m^2)	Not specified	---	---	---	9.5 ± 7.00 (SD) (*n* = 647)	---

[a]Cedergren, 2006 (BMI categories: Underweight = < 20 kg/m^2; Normal weight = 20-24.9 kg/m^2; Obese, Class II = ≥ 35 kg/m^2)
[b]Information contributed to the committee in consultation with Nohr [see Appendix G, Part I]); Obese Class II and III are combined.
[c]P. Dietz, CDC, personal communication January 2009 (states included: AL, AK, FL, ME, NY [excludes NYC], WA, OK, SC, WV)
[d]Information contributed to the committee in consultation with Stein [see Appendix G, Part III]).
[e]Deierlein et al., 2008 (BMI categories: Underweight = < 19.8 kg/m^2; Normal weight = 19.8-26.0 kg/m^2; Overweight = 26.0-29.0 kg/m^2; Obese = > 29.0 kg/m^2)

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If this analysis is restricted to the most recent (2002-2003) multi-state data from PRAMS, the same conclusions are evident (Figure 8-1). These data provide a strong reason to assume that interventions will be needed to assist women, particularly those who are overweight or obese at the time of conception, in meeting the new GWG guidelines. Although the committee recognizes that developing graphical representations to assist caregivers and their clients in conveying the importance of appropriate weight gain during pregnancy is important, the type of expertise represented on the committee as well as the commitment of time and resources limited the extent to which it could develop such material into a format that could be readily disseminated. The review of interventions that were conducted based on IOM (1990) provides a preview of the challenges that will be faced in implementing the new guidelines in this report.

These data provide a strong reason to assume that interventions will be needed to assist women, particularly those who are overweight or obese at the time of conception, in meeting the new GWG guidelines. The review of interventions that were conducted based on *Nutrition During Pregnancy* (IOM, 1990) (see below) provide a preview of the challenges that will be faced in implementing the new guidelines in this report.

Data from observational studies have been consistent in showing an association between gaining within the IOM (1990) guidelines and having a lower risk of adverse outcomes (Carmichael et al., 1997; Abrams et al., 2000; Langford et al., 2008; Olson, 2008), This does not mean, however, that women who gain outside the guidelines will have a bad outcome (Parker and Abrams, 1992). This is because many factors other than GWG are related to the short- and long-term outcomes of pregnancy. Nonetheless, monitoring GWG is useful for identifying women who might benefit from intervention (Parker and Abrams, 1992), and some interventions have been beneficial (see below).

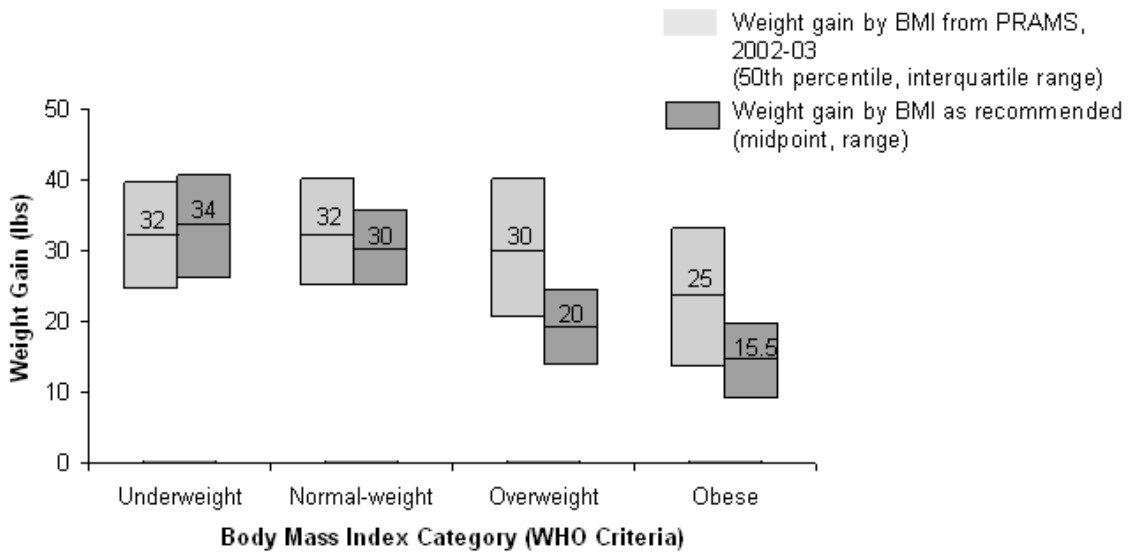

FIGURE 8-1 Comparison of weight gain by BMI category between data reported in the Pregnancy Risk Assessment Monitoring System (PRAMS), 2002-2003, and weight gain as recommended in the new guidelines.

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REVIEW OF INTERVENTION STRATEGIES

The report *Nutrition During Pregnancy* (IOM, 1990) made specific suggestions to improve the utility and success of its guidelines. These included providing guidance on measurement of GWG as well as on counseling of pregnant women. In particular, it was recommended that women and their care providers "set a weight gain goal together" early in pregnancy and that women's progress toward that goal should be monitored regularly. Two additional publications from the Committee on Nutritional Status During Pregnancy and Lactation also provided guidance on how to achieve the weight gain guidelines. *Nutrition Services in Perinatal Care* (IOM, 1992b) called for integrating "basic, patient-centered, individualized nutrition care" into the medical care of every woman beginning before conception and continuing until the end of the breastfeeding period. *Nutrition During Pregnancy and Lactation: An Implementation Guide* (IOM, 1992a) called for a dietary assessment of pregnant women early in gestation with a referral to a dietitian if needed. Such services are not uniformly available today and may not be covered by medical insurance plans. As noted in Chapter 7, the committee endorses these recommendations as they have only become more important as childbearing women have become heavier. The American College of Obstetricians and Gynecologists (ACOG) recently made similar recommendations for nutrition counseling specifically for obese women (ACOG, 2005).

Only limited information is available to determine what advice women have been given about GWG since the publication of the IOM (1990) guidelines. In the two studies that have been conducted (see Chapter 4), both Cogswell et al. (1999) and Stotland et al. (2005) reported that a high proportion of women were either given no advice on how much weight to gain during pregnancy or were advised to gain outside of the recommended range for their prepregnant BMI value. These investigators called for greater effort to educate health care providers about the IOM (1990) guidelines. This issue has also been considered from the physician's perspective. Power et al. (2006) reported that the majority of the 900 obstetrician-gynecologists who responded to a mailed questionnaire used BMI to screen for obesity and counseled their patients about weight control, diet and physical activity. Taken together, these studies suggest that there is a discrepancy between what physicians say they are doing and what women say they are receiving. As a result, there is room for improvement in the process of advising women about GWG.

Status of Interventions to Meet the IOM (1990) Guidelines

The IOM (1990) report called for testing the recommended ranges of GWG against outcomes as well as the effectiveness of specific interventions that are used to improve weight gain. To date, only a limited number of investigators have developed interventions to help women gain within the guidelines (reviewed in Olson, 2008). Few studies are available to provide guidance on helping women gain more weight during pregnancy. In their review, Kramer and Kakuma (2003) found that advice to increase energy and protein intake was successful in achieving those goals but not in increasing GWG. Balanced energy and protein intake were associated with very small (21 g/week) increases in GWG. In contrast, high-protein supplements were not associated with an increase in GWG.

Kramer and Kakuma (2003) also reviewed studies of energy/protein restriction in overweight women or those with high GWG. They found that this approach was associated with reduced weekly weight gain. The most recent studies have been focused on various ways to help women

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to limit their weight gain during pregnancy. None of the four trials that have been conducted in North American populations was completely successful in helping women to limit GWG and adhere to the IOM (1990) guidelines. One study by Gray-Donald et al. (2000) used a pre-post design and included 107 women in the control and 112 women in the intervention groups. This study of Cree women from Quebec showed that the subjects were obese before conception and at high risk of developing gestational diabetes mellitus. They were "offered regular, individual diet counseling, physical activity sessions and other activities related to nutrition." The intervention had only a "minor impact" on the subjects' diets and no effect on GWG, plasma glucose concentration, birth weight, the rate of cesarean delivery or postpartum weight. Olson and her coworkers (2004) also used a pre-post design in their study of normal- and overweight white women from rural community in New York. The intervention included monitoring of weight gain by health care providers and patient education by mail. Overall, there was no difference between the control (n = 381) and intervention (n = 179) groups in GWG or postpartum weight retention at one year. Among the low-income women in the sample, however, those in the intervention group gained less than those in the control group. Polley et al. (2002) randomized 120 normal or overweight women recruited from a hospital clinic that served low-income women to a stepped-care behavioral intervention or usual care. This intervention was successful in reducing the proportion of normal weight but not overweight women who exceeded the IOM (1990) guidelines for GWG. It did not, however, affect weight retention measured at eight weeks postpartum.

In the most recent report, Asbee et al. (2009) randomized women to receive either an organized, consistent program of intensive dietary and lifestyle counseling or routine prenatal care. Among the 100 women who completed the trial, those randomized to the intervention group gained less weight during pregnancy (29 pounds) than those randomized to routine care (36 pounds), but they were not more successful in adhering to the recommended guidelines.

In contrast, women in two of the three studies that have been conducted in Scandinavian populations were successful in reducing GWG. The exception was the pilot study in Finland by Kinnunen et al. (2007), in which primiparous pregnant women were recruited from six public health clinics. Most of these women had a normal prepregnant body mass index (BMI). The 49 women in the intervention group received five individual counseling sessions on diet and leisure-time physical activity; the 56 controls received usual care. Although the intervention improved various aspects of the subjects' diets, it did not prevent excessive GWG. In Sweden, Claesson et al. (2008) offered 160 pregnant women additional visits with a midwife that were designed to motivate them to change their behavior and obtain information relevant to their needs. Those who attended the program were also invited to an aqua aerobic class once or twice a week that was specially designed for obese women. The 208 obese pregnant women in the control group received usual care. Compared to the control group, women in the intervention group gained 2.6 kg less weight during pregnancy and 2.8 kg less between early pregnancy and the postnatal check-up. There were no differences between the groups in type of delivery or infant weight at birth. In Denmark, Wolff et al. (2008) randomized 50 obese pregnant women to receive 10 one-hour dietary consultations that were designed to help them restrict their GWG to 6-7 kilograms or usual care. The women in the intervention group were successful in limiting both their energy intake and their gestational weight gain compared to those in the control group.

The studies in Sweden (Claesson et al., 2008) and Denmark (Wolff et al., 2008) demonstrate that it is possible to motivate obese pregnant women to limit their weight gain during pregnancy to 6-7 kilograms. Achieving this goal required a substantial investment in individual dietary or

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motivational counseling and, in Sweden, also the provision of specially designed aqua aerobics classes. However, some measure of individualized attention was provided in all of the studies—but not all of them were successful. Only normal weight and/or overweight women were enrolled in three of the studies (Polley et al., 2002; Olson et al., 2004; Kinnunen et al., 2007). None of these studies was uniformly successful. Only obese women were enrolled in the other three studies (Gray et al., 2000; Claesson et al., 2008; Wolff et al., 2008); two of these were successful.

The individualized attention that characterized the successful interventions would be expensive to duplicate on a wide scale. However, the significant improvement in serum insulin concentrations seen in the study of obese Danish women by Wolff and her coworkers (2008) might provide adequate justification for this expenditure. It is noteworthy that none of these trials had sufficient statistical power to establish that those whose weight gain stayed within the IOM guidelines or reached the investigators' target had better obstetric outcomes than those who did not. In contrast, there is evidence that these interventions helped some of the subjects reduce postpartum weight retention (Olson, 2004; Kinnunen et al., 2007; Claesson et al., 2008).

For the first time, these new guidelines provide a specific weight-gain range for obese women. This specificity should assist researchers in developing targeted interventions to determine how best to help women to gain within this range as well as to evaluate whether those who do gain appropriately have better short- and long-term outcomes for themselves and their infants than those who do not.

IMPLEMENTATION STRATEGIES

The committee worked from the perspective that the reproductive cycle begins before conception and continues through the first year postpartum. Opportunities to influence maternal weight status are available through the entire cycle. Although it is beyond the scope of this report to consider the evidence associated with timing, duration or strength of specific strategies or interventions, the committee offers a basic framework for possible approaches to the implementation guidelines, with a particular focus on consumer education and strategies to assist practitioners and public health programs. A basic goal of this framework is to help women improve the quality of their dietary intake and increase their physical activity to be able to meet these new guidelines. These behavioral changes will need to be supported by both individualized care and community-level actions to alter the physical and social environments that influence dietary behaviors. A comprehensive review of the evidence associated with such actions, and guidelines for their use, will require future analyses, as was done in the report *Nutrition During Pregnancy and Lactation: An Implementation Guide* (IOM, 1992).

Conceiving at a Normal BMI Value

To meet the recommendations of this report fully, two different challenges must be met. First, a higher proportion of American women should conceive at a weight within the range of normal BMI values. Meeting this first challenge requires preconceptional counseling and, for many women some weight loss. Such counseling may need to include additional contraceptive services (ACOG, 2005) to assist women in planning the timing of their pregnancies. Such counseling also may need to include services directed toward helping women to improve the quality of their diets (Gardiner et al., 2008) and increase their physical activity. This is because only a small proportion of women who are planning a pregnancy—and even fewer of those who

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are not planning a pregnancy but become pregnant nonetheless—comply with recommendations for optimal nutrition and lifestyle (Inskip et al., 2009).

Preconception counseling is an integral part of the recommendations from the Centers for Disease Control and Prevention (CDC) (Johnson et al., 2006) that are designed to enable women to enter pregnancy in optimal health, avoid adverse health outcomes associated with childbearing and reduce disparities in adverse pregnancy outcomes. Practical guidelines for preconceptional care are provided in *Nutrition During Pregnancy and Lactation: An Implementation Guide* (IOM, 1992a). It is noteworthy that few intervention studies have evaluated ways to improve the nutritional choices of women of childbearing age (McFadden and King, 2008), so this is an area in which further investigation is necessary. There is, however, evidence that preconceptional counseling improves women's knowledge about pregnancy-related risk factors as well as their behaviors to mitigate risks (Elsinga et al., 2008). In addition, there is also evidence that pre- and interconceptional counseling will improve attitudes and behavior about nutrition and physical activity in response to a behavioral intervention (Hillemeier et al., 2008). Women with the highest BMI values may even require bariatric surgery to achieve a better weight before conception. Recent systematic reviews suggest women who undergo such surgery have better pregnancy outcomes than women who remain obese (reviewed in Maggard et al., 2008; Guelinckx et al., 2009).

Gaining Weight During Pregnancy Within the New Guidelines

Second, a higher proportion of American women should limit their weight gain during pregnancy to the range specified in these guidelines for their prepregnant BMI. Meeting this second challenge requires a different set of services**.** The first step in assisting women to gain within these guidelines is letting them know that they exist, which will require educating their healthcare providers as well as the women themselves. Government agencies, those who provide healthcare to pregnant women or those who are planning pregnancies as well as private voluntary organizations could provide this education as well as medical societies that have adopted these guidelines as their standard of care.

Women who know about the guidelines and have developed a weight gain goal with their care provider may need additional assistance to achieve their goal. Individualized attention is called for in the IOM (1990) guidelines and was an element in all of the recent interventions that have been successful in helping women to gain within their target range. As noted above, however, not every intervention with individualized attention was successful, so additional services clearly are needed. The IOM report, *Nutrition Services in Perinatal Care* (1992b) calls for "basic, patient-centered individualized nutritional care" to be integrated into the primary care of every woman, beginning before conception and continuing throughout the period of breastfeeding. Guidelines on providing such care are provided in *Nutrition During Pregnancy and Lactation: An Implementation Guide* (IOM, 1992a). The increase in prevalence of obesity that has occurred since 1990 suggests that this recommendation has only become more important.

In offering women individualized attention, a number of kinds of services could be considered. As noted in Chapter 7, health care providers should chart women's weight gain and share the results with them so that they become aware of their progress toward their weight-gain goal. To assist healthcare providers in doing this, the committee has prepared charts (see Figures 8-2 through 8-5) that could be used as a basis for this discussion with the pregnant woman and could also be included in her medical record. These charts reflect the fact that some weight gain

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usually occurs in the first trimester and that weight gain is close to linear in the second and third trimesters (see Chapter 7 for the rates used in preparing these charts). The range around the target line in the second and third trimesters reflects the final width of the target range. In presenting these graphics, the committee emphasizes that graphical formats should be carefully and empirically tested before adoption to insure that the final product effectively communicates the intended messages to women about GWG.

These charts are meant to be used as part of the assessment of the progress of pregnancy and a woman's weight gain, looking beyond the gain from one visit to the next and toward the overall pattern of weight gain. This is because the pattern of GWG, like that of total GWG, is highly variable even among women with good outcomes of pregnancy (Carmichael et al., 1997). Carmichael et al. (1997) have recommended that women should be evaluated for modifiable factors (e.g. lack of money to buy food, stress, infection, medical problems, etc.) that might be causing them to have excessively high or low gains before any corrective action is recommended; the committee endorses this approach.

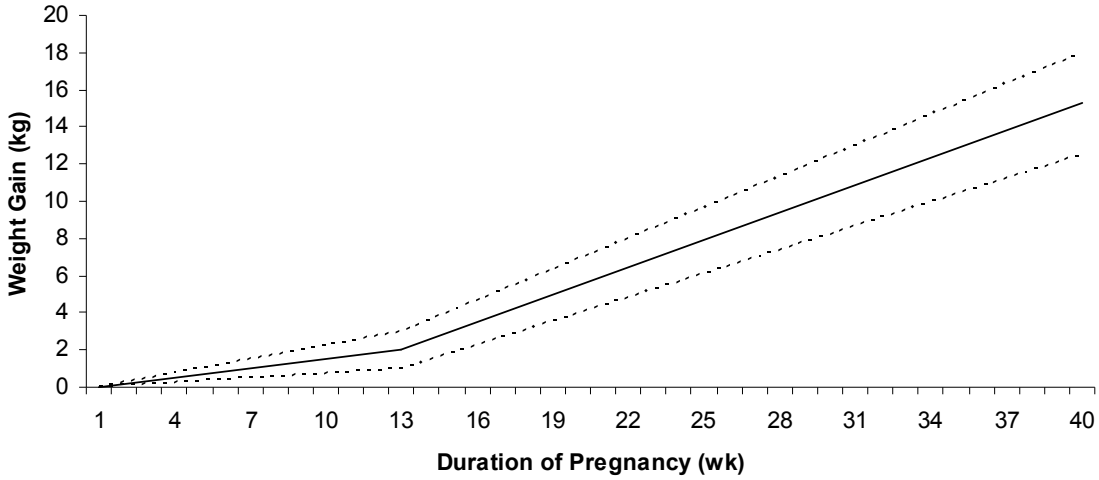

FIGURE 8-2 Recommended weight gain by week of pregnancy for underweight (BMI: < 18.5 kg/m^2) women (dashed lines represent range of weight gain).
NOTE: First trimester gains were determined using three sources (Siega-Riz et al., 1994; Abrams et al., 1995; Carmichael et al., 1997).

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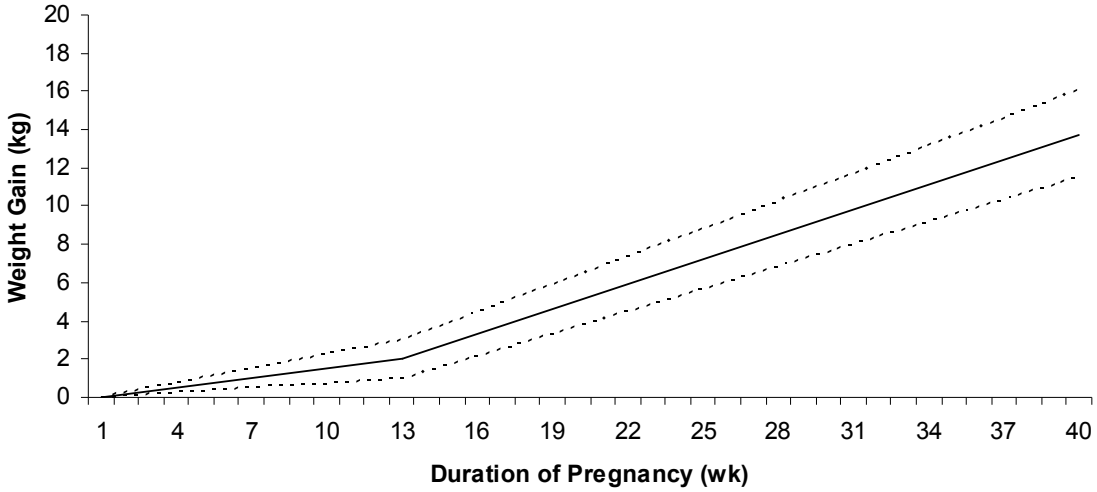

FIGURE 8-3 Recommended weight gain by week of pregnancy for normal weight (BMI: 18.5 - 24.9 kg/m^2) women (dashed lines represent range of weight gain).
NOTE: First trimester gains were determined using three sources (Siega-Riz et al., 1994; Abrams et al., 1995; Carmichael et al., 1997).

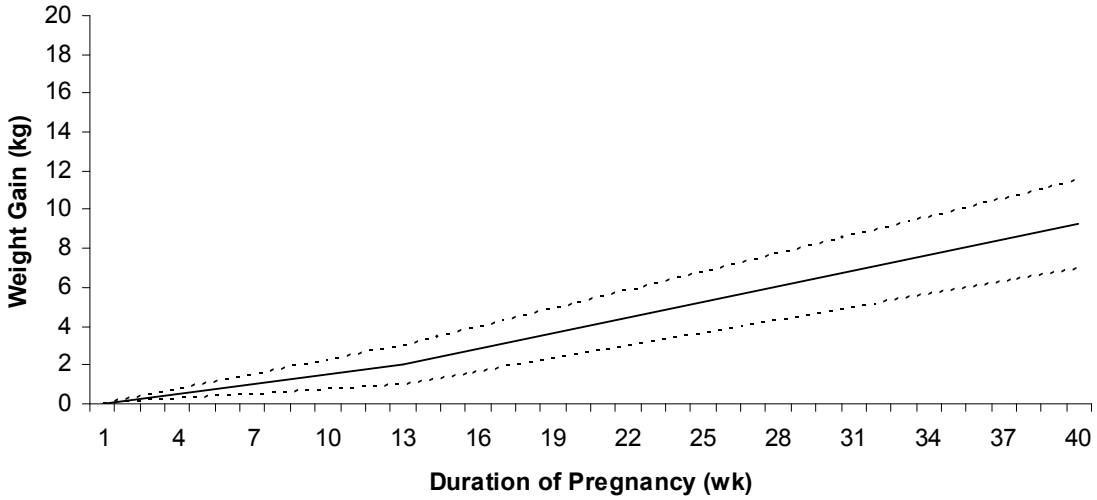

FIGURE 8-4 Recommended weight gain by week of pregnancy for overweight (BMI: 25.0-29.9 kg/m^2) women (dashed lines represent range of weight gain).
NOTE: First trimester gains were determined using three sources (Siega-Riz et al., 1994; Abrams et al., 1995; Carmichael et al., 1997).

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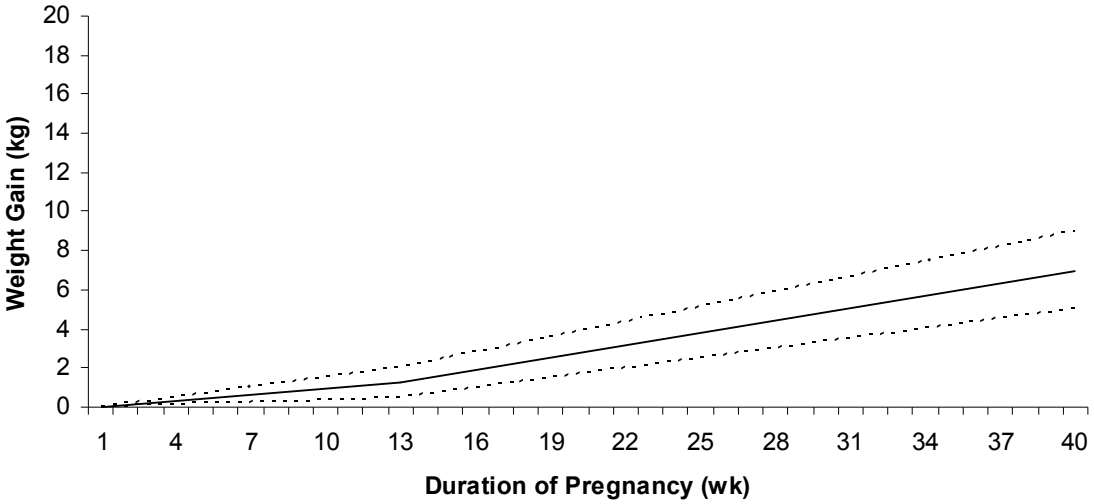

FIGURE 8-5 Recommended weight gain by week of pregnancy for obese (BMI: ≥ 30 kg/m^2) women (dashed lines represent range of weight gain).
NOTE: First trimester gains were determined using three sources (Siega-Riz et al., 1994; Abrams et al., 1995; Carmichael et al., 1997).

In addition, women should be provided with advice about both diet and physical activity (ACOG, 2002). This may require referral to a dietitian as well as other appropriately qualified individuals, such as those who specialize in helping women to increase their physical activity. These services may need to continue into the postpartum period to give women the maximum support to return to their prepregnant weight within the first year and, thus, to have a better chance of returning to a normal BMI value at the time of a subsequent conception.

Individualized nutrition services for pregnant women can be provided by a dietitian, as recommended in *Nutrition Services in Perinatal Care* (IOM, 1992b). Individualized dietary advice is also available for pregnant women on the internet [see, for example, MyPyramid.gov (http://mypyramid.gov/mypyramidmoms/index.html. Accessed February 18, 2009)].

Individualized assessment of physical activity patterns and recommendations for improvement can be provided by a woman's health care provider or by trained practitioners in many health clubs and community-based exercise facilities. General advice on increasing physical activity is available on the internet [see, for example, MyPyramid.gov (http://mypyramid.gov/pyramid/physical_activity_tips.html. Accessed February 18, 2009)] as is advice specifically designed for pregnant women (http://www.acog.org/publications/patient_education/bp045.cfm. Accessed February 18, 2009). According to ACOG (2002), in the absence of either medical or obstetric complications, 30 minutes or more of moderate exercise a day on most, if not all, days of the week is recommended for pregnant women. Participation in a wide range of recreational activities appears to be safe for pregnant women, including pregnant women with diabetes (Kitzmiller et al., 2008). The recent report of the Physical Activity Guidelines Advisory Committee (DHHS, 2008) also offered support for physical activity during pregnancy. Based on the limited number of studies available, this group concluded that "unless there are medical reasons to the contrary, a pregnant woman can begin or continue a regular physical activity program throughout gestation, adjusting the frequency, intensity and time as her condition warrants." This committee added that "in the

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absence of data, it is reasonable for women during pregnancy and the postpartum period to follow the moderate-intensity recommendations set for adults unless specific medical concerns warrant a reduction in activity." However, it is recognized that adequately powered randomized, controlled intervention studies on the potential benefits and risks of regular physical activity at various doses in pregnant women are urgently needed.

Individualized attention is likely to be necessary but not sufficient to enable most women to gain within the new guidelines. For example, pregnant or postpartum women will have difficulty following advice to increase their physical activity by walking unless there is a safe place to walk in their community. Similarly, pregnant or postpartum women will have difficulty following advice to improve the quality of their diets unless healthy foods are available at local markets at prices they can afford. Only limited information is available on the link between community factors and GWG, but it suggests characteristics of neighborhoods influence women's ability to gain weight appropriately during pregnancy (Laraia et al., 2007). The behavior changes that will be required for the majority of pregnant women to gain within the guidelines are difficult and complex. As noted in the report *Promoting Health: Intervention Strategies from Social and Behavioral Sciences* (IOM, 2000), "It is unreasonable to expect that people will change their behavior easily when so many forces in the social, cultural, and physical environment conspire against such change." As a result, these factors must also be addressed if women are to succeed in gaining within these guidelines. For example, hospital-based obstetric programs could link to community facilities with exercise programs for pregnant or postpartum women. The family, and especially the partner, can have a strong influence on maternal behaviors during pregnancy. Yet, at present, their influence on GWG is understudied and underutilized. Further research on these kinds of multilevel, ecological determinants of GWG (see Chapter 4) is needed to guide the development of comprehensive and effective implementation strategies to achieve these guidelines.

Special attention should be given to low-income and minority women, who are at risk of being overweight or obese at the time of conception, consuming diets of lower nutritional value, and of performing less recreational physical activity. The low health literacy levels that characterize this group also represent a major barrier for understanding and acting upon health recommendations (IOM, 2004). The use of culturally appropriate channels and approaches to convey this information at both the individual and population level is essential (Huff and Kline, 1999; Glanz et al., 2002). Approaches considered should range from social marketing (Siegel and Lotenberg, 2007) to improving the cultural skills of the health care providers (Haughton and George, 2008), who will convey the GWG recommendation at an individual level. The community has a particularly important role to play in fostering appropriate GWG in low-income women.

CONCLUDING REMARKS

Although the guidelines developed as part of this committee process are not dramatically different from those published previously (IOM, 1990), fully implementing them would represent a radical change in the care women of childbearing age. In particular, the committee recognizes that full implementation of these guidelines would mean:

- Offering preconceptional services, such as counseling on diet and physical activity as well as access to contraception, to all overweight or obese women to help them reach a

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healthy weight before conceiving. This may reduce their obstetric risk and normalize infant birth weight as well as improve their long-term health.

- Offering services, such as counseling on diet and physical activity, to all pregnant women to help them achieve the guidelines on GWG contained in this report. This may also reduce their obstetric risk, reduce postpartum weight retention, improve their long-term health, normalize infant birth weight and offer an additional tool to help reduce childhood obesity.
- Offering services, such as counseling on diet and physical activity, to all postpartum women. This may help them to eliminate postpartum weight retention and, thus, to be able to conceive again at a healthy weight as well as improve their long-term health.

The increase in overweight and obesity among American women of childbearing age and failure of most pregnant women to gain within the IOM (1990) guidelines alone justify this radical change in care as women clearly require assistance to achieve the recommendations in this report in the current environment. However, the reduction in future health problems among both women and their children that could possibly be achieved by meeting the guidelines in this report provide additional justification for the committee's recommendations.

These new guidelines are based on observational data, which consistently show that women who gained within the IOM (1990) guidelines experienced better outcomes of pregnancy than those who did not (see Chapters 5 and 6). Nonetheless, these new guidelines require validation from experimental studies. To be useful, however, such validation through intervention studies must have adequate statistical power not only to determine if a given intervention helps women to gain within the recommended range but also to determine if doing so improves their outcomes. In the future, it will be important to reexamine the trade-offs between women and their children in pregnancy outcomes related to prepregnancy BMI as well as GWG, and also to be able to estimate the cost-effectiveness of interventions designed to help women meet these recommendations.

FINDING AND RECOMMENDATIONS

Finding

The committee found that:

1. Existing research is inadequate to establish the characteristics of interventions that work reliably to assist women in meeting the 1990 guidelines for GWG or to avoid postpartum weight retention.

Recommendations for Action

Action Recommendation 8-1: The committee recommends that appropriate federal, state and local agencies as well as health care providers inform women of the importance of conceiving at a normal BMI and that all those who provide health care or related services to women of childbearing age include preconceptional counseling in their care.

Action Recommendation 8-2: To assist women to gain within the guidelines, the committee recommends that those who provide prenatal care to women should offer them counseling,

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such as guidance on dietary intake and physical activity, that is tailored to their life circumstances.

Recommendation for Research

Research Recommendation 8-1: The committee recommends that the Department of Health and Human Services provide funding for research to aid providers and communities in assisting women to meet these guidelines, especially low-income and minority women. The committee also recommends that the Department of Health and Human Services provide funding for research to examine the cost-effectiveness (in terms of maternal and offspring outcomes) of interventions designed to assist women in meeting these guidelines.

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9

Open Session and Workshop Agendas

REEXAMINATION OF IOM PREGNANCY WEIGHT GUIDELINES

Institute of Medicine | National Research Council
Food and Nutrition Board
Board on Children, Youth, and Families

The National Academy of Sciences Building
2100 C Street, NW
Washington, DC

January 17, 2008

Open Session Agenda

1:00 p.m. Welcome, Introductions, and Purpose of the Session
Kathleen Rasmussen

1:10 Perspectives from Sponsors:

Michele Lawler, Deputy Director, Division of State and Community Health, Maternal and Child Health Bureau, U.S. Department of Health and Human Services Health Resources and Services Administration

Catherine Spong, Chief, Pregnancy and Perinatology Branch, National Institutes of Health, National Institute of Child Health and Human Development

Michael Katz, Senior Vice President for Research and Global Programs, March of Dimes

Van S. Hubbard, Director, Nutrition Research Coordination National Institutes of Health, Division of Nutrition Research Coordination

Andrea J. Sharma, Lieutenant Commander-USPHS Commissioned Corps Senior Research Scientist Officer-Epidemiologist, Centers for Disease Control and Prevention, Division of Nutrition, Physical Activity, and Obesity

Mary Horlick, Director, Pediatric Obesity Program, National Institutes of Health, Division of Digestive Diseases and Nutrition

Jonelle Rowe, Senior Medical Advisor for Adolescent Women's Health, U.S. Department of Health and Human Services, Office of Women's Health

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Wendy Braund, 11th Luther Terry Fellow and Senior Clinical Advisor, U.S. Department of Health and Human Services, Office of Disease Prevention and Health Promotion

3:10 Break

3:20 Analysis of Data from the Pregnancy Risk Assessment Monitoring System (PRAMS)
Patricia Dietz, Epidemiologist, Centers for Disease Control and Prevention, Division of Reproductive Health

3:40 Update on AHRQ Evidence-Based Review on Outcomes of Maternal Weight Gain
Carmen Kelly, LCDR US Public Health Service, Agency for Healthcare Research and Quality

4:00 Committee Discussion with Sponsors

4:30 Adjourn Open Session

DETERMINANTS OF
GESTATIONAL WEIGHT GAIN AND PREGNANCY OUTCOME

Arnold and Mabel Beckman Center of the National Academies
100 Academy Way
Irvine, CA

March 10, 2008

Open Session Agenda

8:45 am Welcome to Beckman Center and Open Session
Kathleen Rasmussen

9:00 Presentations from Invited Speakers:

Total Weight Gain and Pattern of Weight Gain in Pregnancy
Marie Cedergren, Linkoping University, Sweden

Developmental Programming Determinants of Chronic Disease
Lucilla Poston, King's College, London

Biological Determinants of Gestational Weight Gain
Theresa Scholl, University of Medicine and Dentistry of New Jersey

11:00 Q&A with Committee Members

12 noon Adjourn open session

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IMPLICATIONS OF WEIGHT GAIN FOR PREGNANCY OUTCOMES: ISSUES AND EVIDENCE

The Keck Center of the National Academies
500 Fifth Street, NW
Washington, DC

June 5, 2008

Open Session Agenda

INTRODUCTION

9:00 am Welcome
Kathleen Rasmussen, Sc.D., Chair, Committee to Reexamine IOM Pregnancy Weight Guidelines

SESSION 1: TRENDS IN GESTATIONAL WEIGHT GAIN

9:10 Trends in Distribution of Prepregnancy Body Mass Index
Andrea Sharma, Ph.D., M.P.H., Division of Nutrition, Physical Activity, and Obesity, CDC, Atlanta, GA

9:30 New Analyses from the Pregnancy Risk Assessment Monitoring System
Patricia Dietz, Dr.P.H., M.P.H., Division of Reproductive Health, CDC, Atlanta, GA

9:50 Pregnancy's Effects on Overall and Central Obesity in Women: Influence of Race/Ethnicity
Erica P. Gunderson, Ph.D., Kaiser Permanente, Oakland, CA

10:10 Q&**A**

10:30 BREAK

SESSION 2: DETERMINANTS OF GESTATIONAL WEIGHT GAIN

11:00 Psychosocial and Behavioral Influences on Obesity: Application to Pregnancy
Suzanne Phelan, Ph.D., Brown University

11:20 Biological Determinants: Developmental Origins
Peter Nathanielsz, M.D., Ph.D., University of Texas Health Sciences Center, San Antonio

11:40 Q&A

12:00 noon Break for Lunch

SESSION 3: GESTATIONAL WEIGHT GAIN AND PREGNANCY OUTCOMES

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1:00 pm Gestational Weight Gain: Clinician Survey and Consequences for Mother and Child
Emily Oken, M.D., M.P.H., Harvard University

1:20 Consequences of Gestational Weight Gain: Outcomes for the Mother and Infant
Ellen A. Nøhr, Ph.D., Aarhus University, Denmark

1:40 Disparities in Fetal Growth and Ethnic-Specific Growth Standards
Michael Kramer, M.D., McGill University

2:00 Q&A

2:20 Clinic and Community-Based Intervention Programs: Impact on Gestational Weight Gain
Christine Olson, Ph.D., Cornell University

2:40 Q&A

2:50 BREAK

SESSION 4: PANEL DISCUSSION

3:15 Determinants and Consequences of Gestational Weight Gain: Clinical and Community Perspectives
Moderator:
Esa Davis, M.D., M.P.H., Case Western Reserve University, Case Western Medical Center
Panelists:
Helen Jackson, Ph.D., R.D., L.D./N., Duval County Health Department, Jacksonville, FL
Margie Tate, M.S., R.D., Arizona Department of Health Services, Phoenix
Cheryl Harris, M.P.H., R.D., WIC State Agency, Washington, DC
Deborah Bowers, M.D., Physician and Midwife Collaborative Practice, Alexandria, VA

4:15 Open Discussion

4:45 Adjourn

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Committee Member Biographical Sketches

KATHLEEN M. RASMUSSEN, Sc.D. (*chair*), is professor of nutrition, Division of Nutritional Sciences, at Cornell University. Dr. Rasmussen is internationally known for her research on maternal and child nutrition, particularly in the areas of pregnancy and lactation. She has served as program director for Cornell's National Institutes of Health (NIH) sponsored training grant in maternal and child nutrition since 1986 and has also directed a training grant in international maternal and child nutrition. Dr. Rasmussen has taught a nationally recognized course in maternal and child nutrition for graduate students since 1980 and has co-taught a unique course on public health nutrition for undergraduate students since 1998. Continuing her interest in mentoring the future leaders in nutrition, Dr. Rasmussen serves as the principal faculty member at the Dannon Nutrition Leadership Institute, which she helped to develop in 1998. In 2006, she received the first Excellence in Nutrition Education Award to be given by the American Society for Nutrition. Dr. Rasmussen has served as secretary and then president of the American Society of Nutritional Sciences and also as president of the International Society for Research on Human Milk and Lactation. She has previously been associate dean and secretary of the University Faculty and served a 4-year term on Cornell's Board of Trustees as one of its faculty-elected members. Dr. Rasmussen was a member of the recent DBASSE-IOM (Division of Behavioral and Social Sciences and Education-Institute of Medicine) Committee on the Impact of Pregnancy Weight on Maternal and Child Health and served on the IOM Committee on Nutritional Status During Pregnancy and Lactation and its Subcommittee on Nutrition During Lactation, as well as the Committee on Scientific Evaluation of the WIC (Women, Infants, and Children) Nutrition Risk Criteria. She received her A.B. degree from Brown University in molecular biology and both her Sc.M. and Sc.D. degrees from Harvard University in nutrition.

BARBARA ABRAMS, Dr.P.H., R.D., is professor of epidemiology, maternal and child health, and public health nutrition in the School of Public Health at the University of California, Berkeley. Her expertise includes weight and weight gain in women during pregnancy, post partum, and during menopause; maternal weight, nutrition, social factors, and perinatal health outcomes; and HIV and breastfeeding. She has previously served on the IOM Committee on the Impact of Pregnancy Weight on Maternal and Child Health, the Committee on the Scientific Evaluation of WIC Nutrition Risk Criteria, the Committee on Nutritional Status During Pregnancy and Lactation, and the Subcommittee on Clinical Application Guide. She was awarded the March of Dimes Agnes Higgins Award for her contributions to the field of maternal-fetal nutrition. Dr. Abrams received her B.S. in nutrition and dietetics from Simmons College in Boston. She earned her M.P.H. in nutrition, M.S. in epidemiology, and Dr.P.H in nutrition from the University of California, Berkeley. Dr. Abrams is a member of the American Dietetic Association, the American Society for Nutrition, the Society for Epidemiologic Research, and the Society for Pediatric and Perinatal Epidemiologic Research and an affiliate member of the American College of Obstetrics and Gynecology.

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LISA M. BODNAR, Ph.D., M.P.H., R.D., is assistant professor in the Department of Epidemiology at the University of Pittsburgh Graduate School of Public Health and assistant professor of obstetrics, gynecology, and reproductive sciences at the University of Pittsburgh School of Medicine. Her research interests include nutritional status and birth outcomes, nutritional psychiatry in the perinatal period, the reproductive consequences of obesity, and the use of causal modeling and longitudinal data analysis in reproductive epidemiology. Dr. Bodnar is principal investigator of two NIH grants on nutrition in pregnancy. She recently participated in the 53rd Royal College of Obstetricians and Gynaecologists Work Group on Obesity and Reproductive Health Outcomes in London. Dr. Bodnar graduated with honors from the University of North Carolina, Chapel Hill, where she also received her M.P.H. and Ph.D. in nutritional epidemiology. Dr. Bodnar is a registered dietitian, a member of the American Dietetic Association, and a licensed nutritionist. She also holds membership in the American Society for Nutrition, the Society for Epidemiologic Research, and the Society for Pediatric and Perinatal Epidemiologic Research.

CLAUDE BOUCHARD, Ph.D., is the executive director of the Pennington Biomedical Research Center and the George A. Bray Chair in Nutrition. He holds a B.Ped. (Laval), an M.Sc. (University of Oregon, Eugene) in exercise physiology, and a Ph.D. (University of Texas, Austin) in population genetics. His research deals with the genetics of adaptation to exercise and to nutritional interventions as well as the genetics of obesity and its comorbidities. He has authored and coauthored several books and more than 900 scientific papers. Dr. Bouchard is the recipient of many awards and of an honoris causa doctorate in science from the Katholieke Universiteit Leuven. He has been a foreign member of the Royal Academy of Medicine of Belgium since 1996 and was the Leon Mow Visiting Professor at the International Diabetes Institute in Melbourne in 1998. In 2001, he became a member of the Order of Canada as well as professor emeritus, Faculty of Medicine, Laval University. In 2003 he received the Alumnus of the Year Award from Laval University, and in 2004 he received the Friends of Albert J. Stunkard Award from the North American Association for the Study of Obesity. Dr. Bouchard became a knight in the Ordre National du Quebec in 2005 and also received the Earle W. Crampton Award in Nutrition from McGill University that same year. Dr. Bouchard is past president of the North American Association for the Study of Obesity and the immediate past president of the International Association for the Study of Obesity. Prior to coming to Pennington, he held the Donald B. Brown Research Chair on Obesity at Laval University where he directed the Physical Activity Sciences Laboratory for about 20 years. His research has been funded by agencies in Canada and the United States, primarily the National Institutes of Health.

NANCY BUTTE, Ph.D., M.P.H., is professor of pediatrics at the Children's Nutrition Research Center at Baylor College of Medicine. Her expertise includes energy requirements of infants, children, and women during pregnancy and lactation, as well as the environmental and genetic determinants of childhood obesity, and the contribution of food intake, total energy expenditure, basal metabolic rate (BMR), substrate utilization, physical activity, and fitness to the development of obesity in children. She holds membership in the American Society for Nutrition, the Obesity Society, and the Society of Pediatric Research. Dr. Butte has previously served on the IOM Panel on Dietary Reference Intakes for Macronutrients; the Committee on Body Composition, Nutrition, and Health of Military Women; and the Subcommittee on

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Nutritional Status and Weight Gain During Pregnancy (1988-1990). Dr. Butte received her M.P.H. in public health nutrition and her Ph.D. in nutrition from the University of California, Berkeley.

PATRICK M. CATALANO, M.D., F.A.C.O.G., is professor and chair of the Department of Reproductive Biology at Case Western Reserve University at MetroHealth Medical Center. Dr. Catalano also serves on the Management Council and Executive Committee at MetroHealth Medical Center. He has published more than 130 articles in peer-reviewed journals and served on the editorial boards of the *Journal of Clinical Endocrinology* and *Metabolism and Diabetes*. He holds membership in the American College of Obstetricians and Gynecologists, the American Diabetes Association, the Perinatal Research Society, and the American Gynecological and Obstetrical Society. Dr Catalano is a member of the Maternal-Fetal Medicine Division of the American Board of Obstetrics and Gynecology. Dr. Catalano's research focus is insulin resistance and glucose metabolism in pregnancy and the role of placental cytokines in the regulation of fetal growth and adiposity. He has had research support from the National Institute of Child Health and Human Development (NICHD) for more than 20 years. Dr. Catalano received his M.D. from the University of Vermont, Burlington. He served his internship at the University of California, San Francisco, and residency and postdoctoral fellowship at the University of Vermont, Burlington. Dr. Catalano is certified by the American Board of Obstetrics and Gynecology in maternal and fetal medicine.

MATTHEW W. GILLMAN, M.D., S.M., is professor in the Department of Ambulatory Care and Prevention (DACP) at Harvard Medical School and Harvard Pilgrim Health Care. At the DACP, Dr. Gillman directs the Obesity Prevention Program, whose goal is to lessen obesity-related morbidity and mortality through epidemiologic, health services, and intervention research. Dr. Gillman conducts epidemiologic studies across the age spectrum. He has published widely and has obtained numerous federal and other grants in the areas of developmental origins of health and disease; determinants of dietary and physical activity habits; and interventions to prevent childhood overweight. He is the principal investigator of Project Viva, a prospective cohort study of pregnant women and their children whose goal is to examine pre- and perinatal determinants of offspring health. He is co-principal investigator of the Coordinating Center of the U.S. National Children's Study and a member of the Council of the International Society for Study of the Developmental Origins of Health and Adult Disease. He previously served on the National Research Council-IOM Committee on the Impact of Pregnancy Weight on Maternal and Child Health. Dr. Gillman earned his A.B. and S.M. from Harvard and his M.D. from Duke University. He served a medicine-pediatrics internship and residency at North Carolina Memorial Hospital. Dr. Gillman is a fellow of the American Academy of Pediatrics, American College of Physicians, and the American Heart Association Council on Epidemiology and Prevention.

FERNANDO A. GUERRA, M.D., M.P.H., is director of health for the San Antonio Metropolitan Health District. He is a member of the Institute of Medicine. Dr. Guerra's career reflects a long-standing interest and involvement in pediatric care, public health, and health policy. His expertise is improving access to healthcare systems for infants, women, children, and the elderly and improving access to health care for migrant children. He is also active with local, national, and international forums on a variety of health issues. Dr. Guerra has served on the

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Committee on Ethical Issues in Housing-Related Health Hazard Research Involving Children; the Frontiers of Research on Children, Youth, and Families Steering Committee; the Committee on Using Performance Monitoring to Improve Community Health; and the Committee on Overcoming Barriers to Immunization. He is a former member of the Board on Children, Youth, and Families and has participated as a member of the Roundtable on Head Start Research. Dr. Guerra is recipient of the James Peavey Award from the Texas Public Health Association and the Job Lewis Smith Award from the American Academy of Pediatrics; he is a Kellogg fellow of the Harvard School of Public Health, among many other awards and honors. Dr. Guerra holds a B.A. from the University of Texas at Austin, an M.P.H. from the Harvard School of Public Health, and an M.D. from the University of Texas Medical Branch at Galveston.

PAULA A. JOHNSON, M.D., M.P.H., is executive director of the Connors Center for Women's Health at Brigham and Women's Hospital. Her expertise is in disparities in health care for women and minorities and public health efforts to address affordable and healthy foods for low-income populations. Dr. Johnson has been an active participant in the Disparities Project, an effort to eliminate racial and ethnic inequalities in health in Boston. She is also a leader in public health efforts to address the issue of affordable, healthy food for low-income residents of the city. Her efforts contributed to a major policy conference on Food in the Hub, which provided a set of recommendations regarding food and nutrition policies in Boston. She also has a clinical interest in cardiovascular disease in women, congestive heart failure, and heart transplantation. Dr. Johnson was named to serve as public health commissioner of Boston in 2007. Dr. Johnson received her M.D. and M.P.H. from Harvard Medical School. She served her internship and residency in internal medicine at Brigham and Women's Hospital. She also served a postdoctoral fellowship in cardiology at Brigham and Women's Hospital. Dr. Johnson is board certified in internal medicine and cardiovascular disease.

MICHAEL C. LU, M.D., M.P.H., M.S., is associate professor in the Department of Obstetrics and Gynecology at the University of California, Los Angeles (UCLA), School of Medicine and the Department of Community Health Sciences at UCLA School of Public Health. His research focuses on racial-ethnic disparities in birth outcomes from a life course perspective. Dr. Lu is widely recognized for his research, teaching, and clinical care. Dr. Lu received the 2003 National Maternal and Child Health Epidemiology Young Professional Award and the 2004 American Public Health Association Maternal and Child Health Young Professional Award for his research on health disparities. Dr. Lu has previously served on the IOM Committee on Understanding Premature Birth and Assuring Health Outcomes. He has also received numerous awards for his teaching, including excellence in teaching awards from the Association of Professors of Gynecology and Obstetrics. Dr. Lu also maintains an active clinical practice in obstetrics and gynecology at UCLA Medical Center and has been selected as one of the best doctors in America since 2005. Dr. Lu received a B.A. in human biology and political science from Stanford University, an M.S. in health and medical sciences, an M.P.H. in epidemiology from the University of California, Berkeley, and an M.D. from the University of California, San Francisco, School of Medicine.

ELIZABETH R. MCANARNEY, M.D., is professor and chair emerita of the Department of Pediatrics at the University of Rochester School of Medicine and Dentistry, having served as chair for 13 years. Dr. McAnarney is a member of the IOM. In addition, she has served as the

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president of the Society for Adolescent Medicine, the Association of Medical School Pediatric Department Chairs, and the American Pediatric Society. Dr. McAnarney is interested in the role of nutrition and gestational weight gain as risk factors for adolescent postpartum weight retention. She also has studied the etiology of obesity and age-related differences in the composition of the weight gained by pregnant adolescents and optimal nutrition for pregnant adolescents. Dr. McAnarney served as director of the Rochester Adolescent Maternity Program (RAMP) and the university's Division of Adolescent Medicine for 22 years, prior to becoming chair. She is recipient of the 18th annual Athena Award from the Women's Council of the Rochester Business Alliance, in recognition for her career accomplishments and her role in mentoring women. Dr. McAnarney is a graduate of Vassar College and received her M.D. and an honorary D.Sc. from the State University of New York Upstate Medical Center, Syracuse, and served a postdoctoral fellowship at the University of Rochester.

RAFAEL PEREZ-ESCAMILLA, Ph.D., is professor of nutritional sciences and public health and director of the Connecticut NIH EXPORT Center for Eliminating Health Disparities among Latinos at the University of Connecticut. Dr. Pérez-Escamilla is an internationally recognized scholar in community nutrition. His research includes studies on health disparities and inequalities, maternal nutrition during pregnancy, household food insecurity measurement, nutrition and food safety education, and domestic and international community nutrition program design and evaluation. He is nutrition extension scientist for the State of Connecticut and holds a joint appointment with the Department of Community Medicine and Health Care at the University of Connecticut Health Center in Farmington. Dr. Pérez-Escamilla is leading or co-leading four nutrition capacity building and translational research programs in Connecticut, Ghana, and Brazil in the fields of nutrition-related health disparities, breastfeeding, maternal HIV, and household food security. He is a member of the 2010 Dietary Guidelines Advisory Committee. He is past chair of the Community Nutrition and Public Health Research Interest Section of the American Society for Nutrition and serves on the editorial boards of the *Journal of Human Lactation* and the *Journal of Hunger and Environmental Nutrition*. Dr. Pérez-Escamilla received a B.S. in chemical engineering from the Universidad Iberoamericana in Mexico City, Mexico. He earned an M.S. in food science and a Ph.D. in nutrition from the University of California, Davis.

DAVID SAVITZ, Ph.D., is Charles W. Bluhdorn Professor of Community and Preventive Medicine and director of the Disease Prevention and Public Health Institute at Mount Sinai School of Medicine. He was assistant professor in the Department of Preventive Medicine and Biometrics at the University of Colorado School of Medicine and moved to the University of North Carolina School of Public Health in 1985. He served as the Carey C. Boshamer Distinguished Professor and Chair of the Department of Epidemiology until the end of 2005. His teaching is focused on epidemiologic methods, and he recently authored a book entitled *Interpreting Epidemiologic Evidence*. He directed 29 doctoral dissertations at the University of North Carolina and 13 master's theses. He has served as editor at the *American Journal of Epidemiology* and as a member of the NIH Epidemiology and Disease Control-1 study section and currently is an editor at *Epidemiology*. He was president of the Society for Epidemiologic Research and the Society for Pediatric and Perinatal Epidemiologic Research and North American Regional councilor for the International Epidemiological Association. His primary research activities and interests are in reproductive, environmental, and cancer epidemiology. Dr.

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Savitz received his undergraduate training in psychology at Brandeis University, a master's degree in preventive medicine at Ohio State University in 1978, and his Ph.D. in epidemiology from the University of Pittsburgh Graduate School of Public Health in 1982. He was elected to membership in the Institute of Medicine in 2007.

ANNA MARIA SIEGA-RIZ, Ph.D., is associate professor in the Department of Epidemiology with a joint appointment in the Department of Nutrition in the School of Public Health at the University of North Carolina (UNC), Chapel Hill. Dr. Siega-Riz is a fellow at the Carolina Population Center and serves as the associate chair of epidemiology and director of the Nutrition Epidemiology Core for the Clinical Nutrition Research Center in the Department of Nutrition at UNC. She is also the program leader for the Reproductive, Perinatal and Pediatric Program in the Department of Epidemiology. She has expertise in gestational weight gain, maternal nutritional status and its effects on birth outcomes, obesity development, and trends and intakes among children and Hispanic populations. Dr. Siega-Riz uses a multidisciplinary team perspective as a way to address complex problems such as prematurity, fetal programming, and racial disparities and outcomes. She received the March of Dimes Agnes Higgins Award for Maternal and Fetal Nutrition in 2007, which recognizes professional contributions and outstanding service in the area of maternal and fetal nutrition. Dr. Siega-Riz earned a B.S.P.H. in nutrition from the School of Public Health at UNC, Chapel Hill; an M.S. in food, nutrition, and food service management from UNC, Greensboro; and a Ph.D. in nutrition and epidemiology from the School of Public Health at UNC, Chapel Hill.

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Appendix A

Glossary[1] and Supplemental Information

ACRONYMS AND ABBREVIATIONS

ACTH	Adrenocorticotrophic hormone
ADHD	Attention deficit hyperactivity disorder
ADRB3	Beta 3 adrenergic receptor gene
aFIt1	Fms-like tyrosine kinase 1
AHRQ	Agency for Healthcare Research and Quality
ALL	Acute lymphocytic leukemia
AML	Acute myeloid leukemia
BIA	Bioimpedance analysis
BMI	Body mass index
BMR	Basal metabolic rate
BRFSS	Behavioral Risk Factor Surveillance System
CBG	Corticosteroid-binding globulin
CDC	Centers for Disease Control and Prevention
CI	Confidence interval
CVD	Cardiovascular disease
DDE	Dichlorodiphenyl dichloroehtylene
DHEAS	Dehydroepiandrosterone sulfate
DHHS	U.S. Department of Health and Human Services
DNA	Deoxyribonucleic acid
DOHaD	Developmental origins of health and disease
DXA	Dual energy X-ray absorptiometry
EE	Energy expenditure
ECF	Extracellular fluid
FFA	Free fatty acid

[1] The following sources were used: Thomas C. L., Ed. 1985. Taber's Cyclopedic Medical Dictionary. Philadelphia, PA, F.A. David Company; The American Heritage Stedman's Medical Dictionary. 1995. Boston, MA. Houghton Mifflin Company. Merriam-Webster's Medical Dictionary Online (available at http://medical.merriam-webster.com/medical). Dorland's Medical Dictionary 29th Ed. Medline Plus (available online at http://www.nlm.nih.gov/medlineplus). USDA Dietary Guidelines for Americans 2005 (available at http://www.health.gov/dietaryguidelines/dga2005/document/html/appendixC.htm). Mann and Truswell 2nd Ed 2002. Essentials of Human Nutrition, Oxford University Press, Oxford, UK.

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FFM	Fat-free mass
FM	Fat mass
GDM	Gestational diabetes mellitus
GFR	Glomerular filtration rate
GHQ	General health questionnaire
GWG	Gestational weight gain
HHANES	Hispanic Health and Nutrition Examination Survey
HCS	Human chorionic somatomammotropin
HDL	High density lipoprotein
HPL	Human placental lactogen
ICW	Intracellular water
IGF-I	Insulin-like growth factor I
IGT	Impaired glucose tolerance
IOM	Institute of Medicine
IR-β	Insulin receptor-beta
IRS-1	Insulin receptor substrate 1
IUGR	Intrauterine growth restriction
LBW	Low birth weight
LDL	Low density lipoprotein
LGA	Large-for-gestational-age
LVM	Left ventricular mass
mEQ	Millequivalent
MLL	Mixed lineage leukemia
mRNA	Messenger ribonucleic acid
MUAC	Mid-upper arm circumference
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NIDDM	Non-insulin-dependent diabetes mellitus
NMIHS	National Maternal and Infant Health Survey
NPRQ	Non-protein respiratory quotient
NRC	National Research Council
OR	Odds ratio
P_{AO2}	Partial pressure of oxygen
P_{ACO2}	Partial pressure of carbon dioxide
PAL	Physical activity level
PCB	Polychlorinated biphenyls
PlGF	Placental growth factor

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PPARG2	Peroxisome proliferator-activated receptor gamma 2
PRAMS	Pregnancy Risk Assessment Monitoring System
PSS	Perceived stress scale
PTB	Preterm birth
RBC	Red blood cell
RQ	Respiratory quotients
SD	Standard deviation
SGA	Small-for-gestational-age
SHGB	Sex hormone binding globulin
SNP	Single Nucleotide Polymorphism
SPAWN	Stockholm Pregnancy and Women's Nutrition
SSRI	Selective serotonin reuptake inhibitors
STAI	State Trait Anxiety Inventories
TBK	Total body potassium
TBN	Total body nitrogen
TBW	Total body water
TEE	Total energy expenditure
TNF-α	Tumor necrosis factor alpha
TOBEC	Total body electrical conductivity
VEGF	Vascular endothelial growth factor
VLBW	Very low birth weight
VLDL	Very low density lipoprotein
VO_2	Oxygen consumption
WIC	Special Supplemental Nutrition Program for Women, Infants, and Children
YRBS	National Youth Risk Behavior Survey
±	plus or minus

GLOSSARY

Abruptio placentae
Also called ablatio placentae. Premature detachment of the placenta from the wall of the uterus.

Acetylation
The introduction of one or more acetyl groups into an organic compound.

Adenylate cyclase
An enzyme that catalyzes the synthesis of adenosine 3', 5'-cyclic monophosphate (cyclic AMP) from adenosine triphosphate (ATP)

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Adiponectin
Also called adipocyte complement-related protein of 30 kDa (Acrp30). Protein produced in adipose (fat) tissue that accentuates sensitivity to insulin and is involved in the body's regulation of weight. Low levels of adiponectin are found when obesity and its associated health complications are present. *See also* Adipokines.

Adipokines
Also called adipose cytokines. A variety of proteins released into the systemic circulation by adipose (fat) tissue in response to changes in the metabolic status. Dysregulation of adipokine secretion (either abnormally increased or decreased levels) may be one of the mechanisms by which insulin resistance is tied to obesity. Adipokines implicated in insulin resistance include leptin, resistin, and adiponectin.

Adrenocorticotrophic hormone (ACTH)
A hormone that is produced by the anterior lobe of the pituitary gland and that stimulates the secretion of cortisone, aldosterone, and other hormones by the adrenal complex.

Aldosterone
A steroid hormone secreted by the adrenal cortex that functions in the regulation of the salt and water balance (metabolism of sodium, chloride and potassium) in the body.

Allele
A set of alternate forms of a gene that may occur at a given locus.

Amnion
Also called amniotic sac. The thin membrane forming a closed sac about the embryo/fetus and containing the amniotic fluid.

Amniotic fluid
Liquid contained in the amnion that protects the fetus from injury, helps maintain an even temperature, prevents formation of adhesions between the amnion and the skin of the fetus, and prevents conformity of the sac to the fetus.

Anemia
A condition in which red blood cells, hemoglobin, or total volume content of the blood is less than that required to provide the oxygen demands of the body.

Angiotensin
A vasopressor (increases blood pressure by exerting a vasoconstrictor effect) protein that is formed in the body by interaction of chymosin and serum globulin fraction. The synthetic amide derivative of its physiologically active form, angiotensin II, is used to treat some forms of hypotension.

Angiotensinogen
Also called hypertensinogen. A serum globulin formed by the liver that is cleaved by renin to produce angiotensin I, the physiologically inactive form of angiotensin.

Anion
A negatively charged ion.

Anorexia nervosa
A psychophysiological disorder usually occurring in teenage women that is characterized by fear of becoming obese, a distorted self-image, a persistent aversion to food, and severe weight loss, and that is often marked by hyperactivity, self-induced vomiting, amenorrhea, and other physiological changes.

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Antipyrine
Also called phenazone. An analgesic (pain reducer) and antipyretic (fever reducer) that was formerly widely used, but is now largely replaced in oral use by less toxic drugs such as aspirin.

Attention deficit hyperactivity disorder (ADHD)
A childhood syndrome that is characterized by impulsiveness and short attention span and sometimes by hyperactivity, and that often leads to learning disabilities and various behavioral problems.

Basal Energy Expenditure (BEE)
The amount of energy expended while resting and fasting, extrapolated to 24 hrs that is expressed as kcal/24h. It is used to determine an individual's caloric needs.

Basal metabolic rate (BMR)
The rate of energy expenditure that occurs in the post-absorptive state, defined as the particular condition that prevails after an overnight fast (the subject not having consumed food for 12-14 hours) and resting comfortably, supine, awake, and motionless in a thermoneutral environment. This standardized metabolic state corresponds to the situation in which food and physical activity have minimal influence on metabolism.

Bioimpedance analysis (BIA)
Also called Bioelectrical Impedance Analysis. Method of body composition measurement by which a weak electric current is applied to the subject's wrist and ankle through electrodes. Several prediction equations are then used to calculate lean weight and density, total body water and ^{40}K.

Body mass index (BMI)
Also called Quetelet index. An expression of body weight-for-height used for children and adults, using the formula weight/height^2 x 100. In this report, metric units are used, namely: BMI= kg/m^2 x 100

Bulimia
A chronic eating disorder involving repeated and secretive episodes of eating, characterized by uncontrolled rapid ingestion of large quantities of food over a short period of time, followed by self-induced vomiting, purging and anorexia and accompanied by feelings of guilt, depressions, or self-disgust.

Case-control study
Also called a retrospective study or case referent study. An epidemiological and observational study in which persons are selected because they have a specific disease or other outcome (cases) and are compared to a control (referent comparison) group without the disease to evaluate whether there is a difference in the frequency of exposure to possible disease risk factors.

Congenital anomalies
Birth defects.

Consequences
Health outcomes (effects) caused by the determinants.

Corticosteroids
Any number of hormonal steroid substances obtained from the cortex of the adrenal gland.

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Cortisol
Also called hydrocortisone. A hormone produced by the adrenal cortex upon stimulation by ACTH that mediates various metabolic processes such as gluconeogenesis (formation of glucose from precursors other than carbohydrates), and has anti-inflammatory and immunosupressive properties. Cortisol levels in the blood may become elevated in response to physical or psychological stress.

Creatinine
One of the non-protein constituents of blood, a breakdown product of creatine (protein used to make ATP). Increased quantities of serum creatinine are found in advanced stages of renal disease.

Decidua
The mucous membrane lining the uterus modified during pregnancy, and cast off at parturition or during menstruation. The human decidua is made up of a part lining the uterus (parietalis), a part enveloping the embryo (capsularis), and a part participating with the chorion in the formation of the placenta (basalis).

Dehydroepiandrosterone sulfate (DHEAS)
A weak androgen (male hormone) produced by the adrenal cortex in both men and women that is measured in women showing symptoms of virulism (male body characteristics) or hirsutism (excessive hair growth). It is also measured in children who are maturing too early (precocious puberty).

Deoxycorticosterone
A steroid hormone from the adrenal gland that acts principally on salt and water metabolism.

Deoxyribonucleic acid (DNA)
A nucleic acid that consists of long chains of nucleotides twisted together into a double helix and joined by hydrogen bonds between complementary bases adenine and thymine or cytosine and guanine. DNA carries the cell's genetic information and heredity characteristics via its nucleotides and their sequence, and is capable of self-replication and RNA synthesis.

Determinants
Causal (etiologic) factors.

Deuterium
An isotope of hydrogen with one proton and one neutron in the nucleus.

Deuterium
An isotope of hydrogen with one proton and one neutron in the nucleus having a heavy atomic weight (2.014).

Doxylamine
An antihistamine derived from pyridine and usually administered in the form of its succinate, occurring as a white or creamy powder, that reduces the natural chemical histamine in the body, and that is sometimes combined with other medicines to treat sneezing, itching, watery eyes, and runny nose caused by allergies or the common cold.

Dyspnea
Difficulty in breathing, often associated with lung or heart disease, and resulting in shortness of breath.

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Edema
Also called dropsy, oedema. A local or generalized condition in which the body tissues contain an excessive amount of tissue fluid.

Effect modifier
A factor that increases or decreases the magnitude of the effect of a determinant on a particular consequence.

Embryo34+
The developing organism from 1 week after conception to the end of the second month of gestation.

Energy Expenditure (EE)
Calories used to support the body's basal metabolic needs plus those used for thermogenesis, growth, and physical activity.

Epidemiology
The study of the distribution and determinants of health-related states and events in populations and the control of health problems.

Epigenetic
Mechanisms, processes, and/or biological compounds that affect a cell, organ, or individual without changing or perturbing DNA.

Estrogen
Female sex hormones responsible for the development of secondary sexual characteristics and for cyclic changes in the vaginal epithelium and endothelium of the uterus.

Etiology
Cause and origin of a diseases.

Evans blue
A water-soluble dye that upon injection into the bloodstream combines with serum albumin, and is used to determine blood volume colorimetrically. In pregnancy, evans blue is used to measure plasma volume expansion.

Experimental trials
A type of study in which human or animal exposure to a substance occurs in a controlled environment for the purpose of studying its effects. In humans, experimental trials are only ethical when there is equipoise between the two arms of the trial.

Extracellular fluid (ECF)
Fluid outside of the cell.

Fat-free mass (FFM)
Component of total body mass that includes skeletal muscle, nonskeletal muscle and soft lean tissues, and the skeleton.

Fat mass (FM)
Adipose tissue mass in the body.

Fetus
The developing organism in the human uterus after the second month of gestation.

Fluoxetine
An antidepressant drug that is administered in the form of its hydrochloride $C_{17}H_{18}F_3NO{\cdot}HCl$ and enhances serotonin activity.

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Food insecurity
Whenever the availability of nutritionally adequate and safe food or the ability to acquire acceptable foods in socially acceptable ways is limited or uncertain.

Free fatty acid (FFA)
An uncombined fatty acid.

Genotype
Genetic characteristics of an individual determined by a set of alleles that make up the genome.

Gestational Diabetes Mellitus (GDM)
Metabolic derangement in glucose metabolism and profound abnormalities in the metabolism of fat, protein, and other substances; characterized by hyperglycemia and glycosuria and resulting from inadequate production or utilization of insulin. The body's need for insulin increases dramatically throughout pregnancy, making GDM the most common medical disorder affecting pregnancy.

Gestational weight gain (GWG)
The amount of weight a pregnant woman gains between the time of conception and the onset of labor.

Globulin
One of a group of proteins insoluble in pure water but soluble in neutral solutions of salts of strong acids.

Glomerular filtration rate (GFR)
The volume of water filtered out of the plasma through glomerular capillary walls into Bowman's capsules per unit of time **significance?

Glucagon
A polypeptide hormone that has the property of increasing the concentration of glucose in the blood

G-protein
Any of a class of GTP (energy-rich nucleotide analogous to ATP)-binding proteins that undergo GTP hydrolysis to activate signal transduction pathways in response to extracellular signals such as growth factor stimulation.

Heterozygous
Two different alleles, each at the same position on homologous chromosomes.

High density lipoprotein (HDL)
A complex of lipids and proteins that functions as a transporter of cholesterol in the blood and which, in high concentrations, is associated with a decreased risk of atherosclerosis and coronary heart disease.

Histone
Any of several small simple proteins that are most commonly found in association with DNA in chromatin and are rich in the basic amino acids lysine and arginine.

Homozygous
Two identical alleles, each at the same position on homologous chromosomes.

Human chorionic somatomammotropin (HCS)
See HPL

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Human placental lactogen (HPL)
Also called human chorionic somatomammotropin (HCS). A hormone that is isolated from human placentas, that has a biological activity that weakly mimics the activity of somatotropin and prolactin, and that is secreted into maternal circulation.
Also Hormone produced by the placenta that breaks down fats from the mother to provide fuel for the growing embryo/fetus. It can lead to insulin resistance and carbohydrate intolerance in the mother. A test can be done to measure the amount of hPL in the blood to show how well the placenta is functioning.

Hydrodensitometry
Method of body composition measurement applying Archimedes' principle by submerging subject in water **explain the whole process?

Hyperemesis gravidarum
Severe and prolonged vomiting during pregnancy.

Hyperinsulinemia
Also spelled hyperinsulinaemia. The presence of excess insulin in the blood.

Hypertension
Abnormally high arterial blood pressure that is usually indicated by an adult systolic blood pressure of 140 mm Hg or greater or a diastolic blood pressure of 90 mm Hg or greater, is chiefly of unknown cause but may be attributable to a preexisting condition (such as a renal or endocrine disorder), that typically results in a thickening and inelasticity of arterial walls and hypertrophy of the left heart ventricle, and that is a risk factor for various pathological conditions or events (such as heart attack, heart failure, stroke, end-stage renal disease, or retinal hemorrhage).

Hypertriglyceridemia
An elevated triglyceride concentration in the blood.

Hypoxia
Insufficient levels of oxygen in blood or tissue.

Intracellular water (ICW)
The water within the tissue cells.

Intrauterine growth restriction (IUGR)
Also called intrauterine growth retardation. A condition resulting in a fetal weight less than the tenth percentile of predicted weight for gestational age, in which there is a pathological process present that prevents expression of normal growth potential.

In utero
In the uterus.

Ketone
Any of a class of organic compounds having a carbonyl group linked to a carbon atom in each of two hyrdrocarbon radicals.

Ketonemia
Also called hyperketonemia. A condition marked by an abnormal increase of ketone bodies in the circulating blood.

Large-for-gestational age (LGA)
Usually defined as birth weight above the 90th percentile for gestational age, based on a given reference population.

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Lipolysis
The decomposition of lipids by a reaction with water.

Low birth weight (LBW)
Infant birth weight less than 2,500 grams.

Low density lipoprotein (LDL)
A complex of lipids and proteins that functions as a transporter of cholesterol in the blood, and which, in high concentrations, is associated with an increased risk of atherosclerosis and coronary heart disease.

Macrosomia
Abnormally large size of the body; in this report it is defined as an infant being born at a weight larger than 4,500 g.

Menarche
Initiation of menstruation.

Metabolic syndrome
Also called insulin resistance syndrome, Metabolic Syndrome X. A group of conditions that increase risk of heart disease, diabetes, and stroke. The five conditions are: high blood pressure, high blood sugar levels, high levels of circulating triglycerides, low levels of circulating HDL, and excess fat in the abdominal area.

Methylation
One of the primary mechanisms of regulating gene expression; hyper- or hypo-methylation of a gene promoter region enhances or suppresses gene expression.

Monozygotic twins
Originating from a single fertilized ovum, applied to identical twins.

Multiple pregnancy
Carrying more than one fetus, e.g., twins or triplets.

Net weight gain
Total gestational weight gain minus the infant's birth weight.

Non-protein respiratory quotient (NPRQ)
The component of the respiratory quotient (ratio of oxygen consumption to carbon dioxide production; reflects substrate utilization) not associated with protein oxidation. When NPRQ is greater than 1, net fat synthesis occurs, and carbohydrate is used both for storage and for oxidation.

Nullipara
A woman who has never given birth

Obesity
Increased body weight caused by excessive accumulation of fat.

Observational studies
Study types that follow a population (either prospectively or retrospectively) to examine how exposure to risk factors influences one's probability of developing a disease in the absence of intervention; includes cross-sectional studies, cohort studies, and case-control studies.

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Odds Ratio (OR)
In a case-control study (see above), the exposure odds among cases compared to the exposure odds among controls, where the exposure odds are the number of individuals with the exposure relative to the number of individuals without the exposure (e.g., if 3 out of 10 people are exposed, then the exposure odds are 3:7).

Osmolarity
The osmotic concentration of a solution expressed as osmoles of solute per liter of solution.

Parity
The number of children previously born to a woman.

Phenothiazine
Also called *thiodiphenylamine*. A greenish yellow crystalline compound used as an anthelmintic and insecticide. Various phenothiazine derivatives (such as chlorpromazine) are used as tranquilizing agents, especially in the treatment of schizophrenia.

Phenotype
Physical, biochemical, and physiologic makeup of an individual; determined by genetic and environmental factors.

Physical Activity Level (PAL)
(as an energy component) the ratio of total energy expenditure (TEE) to basal daily energy expenditure (BEE).

Placenta
The membranous vascular organ in female mammals that permits metabolic interchange between fetus and mother. It develops during pregnancy from the chorion of the embryo and the decidua basalis of the maternal uterus, and permits the absorption of oxygen and nutritive materials into the fetal blood and the release of carbon dioxide and nitrogenous waste from it, without the direct mixing of maternal and fetal blood.

Plasma volume
Measure of volume of plasma in the blood.

Postpartum
Of or occurring in the period shortly after childbirth.

Postterm birth
Birth occurring after a gestation of 42 or more weeks.

Preconceptional period
A period from 1 to 3 months prior to gestation through the first 6 weeks of gestation.

Preeclampsia
A toxic condition developing in late pregnancy characterized by a sudden rise in blood pressure, generalized edema, proteinuria, severe headache, and visual disturbances that may result in eclampsia (convulsive or coma state) if untreated.

Pregnancy-induced Hypertension
Encompasses isolated non-proteinuric hypertension, pre-eclampsia or proteinuric, hypertension, and eclampsia; PIH occurs in 5-15 percent of pregnancies, and is a major cause of obstetric and perinatal morbidity and mortality.

Pregravid
Preceding pregnancy.

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Preterm birth
Birth occurring after a gestation of less than 37 weeks.

Prenatal
Preceding birth.

Progesterone
A steroid hormone secreted by the corpus luteum and by the placenta that acts to prepare the uterus for implantation of the fertilized ovum, to maintain pregnancy, and to promote the development of the mammary glands.

Prolactin (PRL)
Also called luteotropic hormone; luteotropin; mammotropin. A pituitary hormone that induces and maintains lactation in the mammary glands.

Prospective Cohort Study
Also called prospective observational study; follow-up study; incidence study. An epidemiological and observational study in which a defined group of persons known to be exposed to a potential disease risk factor is followed over time and compared to a group of persons who were not known to be exposed to the potential risk factor, in order to evaluate the differences in rates of the outcome.

Proteinuria
Excessive amounts of protein in the urine.

Renin
A proteolytic enzyme of the blood that is produced and secreted by the juxtaglomerular cells of the kidney and hydrolyzes angiotensinogen to angiotensin I (the physiologically active form of angiotensin).

Rennin
Also called chymosin. A crystallizable milk-coagulating enzyme found especially in the gastric juice of the fourth stomach of young ruminants, is obtained as a yellowish powder, grains, or scales, and is used chiefly in making cheese and casein for plastics.

Resistin
A cysteine-rich peptide hormone found generally as an oligomer and produced in adipocytes (fat cells) during their differentiation. Resistin production in adipose tissue increases during lipolytic conditions often associated with insulin resistance.

Respiratory quotient (RQ)
The ratio of carbon dioxide produced by tissue metabolism to oxygen consumed in the same metabolism.

Selective serotonin reuptake inhibitor (SSRI)
Any of a class of antidepressants (such as fluoxetine or sertraline) that inhibit the inactivation of serotonin by blocking its reuptake by presynaptic nerve cell endings.

Sequence variation
Variants in the 3-letter codons that comprise a DNA sequence, that can translate to either the same protein as non-variant codons or to a mutated protein.

Serum cholesterol
Cholesterol that travels in the blood in distinct particles containing both lipids and proteins. Three major classes of lipoproteins are found in the serum of a fasting individual: low-density lipoprotein (LDL), high-density lipoprotein (HDL), and very-

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low-density lipoprotein (VLDL). Another lipoprotein class, intermediate-density lipoprotein (IDL), resides between VLDL and LDL; in clinical practice, IDL is included in the LDL measurement.

Sex hormone binding globulin (SHBG)
Glycoprotein possessing high affinity binding for 17 beta-hydroxysteriod hormones such as testosterone and oestradiol.

Shoulder dystocia
Difficulty in delivering the shoulders of the fetus through the birth canal after its head has emerged.

Single Nucleotide Polymorphism (SNP)
Points in the genome sequence where a single nucleotide variant that occurs within a population or group.

Skinfold thickness measurements
Method of assessing the size of the subcutaneous fat depot. Measurements are usually taken in the triceps, the biceps, the subscapular (just below and laterally to the inferior angle of the left scapula), or the suprailiac area (the midaxillary line immediately superior to the iliac crest).

Small-for-gestational age (SGA)
Smaller in size than is normal for the embryo/fetus' gender and gestational age; occurs when an embryo/fetus undergoes intrauterine growth restriction.

Standard Deviation
A statistic that shows how tightly all the various data points are clustered around the mean in a set of data.

Thyroxine
An iodine-containing hormone that is produced by the thyroid gland, increase the rate of cell metabolism, regulates growth, and is made synthetically for treatment of thyroid disorders.

Total Energy Expenditure (TEE)
The sum of basal energy expenditure, thermic effect of food (energy expended during food consumption), physical activity, thermoregulation (body's regulation of heat), and the energy expended in depositing new tissues and in producing milk.

Triglyceride
Also called neutral fat. Any of a group of lipids that are esters formed from one molecule of glycerol and three molecules of one or more fatty acids. Triglycerides are widespread in adipose tissue, commonly circulate in the blood in the form of lipoproteins, and are the chief constituent of fats and oils.

Trophoblast
Also called trophoderm. The outer layer of the blastocyst that attaches the fertilized ovum to the uterine wall and serves as a nutritive pathway for the embryo.

Tyrosine (Tyr)
An amino acid that is derived form the hydrolysis of proteins and is a precursor of epinephrine, thyroxine, and melanin.

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Urea
Also called carbamide. A soluble weakly basic nitrogenous compound that is the chief nitrogenous component of mammalian urine and an end product of protein metabolism and decomposition and that is administered intravenously as a diuretic drug.

Uterus
Also called womb. A hollow muscular organ in the pelvic cavity of female mammals that functions to contain and usually nourish the young during development prior to birth, and that consists of a body, fundus, isthmus, and cervix, and in which the fertilized egg implants and develops into the fetus.

Vascular endothelial growth factor (VEGF)
A growth factor belonging to a structural superfamily of glycoproteins containing a characteristic cysteine knot region, that primarily functions to stimulate rearrangement and reconstitution of connective tissue in embryonic development, organogenesis, or wound healing, and that induces production of extracellular matrix and proliferation of blood vessels and smooth muscle cells.

Vasculogenesis
The development of the vascular system.

Very low birth weight (VLBW)
Infant birth weight less than 1,500 grams.

Very low density lipoproteins (VLDL)
A lipoprotein containing a very large proportion of lipids to protein and acting as the primary carrier of cholesterol from the liver to the tissues.

Wernicke's encephalopathy
A disease of the central nervous system caused by a deficiency of thiamine and characterized by abnormal eye movements, a loss of muscle coordination, tremors, and confusion.

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SUPPLEMENTAL INFORMATION ON PUBLIC HEALTH SURVEYS OF PREGNANT WOMEN, INFANTS AND CHILDREN

TABLE A-1 Description and Comparison of Public Health Surveys of Pregnant Women, Infants, and Children

Survey	Objectives	Population/Data Collection	Data Source	Available Data	Strengths/Limitations
Infant Feeding Practices Survey II (IFPS-II)	To understand and improve the health of mothers and children by collecting information on infant feeding behaviors and factors influencing infant feeding choices.	Approximately 4,000 pregnant women from across the nation began their participation in the Infant Feeding Practices Study II (IFPS-II) between May and December 2005 and approximately 2,000 continued their participation through their infant's first year. To qualify, a healthy women gave birth to one healthy, full-term or near-term infant weighing at least 5 pounds at birth. Data were collected using mailed questionnaires, with the exception of a brief telephone interview near the time of the infant's birth.	A nationally distributed consumer opinion panel of more than 500,000 U.S. households.	• Foods fed to infants, including breast milk and infant formula • Factors that may contribute to infant feeding practices and to breastfeeding success • Mothers' intrapartum hospital experiences, sources of support, and postpartum depression • Mothers' employment status and child care arrangements • Infant sleeping arrangements • Other issues such as food allergies, experiences with breast pumps, and WIC participation • Diets of pregnant and postpartum women	Strengths: • Large sample size • Prospective design • Extensive testing of survey question • Data collected on infants' feeding pattern were extremely detailed • Frequency of questionnaires • Coverage of a wide number of issues • Maternal dietary data available on prenatal and month 3 survey respondents Limitations: • Sample was not representative of the U.S. population • Nutrient intake not formally assessed • Data were self-reported
Pediatric Nutrition Surveillance System (PedNSS)	To collect, analyze, and disseminate data to guide public health policy and action	Data are collected for infants, children, and adolescents from birth to 20 years of age who go to public health clinics for routine care, nutrition education, and supplemental foods.	Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) Early and Periodic Screening,	• Demographic information (clinic, county, date of birth, date of visit, race/ethnicity, sex, zip code, household income, migrant status, and source of data) • Anthropometry (birth	Strengths: • Because it is representative of the population served by the public health program submitting the surveillance data, it is essential for use in planning, implementing, monitoring, and evaluating

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		Data is collected at the clinic level then aggregated at the state level and submitted to CDC for analysis. Forty states, 1 U.S. Territory, 5 Indian Tribal Organizations, and the District of Columbia participated in 2007.	Diagnosis, and Treatment Program (EPSDT) Title V Maternal and Child Health Program (MCH)	weight, length/height, and weight) • Anemia (hemoglobin and hematocrit) • Infant feeding practices (breastfeeding initiation, duration, and exclusivity) • Health risk behaviors (TV/Video viewing, smoking in the household)	the nutritional status of children served by a specific public health program. Limitations: • Not all contributors for a specific public health program participate and therefore data is not representative of all WIC programs • Not representative of all low-income children or children in the general population
Pregnancy Nutrition Surveillance System (PNSS)	To collect, analyze, interpret, and disseminate data to guide public health policy and action	Low-income pregnant women who participate in federally funded public health programs. Data are collected at prenatal and postpartum clinic visits, and are aggregated at the contributor or state level and then submitted to CDC on a quarterly basis. Twenty-six states, 1 U.S. territory and 5 Indian Tribal Organizations participated in 2006.	Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) Title V Maternal and Child Health Program (MCH)	• Demographic Indicators (Source of data, race/ethnicity, woman's age, education, % poverty level, program participation and migrant status) • Maternal Health Indicators (prepregnancy BMI, maternal weight gain anemia, parity, interpregnancy interval, diabetes during pregnancy and hypertension during pregnancy) • Maternal Behavioral Indicators (medical care, WIC enrollment and multivitamin consumption) • Smoking/Drinking Indicators (smoking, smoking changes, smoking in household and drinking)	Strengths: • Because it is representative of the population served by the public health program submitting the surveillance data, it is essential data for use in planning, implementing, monitoring, and evaluating the nutritional status of women served by a specific public health program. • Height and weight at first prenatal visit measured by clinician Limitations: • Contributors voluntarily participate in PNSS and not all contributors for a specific public health program participate in PNSS, therefore it is not representative of all WIC programs. • Not representative of all low-income pregnant women or pregnant women in the

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				• Infant Health Indicators (birth weight, preterm birth, full term low birth weight and breastfeeding initiation)	general population. • Prepregnancy weight and gestational weight gain are self-reported.
Pregnancy Risk Assessment Monitoring System (PRAMS)	To improve the health of mothers and infants by reducing adverse outcomes such as low birth weight, and maternal and infant morbidity or mortality	Women who have had a recent live birth (drawn from state birth certificate file) Selected women are first contacted by mail or interviewed by telephone. Thirty-seven states, New York City and the Yankton Sioux Tribe currently participate.	State-specific, population-based data from birth certificate files on maternal attitudes and experiences before, during, and after pregnancy	• Changes in maternal and child health indicators (unintended pregnancy, prenatal care, breast-feeding, smoking, drinking, infant health)	Strengths: • Provides data not available from other sources about pregnancy and the first few months after birth. • Gestational weight gain is recorded from the birth certificate. Limitations: • Height and prepregnancy weight are self-reported

SOURCES: CDC, Infant Feeding Practices Survey 2 (http://www.cdc.gov/ifps/); Fein et al., 2008; CDC, Pediatric and Pregnancy Nutrition Surveillance System (http://www.cdc.gov/pednss/index.htm); CDC, Pregnancy Risk Assessment Monitoring System (http://www.cdc.gov/prams/).

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Websites:
http://www.cdc.gov/ifps/
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Appendix B

Supplementary Information on Nutritional Intake

DIETARY REFERENCE INTAKES FOR PREGNANCY

TABLE B-1A Equations to Estimate Energy Requirement for Pregnant Women by Trimester

Estimated Energy Requirement (kcal/day) = Nonpregnant EER + Pregnancy Energy Deposition	
1st trimester	EER = Nonpregnant (adolescent or adult) EER + 0
2nd trimester	EER = Nonpregnant (adolescent or adult) EER + 340
3rd trimester	EER = Nonpregnant (adolescent or adult) EER + 452

NOTE: EER = Estimated Energy Requirement;. Use equations in Table B-1B to calculate nonpregnant EER. EER for adult women should be used for ages 19-50 years; EER for adolescent girls should be used for ages 9-18 years

SOURCE: IOM, 2006.

TABLE B-1B Equations to Calculate Estimated Energy Requirement (EER) for Nonpregnant Adolescents or Adult Women

14-18 Years of Age Estimated Energy Requirement (kcal/day) = Total Energy Expenditure + Energy Deposition EER = 135.3 – (30.8 x age [y]) + PA x [(10.0 x weight [kg]) + (934 x height [m])] + 25
19 Years and Older Estimated Energy Requirement (kcal/day) = Total Energy Expenditure EER = 354 – (6.91 x age [y]) + PA x [(9.36 x weight [kg]) + (726 x height [m])]

NOTE: PA = Physical Activity Coefficient. Use equations in Table B-1C to calculate PA.

SOURCE: IOM, 2006.

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TABLE B-1C Physical Activity Coefficients (PA values) for Use in EER Equations for Adolescents or Adult Women

	Sedentary [a] (PAL 1.0-1.39)	**Low Active [b] (PAL 1.4-1.59)**	**Active [c] (PAL 1.6-1.89)**	**Very Active [d] (PAL 1.9-2.5)**
14-18 Years of Age	1.0	1.16	1.31	1.56
19 Years and Older	1.0	1.12	1.27	1.45

NOTE: PAL = Physical Activity Level.

[a] E.g., Typical daily living activities (e.g., household tasks, walking to the bus)
[b] E.g., Typical daily living activities PLUS 30-60 minutes of daily moderate activity (e.g., walking at 5-7 km/h)
[c] E.g., Typical daily living activities PLUS at least 60 minutes of daily moderate activity
[d] E.g., Typical daily living activities PLUS at least 60 minutes of daily moderate activity PLUS an additional 60 minutes of vigorous activity or 120 minutes of moderate activity

SOURCE: IOM, 2006.

TABLE B-2 Dietary Reference Intakes for Pregnant Women: Vitamins, Elements, Total Water, and Macronutrients in Alphabetical Order

Nutrient	**EAR[a]**	**RDA[b]** / AI[c]	**UL[d]**
Biotin			
14-18 y	---	30 µg/day	---
19-30 y	---	30 µg/day	---
31-50 y	---	30 µg/day	---
Boron			
14-18 y	---	---	17 mg/day
19-30 y	---	---	20 mg/day
31-50 y	---	---	20 mg/day
Calcium			
14-18 y	---	1,300 mg/day	2.5 g/day
19-30 y	---	1,000 mg/day	2.5 g/day
31-50 y	---	1,000 mg/day	2.5 g/day
Carbohydrate			
14-18 y	135 g/day	**175 g/day**	---
19-30 y	135 g/day	**175 g/day**	---
31-50 y	135 g/day	**175 g/day**	---
Chloride			
14-18 y	---	2.3 g/day	3.6 g/day
19-30 y	---	2.3 g/day	3.6 g/day
31-50 y	---	2.3 g/day	3.6 g/day
Choline			
14-18 y	---	450 mg/day	3.0 g/day
19-30 y	---	450 mg/day	3.5 g/day
31-50 y	---	450 mg/day	3.5 g/day
Chromium			
14-18 y	---	29 µg/day	---
19-30 y	---	30 µg/day	---

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Nutrient	EAR[a]	RDA[b] / AI[c]	UL[d]
31-50 y	---	30 µg/day	---
Copper			
14-18 y	785 µg/day	**1,000 µg/day**	8,000 µg/day
19-30 y	800 µg/day	**1,000 µg/day**	10,000 µg/day
31-50 y	800 µg/day	**1,000 µg/day**	10,000 µg/day
Fiber (Total)			
14-18 y	---	28 g/day	---
19-30 y	---	28 g/day	---
31-50 y	---	28 g/day	---
Flouride			
14-18 y	---	3.0 mg/day	10 mg/day
19-30 y	---	3.0 mg/day	10 mg/day
31-50 y	---	3.0 mg/day	10 mg/day
Folate[e]			
14-18 y	520 µg/day	**600 µg/day**	800 µg/day
19-30 y	520 µg/day	**600 µg/day**	1,000 µg/day
31-50 y	520 µg/day	**600 µg/day**	1,000 µg/day
Iodine			
14-18 y	160 µg/day	**220 µg/day**	900 µg/day
19-30 y	160 µg/day	**220 µg/day**	1,100 µg/day
31-50 y	160 µg/day	**220 µg/day**	1,100 µg/day
Iron			
14-18 y	23 mg/day	**27 mg/day**	45 mg/day
19-30 y	22 mg/day	**27 mg/day**	45 mg/day
31-50 y	22 mg/day	**27 mg/day**	45 mg/day
Linoleic acid			
14-18 y	---	13 g/day	---
19-30 y	---	13 g/day	---
31-50 y	---	13 g/day	---
α-Linolenic Acid			
14-18 y	---	1.4 g/day	---
19-30 y	---	1.4 g/day	---
31-50 y	---	1.4 g/day	---
Magnesium[f]			
14-18 y	335 mg/day	**400 mg/day**	350 mg/day
19-30 y	290 mg/day	**350 mg/day**	350 mg/day
31-50 y	300 mg/day	**360 mg/day**	350 mg/day
Manganese			
14-18 y	---	2.0 mg/day	9 mg/day
19-30 y	---	2.0 mg/day	11 mg/day
31-50 y	---	2.0 mg/day	11 mg/day
Molybdenum			
14-18 y	40 µg/day	**50 µg/day**	1,700 µg/day
19-30 y	40 µg/day	**50 µg/day**	2,000 µg/day
31-50 y	40 µg/day	**50 µg/day**	2,000 ug/day
Niacin[g]			
14-18 y	14 mg/day	**18 mg/day**	30 mg/day
19-30 y	14 mg/day	**18 mg/day**	35 mg/day
31-50 y	14 mg/day	**18 mg/day**	35 mg/day
Nickel			

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Nutrient	EAR[a]	RDA[b] / AI[c]	UL[d]
14-18 y	---	---	1.0 mg/day
19-30 y	---	---	1.0 mg/day
31-50 y	---	---	1.0 mg/day
Pantothenic Acid			
14-18 y	---	6.0 mg/day	---
19-30 y	---	6.0 mg/day	---
31-50 y	---	6.0 mg/day	---
Phosphorus			
14-18 y	1,055 mg/day	**1250 mg/day**	3.5 g/day
19-30 y	580 mg/day	**700 mg/day**	3.5 g/day
31-50 y	580 mg/day	**700 mg/day**	3.5 g/day
Potassium			
14-18 y	---	4.7 g/day	---
19-30 y	---	4.7 g/day	---
31-50 y	---	4.7 g/day	---
Protein[h]			
14-18 y	0.88 g/kg/day	**71 g/day**	---
19-30 y	0.88 g/kg/day	**71 g/day**	---
31-50 y	0.88 g/kg/day	**71 g/day**	---
Riboflavin			
14-18 y	1.2 mg/day	**1.4 mg/day**	---
19-30 y	1.2 mg/day	**1.4 mg/day**	---
31-50 y	1.2 mg/day	**1.4 mg/day**	---
Selenium			
14-18 y	49 µg/day	**60 µg/day**	400 µg/day
19-30 y	49 µg/day	**60 µg/day**	400 µg/day
31-50 y	49 µg/day	**60 µg/day**	400 µg/day
Sodium			
14-18 y	---	1.5 g/day	2.3 g/day
19-30 y	---	1.5 g/day	2.3 g/day
31-50 y	---	1.5 g/day	2.3 g/day
Thiamin			
14-18 y	1.2 mg/day	**1.4 mg/day**	---
19-30 y	1.2 mg/day	**1.4 mg/day**	---
31-50 y	1.2 mg/day	**1.4 mg/day**	---
Vitamin A[i]			
14-18 y	530 µg RAE/day	**750 µg RAE/day**	2,800 µg/day
19-30 y	550 µg RAE/day	**770 µg RAE/day**	3,000 µg/day
31-50 y	550 µg RAE/day	**770 µg RAE/day**	3,000 µg/day
Vitamin B_6			
14-18 y	1.6 mg/day	**1.9 mg/day**	80 mg/day
19-30 y	1.6 mg/day	**1.9 mg/day**	100 mg/day
31-50 y	1.6 mg/day	**1.9 mg/day**	100 mg/day
Vitamin B_{12}			
14-18 y	2.2 µg/day	**2.6 µg/day**	---
19-30 y	2.2 µg/day	**2.6 µg/day**	---
31-50 y	2.2 µg/day	**2.6 µg/day**	---
Vitamin C			
14-18 y	66 mg/day	**80 mg/day**	1,800 mg/day
19-30 y	70 mg/day	**85 mg/day**	2,000 mg/day

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Nutrient	**EAR**[a]	**RDA**[b] / AI[c]	**UL**[d]
31-50 y	70 mg/day	**85 mg/day**	2,000 mg/day
Vitamin D			
14-18 y	---	5.0 µg/day[j]	50 µg/day
19-30 y	---	5.0 µg/day[j]	50 µg/day
31-50 y	---	5.0 µg/day[j]	50 µg/day
Vitamin E[k]			
14-18 y	12 mg/day	**15 mg/day**	8,00 mg/day
19-30 y	12 mg/day	**15 mg/day**	1,000 mg/day
31-50 y	12 mg/day	**15 mg/day**	1,000 mg/day
Vitamin K			
14-18 y	---	75 µg /day	---
19-30 y	---	90 µg /day	---
31-50 y	---	90 µg /day	---
Water (Total)[l]			
14-18 y	---	3.0 L/day	---
19-30 y	---	3.0 L/day	---
31-50 y	---	3.0 L/day	---
Zinc			
14-18 y	10.5 mg/day	**12 mg/day**	34 mg/day
19-30 y	9.5 mg/day	**11 mg/day**	40 mg/day
31-50 y	9.5 mg/day	**11 mg/day**	40 mg/day

NOTE: This table (taken from the DRI reports; see www.nap.edu) presents Recommended Dietary Allowances (RDA) in **bold type** or Adequate Intakes (AI) in ordinary type in Column 3.

[a] EAR = Estimated Average Requirement. An EAR is the average daily nutrient intake level estimated to meet the requirements of half of the healthy individuals in a group.

[b] RDA = Recommended Dietary Allowance. An RDA is the average daily dietary intake level sufficient to meet the nutrient requirements of nearly all (97-98 percent) healthy individuals in a group. It is calculated from an EAR.

[c] AI = Adequate Intake. If sufficient scientific evidence is not available to establish an EAR, and thus calculate and RDA, an AI is usually developed. The AI for breast-fed infants is the mean intake. The AI for other life-stage groups and gender groups (except healthy breast-fed infants) is believed to cover the needs of all healthy individuals in the group, but a lack of data or uncertainty in the data prevents being able to specify with confidence the percentage of individuals covered by this intake. In the absence of a UL, extra caution may be warranted in consuming levels above recommended intakes. Members of the general population should be advised not to routinely exceed the UL. The UL is not meant to apply to individuals who are treated with the nutrient under medical supervision or to individuals with predisposing conditions that modify their sensitivity to the nutrient

[d] UL = Tolerable Upper Intake Level. The UL is the highest level of daily nutrient intake that is likely to pose no risk of adverse health effects to almost all individual in the general population. Unless otherwise specified, the UL represents total intake from food, water, and supplements.

[e] As dietary folate equivalents (DFEs). 1 DFE = 1 µg food folate = 0.6 µg of folic acid from fortified food or as a supplement consumed with food = 0.5 µg of folic acid from a supplement taken on an empty stomach. In view of evidence linking folate intake with neural tube defects in the fetus, it is recommended that all women capable of becoming pregnancy consume 400 µg from supplements or fortified foods in addition to intake of food folate from a varied diet. It is assumed that women will continue consuming 400 µg from supplements or fortified food until their pregnancy is confirmed and they enter prenatal care, which ordinarily occurs after the end of the periconceptional period—the critical time for formation of the neural tube. The UL for folate applies to synthetic forms obtained from supplements, fortified foods, or a combination of the two.

[f] The ULs for magnesium represent intake from pharmacological agents only and do not include intake from food and water.

[g] For EAR and RDA: as niacin equivalents (NE). 1 mg of niacin = 60 mg of tryptophan. The UL for niacin applies to synthetic forms obtained from supplements, fortified foods, or a combination of the two.

[h] Based on g protein per kg of body weight for the reference body weight, e.g., for adults 0.8 g/kg body weight for the reference body weight.

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[i] RAE = Retinol activity equivalent. 1 µg RAE = 1 µg retinol, 12 µg β-carotene, and 24 µg α-carotene or β-cryptoxanthin. The RAE for dietary provitamin A carotenoids in foods is twofold greater than retinol equivalents (RE), whereas the RAE for preformed vitamin A in foods is the same as RE. The UL for vitamin A is for preformed vitamin A only.

[j] In the absence of adequate exposure to sunlight, and as cholecalciferol. 1 µg cholecalciferol = 40 IU vitamin D.

[k] As α-tocopherol. For EAR and RDA: α-tocopherol includes *RRR*-α-tocopherol, the only form of α-tocopherol that occurs naturally in foods, and the 2*R*-stereoisomeric forms of α-tocopherol (*RRR*-, *RSR*-, *RRS*-, and *RSS*-α-tocopherol) that occur in fortified foods and supplements. This does not include the 2*S*-stereoisomeric forms of α-tocopherol (*SRR*-, *SSR*-, *SRS*-, and *SSS*-α-tocopherol), also found in fortified foods and supplements. The 2*S*-stereoisomers are not stored in the body. For UL: applies to any form of supplemental α-tocopherol.

[l] *Total* water includes all water contained in food, beverages, and drinking water.

SOURCE: IOM, 2006.

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TABLE B-3 Acceptable Macronutrient Distribution Ranges

Macronutrient	Range (percent of energy) 4-18 y	Adults
Fat	25-35	20-35
n-6 Polyunsaturated fatty acids[a] (linoleic acid)	5-10	5-10
n-3 Polyunsaturated fatty acids[a] (α-linolenic acid)	0.6-1.2	0.6-1.2
Carbohydrate	46-65	46-65
Protein	10-30	10-35

[a] Approximately 10 percent of the total can come from longer-chain *n*-3 or *n*-6 fatty acids.

SOURCE: IOM, 2006.

TABLE B-4 Additional Macronutrient Recommendations

Macronutrient	**Recommendation**
Dietary cholesterol	As low as possible while consuming a nutritionally adequate diet
Trans fatty acids	As low as possible while consuming a nutritionally adequate diet
Saturated fatty acids	As low as possible while consuming a nutritionally adequate diet
Added sugars[a]	Limit to no more than 25% of total energy

[a]Not a recommended intake. A daily intake of added sugars that individuals should aim for to achieve a healthful diet was not set.

SOURCE: IOM, 2006.

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TRENDS IN ENERGY INTAKE AND MARKERS OF ENERGY DENSITY

As the prevalence of obesity rises among childbearing-aged women and women entering pregnancy, important shifts in diet and physical activity have also occurred. In a recent study, Nielsen and colleagues (2002) used nationally representative data from the 1977-1978 Nationwide Food Consumption Survey and the 1989-1991 and 1994-1996 Continuing Surveys of Food Intake by Individuals to investigate the trends in total energy intake and energy intake by meal pattern type (Figure B-1). Data were stratified by age but not sex. These investigators found that among U.S. adults aged 19-39 years, there was an 18 percent increase in total energy intake over the 20-year period (1856 to 2198 kcal/d). When separated into energy from meal pattern type, the major contributor to this overall increase in energy intake was a sharp 58 percent increase in energy from snacks (244-387 kcal/d). Additionally, the percent of total energy from key food groups, such as salty snacks, sweetened beverages, candy, pizza, French fries, cheeseburgers, and Mexican-style food, increased between survey years 1977-78 and 1994-96.

These authors further investigated trends in beverage intake using the aforementioned data sources plus the 1999–2001 nationally-representative NHANES. For all age groups, including adults aged 19 to 39 years, sweetened beverage intake increased and milk consumption decreased between survey years 1977-78 and 1999-2001 (Figure B-2) (Nielsen and Popkin, 2004). Specifically, energy from soft drinks nearly tripled (2.8 to 7.0 percent [approximately 50 to 144 kcal per person per day]), energy from fruit drinks doubled (1.1 to 2.2 percent [from 20 to 45 kcal per person per day]), and energy from milk decreased (8.0 to 5.0 percent [from 143 to 99 kcal per person per day]).

Taken together, these findings illustrate an increase in consumption of foods of low nutrient density in the general population. This has special implications for pregnancy and lactation, which require modest increases in energy intake but proportionally greater increases in vitamin and mineral intake.

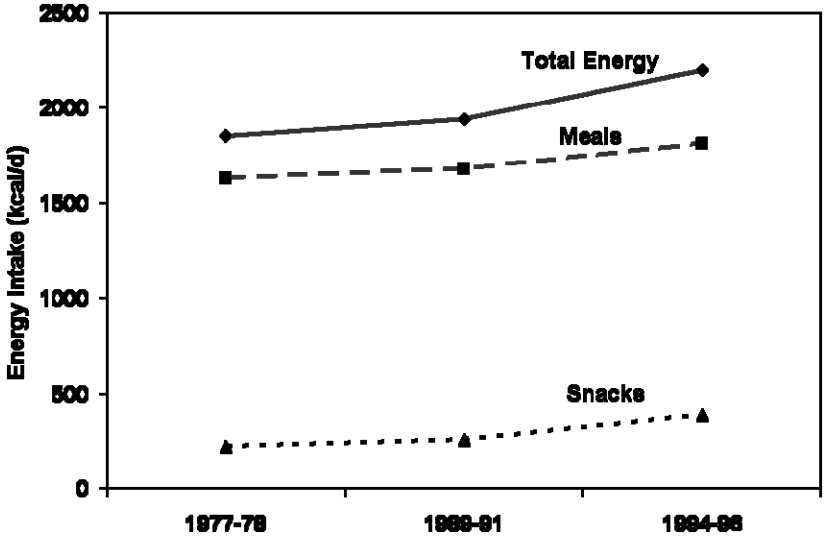

FIGURE B-1 Trends in energy intake and meal pattern type, U.S. adults aged 19-39 years.
SOURCE: Nielsen et al., 2002.

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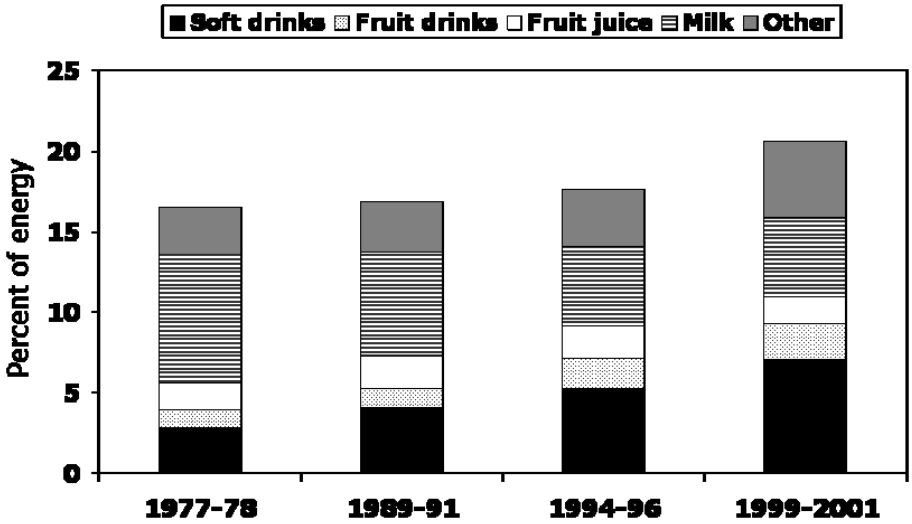

FIGURE B-2 Trends in US beverage consumption 1977-2001: percent of total energy intake
SOURCE: Nielsen and Popkin, 2004.

The U.S. Dietary Guidelines represent federal nutrition policy. The recommendations of the Dietary Guidelines are interpreted for use by healthy Americans over 2 years of age in MyPyramid (Available online: http://www.MyPyramid.gov [accessed October 16, 2008]). The current MyPyramid recommends that females aged 14-18 years and 31-50 years consume 8 combined servings of fruits and vegetables, while 9 combined servings is recommended for females aged 19-30 years. MyPyramid also makes more specific recommendations about types of vegetables, including dark green and orange vegetables, and legumes to ensure consumption of the variety of nutrients available from these foods. When the Guenther et al. (2006) examined sub-groups of vegetables, mean intakes for childbearing-aged women were below the recommended amounts for all subgroups except for starchy vegetables (Figure B-3). These data clearly illustrate that childbearing-aged women failed to meet recommendations for fruits and vegetables.

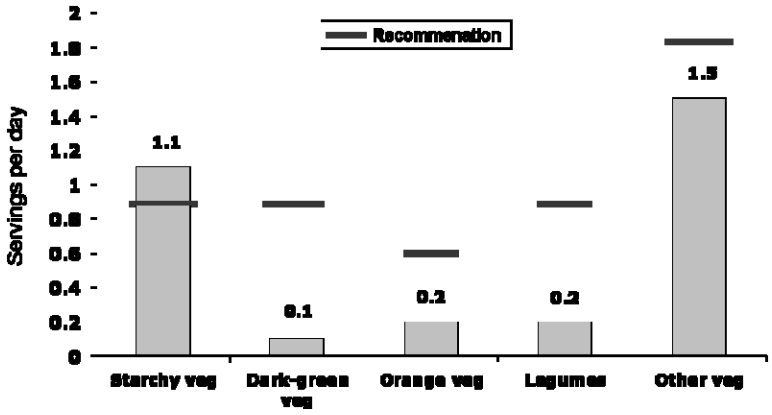

FIGURE B-3 Mean daily intakes of vegetables by subtype among U.S. females 19-30 years of age
SOURCE: Guenther et al., 2006.

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Another method of quantifying the overall quality of American's diets is through the use of the Healthy Eating Index-2005 (HEI-2005), a tool designed to measure compliance of diets with the key, diet-related recommendations of the 2005 Dietary Guidelines for Americans (DHHS/USDA, 2005). The HEI-2005 has 12 components, as seen in Table B-3 (Guenther et al., 2006). For most components, higher intakes result in higher scores. Note, however, that for three components, saturated fat, sodium, and calories from solid fats, alcoholic beverages, and added sugars (SoFAAS), lower intake levels result in higher scores because lower intakes are more desirable. Monitoring changes in the HEI-2005 scores can provide a method for nutrition surveillance in the population.

In a recent analysis, trends in the HEI-2005 scores and its components were published for all Americans two years of age and older (subgroups of the population were not studied). From 1994-96 to 2001-02, there was little change in either overall HEI-2005 score or its components. The average HEI-2005 score was 58.2 out of 100 possible points in both time periods. American's diets consistently met recommendations for the groups "Total Grains" and "Meat and Beans", but were far below the maximum score for the groups, "Dark Green and Orange Vegetables" and "Legumes" as well as whole grains. Intakes from SoFAAS were well below the recommendations, as reflected in low scores on these components. From 1994-96 to 2001-02 the HEI-2005 score declined for the groups "Whole Fruit", "Total Vegetables", and "Whole Grains" while the score for the groups "Milk", "Oils", and "Sodium" improved.

TABLE B-3 Estimated Healthy Eating Index-2005 Component and Total Scores, U.S. (1994-96 and 2001-02)

Component (maximum score)	1994-96 Score (95 % CI)	2001-02 Score (95% CI)
Total fruit (5)	3.1 (3.0, 3.3)	3.1 (2.9, 3.3)
Whole fruit (5)	4.5 (4.3, 4.7)	3.4 (3.2, 3.7)*
Total vegetables (5)	3.6 (3.6, 3.7)	3.2 (3.1, 3.4)*
Dark green and orange vegetables and legumes (5)	1.4 (1.4, 1.5)	1.4 (1.2, 1.5)
Total grains (5)	5.0 (5.0, 5.0)	5.0 (5.0, 5.0)
Whole grains (5)	1.2 (1.2, 1.2)	1.0 (1.0, 1.1)*
Milk (10)	5.9 (5.7, 6.2)	6.3 (6.0, 6.5)*
Meat and beans (10)	10.0 (9.9, 10.0)	10.0 (10.0, 10.0)
Oils (10)	6.0 (5.8, 6.2)	6.8 (6.5, 7.1)*
Sodium (10)	3.2 (3.1, 3.3)	4.1 (3.9, 4.2)*
Saturated fat (10)	6.5 (6.4, 6.7)	6.4 (6.1, 6.7)
Calories from solid fat, alcohol, and added sugar (20)	7.8 (7.5, 8.2)	7.5 (6.9, 8.1)
Total HEI-2005 score (100)	58.2 (57.2, 59.2)	58.2 (56.6, 59.9)

NOTES: Excludes children under 2 years of age and breast-fed children

*Significantly different ($p < 0.05$).

SOURCE: Nutrition Insight 37, USDA, available online at http://www.cnpp.usda.gov/Publications/NutritionInsights/Insight37.pdf [accessed April 16, 2009].

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Some of these dietary pattern changes may be a result of the trend toward obtaining a greater proportion of food outside the home. Self-reported dietary data from national surveys was used to show that the percentage of total energy intake obtained from foods consumed at home decreased from 77 in 1977-78 to 65 percent from 1994-96 (Figure B-4) (Nielsen et al., 2002). The amount of energy obtained from foods consumed from restaurants, including fast food establishments, doubled from 9 to 21 percent during this same period.

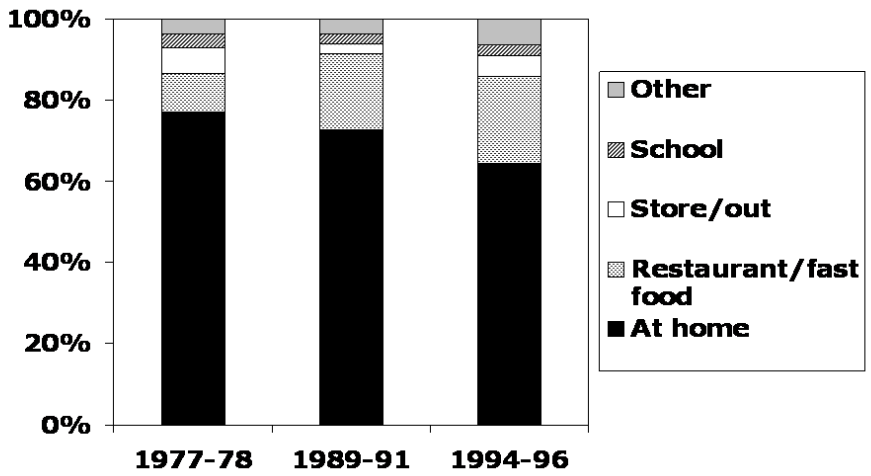

FIGURE B-4 Trends in energy intake (percent energy) by location, Americans aged 2+ years
SOURCE: Nielsen et al., 2002.

The aforementioned analysis relied on dietary intake data obtained from surveys. There is no "gold standard" method of assessing dietary intakes in individuals, and all self-reported dietary intake data have inherent biases. Therefore food supply data, collected directly from food producers and distributors, are often used to examine trends in American dietary patterns. The estimates are adjusted for spoilage, cooking losses, plate waste, and other food losses accumulated throughout the marketing system and the home. Analysis of trends in food supply data provide the same conclusions regarding trends in increasing energy intake and overall diet quality of Americans as self-reported survey data.

The most recent loss-adjusted annual per capita food supply data analyses by the USDA's Economic Research Service suggests a 12 percent increase in total energy intake (~300 kcal per person per day) from 1985 to 2000 (USDA, 2002; available online: http://www.ers.usda.gov/publications/FoodReview/DEC2002/frvol25i3a.pdf [accessed April 16, 2009]) (Figure B-5). Of the 300-kcal increase, grains (mainly refined grains) accounted for 46 percent; added fats, 24 percent; added sugars, 23 percent; fruits and vegetables, 8 percent of the increase. At the same time, energy intake from the meat and dairy groups together declined by 1 percent. When the per capita food supply data in 2000 was compared with the 1992 Food Guide Pyramid recommendations as a marker of overall diet quality, intakes exceeded recommendations for grains, meats, added fats, and added sugars, and fell below recommendations for dairy, fruits, and non-starchy vegetables (USDA, 2002; available online:

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http://www.ers.usda.gov/publications/FoodReview/DEC2002/frvol25i3a.pdf [accessed April 16, 2009])).

FIGURE B-5 Energy from the U.S. per capita food supply (adjusted for losses)
SOURCE: USDA, 2002; available online at
http://www.ers.usda.gov/publications/FoodReview/DEC2002/frvol25i3a.pdf [accessed April 16, 2009].

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Appendix C

Supplementary Information on Composition and Components of Gestational Weight Gain

Tables C-1 through C-6 summarize literature and data that are referenced and/or support the discussion in Chapter 3, *Composition and Components of Gestational Weight Gain: Physiology and Metabolism*.

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STUDIES ON COMPOSITION AND COMPONENTS OF GESTATIONAL GAIN: PHYSIOLOGY AND METABOLISM

TABLE C-1A Maternal Weight Gain in Singleton Pregnancies (by trimester and total weight gain)

Study Description	Population Characteristics	Study Criteria	Weight Gain by Trimester 1st	2nd	3rd	Total Weight Gain
Author, year: Abrams et al., 1995	**Total Study N:** 10,418	*By ethnicity:*				
		Hispanic	0.15 kg/wk	0.54 kg/wk	0.54 kg/wk	
		White	0.19 kg/wk	0.58 kg/wk	0.52 kg/wk	
Country: USA		Black	0.21 kg/wk	0.52 kg/wk	0.49 kg/wk	
		Asian	0.11 kg/wk	0.56 kg/wk	0.52 kg/wk	
		Other	0.14 kg/wk	0.56 kg/wk	0.52 kg/wk	
		By BMI:				
		Low BMI	0.15 kg/wk	0.57 kg/wk	0.49 kg/wk	
		Ideal BMI	0.18 kg/wk	0.58 kg/wk	0.53 kg/wk	
		High BMI	0.18 kg/wk	0.51 kg/wk	0.51 kg/wk	
		Obese	0.14 kg/wk	0.41 kg/wk	0.49 kg/wk	
		By Fetal Size:				
		SGA	0.13 kg/wk	0.48 kg/wk	0.48 kg/wk	
		AGA	0.17 kg/wk	0.56 kg/wk	0.52 kg/wk	
		LGA	0.22 kg/wk	0.62 kg/wk	0.57 kg/wk	
Author, year: Abrams and Selvin, 1995	**Total Study N:** 2,994		2.1 kg	7.7 kg	6.6 kg	16.7 kg
Country: USA	**Group Description:** White nonobese women					
Author, year: Abrams et al., 1989	**Total Study N:** 2,163	Preterm deliveries (n = 118)				11 kg (0.33 kg/wk)
		Term deliveries (n = 2,045)				14.5 kg (0.39 kg/wk)
Country: USA	**Group Description:** Women at high risk for nutritional problems during pregnancy					

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TABLE C-1A Maternal Weight Gain in Singleton Pregnancies (by trimester and total weight gain)

Study Description	Population Characteristics	Study Criteria	Weight Gain by Trimester 1st	2nd	3rd	Total Weight Gain
Author, year:	**Total Study N:**	Obese (n = 110)				7.4 kg
Amador et al., 2008	220	Non-obese (n = 110)				9.4 kg
Country:						
Mexico						
Author, year:	**Total Study N:**					Mean:
Bianco et al., 1998*	11,926	Morbidly obese (n = 613)				9.1 kg
		Non-obese (n = 11,313)				14.2 kg
Country:	**Group Description:**					
USA	Women					
Author, year:	**Total Study N:**		Mean:	Mean:	Mean:	
Carmichael et al., 1997	4,218	Underweight	1.92 kg	0.57 kg/wk	0.48 kg/wk	
		Normal weight	2.19 kg	0.58 kg/wk	0.51 kg/wk	
Country:	**Group Description:**	Overweight	2.16 kg	0.51 kg/wk	0.49 kg/wk	
USA	Women with good pregnancy outcomes	Obese	1.65 kg	0.41 kg/wk	0.47 kg/wk	
Author, year:	**Total Study N:**	Women with GDM (n = 78)				12.6 kg
Catalano et al., 1993	390	Controls (n = 312)				15.0 kg
Country:		Underweight				
USA		GDM (n = 15)	0.24 kg/wk	0.35 kg/wk	0.34 kg/wk	12.2 kg
		Control (n = 92)	0.28 kg/wk	0.38 kg/wk	0.39 kg/wk	15.0 kg
		Average weight				
		GDM (n = 34)	0.29 kg/wk	0.42 kg/wk	0.39 kg/wk	14.6 kg
		Control (n = 172)	0.35 kg/wk	0.40 kg/wk	0.40 kg/wk	15.2 kg
		Overweight				
		GDM (n = 29)	0.27 kg/wk	0.32 kg/wk	0.30 kg/wk	10.5 kg
		Control (n = 48)	0.28 kg/wk	0.36 kg/wk	0.37 kg/wk	14.1 kg
Author, year:	**Total Study N:**	BMI < 20				13.5 kg
Cedergren, 2006*	245,526	BMI 20-24.9				13.8 kg
		BMI 25.29.9				13.2 kg
Country:		BMI 30-34.9				11.1 kg

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TABLE C-1A Maternal Weight Gain in Singleton Pregnancies (by trimester and total weight gain)

Study Description	Population Characteristics	Study Criteria	Weight Gain by Trimester 1st	2nd	3rd	Total Weight Gain
Sweden		BMI > 35				8.7 kg
Author, year: Claesson et al., 2008	**Total Study N:** 348	Obese (n=155)				8.7 kg
		Controls (n=193)				11.3 kg
						Adjusted:
Country: Sweden						7.5 kg
						9.8 kg
Author, year: Gunderson et al., 2001*	**Total Study N:** 985	Pregravid BMI:				*Hispanic:*
		Underweight (n = 266)				12.1 kg
		Normal (n = 595)				12.9 kg
	Group Description:	Overweight (n = 71)				13.5 kg
Country: USA	Healthy women	Obese (n = 53)				11.0 kg
						White:
		Race/ethnicity:				12.6 kg
		Hispanic (n = 130)				13.2 kg
		White (n = 532)				14.7 kg
		Black (n = 100)				10.6 kg
		Asian (n = 223)				*Black:*
						13.5 kg
						13.8 kg
						14.3 kg
						10.9 kg
						Asian:
						12.1 kg
						11.8 kg
						13.4 kg
						11.9 kg
Author, year: Hediger et al., 1989	**Total Study N:** 1,790	Total (n = 1,790)	7.29 kg		7.46 kg	14.75 kg
		Adequate gain (n = 955)	9.12 kg		9.26 kg	18.38 kg
		Early inadequate (n = 304)	1.89 kg		8.89 kg	10.78 kg
Country: USA (Camden, NJ)	**Group Description:** Adolescents	Late inadequate (n = 387)	8.94 kg		3.27 kg	12.21kg
		Both inadequate (n = 144)	2.18 kg *(early gain, 0-24 wks)*		3.69 kg *(late gain, 24 wks-delivery)*	5.87 kg *(0-delivery)*

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TABLE C-1A Maternal Weight Gain in Singleton Pregnancies (by trimester and total weight gain)

Study Description	Population Characteristics	Study Criteria	Weight Gain by Trimester 1st	2nd	3rd	Total Weight Gain
Author, year: Hickey et al., 1995	**Total Study N:** 1,015	Black women, pregravid BMI:				
		Low BMI (n = 255)	2.61 kg	0.49 kg/wk	0.46 kg/wk	
		Normal BMI (n = 422)	2.54 kg	0.46 kg/wk	0.45 kg/wk	
Country: USA	**Group Description:** Nonobese low-income black and white women	White women, pregravid BMI:				
		Low BMI (n = 143)	2.93 kg	0.52 kg/wk	0.42 kg/wk	
		Normal BMI (n = 195)	1.81 kg	0.52 kg/wk	0.45 kg/wk	
Author, year: Johnston et al., 1992	**Total Study N:** 272	Adolescents (n = 123)				15.4 kg
		Adults (n = 149)				14.4 kg *(39.6 wks gestation)*
Country: USA	**Group Description:** Middle class, upper class					
Author, year: Kinnunen et al., 2007	**Total Study N:** 132	Intervention (n = 48)				14.6 kg
		Control (n = 56)				14.3 kg
Country: Finland	**Group Description:** Primiparas					
Author, year: Kramer et al., 1992	**Total Study N:** 9,742	Mean prepregnancy BMI: 22.1 kg/m^2				14.5 kg (0.37 kg/wk)
	Group Description: Mostly Canadian-born, Caucasians	Time of delivery:				
		≥ 37 weeks				14.6 kg
		< 37 weeks				12.5 kg
		< 34 weeks				9.9 kg
		< 32 weeks				9.1 kg
Author, year: Lawton et al., 1988	**Total Study N:** 334	Delivered SGA infant (n = 30)			0.99 kg	12.6 kg
		Delivered AGA infant (n = 128)			1.95 kg (gain between 28-32 weeks)	12.8 kg
Country: UK	**Group Description:** Predominantly white women					

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TABLE C-1A Maternal Weight Gain in Singleton Pregnancies (by trimester and total weight gain)

Study Description	Population Characteristics	Study Criteria	Weight Gain by Trimester 1st	2nd	3rd	Total Weight Gain
Author, year: Loris et al., 1985	**Total Study N:** 145	Pregravid weight/height:				
		75%-90% ideal weight (n = 25)				16 kg
Country: USA	**Group Description:** Teens, aged 13-19 years	91%-110% ideal weight (n = 68)				18 kg
	Delivered at term	111%-166% ideal weight (n = 28)				17 kg
Author, year: Muscati et al., 1996*	**Total Study N:** 371	*Normal weight (90-120% of standard pregravid wt/ht)*	0-20 wks:	21-30 wks:	31-term:	
		PPWR < median	3.8 kg	5.3 kg	4.1 kg	13.2 kg
Country: Canada	**Group Description:** Healthy, white, nonsmoking women	PPWR ≥ median	7.1 kg	6.8 kg	6.2 kg	20.2 kg
		Underweight (< 90% of standard pregravid wt/ht)				
		PPWR < median	3.8 kg	5.4 kg	4.1 kg	13.3 kg
		PPWR ≥ median	7.1 kg	6.4 kg	6.1 kg	19.6 kg
		Overweight (> 120% of standard pregravid wt/ht)				
		PPWR < median	1.4 kg	4.5 kg	3.7 kg	9.6 kg
		PPWR ≥ median	7.6 kg	6.2 kg	5.3 kg	19.1 kg
Author, year: Oken et al., 2007*	**Total Study N:** 1,044	Prepregnancy BMI:				
		< 19.8 (n = 124)				15.7 kg
		19.8-26.0 (n = 622)				16.4 kg
Country: USA		26.1-29.0 (n = 134)				15.3 kg
		> 29.0 (n = 164)				13.1 kg
Author, year: Rees et al., 1992	**Total Study N:** 459	Birth weight:				
		< 3,000 g (n = 88)			0.50 kg/wk	13.8 kg
		3,000-4,000 g (n = 274)			0.58 kg/wk	16.6 kg
Country: USA	**Group Description:** Adolescents					
Author, year: Rosso, 1985	**Total Study N:** 262	Prepregnancy weight:				
		Underweight, < 89% standard weight (n = 62)				12 kg

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TABLE C-1A Maternal Weight Gain in Singleton Pregnancies (by trimester and total weight gain)

Study Description	Population Characteristics	Study Criteria	Weight Gain by Trimester 1st	2nd	3rd	Total Weight Gain
Country: USA	**Group Description:** Mostly black (51%) and Hispanic (41%) women	Normal, 90-110% standard weight (n = 137)				10 kg
		Overweight, >111% standard weight (n = 63)				7 kg
Author, year: Schieve et al., 1999*	**Total Study N:** 266,172				Mean:	Mean:
		Low BMI (n = 45,142)			0.39 kg/wk	15.2 kg
		Average BMI (n = 135,390)			0.38 kg/wk	15.0 kg
Country: USA	**Group Description:** Low income women	High BMI (n = 33,697)			0.35 kg/wk	13.8 kg
		Obese (n = 51,943)			0.29 kg/wk	11.6 kg
Author, year: Scholl et al., 1988	**Total Study N:** 696	Prepregnant relative weight (% ideal):				
		Underweight , < 90% (n = 183)				14.5 kg
Country: USA	**Group Description:** Adolescents Delivered at 37 weeks or more gestation	Ideal, 90-119% (n = 397)				14.6 kg
		Overweight, ≥ 120% (n = 116)				14.7 kg
Author, year: Scholl et al., 1995*	**Total Study N:** 274	Low gain < 34 kg/wk (n = 59)				10.2 kg
		Moderate gain (n = 138)				13.3 kg
Country: USA	**Group Description:** Low SES 12-29 years	Excessive gain > 0.68 kg/wk (n = 77)				20.0 kg
Author, year: Segel and McAnarney, 1994	**Total Study N:** 55	Rapid gain (n = 30)				> 0.40 kg/wk
		Average gain (n = 15)				0.23-0.40 kg /wk
		Slow gain (n = 10)				< 0.23 kg/wk
Country: USA	**Group Description:** Teens, Black					
Author, year: Siega-Riz et al., 1994*	**Total Study N:** 8,736	BMI < 19.8	1.60 kg	0.57 kg/wk	0.52 kg/wk	
		BMI 19.8-26	1.03 kg	0.56 kg/wk	0.54 kg/wk	
		BMI 26-29	0.74 kg	0.49 kg/wk	0.51 kg/wk	

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TABLE C-1A Maternal Weight Gain in Singleton Pregnancies (by trimester and total weight gain)

Study Description	Population Characteristics	Study Criteria	Weight Gain by Trimester 1st	2nd	3rd	Total Weight Gain
Country: USA		BMI > 30	0.78 kg	0.43 kg/wk	0.52 kg/wk	
Author, year: Siega-Riz et al., 1996	**Total Study N:** 9,651	Preterm deliveries (n = 517)	1.4 kg	0.51 kg/wk	0.50 kg/wk	
		Term deliveries (n = 7,072)	1.2 kg	0.53 kg/wk	0.53 kg/wk	
Country: USA	**Group Description:** 80% Hispanic					
Author, year: Stevens-Simon and McAnarney, 1992*	**Total Study N:** 141	Weight Gain:				
		Slow, < 0.28 kg/wk (n = 23)			0.16 kg/wk	6.50 kg
	Group Description: Teens, Black	Average, 0.28-0.45 kg/wk (n = 87)			0.32 kg/wk	13.3 kg
Country: USA		Rapid, >0.45 kg/wk (n = 31)			0.56 kg/wk	22.7 kg
Author, year: Soltani and Fraser, 2000*	**Total Study N:** 77	Normal weight (n = 29)				11.0 kg
		Overweight (n = 23)				11.9 kg
		Obese (n = 25)				9.7 kg
Country: UK						
Author, year: Takimoto, 2006*	**Total Study N:** 46,659					9.9 kg
Country: Japan	**Group Description:** Low risk deliveries					
Author, year: Tsukamoto et al., 2007	**Total Study N:** 3,071	BMI < 18 (n = 493)				10.5 kg
		BMI 18-24 (n = 2,301)				9.8 kg
		BMI > 24.0 (n = 277)				6.6 kg
Country: Japan						

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TABLE C-1A Maternal Weight Gain in Singleton Pregnancies (by trimester and total weight gain)

			Weight Gain by Trimester			
Study Description	**Population Characteristics**	**Study Criteria**	**1st**	**2nd**	**3rd**	**Total Weight Gain**
Author, year:	**Total Study N:**					Predicted weight
Villamor et al., 1998	432	Maternal Height				gain (mean):
		140-155 cm				0.39 kg/wk
Country:		156-160 cm				0.48 kg/wk
Isreal		161-165 cm				0.44 kg/wk
		166-170 cm				0.47 kg/wk
		171-180 cm				0.69 kg/wk

*Indicates that study is included in the systematic literature review conducted by Viswanathan et al., 2008.
NOTES: First trimester, 0 - 13 weeks; Second trimester, 13 - 26 weeks; Third trimester, 27-40 weeks. PPWR = Postpartum weight retention.

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TABLE C-1B Maternal Weight Gain in Singleton Pregnancies (by percent of BMI and gain category)

Study Description	Population Characteristics	Weight/BMI Category	Weight Gain (percent of n)
Author, year:	**Total Study N:**		*Gained <6.8 kg:*
Cogswell et al., 1995	53,541	BMI 19.8-26	6%
		BMI > 26-29	11%
Country:	**Group Description:**	BMI > 29	25%
USA	Low income women		*Gained 6.8-8.6 kg:*
	BMI 19.8-26 (n = 33,809)	BMI 19.8-26	6%
	BMI > 26-29 (n = 7,661)	BMI > 26-29	8%
	BMI > 29 (n = 12,071)	BMI > 29	10%
			Gained 9.1-10.9 kg:
		BMI 19.8-26	11%
		BMI > 26-29	13%
		BMI > 29	13%
			Gained 11.4-13.2 kg:
		BMI 19.8-26	14%
		BMI > 26-29	13%
		BMI > 29	13%
			Gained 13.6-15.4 kg:
		BMI 19.8-26	17%
		BMI > 26-29	16%
		BMI > 29	12%
			Gained 15.9-17.7 kg:
		BMI 19.8-26	14%
		BMI > 26-29	11%
		BMI > 29	8%
			Gained >18.2 kg:
		BMI 19.8-26	31%
		BMI > 26-29	28%
		BMI > 29	19%
Author, year:	**Total Study N:**	Black women, pregravid BMI:	*Gained within IOM recommendations:*
Hickey et al., 1993*	1,168	Low, < 19.8 (n = 221)	37.1%
		Normal, 19.8-26.0 (n = 350)	30.9%
Country:	**Group Description:**	High, > 26.0-29.0 (n = 84)	27.4%
USA	Low income, high risk women		

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TABLE C-1B Maternal Weight Gain in Singleton Pregnancies (by percent of BMI and gain category)

Study Description	Population Characteristics	Weight/BMI Category	Weight Gain (percent of n)
	Black (n = 803)	White women, pregravid BMI:	
	White (n = 365)	Low, < 19.8 (n = 118)	37.3%
		Normal, 19.8-26.0 (n = 168)	35.7%
		High, > 26.0-29.0 (n = 29)	20.7%
Author, year:	**Total Study N:**		*Gain of less than 0.9 kg:*
Kiel et al., 2007*	120,170	BMI 30-34.9	3%
		BMI 35-39.9	8%
Country:	**Group Description:**	BMI ≥ 40	15%
USA	Obese women		*Gain of 0.9-6.4 kg:*
	BMI 30-34.9 (n = 70,536)	BMI 30-34.9	15%
	BMI 35-39.9 (n = 30,609)	BMI 35-39.9	22%
	BMI ≥ 40 (n = 19,025)	BMI ≥ 40	25%
			Gain of 6.5-11.4 kg:
		BMI 30-34.9	26%
		BMI 35-39.9	27%
		BMI ≥ 40	25%
			Gain of > 11.4 kg:
		BMI 30-34.9	56%
		BMI 35-39.9	43%
		BMI ≥ 40	35%
Author, year:	**Total Study N:**		*Gain of 0.28 kg/wk:*
Nohr et al., 2007*	62,167	BMI < 18.5	15.3%
		BMI 18.5-24.9	11.5%
Country:		BMI 25-29.9	19.6%
Sweden		BMI 30+	36.1%
			Gain of 0.28-0.68 kg/wk:
		BMI < 18.5	71.0%
		BMI 18.5-24.9	72.2%
		BMI 25-29.9	62.1%
		BMI 30+	49.6%
			Gain of > 0.68 kg/wk:
		BMI < 18.5	13.7
		BMI 18.5-24.9	16.3

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TABLE C-1B Maternal Weight Gain in Singleton Pregnancies (by percent of BMI and gain category)

Study Description	Population Characteristics	Weight/BMI Category	Weight Gain (percent of n)
		BMI 25-29.9	18.3
		BMI 30+	14.2
Author, year:	**Total Study N:**		*Gained < 0.23 kg/wk:*
Schieve et al., 2000*	3,511	Low BMI	4%
		Average BMI	5%
Country:		High BMI	23%
USA			*Gained 0.23-0.68 kg/wk:*
		Low BMI	78%
		Average BMI	74%
		High BMI	63%
			Gained > 0.68 kg/wk:
		Low BMI	18%
		Average BMI	21%
		High BMI	14%
Author, year:	**Total Study N:**	Low and Normal BMIs	*Gain of < 0.27 kg/wk:*
Stotland et al., 2006*	15,101		11%
			Gain of 0.27-0.52 kg/wk:
Country:			68.2%
USA			*Gain of > 0.27 kg/w:*
			21.1%
Author, year:	**Total Study N:**		*Actual Gain < 10 kg:*
Taffel et al., 1993	1,707	BMI < 19.8	13%
		BMI 19.8-26	16%
Country:	**Group Description:**	BMI > 26	38%
USA	BMI < 19.8 (n = 379)		*Actual Gain 10-12.3 kg:*
	BMI 19.8-26 (n = 1,024)	BMI < 19.8	21%
	BMI > 26 (n = 304)	BMI 19.8-26	19%
		BMI > 26	19%
			Actual Gain > 12.3 kg:
		BMI < 19.8	66%
		BMI 19.8-26	64%
		BMI > 26	42%

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TABLE C-1B Maternal Weight Gain in Singleton Pregnancies (by percent of BMI and gain category)

Study Description	Population Characteristics	Weight/BMI Category	Weight Gain (percent of n)
Author, year: Wen et al., 1990*	**Total Study N:** 17,149	Weights (kg): < 50, 50-60, 61-72, 73-84, ≥ 85.	Gained < 0.24 kg/wk: 12% Gained 0.24-0.57 kg/wk: 54% Gained 0.58-0.74 kg/wk: 19% Gained ≥ 0.75 kg/wk: 14%
Country: USA	**Group Description:** Black and White indigent women		

*Indicates that study is included in the systematic literature review conducted by Viswanathan et al., 2008.

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TABLE C-2 Maternal Weight Gain in Twin and Triplet Pregnancies (by trimester and total weight gain)

Author (Year)	Population Characteristics	Study Criteria	Weight Gain by Trimester 1st	2nd	3rd	Total Weight Gain
Twins						
Author, year: Brown and Schloesser, 1990	**Total Study N:** 203,768	Prepregnancy Weight Status:				
		Twins				
		Underweight				17.9 kg
	Group Description:	Normal weight				16.9 kg
Country:	Twins (n = 1,984)	Overweight				17.0 kg
USA	Singletons (n = 201,784)	Obese				15.2 kg
		Very obese				12.7 kg
		Singletons				
		Underweight				13.5 kg
		Normal weight				13.8 kg
		Overweight				13.9 kg
		Obese				12.6 kg
		Very obese				11.0 kg
Author, year: Fenton et al., 1994	**Total Study N:** 100				*To 34 wks*	
		Birth weight:				
		> 3 kg			15.50 kg	
Country:	**Group Description:**	Intermediate			13.37 kg	
Canada	Normal weight women (BMI = 19-28), aged 20-35	SGA			14.66 kg	
Author, year: Lantz et al., 1996	**Total Study N:** 163	BMI Categories:				
		Overall				14.7 kg
Country:		< 19.8				17.7 kg
USA	**Group Description:**	19.8-26.0				15.6 kg
	White women (n = 112)	> 26.0				14.8 kg
	Black women (n = 51)					
Author, year: Luke et al., 1991	**Total Study N:** 275	Parity and Age:				
		Primiparas				
		18-24 years				19.4

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TABLE C-2 Maternal Weight Gain in Twin and Triplet Pregnancies (by trimester and total weight gain)

Author (Year)	Population Characteristics	Study Criteria	Weight Gain by Trimester 1st	2nd	3rd	Total Weight Gain
Country: USA	**Group Description:** White women, delivered twins (term and preterm)	25-34 years				18.5
		35-44 years				22.8
		Multiparas				
		18-24 years				22.3
		25-34 years				20.4
		35-44 years				16.8
Author, year: Luke et al., 1992	**Total Study N:** 270	Pregravid BMI and birth weight categories:		*Before 24 wks*	*After 24 wks*	
Country: USA		*All BMIs*				
		< 2,500 g (n = 105)		0.41 kg/wk	0.49 kg/wk	15.01 kg
		≥ 2,500 g (n = 58)		0.43 kg/wk	0.65 kg/wk	19.28 kg
		Underweight BMI < 19.8				
		< 2,500 g (n = 26)		0.39 kg/wk	0.49 kg/wk	14.74 kg
		≥ 2,500 g (n = 14)		0.44 kg/wk	0.66 kg/wk	19.78 kg
		Normal weight BMI 19.8-26.0				
		< 2,500 g (n = 63)		0.39 kg/wk	0.51 kg/wk	14.70 kg
		≥ 2,500 g (n = 36)		0.43 kg/wk	0.65 kg/wk	19.01 kg
		Overweight BMI > 26.0				
		< 2,500 g (n = 16)		0.51 kg/wk	0.42 kg/wk	16.65 kg
		≥ 2,500 g (n = 8)		0.42 kg/wk	0.68 kg/wk	19.73 kg
Author, year: Luke and Leurgans, 1996	**Total Study N:** 924	Ideal outcome[a] (n = 148)				20.3 kg
		Non-ideal outcome (n = 776)				18.6 kg
Country: USA	**Group Description:** Data collected during Twins Days Festival					
Author, year: Pederson et al., 1989	**Total Study N:** 217	*Optimum Outcome*[b]				Mean:
		Percent ideal body weight:				
		< 90%				19.1 kg
Country:	**Group Description:**	90-120%				21.8 kg

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TABLE C-2 Maternal Weight Gain in Twin and Triplet Pregnancies (by trimester and total weight gain)

Author (Year)	Population Characteristics	Study Criteria	Weight Gain by Trimester 1st	2nd	3rd	Total Weight Gain
USA	Mostly white women, all >18 years of age	> 120%				15.0 kg
		Less-than-optimum Outcome				
		Percent ideal body weight:				
		< 90%				18.2 kg
		90-120%				17.3 kg
		> 120%				12.3 kg
Author, year:	**Total Study N:**	Prepregnancy BMI:				
Yeh and Shelton, 2007	1,342	< 19.8 (n = 127)				19.1 kg
		19.8-26.0 (n = 649)				18.1 kg
Country:		26.1-29.0 (n = 185)				17.2 kg
USA		> 29.0 (n = 381)				14.3 kg
Triplets						
Author, year:	**Total Study N:**	Prepregnancy BMI:				Median:
Eddib et al., 2007	56	< 19.8 (n = 5)				15.5 kg
		19.8-26.0 (n = 24)				21.8 kg
Country:	**Group Description:**	> 26.0 (n = 27)				15.0 kg
USA	Triplet gestations					
		Adverse Maternal Outcome present (yes/no)?				
		Gestational diabetes:				
		No (n = 53, BMI 25.8)				17.3 kg
		Yes (n = 3, BMI 37.6)				15.5 kg
		Gestational hypertension:				
		No (n = 51, BMI 25.8)				17.7 kg
		Yes (n = 5, BMI 36.8)				15.5 kg
		Preeclampsia:				
		No (n = 49, BMI 25.8)				15.9 kg
		Yes (n = 7, BMI 28.1)				17.3 kg
Author, year:	**Total Study N:**					19.91 kg
Luke et al., 1995	38	Gestation categories:		*Before 24 wks*	*After 24 wks*	

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TABLE C-2 Maternal Weight Gain in Twin and Triplet Pregnancies (by trimester and total weight gain)

Author (Year)	Population Characteristics	Study Criteria	Weight Gain by Trimester 1st	2nd	3rd	Total Weight Gain
		27-30 wks		0.51kg/wk	0.77kg/wk	
Country:		31-34 wks		0.50kg/wk	1.08kg/wk	
USA		35-37 wks		0.44kg/wk	0.90kg/wk	
		Total		0.48kg/wk	0.96kg/wk	

[a]Ideal outcome is defined as mean birth weight between 2,000 and 2,800 g and gestation between 35-38 weeks.
[b]Optimum outcome was defined as a pregnancy resulting in two living infants with birth weights ≥ 2,500g, Apgar scores ≥ 7 at 5 minutes, and Dubowitz scores > 37 weeks.
NOTE: First trimester, 0 - 13 weeks; Second trimester, 13 - 26 weeks; Third trimester, 27-40 weeks.

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TABLE C-3A Summary of Adjusted and Unadjusted* Rates of Maternal Weight Gain by Trimesters, by Pregravid BMI Status for Mothers of Twins at Gestational Ages 37-42 Wk, and with Average Twin Birth weight > 2,500 g

	Rates of Weight Gain					
	2-13 wks		14-26 wks		27-delivery	
Pregravid BMI	**kg/wk**	**lb/wk**	**kg/wk**	**lb/wk**	**kg/wk**	**lb/wk**
Normal weight[a] (n = 409)	0.33 ± 0.02 (0.32 ± 0.32)*	0.73 ± 0.05 (0.71 ± 0.70)*	0.77 ± 0.02 (0.78 ± 0.24)*	1.70 ± 0.04 (1.72 ± 0.54)*	0.67 ± 0.01 (0.68 ± 0.27)*	1.47 ± 0.03 (1.49 ± 0.60)*
Overweight[b] (n= 154)	0.20 ± 0.04 (0.20 ± 0.32)*	0.43 ± 0.09 (0.43 ± 0.70)*	0.72 ± 0.03 (0.71 ± 0.20)*	1.58 ± 0.06 (1.56 ± 0.43)*	0.61 ± 0.02 (0.61 ± 0.28)*	1.35 ± 0.05 (1.35 ± 0.62)*
Obese[c] (n = 143)	0.18 ± 0.04 (0.20 ± 0.44)*	0.39 ± 0.10 (0.44 ± 0.97)*	0.45 ± 0.03 (0.44 ± 0.27)*	1.0 ± 0.07 (0.98 ± 0.60)*	0.58 ± 0.03 (0.57 ± 0.33)*	1.28 ± 0.06 (1.26 ± 0.73)*

NOTES: Results are presented as least square means ± SEM from models controlling for diabetes and gestational diabetes, preeclampsia, smoking during pregnancy, primiparity, and placental membranes (monochorionicity and missing chorionicity). Rates for the 1st trimester are calculated post-conception, using 2 post-menstrual weeks as the average for time of conception. Results in parentheses are the unadjusted means ± SD.

[a] BMI = 18.5-24.9 kg/m^2
[b] BMI = 25.0-29.9 kg/m^2
[c] BMI = ≥ 30 kg/m^2

SOURCE: Historical cohort of twin births delivered at John Hopkins Hospital, Baltimore, Jackson Memorial Hospital, Miami, Medical University of South Carolina, Charleston, and University of Michigan, Ann Arbor provided by Barbara Luke, Sc.D., M.P.H., R.D., and Mary L. Hediger, Ph.D. For more details on this historical cohort, see Luke et al. (2003).

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TABLE C-3B Summary of Adjusted and Unadjusted* Cumulative Gain by Trimesters, by Pregravid BMI Status for Mothers of Twins at Gestational Ages 37-42 Wk, and with Average Twin Birth weight > 2,500 g

	Cumulative Weight Gain					
	To 13 wks		To 26 wks		To 37-42 wks	
Pregravid BMI	**kg/wk**	**lb/wk**	**kg/wk**	**lb/wk**	**kg/wk**	**lb/wk**
Normal weight[a] (n = 409)	3.6 ± 0.3 (3.5 ± 3.5)*	8.0 ± 0.6 (7.8 ± 7.7)*	13.1 ± 0.3 (13.2 ± 4.9)*	28.8 ± 0.6 (29.1 ± 10.7)*	20.9 ± 0.3 (21.0 ± 6.1)*	45.9 ± 0.7 (46.2 ± 13.4)*
Overweight[b] (n= 154)	2.1 ± 0.4 (2.2 ± 3.5)*	4.7 ± 1.0 (4.8 ± 7.7)*	11.3 ± 0.4 (11.1 ± 5.5)*	24.8 ± 1.0 (24.4 ± 12.1)*	18.9 ± 0.5 (18.7 ± 7.0)*	41.6 ± 1.1 (41.1 ± 15.5)*
Obese[c] (n = 143)	2.0 ± 0.5 (2.2 ± 4.8)*	4.3 ± 1.1 (4.8 ± 10.6)*	8.5 ± 0.4 (8.4 ± 5.9)*	18.8 ± 1.0 (18.6 ± 12.9)*	15.7 ± 0.5 (15.4 ± 7.2)*	34.6 ± 1.2 (34.0 ± 15.9)*

NOTES: Results are presented as least square means ± SEM from models controlling for diabetes and gestational diabetes, preeclampsia, smoking during pregnancy, primiparity, and placental membranes (monochorionicity and missing chorionicity). Total cumulative gain is also adjusted for length of gestation. Results in parenthese are the unadjusted means ± SD.

[a] BMI = 18.5-24.9 kg/m^2
[b] BMI = 25.0-29.9 kg/m^2
[c] BMI = ≥ 30 kg/m^2

SOURCE: Historical cohort of twin births delivered at John Hopkins Hospital, Baltimore, Jackson Memorial Hospital, Miami, Medical University of South Carolina, Charleston, and University of Michigan, Ann Arbor provided by Barbara Luke, Sc.D., M.P.H., R.D., and Mary L. Hediger, Ph.D. For more details on this historical cohort, see Luke et al. (2003).

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TABLE C-3C Interquartile Ranges of Rates of Maternal Weight Gain by Trimesters, by Pregravid BMI Status for Mothers of Twins at Gestational Ages 37-42 Weeks, and with Average Twin Birth weight > 2,500 g

	Rates of Weight Gain					
	2-13 wks		14-26 wks		27-delivery	
Pregravid BMI	**kg/wk**	**lb/wk**	**kg/wk**	**lb/wk**	**kg/wk**	**lb/wk**
Normal weight[a] (n = 409)	0.12 – 0.49	0.27 – 1.07	0.64 – 0.94	1.40 – 2.06	0.50 – 0.83	1.09 – 1.83
Overweight [b] (n= 154)	0.03 – 0.39	0.06 –0.85	0.57 – 0.87	1.25 – 1.91	0.42 – 0.81	0.92 – 1.79
Obese [c] (n = 143)	0.08 – 0.34	0.18 – 0.76	0.24 – 0.63	0.54 – 1.39	0.34 – 0.70	0.75 – 1.55

NOTES: Results are presented as the 25th – 75th percentiles for the rates or cumulative gain over the interval. Rates for the 1st trimester are calculated post-conception, using 2 post-menstrual weeks as the average for time of conception.

[a] BMI = 18.5-24.9 kg/m^2
[b] BMI = 25.0-29.9 kg/m^2
[c] BMI = ≥ 30 kg/m^2

SOURCE: Historical cohort of twin births delivered at John Hopkins Hospital, Baltimore, Jackson Memorial Hospital, Miami, Medical University of South Carolina, Charleston, and University of Michigan, Ann Arbor provided by Barbara Luke, Sc.D., M.P.H., R.D., and Mary L. Hediger, Ph.D. For more details on this historical cohort, see Luke et al. (2003).

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TABLE C-3D Interquartile Ranges of Cumulative Gain by Trimesters, by Pregravid BMI Status for Mothers of Twins at Gestational Ages 37-42 Weeks, and with Average Twin Birth weight > 2,500 g

	Cumulative Weight Gain					
	To 13 wks		**To 26 wks**		**To 37-42 wks**	
Pregravid BMI	**kg/wk**	**lb/wk**	**kg/wk**	**lb/wk**	**kg**	**lbs**
Normal weight[a] (n = 409)	1.4 – 5.4	3.0 – 11.8	10.0 – 16.4	22.0 – 36.0	16.8 – 24.5	37 – 54
Overweight [b] (n= 154)	0.3 – 4.3	0.7 – 9.4	7.7 – 14.1	17.0 – 31.0	14.1 – 22.7	31 – 50
Obese [c] (n = 143)	0.9 – 3.8	2.0 – 8.4	4.9 – 11.4	10.7 – 25.0	11.4 – 19.1	25 – 42

NOTES: Results are presented as the 25th – 75th percentiles for the rates or cumulative gain over the interval.

[a] BMI = 18.5-24.9 kg/m^2
[b] BMI = 25.0-29.9 kg/m^2
[c] BMI = ≥ 30 kg/m^2

SOURCE: Historical cohort of twin births delivered at John Hopkins Hospital, Baltimore, Jackson Memorial Hospital, Miami, Medical University of South Carolina, Charleston, and University of Michigan, Ann Arbor provided by Barbara Luke, Sc.D., M.P.H., R.D., and Mary L. Hediger, Ph.D. For more details on this historical cohort, see Luke et al. (2003).

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TABLE C-4 Maternal Weight Gain and Body Composition

Author (Year)	Population/ Study Characteristics	Study Criteria	Body Composition Measurements (FFM, FM, TBW)	Pregravid Weight/ Body Comp	Weight Gain/Body Composition by Trimester			Total Weight Gain	Postpartum Weight/ Body Composition
					1st	2nd	3rd		
Author, year: Bronstein et al., 1996	**Total Study N:** 33	Pregnant women (n = 16)	FFM at 31-35 weeks: 55.5 kg FM at (31-35 weeks: 32.8 kg	75.7 kg					
Country: USA	**Group Description:** Non-pregnant and pregnant women [BC by densitometry}	Non-pregnant women (n = 17)	FFM at study: 50.1 kg FM at study: 29.8 kg	79.9 kg					
Author, year: Butte et al., 2003*	**Total Study N:** 63		GWG		*9 wks*	*22 wks*	*36 wks*		
		Low BMI		49.9 kg	51.9 kg	57.7 kg	63.0 kg	15.0 kg	
		Normal BMI		59.3 kg	60.2 kg	65.1 kg	72.2 kg	14.5 kg	
	Group Description:	High BMI		77.3 kg	81.8 kg	85.8 kg	93.8 kg	17.9 kg	
	BMI Categories:		TBW						
Country: USA	Low, BMI < 19.8 (n = 17)	Low BMI		28.7 kg	28.7 kg	32.2 kg	34.7 kg		
		Normal BMI		32.0 kg	31.6 kg	34.1 kg	38.7 kg		
	Normal, BMI 19.8-26 (n = 34)	High BMI		35.6 kg	35.7 kg	37.9 kg	42.8 kg		
			FFM						
	High, BMI > 26 (n = 12)	Low BMI		39.0 kg	39.6 kg	42.6 kg	46.9 kg		
		Normal BMI		43.1 kg	43.0 kg	46.0 kg	51.4 kg		
		High BMI		47.8 kg	48.9 kg	51.0 kg	56.9 kg		
	[BC by TBK 4 compartment method]		FM						
		Low BMI		10.9 kg	12.4 kg	15.1 kg	16.1 kg		
		Normal BMI		16.8 kg	17.2 kg	19.1 kg	21.0 kg		
		High BMI		309 kg	33.0 kg	34.8 kg	37.0 kg		
			Net GWG						
		Low BMI						11.6 kg	
		Normal BMI						11.0 kg	
		High BMI						14.1 kg	

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TABLE C-4 Maternal Weight Gain and Body Composition

Author (Year)	Population/ Study Characteristics	Study Criteria	Body Composition Measurements (FFM, FM, TBW)	Pregravid Weight/ Body Comp	Weight Gain/Body Composition by Trimester 1st	2nd	3rd	Total Weight Gain	Postpartum Weight/ Body Composition
			GWG per wk						
		Low BMI						0.40 kg/wk	
		Normal BMI						0.37 kg/wk	
		High BMI						0.45 kg/wk	
Author, year: Catalano et al., 1998	**Total Study N:** 16	Ab GT (n = 6)	Pregravid measurements		12-14 wks *Weight:*		34-36 wks *Weight:*		
		Controls (n = 10)		59.2 kg	60.5 kg		71.6 kg		
	Group Description:			56.5 kg	59.9 kg		70.0 kg		
Country: USA	Women with normal and abnormal glucose tolerance (Ab GT); Ab GT (n = 6), Controls (n = 10) [BC by densitometry]		*FFM:* 46.4 kg 46.3 kg *FM:* 12.8 kg 10.2 kg		*FFM:* 46.4 kg 46.8 kg *FM:* 14.1 kg 13.2 kg		*FFM:* 53.4 kg 53.6 kg *FM:* 18.2 kg 16.5 kg		
			Sum of 7 site skinfolds: 88.7 mm 74.0 mm		*Sum of 7 site skinfolds:* 93.5 mm 89.0 mm		*Sum of 7 site skinfold:* 108.9 mm 109.8mm		
Author, year: Ehrenberg et al., 2003	**Total Study N:** 52	Lean (n = 17)		56.2 kg				12.3 kg	*FM gain* 4.7 kg
		Obese (n = 35)		67.6 kg				13.0 kg	4.2 kg
	Group Description:	GDM/obese (n = 14)		69.7 kg				12.0 kg	3.2 kg
Country: USA	Women with GDM (lean, n = 5; obese, n = 14)	CTLl/obese (n = 21)		66.3 kg				13.7 kg	4.8 kg
	Women with normal								

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TABLE C-4 Maternal Weight Gain and Body Composition

Author (Year)	Population/ Study Characteristics	Study Criteria	Body Composition Measurements (FFM, FM, TBW)	Pregravid Weight/ Body Comp	Weight Gain/Body Composition by Trimester 1st	2nd	3rd	Total Weight Gain	Postpartum Weight/ Body Composition
	glucose tolerance (lean, n = 12; obese, n = 21) [Underwater weighing]								
Author, year: Forsum et al., 2006 **Country:** Sweden	**Total Study N:** 23 **Group Description:** Adults [BC of mothers by 2 compartment method (TBW)]	Healthy women, parity 0-2 and planning pregnancy					*32 wks:*		*2 wks pp:*
			Body weight	67.4 kg			79.3 kg	18.1 kg	73.5 kg
			Total body fat	22.6 kg			27.0 kg	3.9 kg	26.7 kg
			FFM	44.6 kg					
Author, year: Kopp-Hoolihan et al., 1999 **Country:** USA	**Total Study N:** 9 **Group Description:** Healthy, non-smokers planning a pregnancy				*8-10 wks*	*24-26 wks*	*34-36 wks*		4-6 wks
			Body weight	64.7 kg	64.9 kg	72.1 kg	75.9 kg		68.0 kg
			TBW	33.5 kg	33.9 kg	36.5 kg	39.1 kg		33.8 kg
			TBW/FFM	0.72	0.73	0.74	0.74		0.72
			TBBM	2525 g	---	---	---		2463 g
			FFM	46.3 kg	46.7 kg	49.7 kg	52.8 kg		46.7 kg
			FM	20.2 kg	20.3 kg	24.4 kg	24.3 kg		22.0 kg
			Mean wt gain	---	0.19 kg	7.23 kg	3.76 kg	11.2 kg	---
			Mean fat gain	---	0.10 kg	4.10 kg	-0.10 kg	4.20 kg	---
			% wt as FM	---	53.00	57.00	-3.00	---	---
Author,	**Total Study N:**	BMI/Gain	Body water gain:		63.4 kg				

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TABLE C-4 Maternal Weight Gain and Body Composition

Author (Year)	Population/ Study Characteristics	Study Criteria	Body Composition Measurements (FFM, FM, TBW)	Pregravid Weight/ Body Comp	Weight Gain/Body Composition by Trimester 1st	2nd	3rd	Total Weight Gain	Postpartum Weight/ Body Composition
year:	196	Categories		Fat gain:					
Lederman et al., 1997*	**Group Description:**	*BMI < 19.8*							
	Hispanic, White and	Total (n=21)	6.1 L	4.8 kg					12.60 kg
Country:	Black women	< rec (n=6)	6.4 L	0.6 kg					7.90 kg
USA	Aged 18-35 years	rec (n=7)	5.9 L	6.0 kg					12.60 kg
	[BC by 4-	> rec (n=8)	6.1 L	6.9 kg					16.10 kg
	compartment method]	*BMI 19.8-26*							
		Total (n=118)	7.0 L	3.9 kg					12.2 kg
		< rec (n=31)	6.2 L	1.3 kg					8.60 kg
		rec (n=46)	6.9 L	3.8 kg					12.1 kg
		> rec (n=41)	7.6 L	6.0 kg					15.2 kg
		BMI >26-29							
		Total (n=29)	7.8 L	2.8 kg					11.0 kg
		< rec (n=7)	6.9 L	0.3 kg					8.50 kg
		rec (n=9)	5.7 L	2.8 kg					9.10 kg
		> rec (n=13)	9.7 L	4.2 kg					13.6 kg
		BMI > 29							
		Total (n=28)	7.3 L	0.2 kg					8.70 kg
		< rec (n=7)	7.8 L	-5.2kg					3.20 kg
		rec (n=6)	6.0 L	-0.6kg					6.9 kg
		> rec (n=15)	7.6 L	3.1 kg					12.0 kg
Author, year:	**Total Study N:**	Pregnant women				*Week 14*	*Week 32*		*2 wk pp:*
Lof and	17		Body weight	66.6 kg		68.4 kg	77.3 kg	16.7 kg	71.5 kg
Forsum,			TBW	31.5 kg		32.5 kg	38.1 kg		33.6 kg
2004	**Group Description:**		FFM	43.9 kg		44.9 kg	51.0 kg		45.7 kg
	Adults		FM	22.7 kg		23.5 kg	26.3 kg		25.8 kg
Country:	[BC by deuterium								
Sweden	dilution underwater								

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TABLE C-4 Maternal Weight Gain and Body Composition

Author (Year)	Population/ Study Characteristics	Study Criteria	Body Composition Measurements (FFM, FM, TBW)	Pregravid Weight/ Body Comp	Weight Gain/Body Composition by Trimester 1st	2nd	3rd	Total Weight Gain	Postpartum Weight/ Body Composition
	weighing]								
Author, year: Okereke et al., 2004	**Total Study N:** 15		WT		*12-14 wks*		*34-36 wks*		
		NGT (n =8)		71.4 kg	73.2 kg		84.1 kg		
		GDM (n = 7)		78.2 kg	79.6kg		89.5 kg		
	Group Description: Obese women		FFM						
		NGT		49.4 kg	50.1 kg		55.2 kg		
Country: USA		GDM		51.7 kg	52.1 kg		56.8 kg		
			FM						
		NGT		22.0 kg	23.1 kg		28.9 kg		
		GDM		26.5 kg	27.5 kg		32.7 kg		
Author, year: Stevens-Simon et al., 1997	**Total Study N:** 108					*15-16 wks*	*34-35 wks*		
		Teens < 16y	WT	59.7 kg		61.6 kg	68.3 kg		
	Group Description: Teens, Black		LBM			46.6 kg	50.8 kg		
			FM			15.0 kg	17.50 kg		
Country: USA	[All weights are net wt Total body potassium]	Teens 16-18 yrs	WT	61.7 kg		63.9 kg	69.6 kg		
			LBM			47.8 kg	51.2 kg		
			FM			16.1 kg	18.3 kg		

*Indicates that study is included in the systematic literature review conducted by Viswanathan et al., 2008.
NOTE: First trimester, 0 - 13 weeks; Second trimester, 13 - 26 weeks; Third trimester, 27-40 weeks. GDM= gestational diabetes mellitus; FFM= fat-free mass; FM= fat mass; TBW=; NGT= Normal glucose tolerance; WT= weight.

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TABLE C-5 Mean Weights and Percentiles for Placentas (singletons, twins, and triplets)

	90th Percentile			Mean Placental Weight			10th Percentile			Cases (*n*)		
Gestational age (weeks)	**Singletons**	**Twins**	**Triplets**	**Singletons**	**Twins**	**Triplets**	**Singletons**	**Twins**	**Triplets**	**Singletons**	**Twins**	**Triplets**
19	—	263	—	—	212	—	—	161	—	—	2	—
20	—	270	285	—	218	253	—	166	226	—	3	3
21	172	286	320	143	231	284	114	176	257	3	2	2
22	191	310	345	157	251	319	122	191	289	6	5	2
23	211	343	400	172	276	361	133	210	331	7	2	3
24	233	382	445	189	307	406	145	232	371	9	3	5
25	256	426	498	208	341	456	159	257	408	19	5	6
26	280	475	558	227	380	509	175	284	444	14	4	6
27	305	528	630	248	421	564	192	314	480	9	8	4
28	331	584	697	270	464	621	210	345	516	16	7	5
29	357	641	772	293	509	679	229	377	553	11	12	6
30	384	700	849	316	554	738	249	409	591	12	17	10
31	411	758	925	340	600	797	269	441	631	14	13	15
32	438	815	1,000	364	644	855	290	472	674	24	29	7
33	464	870	1,072	387	687	911	311	503	719	30	27	14
34	491	923	1,139	411	727	965	331	531	768	32	53	43
35	516	971	1,200	434	764	1,017	352	558	821	44	52	33
36	542	1,014	1,253	457	798	1,065	372	582	878	36	66	19
37	566	1,051	1,297	478	827	1,108	391	602	940	32	58	8
38	589	1,082	1,330	499	850	1,147	409	619	1,007	62	54	5
39	611	1,105	—	519	868	—	426	631	—	103	38	—
40	632	1,118	—	537	879	—	442	639	—	193	47	—
41	651	1,123	—	553	882	—	456	642	—	87	12	—

SOURCES: *Pediatric Pathology & Laboratory* Medicine by Pinar H., C. J. Sung, C. E. Oyer and D. B. Singer. Copyright 1996 by Informa Clinical Medicine - Journals. Reproduced with permission of Informa Clinical Medicine - Journals via Copyright Clearance Center; Reprinted from Pinar H., M. Stephens, D. B. Singer, T. K. Boyd, S. M. Pflueger, D. L. Gang, D. J. Roberts and C. J. Sung. 2002. Triplet placentas: reference values for weights. *Pediatric and Developmental Pathology* 5(5): 495-498 with kind permission from Springer Science and Business Media.

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TABLE C-6 DNA, Glycogen, and Lipid Content in Placentas from Normal and Diabetic Human Pregnancies

		Placental DNA		Placental Glycogen			Placental Lipid Triglycerides		
Pregnancy	**Placental Weight (g)**	**(mg/g)**	**(g/ total placenta)**	**(mg/g)**	**(mg/ mg DNA)**	**(g/ total placenta)**	**(µmol/g)**	**(µmol/mg DNA)**	**(mmol/ total placenta)**
Normal (n = 50)	550 ± 28	2.54 ± 0.13	1.40 ± 0.07	8.4 ± 0.5	3.3 ± 0.3	4.67 ± 0.29	2.51 ± 0.16	0.98 ± 0.15	1.38 ± 0.10
Gestational diabetes (n = 23)	664 ± 60	2.58 ± 0.24	1.71[a] ± 0.12	9.9 ± 0.8	3.8 ± 0.4	6.57[a] ± 0.60	3.32[a] ± 0.31	1.29 ± 0.20	2.20[a] ± 0.25
Insulin-treated diabetes (n = 12)	615 ± 110	3.04 ± 0.32	1.87[a] ± 0.20	14.9[b] ± 1.9	4.9[a] ± 0.3	9.16[b] ± 0.88	4.56[b] ± 0.70	1.50 ± 0.16	2.80[b] ± 0.41

NOTE: Values in the table are means ± SE for number of patients indicated in parentheses.

[a]Significant difference from value for normal pregnancy ($p < 0.05$)
[b]Significant difference from value for normal pregnancy ($p < 0.01$)

SOURCE: Modified from Diamant et al., 1982. This information was published in the *American Journal of Obstetrics and Gynecology*, Vol 144, Diamant Y. Z., B. E. Metzger, N. Freinkel and E. Shafrir, Placental lipid and glycogen content in human and experimental diabetes mellitus, Pages 5-11, Copyright Elsevier (1982).

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Appendix D

Summary of Determinants of Gestational Weight Gain

Table D-1 summarizes the literature that is referenced and discussed in Chapter 4, *Determinants of Gestational Weight Gain*.

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TABLE D-1 Summary of Literature on Determinants of Gestational Weight Gain

Determinants	Findings/Interpretations	Comments	References
Societal/Institutional			
Media	The committee was unable to identify studies that examined specifically the media's influence on gestational weight gain.	Media may exert its effects on gestational weight gain indirectly by influencing prepregnancy BMI and other biological determinants, as well as eating habits and sedentary behaviors that are established well before pregnancy.	Gortmaker et al., 1996 Gortmaker et al., 1999 Robinson, 1999 Kunkel, 2001 Hastings et al., 2003 Epstein et al., 2008
Culture and Acculturation	The committee was unable to identify studies that examined specifically the effects of culture and acculturation factors on gestational weight gain.	Cultural norms and beliefs can influence dietary behavior and physical activities, thereby affecting energy balance and gestational weight gain. Acculturation is generally associated with more unhealthy behaviors, including dietary intake, and higher rates of overweight and obesity.	Ventura and Taffel, 1985 Scribner and Dwyer, 1989 Cabral et al., 1990 Kleinman et al., 1991 Rumbaut and Weeks, 1996 Singh and Yu, 1996 Fuentes-Afflick and Lurie, 1997 Schaffer et al., 1998 Jones and Bond, 1999 King, 2000 Callister and Birkhead, 2002 Lizarzaburu and Palinkas, 2002 Hubert et al., 2005 Baker and Hellerstedt, 2006 Hernandez-Valero et al., 2007 Fuentes-Afflick and Hessol, 2008
Health Services	The committee found insufficient evidence to evaluate the influence of prenatal weight gain advice on actual gestational weight gain.	Studies limited by self-selection bias, recall bias, differences in time during gestation when nutrition advice was given, variation in content and frequency of advice, the pairing of advice with other food or nonfood interventions, individual and social characteristics of the provider as contrasted with those of the pregnant woman, and racial-ethnic and socioeconomic disparities in weight gain advice given to women.	Rush, 1981 Orstead et al., 1985 Olds et al., 1986 Bruce and Tchabo, 1989 Brown et al., 1992 Morris et al., 1993 Hickey, 2000
Policy	Examples include IOM guidelines, WIC programs, and policy recommendations to restrict food/beverage marketing to young children. IOM guidelines appear to influence what women believe to be	The impact of the IOM guidelines on actual gestational weight gain may be limited in part because many health professionals are providing no or inappropriate advice about weight gain during pregnancy. More	Rush et al., 1988 Cogswell et al., 1999 ACOG, 2005 Power et al., 2006 Stotland et al., 2005

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Determinants	Findings/Interpretations	Comments	References
	appropriate weight gain during pregnancy. A national evaluation of WIC programs found a reversal of low weight gain in early pregnancy and greater total weight gain during pregnancy among women who enrolled in WIC compared with controls.	research on implementation of gestational weight gain guidelines is needed.	Joyce et al., 2008
Environmental			
Altitude	Jensen and Moore (1997) did not find any significant difference in gestational weight gain among women residing at 3,000 to 5,000 feet, 5,000 to 7,000 feet, 7,000 to 9,000 feet, and 9,000 to 11,000 feet.	The decline in birth weight associated with increase in altitude appears to be independent of gestational weight gain.	Jensen and Moore, 1997
Environmental Toxicants	The committee was unable to identify studies that examined specifically the effects of exposures to environmental toxicants on gestational weight gain.	More research is needed on the relationships among environmental toxicants, gestational weight gain, and fetal growth.	Dar et al., 1992 Wolff et al., 2007
Natural and Man-made Disasters	The committee was unable to identify studies that examined specifically the effects of natural or man-made disasters on gestational weight gain.	Disasters can affect gestational weight gain by influencing resource availability (including food supply), healthcare access, and stress levels.	Weissman et al., 1989 Cordero, 1993 Glynn et al., 2001 Lederman et al., 2004 Eskenazi et al., 2007 Landrigan et al., 2008
Neighborhood/Community			
Access to Healthy Foods	The committee found no direct evidence for the influence of neighborhood or community factors such as access to healthy foods on gestational weight gain.	Laraia et al. found pregnant women who live more than four miles from a supermarket had a two-fold greater risk of falling into the lowest Diet Quality Index quartile compared to women who lived ≤ 2 miles from a supermarket, but the authors also did not report on gestational weight gain.	Laraia et al., 2004 Lane et al., 2008
Opportunities for Physical Activity	Laraia et al. found that social spaces were associated with decreased odds for inadequate or excessive gestational weight gain.	Neighborhood environments can influence gestational weight gain by providing access to healthy foods and opportunities for physical activities.	Laraia et al., 2007
Interpersonal/Family Determinants			
Family Violence	Several studies demonstrated lower weight gain and greater risk of inadequate weight	More research is needed.	McFarlane et al., 1996 Siega-Riz and Hobel, 1997

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Determinants	Findings/Interpretations	Comments	References
	gain among abused pregnant women.		Boy and Salihu, 2004 Moraes et al., 2006
Marital Status	Several studies found married women were more likely to gain within the IOM recommended weight gain range than single or separated/divorced women.	More research is needed.	Kleinman et al., 1991 Ventura, 1994 Olsen and Strawderman, 2003
Partner and Family Support	The committee found insufficient evidence at this time to establish a relationship between partner support and gestational weight gain.	More research is needed.	Stevens-Simon et al., 1993 Parker et al., 1994 Gutierrez, 1999 Dipietro et al., 2003
Non-modifiable Maternal Factors			
Genetic Characteristics	The committee was unable to identify studies of familial aggregation or heritability of gestational weight gain. Presently it is not possible to conclude firmly on the role of specific genes and alleles on gestational weight gain.	Several studies have examined the effects of ADRB3 and Pro12Ala gene polymorphisms on gestational weight gain with mixed results. Studies lacked adequate statistical power to identify the effects of alleles or genotypes with a small effect size.	Festa et al., 1999 Yanagisawa et al., 1999 Alevizaki et al., 2000 Dishy et al., 2003 Tsai et al., 2004 Fallucca et al., 2006 Tok et al., 2006
Epigenetics and Developmental Programming	The committee was unable to identify studies that examined specifically the influence of epigenetic events and developmental programming on gestational weight gain.	More research is needed.	Fraga et al., 2005
Prepregnancy BMI	Gestational weight gain is generally inversely proportional to prepregnancy BMI. Chu et al found that maternal pre pregnancy obesity was the strongest factor affecting maternal weight gain in pregnancy, with obese women reporting the lowest gestational weight gain.	An important strategy for optimizing gestational weight gain is to help women achieve a healthy weight *before* pregnancy.	Voigt et al., 2007 Chu et al., 2009
Preexisting Morbidities	The committee found no direct evidence for pre-existing morbidities as determinants of gestational weight gain.	More research is needed on the influence of preconceptional health status of woman on gestational weight gain.	Fonager et al., 1998
Non-modifiable Maternal Factors			
Sociodemographic Factors: Adolescents	Several studies have demonstrated higher gestational weight gain among adolescents than adults; the relationships of adolescent gestational weight gain to birth outcomes, post-partum weight retention, and	The committee found that data generated between the IOM (1990) report and the present support the recommendation that "until more is known, adolescents less than two years post-menarche should be advised	Hediger et al., 1990 Howie et al., 2003 Nielsen et al., 2006 Chen et al., 2007 Groth, 2007

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Determinants	Findings/Interpretations	Comments	References
	subsequent risk for overweight/obesity remain unclear and requires further research.	to stay within the IOM-recommended, BMI-specific weight range without either restricting weight gain or encouraging weight gain at the upper end."	
Sociodemographic Factors: Older Women	Several studies reported higher prepregnancy BMI and lower gestational weight gain among older women.	The contributions of gestational weight gain to birth outcomes, postpartum weight retention and subsequent overweight/obesity among older women remain unclear and require further research.	Gross et al., 1980 Endres et al., 1987 Cnattingius et al., 1992 Prysak et al., 1995 Fretts, 2005 Joseph et al., 2005 Reddy et al., 2006 Delpisheh et al., 2008
Sociodemographic Factors: Race/Ethnicity	The committee found that few of the studies reviewed considered the influence of the many possible determinants of gestational weight gain among different racial/ethnic groups or alternatively, adjusted for race/ethnicity in their analyses.		
Sociodemographic Factors: Food Insecurity	Several studies have reported an association between food insecurity and overweight/ obesity. Olson and Strawderman (2008) found that women who were both obese and food insecure in early pregnancy were at greatest risk of major gestational and postpartum weight gain, suggesting that food insecurity may play a role in gestational weight gain.	Food restriction or deprivation, whether voluntary or involuntary, results in a variety of changes including the preoccupation with food and eating. Food-insecure households also tend to purchase calorie-dense foods that are often high in fats and added sugars in adaptation to their food insecurity	Anderson, 1990 Polivy, 1996 Frongillo et al., 1997 Olson, 1999 Townsend et al., 2001 Adams et al., 2003 Basiotis and Lino, 2003 Crawford et al., 2004 Drewnowski and Darmon, 2005 Wilde and Peterman, 2006 Jones and Frongillo, 2007 Olson and Strawderman, 2008
Potentially Modifiable Maternal Factors			
Physiological Factors: Insulin, Leptin, and Hormonal Milieu	Gestational weight gain may be related to changes in insulin sensitivity during pregnancy which depends on maternal pregravid metabolic status. Both leptin and adiponectin are correlated with various components of maternal metabolism such as energy expenditure and adiposity, and may influence gestational weight gain by		Swinburn et al., 1991 Catalano et al., 1993 Catalano et al., 1998 Highman et al., 1998 Catalano et al., 1999 Kirwan et al., 2002 Cnop, 2003 Okereke et al., 2004

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	affecting maternal insulin sensitivity.		Retnarkaran et al., 2004 Williams et al., 2004 Winzer et al., 2004 Hauguel-deMouzon et al., 2006 Pinar et al., 2008
Physiological Factors: Basal Metabolic Rate (BMR)	BMR can influence total energy expenditure and gestational weight gain. Several studies have shown increased BMR during pregnancy, but change in BMR varies according to pregravid BMI [Claude to edit]	Wide variability in BMR change during pregnancy has been observed and makes recommendations related to energy intake difficult	Prentice et al., 1983 Forsum et al., 1985 Lawrence et al., 1985 Durnin, 1991 Goldberg et al., 1993 Butte et al., 2004
Medical Factors: Hyperemesis Gravidarium	Several studies have demonstrated lower gestational weight gain and birth weight among women with hyperemesis gravidarum.		Gross et al., 1989 Goodwin et al., 1992 Vilming and Nesheim, 2000 Furneaux et al., 2001 Goodwin, 2002 Jewell and Young, 2003
Medical Factors: Anorexia Nervosa and Bulimia	Sollid et al. found increased preterm delivery & SGA among women with eating disorders but did not obtain information on gestational weight gain. Kouba et al. found anorexic women gained less weight and had lower birth weight infants.		Sollid et al., 2004 Kouba et al., 2005 Wisner et al., 2007 Bulik et al., 2008
Medical Factors: Bariatric Surgery	Three studies reported a decrease in gestational weight gain during a subsequent pregnancy in women who had bariatric surgery.	Incidence of gestational diabetes and hypertensive disorders is lower in pregnancy following bariatric surgery; The effect of bariatric surgery on the risk of fetal macrosomia and birth weight are inconclusive.	Gurewitsch et al., 1996 Marceau et al., 2004 Skull et al., 2004 Dixon et al., 2005 Santry et al., 2005 Davis et al., 2006 Ducarme et al., 2007
Psychological Factors: Depression	The committee found that evidence in support of a relationship between depressive symptoms and gestational weight gain is inconclusive.	Studies limited by lack of generalizability and control for confounding. Difficult to establish cause-effect from cross-sectional studies.	Cameron et al., 1996 Casanueva et al., 2000 Abraham et al., 2001 Walker and Kim, 2002 Dipietro et al., 2003 Bodnar, In press
Psychological Factors: Stress	The committee found no robust association between stress and gestational weight gain. [Ann Marie to review]		Picone et al., 1982 Orr et al., 1996 Rondo et al., 2003

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			Brawarksy et al., 2005 Dominguez et al., 2005
Psychological Factors: Social Support	The committee found inconclusive evidence for the role of social support in gestational weight gain.		Casanueva et al., 1994 Hickey et al., 1995 Olson and Strawderman, 2003
Psychological Factors: Attitude Toward Low Weight Gain	The committee found mixed evidence for the influences of maternal attitude on actual gestational weight gain.	This relationship may vary according to maternal prepregnancy BMI	Palmer et al., 1985 Stevens-Simon et al., 1993 Copper et al., 1995
Behavioral Factors: Dietary Intake	Several studies have demonstrated a relationship between energy intake and gestational weight gain.	Dietary intake of certain types of foods may also influence gestational weight gain, but more research is needed.	Campbell, 1975 Campbell, 1983 Bergmann, 1997 Clapp, 2002 Lagiou et al., 2004 Olafsdottir et al, 2006 Deierlein et.al., 2008
Behavioral Factors: Physical Activity	Several studies have demonstrated an inverse relationship between the level of physical activity and gestational weight gain.	There is a need for appropriately powered randomized clinical trials designed to clarify the relationship between volume and intensity of physical activity regimens (dose) and maternal weight gain in women with various levels of pre-pregnancy BMI.	Lokey et al., 1991 Ohlin and Rossner, 1994 Clapp and Little, 1995 Sternfeld et al., 1995 Stevenson, 1997 Abrams et al., 2000 Hinton and Olson, 2001 Kramer and Kakuma, 2003 Siega-Riz et al., 2004 Morris and Johnson, 2005 Artal et al., 2007 Haakstad et al., 2007 Lof et al., 2008
Behavioral Factors: Substance Abuse	Several studies examining associations between decreasing GWG and amount of reported smoking show inconclusive results. More recently, Furuno et al. (2004) found no significant difference in mean GWG between smoking and non-smoking mothers but did find an increased risk for low GWG among smokers. Little information is available about effects of alcohol consumption on GWG. Smith et al. found that those who used amphetamine in the	The major effects of substance use (cigarette smoking, alcohol and drug use) on birth outcomes (e.g. birth defects, SGA) appear to be independent of gestational weight gain.	Rush, 1974 Hanson et al., 1978 Garn et al., 1979 Haworth et al., 1980 Papoz et al., 1982 Little et al., 1986 Graham et al., 1992 Stevens-Simon and McAnarney, 1992 Wolff et al., 1993 Jacobsen et al., 1994

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	first two trimesters but ceased use by the third trimester gained significantly more weight than either women who used throughout pregnancy or non-exposed women ($p = 0.019$), suggesting the anorexic effects of methamphetamine are limited to continuous use, and there may be a rebound in weight gain if the mother stops use.		Adriananse et al., 1996 Bagheri et al., 1998 Wagner et al., 1998 Seckler-Walker and Vacek, 2003 Furuno et al., 2004 Ogunyemi and Hernandez-Loera, 2004 Smith et al., 2006 Wells et al., 2006
Behavioral Factors: Unintended Pregnancy	The committee found data concerning the effect of unintended pregnancy on gestational weight gain to be conflicting.		Marsiglio and Mott, 1988 Hickey et al., 1997 Siega-Riz and Hobel, 1997 Kost et al., 1998 Wells et al., 2006
Energy Balance	The committee found that there remains a dearth of information to relate dietary intake or physical activity to gestational weight gain even though they are primary determinants of weight gain in non-pregnant individuals.		
Vulnerable Populations: Seasonal Migrant Workers	Pregnancy Nutrition Surveillance System (PNSS) found that migrant women had lower gestational weight gain than non-migrant women; however, the prevalence for adverse birth outcomes (low birth weight, very low birth weight, preterm birth, and small for gestational age) was similar for both groups.		Reed et al., 2005
Vulnerable Populations: Military	The committee was unable to identify studies that specifically examined gestational weight gain among women in military service.		Magann and Nolan, 1991 O'Boyle et al., 2005 Haas and Pazdernik, 2006
Vulnerable Populations: Incarcerated Women	The committee was unable to identify studies that specifically examined gestational weight gain among women who are incarcerated.	Several studies suggest that birth outcomes of incarcerated pregnant women may be better, suggesting certain aspects of the prison environment, such as shelters and regular meals, may be protective particularly for high-risk pregnant women.	Safyer and Richmond, 1995 Martin et al., 1997a Martin et al., 1997b Bell, 2004

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Appendix E

Results from the Evidence-based Report* on Outcomes of Maternal Weight Gain

The purpose of this systematic evidence-based review, requested by the Agency for Healthcare Research and Quality (AHRQ) and conducted by the RTI International—University of North Carolina at Chapel Hill Evidence-based Practice Center (RTI-UNC EPC), was to review the evidence on outcomes of gestational weight gain with specific attention to five key questions:

- KQ 1. What is the evidence that either total weight gain or rate of weight gain during pregnancy is associated with (1) birth outcomes, (2) infant health outcomes, and (3) maternal health outcomes? Does any evidence suggest that either total weight gain or rate of weight gain is a causal factor in infant or maternal health outcomes?
- KQ 2. What are the confounders and effect modifiers for the association between gestational weight gain (overall and patterns) and birth outcomes? Based on the findings in KQ 1, do these confounders and effect modifiers themselves contribute to antepartum or postpartum complications or to longer-term maternal and fetal complications, including development of adult obesity?
- KQ 3. What is the evidence that weight gains above or below thresholds defined in the 1990 IOM body mass index (BMI) guidelines or weight loss in pregnancy contribute to antepartum or postpartum complications or longer-term maternal and fetal complications? How do these relationships vary by sociodemographic characteristics (i.e., race and age)?
- KQ 4. What are the harms or benefits of offering the same weight gain recommendations to all pregnant women, irrespective of age and body weight considerations (e.g., pregravid weight, actual body weight at a particular time point, or optimal body weight)?
- KQ 5. What are the anthropometric tools for determining adiposity and their appropriateness for the pregnancy state? What are the risks and benefits of measuring adiposity for (1) clinical management of weight gain during pregnancy and (2) evaluation of the relationship between weight gain and outcomes of pregnancy?

The review focused on screening studies from 1990 to October 2007 that were published in English, and excluded studies with low sample size (case series < 100 and cohorts < 40) or failure to control for pregravid weight. In total, 150 studies were systematically reviewed and each was rated on quality and used to assess the strength of evidence for each outcome. The report, including appendices and evidence tables, can be accessed and viewed in its entirety at:

*Viswanathan M., A. M. Siega-Riz, M.-K. Moos, A. Deierlein, S. Mumford, J. Knaack, P. Thieda, L. J. Lux and K. N. Lohr. 2008. Outcomes of Maternal Weight Gain, Evidence Report/Technology Assessment No. 168. (Prepared by RTI International-University of North Carolina Evidence-based Practice Center under contract No. 290-02-0016.) AHRQ Publication No. 08-E-09. Rockville, MD: Agency for Healthcare Research and Quality.

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http://www.ahrq.gov/clinic/tp/admattp.htm. Literature published outside of the scope of the report (prior to 1990 and after October 2007) are reviewed in Appendix C of this report. The methods and results and of the evidence review (Chapter 3 of the report) are provided below.

CHAPTER 2: METHODS

In this chapter, we document the procedures that the RTI International-University of North Carolina Evidence-based Practice Center (RTI-UNC EPC) used to develop this comprehensive evidence report on outcomes of maternal weight gain. The team was led by a senior health services researcher (Meera Viswanathan, PhD, Study Director), a senior epidemiologist (Anna Maria Siega-Riz, PhD, RD, Scientific Director), and a senior nurse-researcher (Merry-K Moos, FNP, MPH, co-Scientific Director).

We first describe our strategy for identifying articles relevant to our five key questions (KQs), our inclusion and exclusion criteria, and the process we used to abstract relevant information from the eligible articles and generate our evidence tables. We also discuss our criteria for grading the quality of individual articles and for rating the strength of the evidence as a whole. Finally, we explain the peer-review process.

Literature Review Methods

Inclusion and Exclusion Criteria

Our inclusion and exclusion criteria are documented in Table 1. As noted in Chapter 1, this systematic review focuses on outcomes of maternal weight gain with respect to the 1990 recommendations from the Institute of Medicine (IOM).[**] Largely for that reason, we limited our searches to articles published in 1990 and thereafter. We also restricted our searches to developed countries so that we could have data generally relevant for maternal weight gain and health outcomes in the United States.

[**]Appendixes and evidence tables cited in this report are provided electronically at http://www.ahrq.gov/downloads/pub/evidence/pdf/admaternal/admaternalapp.pdf.

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Table 1. Inclusion/exclusion criteria for gestational weight gain

Category	**Criteria**
Study population	Women of any age with singleton pregnancies
Study settings and geography	KQ 1, KQ 2, KQ 4: Developed nations: United States, Canada, Western Europe, Japan, Australia, New Zealand KQ 3: United States KQ 5: All countries
Time period	January 1990 through October 2007
Publication languages	English only
Admissible evidence (study design and other criteria)	Admissible designs Controlled trials (n ≥ 40), nonrandomized controlled trials (n ≥ 40), systematic reviews, meta-analyses, prospective trials with historical controls (n ≥ 40), prospective or retrospective observational cohort studies (n ≥ 40), and medium to large case series (n ≥ 100) Other criteria Original research studies must provide sufficient detail regarding methods and results to enable use and adjustment of the data and results. Relevant outcomes must be abstractable from data presented in the papers. Sample sizes must be appropriate for the study question addressed in the paper; single case reports or small case series (fewer than 100 subjects) are excluded. For KQ 1, 2, 3, and 4: prepregnancy body mass index (BMI) or weight must be accounted for in the relationship between maternal weight gain and outcome. Studies limited to women with preexisting health conditions only are excluded.

We excluded studies that (1) were published in languages other than English (given the available time and resources); (2) did not report information pertinent to the key clinical questions; (3) had fewer than 40 subjects for randomized controlled trials (RCTs) or nonrandomized cohorts with comparisons or fewer than 100 subjects for case series; and (4) were not original studies.

For KQ 1, 2, 3, and 4, we required that the reported association between maternal weight gain and health outcomes accounted for prepregnancy body mass index (BMI) or weight, either through stratified univariate analysis or multivariate analysis.

Literature Search and Retrieval Process

Databases We used multifaceted search strategies to include current and valid research on the KQs, which we applied to four standard electronic databases—MEDLINE®, Cochrane Collaboration resources, Cumulative Index to Nursing and Allied Health Literature (CINAHL), and Embase. We also hand-searched the reference lists of relevant articles to make sure that we did not miss any relevant studies. We consulted with our Technical Expert Panel (TEP) about any studies or trials that are currently under way or that may not yet be published.

Search terms. Based on the inclusion/exclusion criteria above, we generated a list of Medical Subject Heading (MeSH) search terms (Table 2 and Appendix A).** Our TEP also reviewed

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these terms to ensure that we were not missing any critical areas, and this list represents our collective decisions as to the MeSH terms used for all searches.

Table 2. MEDLINE® search strategy and unduplicated results for February 2007

Search Terms	**Search Results**
#2 Search "Weight Gain"[MeSH]	13,220
#5 Search pregnancy [MeSH]	577,647
#6 Search #2 AND #5	1,808
#7 Search gestational weight gain	1,725
#8 Search #6 OR #7	3,023
#9 Search #6 OR #7 Limits: English, Humans	1,696
#15 Search ("Outcome Assessment (Health Care)"[MeSH] OR "Outcome and Process Assessment (Health Care)"[MeSH] OR "Pregnancy Outcome"[MeSH]) OR "Reproductive History"[MeSH] OR "birth outcomes" OR "infant health outcomes" OR "maternal health outcomes" Limits: English, Humans	332,914
#16 Search #9 AND #15 Limits: English, Humans	474
#19 Search ("Counseling"[MeSH] OR "Directive Counseling"[MeSH])	23,091
#20 Search #9 AND #19	12
#25 Search "Body Weights and Measures"[MeSH]	279,399
#26 Search #9 AND #25	1,044
#29 Search "Anthropometry"[MeSH]	71,849
#30 Search #26 AND #29	359

Our searches in MEDLINE® produced 715 unduplicated records. Searches in other databases yielded in 190 new records from CINAHL and 4 from Embase. Similar searches in Cochrane did not produce any new citations. Following an update on October 3, 2007, and additional searches for KQ 5, we ultimately identified 1,082 unduplicated records. In addition, peer reviews suggested 3 new citations that met our inclusion criteria.

Figure 2 presents the yield and results from our searches, which we conducted from February through October 3, 2007. Beginning with a yield of 1,085 articles, we retained 150 articles that we determined were relevant to address our KQs and met our inclusion/exclusion criteria (Table 1). We reviewed titles and abstracts of the articles against the basic inclusion criteria above; we retained relevant articles, all published after our search cutoff date of January 1990, and used them as appropriate in the discussion in Chapter 4.

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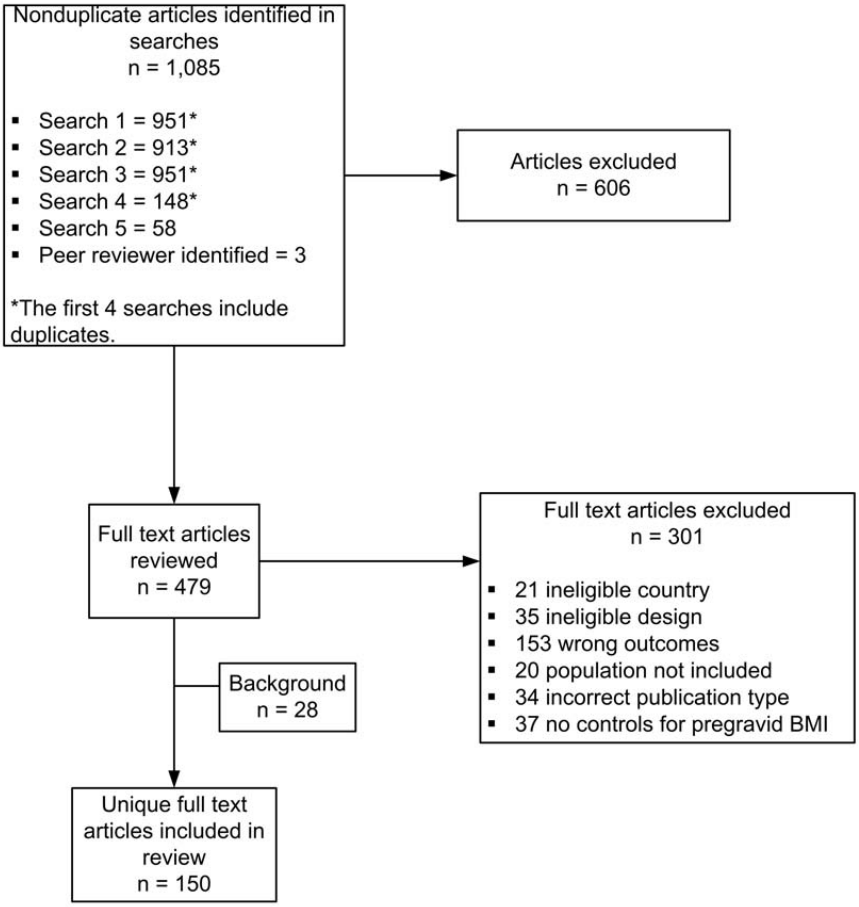
Figure 2. Disposition of articles for gestational weight gain



BMI, body mass index

Article selection process Once we had identified articles through the electronic database searches, review articles, and reference lists, we examined abstracts of articles to determine whether studies met our criteria. Each abstract was independently, dually reviewed for inclusion or exclusion, using an Abstract Review Form (Appendix B). If one reviewer concluded that the article should be included in the review, we retained it.

Of this entire group of 1,085 articles, 479 required full review. For the full article review, one team member read each article and decided whether it met our inclusion criteria, using a Full Text Inclusion/Exclusion Form (Appendix B)**. Reasons for article exclusion are listed in Appendix D.

Literature Synthesis

Development of Evidence Tables and Data Abstraction Process

The senior staff who conducted this systematic review jointly developed the evidence tables. We designed the tables to provide sufficient information to enable readers to understand the studies and to determine their quality; we gave particular emphasis to essential information

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related to our KQs. We based the format of our evidence tables on successful designs that we have used for prior systematic reviews.

We trained abstractors by having them abstract several articles into evidence tables and then reconvening as a group to discuss the utility of the table design. The abstractors repeated this process through several iterations until they decided that the tables included the appropriate categories for gathering the information contained in the articles.

Three junior epidemiologists (Sunni Mumford, SM; Andrea Deierlein, MS, MPH; and Julie K. Knaack, MPH, RD, LDN) shared the task of initially entering information into the evidence tables. Senior staff reviewed the articles and edited all initial table entries for accuracy, completeness, and consistency. Abstractors reconciled all disagreements concerning the information reported in the evidence tables. The full research team met regularly during the article abstraction period and discussed global issues related to the data abstraction process.

The final evidence tables are presented in their entirety in Appendix C. Studies are presented in the evidence tables alphabetically by the last name of the first author. A list of abbreviations and acronyms used in the tables appears at the beginning of that appendix.

Quality Rating of Individual Studies

The evidence for this systematic review is based almost entirely on observational studies. This fact presents a challenge for rating individual studies. Quality rating forms for RCTs have been validated and in use for several years; a similarly well-validated form for observational studies does not exist.

Thus, as a parallel effort, we developed a form to rate observational studies.[35] This form, which can be used to rate the quality of a variety of observational studies, was based on a review of more than 90 AHRQ systematic reviews that included observational studies; we supplemented this review with other key articles identifying domains and scales.[36,37] We structured the resultant form largely on the basis of the domains and subdomains suggested by Deeks and colleagues;[36] we then adapted it for use in this systematic review (Appendix B).**

The form currently includes review of nine key domains: background, sample selection, specification of exposure, specification of outcome, soundness of information, followup, analysis comparability, analysis of outcome, and interpretation. Each of these domains was further evaluated on aspects of quality of the study design or reporting that would influence the reader's perception of internal validity of the journal article (Table 3). We note that variations in reporting could result in different scores for studies drawing from the same sample.

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Table 3. Scoring algorithm for subdomains and overall quality rating for individual studies

Definition and Scoring Algorithm	**Rating**
Score algorithm for background (presented in the context of previous research, hypothesis clearly described)	
• Both elements present	Good
• Neither present	Poor
• One of two elements present	Fair
Score algorithm for sample definition (explicitly stated inclusion/exclusion criteria, uniform application of criteria, clear description of recruitment strategy, clear description of characteristics of the participants, power analysis or some other basis noted for determining the adequacy of study sample size)	
• > Three elements present	Good
• < Two elements present	Poor
• Two or three elements present	Fair
Score algorithm for exposure (clear definition of weight gain, check for plausibility of pregravid weight, clear explanation of actions taken on outliers)	
• All three elements present or clearly defined	Good
• Poor definition of weight gain	Poor
• Moderate or very clear definition of weight gain, one or more other elements present	Fair
Score algorithm for outcome (clear description of primary outcomes)	
• All essential details described	Good
• Few or no essential details described	Poor
• Some essential details described	Fair
Score algorithm for soundness of information (quality of source of information on exposure, confounders, and outcome)	
• Good for all three	Good
• Poor on source of information for exposure	Poor
• Any other score	Fair
Score algorithm for followup (adequate reporting of reasons for loss to followup)	
• Retrospective or prospective study with clear reporting on loss to followup	Good
• Prospective study, no reporting on followup	Poor
• Retrospective study with no reporting on loss to followup	Fair
Score algorithm for analysis comparability (comparability of cohorts through design, reasonable choice of control variables, clear description of confounders, adequate adjustment for confounders)	
• All elements present	Good
• Inadequate adjustment for confounding	Poor
• Any other score	Fair
Score algorithm for analysis outcome (withdrawals, lost to followup, and missing data adequately accounted for in the analysis, and appropriate statistical methods used)	
• Both elements clearly present	Good
• Neither element present	Poor
• Any other score	Fair
Score algorithm for interpretation (results interpreted appropriately based on study design and statistics, clinically useful, appropriate presentation, presented in the context of prior research, and conclusion supported by results)	
• All elements clearly present	Good
• Conclusions not supported by results	Poor
• Any other score	Fair
Score algorithm for overall quality	
• 5 or more good ratings and no poor ratings on subdomains	Good
• 3 or more poor ratings on subdomains	Poor
• <5 good ratings and <3 poor ratings on subdomains; 5 or more good ratings and any poor ratings	Fair

As described in Table 3, we combined these elements to generate overall scores. We set the default as fair and then focused on the threshold required for good and poor studies; the algorithm is also described in Table 3. Fair studies, therefore, include studies that were predominantly fair (four to nine fair ratings on domains) and could not be rated either good

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(fewer than five good ratings for subdomains) or poor (fewer than three poor ratings for subdomains). Studies with more than five good ratings for domains that also received one or two poor ratings were downgraded to fair quality.

Key methodological concerns in this literature relate to the source of information on weight gain and the timing of measurement of weight gain. Studies that relied solely on self-reported pregravid and final pregnancy weights suffer from well-documented issues of recall bias. In addition, women tend to misreport their weight, and this bias varies by weight status[38] and ethnicity.[39] The timing of weight measurement (for pregravid weight and final weight) can vary depending on the design of the study; when unreported, the total weight gain during pregnancy cannot be assumed to be collected at similar time points for all women within the study, resulting in further bias. Our rating algorithm, therefore, paid special attention to the source of data on gestational weight gain and the timing of measurement. Studies that relied solely on recalled prepregnancy and total pregnancy weight were rated poor on that domain, but if they defined their gestational weight variable clearly (providing details on the timing of measurement for pregravid and final weight measurements) and either checked for the biological plausibility of pregravid weight status or explained how outliers were dealt with, they could receive an overall fair rating (assuming that they received fewer than three poor ratings overall).

Strength of Available Evidence

Our scheme follows the criteria applied in an earlier RTI-UNC EPC systematic review of systems for rating the strength of a body of evidence.[40] That system has three domains: quality of the research (as evaluated by the quality rating algorithm described above), quantity of studies (including number of studies and adequacy of the sample size), and consistency of findings. Two senior staff members assigned grades by consensus.

We graded the body of literature for each KQ and present those ratings as part of the discussion in Chapter 4. The possible grades in our scheme are as follows:

I. Strong: The evidence is from studies of sound design (good quality); results are both clinically important and consistent with minor exceptions at most; results are free from serious doubts about generalizability, bias, or flaws in research design. Studies with negative results have sufficiently large samples to have adequate statistical power.

II. Moderate: The evidence is from studies of sound design (good quality), but some uncertainty remains because of inconsistencies or concern about generalizability, bias, research design flaws, or adequate sample size. Alternatively, the evidence is consistent but derives from studies of weaker design (fair quality).

III. Weak: The evidence is from a limited number of studies of weaker design (fair or poor quality). Studies with strong design (good quality) either have not been done or are inconclusive.

IV. No evidence: No published literature.

External Peer Review

As is customary for all evidence reports and systematic reviews done for AHRQ, the RTI-UNC EPC requested review of this report from a wide array of individual outside experts in the field, including our TEP, and from relevant professional societies and public organizations. AHRQ also requested review from its own staff. We sent 20 invitations for peer review: 6 TEP members, 6 relevant organizations, and 8 individual experts. Reviewers included clinicians (e.g.,

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obstetrics and gynecology, women's health/general health), representatives of federal agencies, advocacy groups, and potential users of the report.

We charged peer reviewers with commenting on the content, structure, and format of the evidence report, providing additional relevant citations, and pointing out issues related to how we had conceptualized and defined the topic and KQs. We also asked them to complete a peer review checklist. We received comments from 11 of the invited peer reviewers in addition to comments from AHRQ staff. The individuals listed in Appendix E** gave us permission to acknowledge their review of the draft. We compiled all comments and addressed each one individually, revising the text as appropriate.

CHAPTER 3: RESULTS

This chapter presents the results of our evidence review for the following four key questions (KQs): KQ 1, outcomes of gestational weight gain; KQ 3, outcomes of gestational weight gain within or outside the recommendations of the Institute of Medicine (IOM); and KQ 5, anthropometrics of gestational weight gain.

We note that KQ 2, on modifiers of outcomes, is derivative of KQ 1. KQ 4, on recommendations for weight gain, is derivative of KQ 3. Because we framed KQ 2 and KQ4 as synthesis questions, we cover them in Chapter 4.

Appendix C provides the detailed evidence tables for KQs 1, 3, and 5. Our summary tables below feature groups of studies addressing each outcome; we present these text tables only when we have three or more studies pertaining to that particular outcome. These tables are organized by quality (good, then fair, then poor), and then alphabetically.

The summary tables generally provide information to identify the study (author and date), sample size, study quality, definition of gestational weight gain, definition of outcome, results, and confounders and effect modifiers. Unless otherwise noted, we use the metrics (e.g., grams, kilograms, pounds) that each study article used; we did not recalculate measures into the same metric.

KQ 1: Outcomes of Gestational Weight Gain

We present outcomes in the physiological order, beginning with maternal antepartum and intrapartum outcomes, then birth outcomes (neonatal outcomes at the time of birth), infant outcomes (<1 year), child outcomes (≥ 1 year), and finally maternal short- and long-term outcomes. Evidence Tables 1–35 (Appendix C) include studies relevant for KQ 1, listed alphabetically by author. For each outcome, we describe study characteristics and then report an overview of results, followed by detailed results. When meaningful, we present results separately for varied measures of gestational weight gain (categorical measures of weight gain, rate of weight gain, total weight gain, and other). For some bodies of evidence, variations in the definition of the outcome and inconsistencies in the direction of effect may suggest that an overall assessment of the effect is more meaningful than separate assessments of outcomes associated with each measure of gestational weight gain. Summary tables and text include information on the confounders and effect modifiers accounted for in each study.

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Maternal Antepartum Outcomes

Maternal discomforts of pregnancy

Study characteristics Five studies (Evidence Table 1) investigated the relationship between weight gain and diverse maternal discomforts of pregnancy: a composite of pregnancy discomforts,[41] physical energy and fatigue,[42] stretch marks,[43,44] and heartburn.[45]

Overview of results Two fair[41,42] and three poor studies[43–45] found no differences for women who gained an excessive amount of weight compared to those who did not, irrespective of body mass index (BMI) group,[42] a higher frequency of symptoms from midpregnancy through the 36th week of gestation,[46] no association between gestational weight gain and heartburn in gestation,[45] and some increased risk of stretch marks with increased weight gain.[43,44]

Detailed results A prospective cohort study in Sweden examined symptoms across pregnancy and attempted to document the prevalence and frequency of 27 pregnancy symptoms while controlling for biomedical factors.[41] A cohort of 476 nulliparous women was assessed six times during gestation (gestational ages of 10, 12, 20, 28, 32, and 36 weeks). The investigators sought to determine the prevalence of various symptoms in pregnancy and to explore whether psychosocial variables are explanatory while controlling for possible confounding variables such as medical risk, smoking, and weight gain. Pregravid BMIs were calculated from self-reported weight information and women were weighed when they arrived at the hospital to give birth. Total weight gain was associated with a higher frequency of symptoms from midpregnancy through the 36th week of gestation. Reflecting on their findings, the researchers recommend that weight gain be included in future studies exploring the etiology of symptoms during pregnancy.

A secondary analysis of data collected in a US prospective cohort study investigated the relationship of prepregnancy weight and gestational weight gain on levels of physical energy and physical symptoms collected through a series of questionnaires that had been administered in patient homes in early, mid, and late pregnancy.[42] All weight data were self-reported. The researchers found no differences in the number of physical symptoms or level of physical energy reported by women who gained an excessive amount of weight compared with those who did not, irrespective of BMI group. Women whose weight gain was greater than the IOM guidelines reported a lower level of functional status in the third trimester than women whose weight gain was within the guidelines ($P = 0.014$). Women participating in this study were 30.9 years of age on average, married, English-speaking, and of low medical risk. No confounders or effect modifiers were accounted for in the analysis.

The one study (rated poor quality) that investigated the determinants of heartburn in pregnancy undertook a cross-sectional study in the United Kingdom of 602 women of different gestational lengths who self-reported their pregravid weight and completed a questionnaire.[45] The analysis, which considered age, race, parity, and pregravid BMI, found that weight gain in pregnancy was not a risk factor for heartburn in gestation.

Two studies (both rated poor quality) reported on the relationship between stretch marks (striae gravidarum) and weight gain.[43,44] One was a small retrospective cohort ($N = 48$) recruited from one private and one teaching hospital in the United States.[43] Mean total weight gain was significantly greater in women with abdominal striae than women without stretch marks ($P <$

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.05) but the analysis did not account for any confounders or effect modifiers. The other study reported on a cross-sectional sample of 324 primiparous women who were assessed within 48 hours of giving birth in Great Britain.[44] Logistic regression analysis found maternal age, BMI, weight gain, and neonatal birthweight to be independently associated with striae. Weight gain was a weakly significant risk factor (OR, 1.08; 95% CI, 1.02–1.14).

Hyperemesis

Study characteristics A retrospective cohort study compared the experiences of 1,270 women who had an antepartum admission before 24 weeks of gestation for hyperemesis with those of 154,821 women who experienced no antepartum admission related to vomiting (Evidence Table 2).[47] Baseline weight and weight gain were abstracted from the Nova Scotia Atlee Perinatal Database, but the authors did not explain how the weights entered into the database were assessed.

Overview of results One poor study found a correlation between increasing likelihood of total gestational weight gain of < 7 kg with increasing numbers of antenatal admissions for hyperemesis.[47]

Detailed results The study, undertaken to determine the relationship between hyperemesis and a variety of outcomes, used the number of antenatal admissions as a marker for severity of disease. The study found a correlation between increasing likelihood of total gestational weight gain of < 7 kg with increasing numbers of antenatal admissions. Many potential confounders were incorporated into the analysis including previous pregnancy experiences, psychiatric disorders, pregravid weight, and preexisting medical diseases. Weight gain information, however, was missing for approximately 17 percent of the cohort.

Abnormal glucose metabolism

Study characteristics Eleven studies specifically investigated the relationship between weight gain in pregnancy and the development of abnormal glucose metabolism (Evidence Table 3, Table 4).[3,48–57] Of these, four were done outside the United States.[49,50,52,53] Numerous inconsistencies in methodology and definitions, such as differences in criteria used for the diagnosis of gestational diabetes mellitus (GDM), preclude clear summations regarding the research.

TABLE 4. Gestational weight gain and abnormal glucose control

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Kieffer et al., 2006[48] US Michigan federally qualified community health center	Pregravid weight: Self-report; if unknown calculated from wt at ≤ 10 wks gestation Total weight gain: Computed	Note: study aim to determine relationship of anthropometric and metabolic variables on infant outcomes	Women with GDM had significantly lower average weight gain than those without GDM but weight gain was not significantly related to glucose category	Parity, pregravid BMI, weight gain

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
1,041 Latinas All BMIs Good	from last weight recorded within 7 days delivery			
Saldana et al., 2006[3] US North Carolina prenatal study 2,254 All BMIs Good	Pregravid weight: Self-report Total weight gain: Calculated on prenatal measurement to end of second trimester		Weight gain ratio (observed/recommended) IGT OR (95% CI) 0.9 (0.7–1.1) GDM OR (95% CI) 1.2 (0.9–1.4)	Race, maternal age, gestation age of weight measurement
Edwards et al., 1996[55] US Minnesota, births at specific medical center 1,343 divided between obese women (BMI > 29) matched to nonobese (BMI 19.8–26.0) Fair	Pregravid weight: Self-report Total weight gain: Last prenatal assessment	Pregravid wt 19.8–26.0 BMI: G1: < 11.5 kg gain G2: 11.6–16 kg gain G3: > 16 kg gain Pregravid wt > 29 kg G4: lost/gained nothing G5: 0.5–6.5 kg gain G6: 7–11.5 kg gain G7: 12–16 kg gain G8: >16 kg gain	Incidence gestational diabetes: G1: 2.3% G2: 3.3% G3: 2.9% (*P*= .759) G4: 13.3% G5: 24.3% G6: 11.9% G7: 16.7% G8: 17.3% (*P*= .554)	Maternal age, parity, race, prenatal smoking, prenatal alcohol use, prenatal illicit drug use, pregravid health, weight and adequacy of prenatal care
Hackmon, et al., 2007[57] US inner city population 75 All BMIs Fair	Pregravid weight: How determined not described Weight gain: Weight at 24–28 weeks end point for calculations		There was no difference in maternal weight gain during early pregnancy between patients with abnormal versus normal GCT values (mean±SD of 4.13±3.2 and 4.16±1.67, respectively).	Gravidity, parity, ethnicity, BMI
Kieffer et al., 2001[56]	Pregravid weight: Self-report or, if	Wt gain to 28 wk GA:	Multiple logistic regression analyses revealed statistically significant risk	Age, family history diabetes, parity, BMI,

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
US Detroit health care system 1,334 AA and Hispanic All BMIs Fair	unknown, weight assessment in 1st 10 wks gestation Total weight gain: How determined not described	Latinas: G1: < 14 lbs gain G2: 14–28 lbs gain (reference) G3: >28 lbs gain AAs: G4: < 14 lbs gain G5: 14–28 lbs gain G6: > 28 lbs gain	factors for GDM included increasing weight gain during first 28 wks of gestation	weight gain first 28 weeks, ethnicity
Murakami et al., 2005[52] Japan hospital data 633 All BMIs Fair	Pregravid weight: Self-report on first visit Total weight gain: Measured on admission for birth	G1: < 8.5 kg gain G2: 8.5–12.5 kg gain G3: >12.5 kg gain	AOR (95% CI) gestational diabetes G1: 5.14 (0.97–27.20) G2: Reference G3: 3.91 (0.61–24.73)	Maternal age, parity, smoking, weight gain, gestational weeks; pregravid BMI
Thorsdottir, et al., 2002[53] Iceland University Hospital 615 BMI: 19.5–25.5 Fair	Pregravid weight: Self-report Total weight gain: Maternity records (no specifics offered)	G1: <11.5 kg gain G2: 11.5–16.0 kg gain G3: 16.1–20.0 kg gain G4: >20 kg gain	Incidence gestational diabetes G1: 2.9% G2: 0 G3: 0 G4: 0 (P = .015)	Age, parity, height, gestational age
Bianco, et al., 1998[54] US New York Medical Center Database 11,840 Nonobese (BMI 19–27) and Morbidly obese	Pregravid weight: Self-report Total weight gain: Computed from measured weight within 4 weeks of delivery	Reported only for BMI > 35: G1: weight loss or no gain G2: 1–15 lb gain G3: 16–25 lb gain G4: 26–35 lb gain G5: > 35 lb gain	Incidence GDM: G1: 15.7% G2: 15.0% G3: 14.4% G4: 13.4% G5: 12.5% (P = NS)	Race, parity, clinic service, substance abuse, and preexisting medical conditions

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
(BMI > 35) ages 20–34 Poor				
Brennand et al., 2005[49] Quebec, Canada, First Nation's People (Cree) 603 All BMIs ≥18.5 Poor	Pregravid weight: Measured weight ≤14 wk GA used as proxy Total weight: Last recorded weight within 4 wks of giving birth	G1: "Low weight gain" G2: "Acceptable weight gain" G3: "Excessive weight gain" All categories per Canadian Guidelines	Incidence GDM: G1: 38.6% G2: 27.3% G3: 19.3% (P = 0.011) Incidence IGT: G1: 12.0% G2: 15.2% G3: 7.9% (P = 0.249)	None reported
Kabiru and Raynor, 2004[51] US Atlanta public hospital database 5,131 All BMIs ≥ 20 Poor	Pregravid weight: First prenatal visit Total weight gain: Computed on weight at admission for birth	BMI < 25 first assessment: G1: no change BMI category G2: increase 1 category G3: increase > 1 category BMI ≥ 25 first assessment: G4: no change BMI category G5: increase 1 category G6: increase >1 category	Incidence gestational diabetes G1: 0.5% G2: 1.5% G3: 3.7% (P = .005) G4: 1.0% G5: 3.3% G6: 1.9% (P = .005)	Pregravid weight
Seghieri et al., 2005[50] Italy Outpatient Diabetes Unit 1,880 All BMIs Poor	Pregravid weight: How determined not described Total weight gain: How determined not described		Weight gain as predictor of GDM OR (95% CI) 1.024 (0.974–1.077) (P = NS)	Parity, age, pregestational BMI, weight gain, family history diabetes

AA, African American; ACOG, American College of Obstetrics and Gynecology; BMI, body mass index; CHC,

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis

community health center; CI, confidence interval; G, group; GA, gestational age; GDM, gestational diabetes mellitus; GIP, gastric inhibitory polypeptide; IGT, impaired glucose tolerance; IOM, Institute of Medicine; NS, not significant; OR, odds ratio; PNV, prenatal visit.

The diagnostic algorithm for assigning the diagnosis of GDM in most asymptomatic women begins with administration and interpretation of a 1-hour glucose challenge test; those women who have a glucose level following the challenge above a specified level then receive a 3-hour glucose tolerance test (GTT). Abnormalities in the GTT results are considered diagnostic of GDM. The set point for determining if the glucose challenge test is abnormal is not universally agreed upon. Therefore, more women in one setting may be tested for disease than in another setting, not because of an increased prevalence of disease but because of differing definitions of abnormal. In addition, impaired glucose tolerance (IGT) is not clearly defined. Women with an abnormal glucose challenge test who subsequently have a normal GTT are sometimes identified as having IGT; more commonly, women who have one abnormal value in their GTT are designated as having IGT. The lack of standardization in the criteria necessary to be considered to have IGT and GDM hampers the body of research exploring the relationship between weight gain and abnormal glucose tolerance in pregnancy. Further hampering understanding of the relationship is that GDM is generally diagnosed around 28 weeks of gestation and is treated, in part, by dietary counseling and efforts to control weight gain. Similar attention is not directed toward women without this diagnosis. Therefore, using total weight gain as a predictor of disease or as a comparison point to a population without the diagnosis is likely to result in methodologically flawed conclusions.

Overview of results Four studies (1 good,[3 2] fair,[55,56] 1 poor[51]) found that greater weight gains in pregnancy were positively associated with abnormal glucose tolerance. Three studies (1 good quality,[48] 1 fair,[53] 1 poor[49]) found that women having lower than average weight gains had higher likelihood of GDM. Finally, four studies (2 poor,[50,54] 1 fair[52,53,57]) found no significant association.

Detailed results Whether total weight gain or the distribution of the gain across trimester or weeks of pregnancy predicts development of GDM is unclear from the articles we reviewed. As previously noted, treatment of the condition can alter total weight gain. Three studies[3,56,57] analyzed the association between weight gain in the first two trimesters of pregnancy and the diagnosis of GDM. A good-quality study reported that a weight gain ratio at the end of the second trimester of pregnancy that was greater than the IOM recommendations correlated with abnormalities of glucose metabolism.[3] A fair study found no correlation between weight gain in the first 24 to 28 weeks of gestation and an abnormal glucose challenge test, the first step in the testing process to identify GDM.[57] A third study assessed to be of fair quality reported that weight gain in the first 28 weeks of gestation was a significant predictor of the diagnosis of GDM (OR, 1.02; 95% CI, 1.004–1.042; P = 0.015) for their total sample of 987 black and Latina women but that total weight gain was not.[56] The OR for black women was the same (1.02; 95% CI, 1.002–1.044; P = 0.30). However, the range of weight gain included in the reference category

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was large (14–28 pounds) especially given that nearly 50 percent of the sample entered into the reported pregnancies with BMIs > 26.0.

Overall, family history of diabetes,[50,56] maternal age,[3,50,56] parity,[50] and BMI[3,50,56,57] were found to be more predictive of abnormal glucose metabolism than gestational weight gain in the research we reviewed.

Maternal hypertensive disorder

Study characteristics Twelve studies investigated the relationship between weight gain and pregnancy-induced hypertensive disorders (Evidence Table 4, Table 5).[4,25,49,51–55,58–61] Six of the studies were conducted outside the United States;[49,52,53,58,59,61] six studied US cohorts.[4,25,51,54,55,60] While all of these studies reported on blood pressures that became elevated during gestation, the criteria for diagnosing gestational hypertension (also called pregnancy-induced hypertension) and preeclampsia were often poorly defined; in addition, criteria for the various diagnoses lacked consistency between studies.

TABLE 5. Gestational weight gain and pregnancy-induced hypertension

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Cedergren, 2006[58] Swedish Medical Birth Registry 245,526 All BMIs Fair	Pregravid weight: Self-report; if unknown "standardized measurement" used Total weight gain: Computed on weight at presentation for delivery	G1: BMI < 20 G2: BMI 20–24.9 G3: BMI 25–29.9 G4: BMI 30–34.9 G5: BMI > 35	Preeclampsia by BMI for weight gain < 8 kg (reference gain 8–16 kg). OR (95% CI): G1: 0.90 (0.55–1.48) G2: 0.73 (0.61–0.89) G3: 0.64 (0.54–0.76) G4: 0.52 (0.42–0.62) G5: 0.63 (0.51–0.79) Preeclampsia by BMI for weight gain >16 kg (reference weight gain 8–16 kg): Odds ratios (95% CI) G1: 2.23 (1.83–2.71) G2: 2.31 (2.15–2.49) G3: 1.88 (1.72–2.06) G4: 1.65 (1.43–1.92) G5: 1.50 (1.17–1.92)	Age, parity, smoking in early pregnancy, year of birth
DeVader, et al., 2007[25] USA Missouri, birth certificate data 94,696	Pregravid weight: Noted on prenatal record or reported at postpartum stay Total weight gain: As stated on birth certificate data.	G1: Weight gain < 25 lbs G2: Weight gain 25–35 lbs G3: Gained > 35 lbs	AOR for preeclampsia (95% CI) G1: 0.56 (0.49–0.64) G2: 1 G3: 1.88 (1.74–2.04)	Maternal age, race/ethnicity, education, Medicaid status, tobacco and alcohol use, maternal height, adequacy of prenatal care, child's

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
BMI: 19.8–26.0 Fair	Specifics not provided			sex, child's birth year
Edwards et al.,1996[55] USA Minnesota, births at specific medical center 1,343 divided between obese women (BMI > 29) matched to nonobese (BMI 19.8–26.0) Fair	Pregravid weight: Self-reported Total weight gain: Last prenatal assessment	Pregravid wt 19.8–26.0 BMI: G1: < 11.5 kg gain G2: 11.6–16 kg gain G3: > 16 kg gain Pregravid wt > 29 kg G4: lost/gained nothing G5: 0.5–6.5 kg gain G6: 7–11.5 kg gain G7: 12–16 kg gain G8: > 16 kg gain	Preeclampsia: G1: 2.8% G2: 2.9% G3: 6.6% (*P* = .048) G4:10.7% G5: 7.7% G6: 8.3% G7: 7.9% G8: 16.5% (*P* = .076) Gestational HTN: G1: 2.3% G2: 3.8% G3: 3.3% (*P* = .607) G4: 9.3% G5: 8.3% G6: 11.3% G7: 10.3% G8: 9.0% (*P* = .832)	Maternal age, parity, race, prenatal smoking, prenatal alcohol use, prenatal illicit drug use, pregravid health, weight and adequacy of prenatal care
Kiel et al., 2007[4] USA Missouri, birth certificate data 120,251 BMIs ≥ 30.0 Fair	Total weight gain: As stated on birth certificate data. Specifics not provided	Analysis done by each class of obesity and weight changes in gestation including: weight loss ≥ 10 lbs; weight loss 2–9 lbs; no weight change; gain 2–9 lbs; gain 10–14 lbs; gain 15–25 lbs; gain 26–35 lbs; gain > 35 lbs	Data all presented in graph form: Using a gain of 15–25 pounds as reference for each obesity class, OR of preeclampsia lower with less weight gain and higher with more weight gain	Age, education, poverty (defined as participation in one or more subsidized programs) tobacco use, parity, chronic hypertension
Murakami et al., 2005[52] Japan hospital data	Pregravid weight: Self-reported at first visit Total weight gain: Measured on admission for birth	G1: < 8.5 kg gain G2: 8.5–12.5 kg gain G3: >12.5 kg gain	Estimated OR (95% CI) preeclampsia G1: 0.74 (0.37–1.48) G2: 1 G3: 0.57 (0.24–1.32)	Maternal age, parity, smoking, weight gain, gestational weeks; pregravid BMI

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
633 All BMIs Fair				
Ogunyemi et al., 1998,[60] USA, rural Alabama 582 All BMIs Fair	Pregravid weight: Self-reported Total weight gain: Weight at last prenatal visit	G1: "low weight gain" G2: "normal weight gain" G3: "high weight gain"	Incidence preeclampsia: G1: 10% G2: 7% G3: 19% (P = < .01)	Age, parity, pregravid BMI, tobacco use, hypertension
Thorsdottir, et al., 2002[53] Iceland, university hospital 615 BMI: 19.5–25.5 Fair	Pregravid weight: Self-reported Total weight gain: Maternity records (no specifics offered)	G1: <11.5 kg gain G2: 11.5–16.0 kg gain G3: 16.1–20.0 kg gain G4: > 20 kg gain	% gestational HTN G1: 1.5% G2: 4.6% G3: 5.1% G4: 9.2% (P = 0.026) % preeclampsia G1: 1.4% G2: 2.3% G3: 5.4% G4: 4.4% (P = 0.262)	Age, parity, height, gestational age
Bianco, et al., 1998[54] USA, New York Medical Center Database 11,840 Nonobese (BMI 19–27) and morbidly obese (BMI > 35) ages 20–34 Poor	Pregravid weight: Self-reported Total weight gain: Computed on measured weight within 4 weeks of delivery	Reported only for BMI > 35: G1: weight loss or no gain G2: 1–15 lb gain G3: 16–25 lb gain G4: 26–35 lb gain G5: > 35 lb gain	Incidence PIH G1: 11.8% G2: 13.7% G3: 13.7% G4: 12.4% G5: 21.3% (P = NS)	Race, parity, clinic service, substance abuse, and preexisting medical conditions

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Brennand et al., 2005[49] Quebec, Canada, First Nation's People (Cree) 603 BMI ≥ 18.5 Poor	Pregravid weight: Measured weight ≤ 14 wk GA used as proxy Total weight gain: Computed on last recorded weight within 4 wks of giving birth	G1: "Low weight gain" G2: "Acceptable weight gain" G3: "Excessive weight gain" All categories per Canadian Guidelines	HTN disorders G1: 7.3% G2: 12.5% G3: 19.3% (P = 0.051) PIH: G1: 3.7% G2: 6.3% G3: 4.4% (P = 0.698) Preeclampsia G1: 3.7% G2: 6.3% G3: 14.9% (P = 0.013)	None reported
Jensen et al., 2005[59] Danish medical centers 481 BMI ≥ 30 Poor	Pregravid weight: Self-reported Total weight gain: Details not provided	G1: < 5 kg gain G2: 5.0–9.9 kg gain G3: 10.0–14.9 kg gain G4: ≥ 15.0 kg gain	OR (95% CI) gestational HTN G1: 1 G2: 2.1 (0.8–5.7) G3: 3.6 (1.3–9.8) G4: 4.8 (1.7–13.1) (P = 0.001)	Results of 2 hour OGTT, age, pregravid BMI, gestational age, parity, smoking, ethnicity, and site of prenatal care
Kabiru and Raynor, 2004[51] USA Atlanta, public hospital database 5,131 BMI ≥20 Poor	Pregravid weight: First prenatal visit Total weight gain: Computed on weight at admission for birth	BMI < 25 first assessment: G1: no change BMI category G2: increase 1 category G3: increase > 1 category BMI ≥ 25 first assessment: G4: no change BMI category G5: increase 1 category G6: increase > 1 category	Incidence preeclampsia G1: 1.9% G2: 3.2% G3: 1.6% (P = .203) G4: 2.8% G5: 3.7% G6: 3.7% (P = .002)	Pregravid weight
Wataba et al., 2006[61] Japanese medical center	Total weight gain: Computed by delivery weight less pregravid weight (no details on how assessed) divided by	Separate analyses done for low, medium and high pregravid weight groups by following intervals of kg/week gain: < 0.15:	No clear trends for preeclampsia or severe preeclampsia by pregravid weight status and kg/week weight gains. AOR generally	

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
21,718 All BMIs Poor	gestational age	0.15–.20; 0.20–.25; 0.25–.30; 0.30–.35; 0.35–40; >0.40	crossed 1.0 or had wide confidence intervals.	

AOR, adjusted odds ratio; BMI, body mass index; CI, confidence interval; G, group; GA, gestational age; HTN, hypertension; kg, kilogram; lb, pounds; NS, not significant; OGTT, oral glucose tolerance test; OR, odds ratio; PIH, pregnancy-induced hypertension; USA, United States of America; wt, weight; wts, weights.

Overview of results The vast majority of the studies (7 fair,[4,25,53,55,58–60] 3 poor,[49,51,61]) found that increasing weight gain was associated with increasing likelihood of a pregnancy-induced hypertensive disorder. Two studies, one fair[52] and one poor,[54] did not support this association.

Detailed results Six studies specifically examined the impact of weight gain on the development of pregnancy-induced hypertension in women classified as obese by their pregravid weight status.[4,49,54,55,58,59] A prospective cohort study from Sweden examined the relationship of weight gain by pregravid BMI on pregnancy outcomes for 245,526 women who delivered term infants between 1994 and 2002.[58] When compared to a reference gain of 8–16 kg, the researchers found that gains of less than 8 kg were protective against the development of preeclampsia for all pregravid BMI categories. The finding was not significant, however, for those with BMIs < 20. Gaining more than 16 kg increased the likelihood of developing preeclampsia, especially for women who entered pregnancy with lower BMIs. The greatest increased risk was for women entering pregnancy at a BMI of 20 to 24.9 (OR, 2.31; CI, 2.15–2.49); the lowest increased risk was for women who entered pregnancy at a BMI ≥ 35 (OR, 1.50; CI, 1.17–1.92).

One US retrospective cohort study studied 771 women with BMIs of 30 or greater matched by race or ethnicity, delivery date, age categories, and parity categories with women of normal pregravid BMIs (19.8–26.0).[55] For women of normal weight, as weight increased the prevalence of preeclampsia steadily increased (P =.048) but increasing weight was not associated with the prevalence of gestational hypertension. For obese women, weight gain and the development of either gestational hypertension or preeclampsia were not significantly associated.

In a retrospective cohort study of 603 Cree women in Canada (rated poor quality), Brennand et al.[49] found that overweight and obese women had a significant unadjusted OR of 2.25 to 4.25 times higher, respectively, than normal weight women for pregnancy-induced hypertension and 1.25 to 3.45 times higher for preeclampsia.

Three retrospective cohorts were limited to women who entered pregnancy with BMIs ≥ 30.[4,54,59] In a study of 481 Danish women, the authors, using < 5 kg as the reference weight gain, found a statistically significant trend for development of pregnancy-associated hypertension with increasing weight (P = 0.0001).[59] A US study examined birth certificate data for 120,251 obese women classified according to the 1998 National Institute of Health obesity classes.[4] The researchers found that the amount of weight gain associated with minimal risk for preeclampsia differed by class of obesity but that, in all classes, a gain of less than 15 pounds was protective.

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The third study (rated poor quality) specifically investigated pregnancy and neonatal risks associated with BMIs > 35 in 527 morbidly obese women.[54] Although these women were more likely to experience obstetrical complications than a control population (BMIs 19–27), gestational weight gain did not affect the complication rate.

One other study did not support the association between weight gain and pregnancy-induced hypertension.[52] In this study, 633 Japanese women who gave birth to a singleton infant at 24–42 weeks of gestational age were studied. Pregravid BMI categories were those defined by the IOM. At the time of the study (2005) the Japan Society of Obstetrics and Gynecology did not have a recent guideline for weight gain during pregnancy; as a result, researchers used the frequency distributions from their population to set quartiles regarding weight gain and then set the parameters for insufficient and excessive gains accordingly. In this study, insufficient gain was defined as less than 8.5 kg and excessive gain as 12.5 kg. Finding no significant influence on weight gain and various perinatal outcomes of the mother or infant, the research team used other cut-off points and was still unable to find an appropriate criterion for predicting risk. The authors stated that their sample size was not sufficient to prove a lack of significance. Of note, the mean pregravid BMI of the sample was 20.9 ± 2.8 and the mean weight gain was 10.5 kg ± 3.4. While this study was assessed to be of fair quality, it has little, if any, generalizability to the United States because our population of childbearing women is more racially and ethnically diverse and have a higher mean BMI.

Gallstones

Study characteristics Two studies reported on the relationship between weight gain in pregnancy and cholelithiasis (gallstones)[62,63](Evidence Table 5).

Overview of results Two studies (1 poor[62] and 1 fair[63]) suggest a potential relationship between weight gain and cholelithiasis.

Detailed results One study reported on weight and the development of gallstones in a prospective study of 128 northern plains Native American and white women in 2004.[63] Nine independent variables including BMI, prenatal weight gain, prenatal physical activity, dietary fat, iron supplementation, age, parity, history of gallbladder disease, and serum cholesterol were analyzed. Weight assessments during pregnancy were carefully collected; how pregravid weights were determined is not specifically stated. Gestational weight gain had a nonsignificant, partial correlation of 0.09 and a beta coefficient of 0.13. A case-control study (rated poor quality), using data abstracted from birth certificates, reported on 6,211 women from the state of Washington who had a gallstone-related diagnosis at delivery or in the first year postpartum between 1987 and 2001.[62] Four controls were randomly selected for each case and matched for year of delivery. Multiple logistic regression found an inverse relationship between gestational weight gain and gallbladder disease. The OR per kg was. 0.98 (95% CI, 0.97–0.99; P = < 0.001). Maternal age, race, BMI based on self-reported pregravid weight, GDM, and infant gestational age were accounted for in the analysis.

Maternal Intrapartum Outcomes

Premature rupture of membranes (PROM)

Study characteristics Investigators explored the relationship of gestational weight gain and the risks for premature rupture of membranes (PROM) in two studies (Evidence Table 6).[64,65] One involved a total of 1,176 women who had experienced preterm delivery, defined as gestation ≤ 36 weeks, with PROM (n = 220), preterm delivery without PROM (n = 184), full-term delivery

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with PROM, defined as gestation ≥ 37 weeks, with at least 3 hours of PROM before the onset of labor, (n = 184), and 588 controls. Women were recruited following delivery at two academic medical centers in the United States.[64] In another study,[65] the investigators analyzed data for 62,167 women enrolled in the Danish National Birth Cohort who had pregravid weight and total weight gain recorded in the registry. They assessed the impact of obesity and gestational weight gain on the risk of various subtypes of preterm birth, including PROM. Pregravid weight and gestational gains were self-reported.

Overview of results Two fair studies[64,65] suggest that low weight gain (< 21 pounds) or low rate of weight gain (< 275 g per week) is associated with a higher risk of PROM for full-term pregnancies and preterm pregnancies.

Results for categorical measures of weight gain. A retrospective case-control study,[64] published in 1992, found that weight gain below the reference category of 21 pounds to 30 pounds significantly increased the risk of preterm delivery with PROM while weight gain above the reference category significantly reduced the risk of PROM. Similar trends were noted for full-term PROM. However, they were statistically significant only for gestational weight gain of 31 to 40 pounds when compared with women who gained 21 to 30 pounds (OR, 0.56; 95% CI, 0.33–0.94). Many potential confounders and effect modifiers were included in the analyses, including diet quality, BMI, age, race, parity, gestational iron supplementation, various medical conditions such as chlamydia that are considered risks for PROM, and smoking. The authors did not say if they adjusted for gestational age as a continuous variable. All variables, including pregravid weight and total weight gain, were assessed through a questionnaire administered to most of the subjects within 72 hours of giving birth.

Results for rate of weight gain In the Danish cohort study, women with a weekly weight gain of less than 275 grams per week had an adjusted hazards ratio for PROM of 1.5 (95% CI, 1.2–1.7) compared with women gaining between 276 grams and 675 grams weekly. When compared with women with BMIs of 18.5 to 24.9, those with either low (< 18.5) or high (> 30) BMIs had significantly higher rates of preterm delivery with PROM. The authors adjusted for prepregnancy BMI, weight gain, parity, mother's age, socio-occupational status, and lifestyle exposures in early pregnancy including smoking and alcohol exposure.[65]

Preterm labor

Study characteristics One poor study (Evidence Table 7) examined the relationship between gestational weight gain and preterm labor.[66] Preterm labor was not defined. This study, set in the United States, examined data from 11,505 women at the Boston Hospital for Women. The study defined gestational weight gain as pounds gained per week (≤ 0.4, 0.41 to 0.65, 0.66 to 0.9, and > 0.9).

Overview of results One poor study suggested that weight gain below 0.65 to 0.9 pounds per week significantly increased the risk of preterm labor.[66]

Results After controlling for an extensive list of confounders and effect modifiers (race, height, prepregnancy weight, infant sex, maternal age, education, health insurance, marital status, planned pregnancy, parity, previous induced or spontaneous abortion, previous stillbirth, uterine exposure to diethylstilbestrol, incompetent cervix, uterine anomaly, maternal morbidity, substance abuse, caffeine use, and prenatal care), the study found that weight below the reference range of 0.66 to 0.9 pounds per week significantly increased the risk of premature labor (AOR for 0.41–0.65 pounds per week: 1.7, 95% CI, 1.3–2.1; AOR for ≥ 0.4 pounds per week: 3.0; 95%

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CI, 2.2–4.2). Weight gain above 0.9 pounds per week did not have a significant effect on premature labor.

Postterm pregnancy

Study characteristics One study[58] used data from 245,526 pregnancies identified through the Swedish Medical Birth Registry (Evidence Table 8).

Overview of results One fair study found no evidence of association between gestational weight gain and postterm gestation.[58]

Results The author examined the effects of low (< 8 kg) and high weight gain (> 16 kg), compared with the effect of average weight gain (8–16 kg), on deliveries at > 41 weeks of gestation across strata of maternal pregravid BMI strata. After adjusting estimates for maternal age, parity, smoking in early pregnancy, and year of birth, no significant associations emerged between gestational weight gain and postterm gestational age. The study suggests that low or high gestational weight gain has no effect on postterm gestation.

Induction of labor

Study characteristics Five studies examined the relationship between gestational weight gain and labor induction (Table 6, Evidence Table 9) Of these, three were set in the United States,[25,51,67] one in Denmark,[59] and one in Finland.[68] Of these five studies, three were of poor quality.[51,59,68] Three examined induction of labor[59,67,68] and two examined failed induction of labor (defined as a birth that required a cesarean delivery despite induction of labor).[25,51] One of five studies was limited to obese, glucose-tolerant women,[67] and one to women of normal weight;[25] the other studies included women with a range of pregravid BMI. Each of the five studies defined gestational weight gain differently. Three used categories of gestational weight gain, with different cutpoints.[25,59,67] One stratified its sample by weight gain categories, comparing women with normal prepregnancy weight and weight gain during pregnancy with those with abnormal weight gain during pregnancy, defined as ≥ 20 kg or ≤ 5 kg during pregnancy; the study did not specify the prepregnancy weight status of women in these "abnormal" weight gain categories.[68] Another study characterized weight gain as change in BMI class between prepregnancy weight and weight at delivery.[51] The study defined BMI categories as follows: normal, BMI 20 to 24.9; overweight, BMI 25 to 29.9; obese I, BMI 30 to 34.9; obese II, BMI 35 to 39.9; morbid obesity, BMI ≥ 40.51.

TABLE 6. Gestational weight gain and induction of labor

Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
DeVader et al., 2007[25] USA, birth certificate data 94,696	Pregravid weight: Medical record: If missing, obtained from mother during postpartum hospital stay Total weight gain:	G1: < 30 lbs G2: 30–35 lbs G3: > 35 lbs	AOR for failed induction of labor vs. other birth outcomes G1: 0.68 (95% CI, 0.59–0.78) G2: 1.0	Maternal age, maternal race or ethnicity, maternal education, Medicaid status, tobacco use, alcohol use, maternal height, prior pregnancy, adequacy of prenatal care, child's sex, and child's birth year

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Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Normal weight only Fair	Obstetrical records		G3: 1.51 (95% CI, 1.39–1.64)	
Graves et al., 2006[67] USA, midwifery practices 1,500 All weights/BMI Fair	Pregravid weight: Actual prepregnant weight or early first trimester weight documented in medical records Total weight gain: Last prenatal assessment	≤ 45 pounds vs. > 45 pounds	OR induction of labor for > 45 lb: 1.5 (95% CI, 1.0–2.4)	Maternal BMI, infant birthweight, and gestational age at delivery
Ekblad and Grenman, 1992[68] Finland, hospital 357 Normal weight only Poor	Pregravid weight: Data from records, unclear if self-reported Total weight gain: Last prenatal assessment	G1: weight gain ≤ 5 kg G2: weight gain ≥ 20 kg G3: reference (normal prepregnancy weight and normal weight gain [undefined])	Percentage induced G1: 23% G2: 43% G3: 24 $P < 0.05$ for G2 vs. G3	NA
Jensen et al., 2005[59] Denmark, university hospitals 481 Obese only Poor	Pregravid weight: Records or self-report of pregravid BMI Total weight gain: Last prenatal assessment	G1: < 5.0 kg G2: GWG 5.0–9.9 kg G3: GWG 10.0–14.9 kg G4: GWG ≥ 15 kg	OR for induction of labor G1: 1.0 G2: 2.7 (95% CI, 1.3–5.7) G3: 2.8 (95% CI, 1.3–5.9) G4: 3.7 (95% CI, 1.7–8.0) P for trend = 0.002	Age, pregravid BMI, 2 hour OGTT result, smoking, gestational age
Kabiru and	Pregravid weight:	G1: normal BMI, no	Percent failed	NA

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Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Raynor, 2004[51] USA, hospital 5,131 All BMIs > 20 Poor	Weight at first prenatal visit Total weight gain: Weight at admission for birth	change in BMI between first prenatal visit and delivery G2: normal BMI, 1 category increase in BMI between first prenatal visit and delivery G3: normal BMI, > 1 category increase in BMI between first prenatal visit and delivery G4: overweight BMI, no change in BMI between first prenatal visit and delivery G5: overweight BMI, 1 category increase in BMI between first prenatal visit and delivery G6: overweight BMI, > 1 category increase in BMI between first prenatal visit and delivery	induction of labor G1: 4.7 G2: 9.2 G3: 15.9 *P* < 0.001 G4: 7.9 G5: 10.3 G6: 14.6 *P* < 0.001	

AOR, adjusted odds ratio; BMI, body mass index; G, group; GWG: gestational weight gain; OGTT, oral glucose tolerance test.

Overview of results Two fair[25,67] and three poor[51,59,68] studies examined the association of increased gestational weight gain and labor induction[59,67,68] or failure of labor induction,[25,51] and found a risk of labor induction or failure of induction with increased gestational weight gain.

Results The three studies that looked at induction of labor found a statistically significant increase in the risk of labor induction with increases in gestational weight gain.[59,67,68] The magnitude of the effect across all three studies cannot be summarized because of differences in the definition of weight gain and in the nature of confounders controlled for in the analysis. Both studies examining failed induction of labor found a significant association between gestational weight gain and increase in the risk of failed induction compared with all other delivery routes.[25,51]

Length of labor

Study characteristics Three cohort studies, set in Finland and the United States, examined the association between gestational weight gain and labor (Table 7, Evidence Table 10).[68–70] Two

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studies focused on length of labor,[68,69] one on labor abnormalities.[70] The definition of gestational weight gain differed across studies. One study examined an overall increase in weight of > 25 percent or ≤ 25 percent for women with normal pregravid weight (90–120 percent of normal weight for height based on Metropolitan Life Insurance Company Table for 1983).[69] Another reported on categories of gestational weight gain (< 16 pounds, 16–25 pounds, 26–35 pounds, and > 35 pounds) for pregravid BMI categories defined by the IOM.[70] The third study, of poor quality, stratified its sample by weight gain categories, comparing women with normal prepregnancy weight and weight gain during pregnancy with those with abnormal weight gain (≥ 20 kg, or ≤ 5 kg) during pregnancy; the study did not specify the prepregnancy weight status of women in these "abnormal" weight gain categories.[68]

TABLE 7. Gestational weight gain and length of labor

Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Johnson et al., 1992[70] USA, prenatal clinics 3,191 All weights/BMI Fair	Pregravid weight: Self-report collected at first antepartal visit Total weight gain: Last prenatal visit	G1: total weight gain < 16 pounds G2: total weight gain 16–25 pounds G3: total weight gain 26–35 pounds G4: total weight gain > 35 pounds	Elevated odds of labor abnormalities only in the group gaining > 35 pounds compared with women gaining < 16 pounds; not significant when adjusted for confounders Trend analysis showed risk of labor abnormalities with increased weight gain, a difference in 10 lb. corresponds to OR=2 (P< 0.0001) after adjusting for BMI, patient care (private vs. nonprivate), parity, infant sex, hypertension, and macrosomia	Prepregnancy weight quartile, height (tertile), BMI category, race/ethnicity, marital status, private physician, parity, infant sex, maternal age, hypertension, and birthweight
Purfield and Morin, 1994[69] USA, Tertiary care medical center 104 Normal weight women only Fair	Pregravid weight: Self-report as noted in medical chart Total weight gain: Weight at admission to hospital for birth	G1: prepregnant weight increased by 25% or less G2: prepregnant weight increased by more than 25%	Normal weight primigravidas with a low risk pregnancy who gained an excessive amount of weight had a longer mean length of second stage labor than women who gained less weight Minutes of length of second stage in minutes by weight groups (SD): G1: 72.42 (46.69) G2: 93.28 (52.87) t = -2.05 *P* = 0.02	NA
Ekblad and Grenman, 1992[68]	Pregravid weight: Data from records,	G1: weight gain ≤ 5 kg G2: weight gain ≥ 20 kg	Labor pattern - I stage (minutes± SD) G1: 333±208 G2: 374±208	NA

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Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Finland, hospital 357 Normal weight only Poor	unclear if self-reported Total weight gain: Last clinically measured weight prior to delivery	G3: reference (normal prepregnancy weight and normal weight gain [undefined])	G3: 346±188 Labor pattern - II stage (minutes) G1: 15±18) $P < 0.05$ compared to reference category G2: 27±25 G3: 21±18 Labor pattern - III stage (minutes) G1: 13±13 G2: 13±11 G3: 12±12	

BMI, body mass index; G, group; SD, standard deviation.

Overview of results Two of three studies (2 fair,[69,70] 1 poor[68]) suggested that higher weight gain among normal weight women of normal weight was associated with longer labor.[68,69]

Results The two studies that examined length of labor demonstrated significantly longer second stage of labor for women with high weight gain, based on samples of 35,768 and 10,469 respectively. Neither study controlled for confounders or effect modifiers.

The study that reported on labor abnormalities found higher odds of labor abnormalities for women gaining > 35 pounds compared with women gaining < 16 pounds. These odds lost statistical significance when adjusted for confounders. In a trend analysis, the study found a higher risk of labor abnormalities with increased weight gain, suggesting that a difference of 10 pounds corresponds to an OR of 2 (P < 0.0001) after adjusting for BMI, patient care (private vs. nonprivate), parity, infant sex, hypertension, and macrosomia.[70]

Mode of delivery

Study characteristics Twenty-one cohort studies reported on the relationship between gestational weight gain and mode of delivery (Table 8, Evidence Table 11).[4,25,49,51,52,54,58,59,61,67–78] Thirteen studies were set in the United States,[4,25,51,54,67,69–71,73–77] three in Canada,[49,53,72,78] two in Japan,[52,61] one in Sweden,[58] one in Denmark,[59] and one in Finland.[68]

TABLE 8. Gestational weight gain and mode of delivery

Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Cedergren, 2006[58] Sweden, Medical Birth Registry	Pregravid weight: Self-report; if unknown, standardized measurement is	Weight gain < 8 kg, 8–16 kg, and > 16 kg for each BMI class below	AOR for weight gain < 8 kg for cesarean section compared with weight gain 8–16 kg (95% CI) G1: 1.07 (0.89–1.29)	Maternal age, parity, smoking in early pregnancy, and year of birth

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Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
245,526 All weights/BMI Fair	made during first visit to maternity health care center Total weight gain: Measured when woman entered delivery unit	G1: BMI< 20 G2: BMI 20–24.9 G3: BMI 25–29.9 G4: BMI 30–34.9 G5: BMI ≥ 35	G2: 0.98 (0.92–1.05) G3: 0.88 (0.82–0.95) G4: 0.81 (0.73–0.90) G5: 0.75 (0.66–0.87) AOR for weight gain > 16 kg for cesarean section compared with weight gain 8–16 kg (95% CI) G1: 1.29 (1.17–1.43) G2: 1.24 (1.19–1.29) G3: 1.23 (1.17–1.30) G4: 1.22 (1.10–1.35) G5: 1.27 (1.05–1.52) AOR for weight gain < 8 kg for instrumental delivery compared with weight gain 8–16 kg (95% CI) G1: 0.89 (0.71–1.11) G2: 0.88 (0.80–0.96) G3: 0.85 (0.76–0.95) G4: 0.75 (0.63–0.88) G5: 0.83 (0.65–1.03) AOR for weight gain > 16 kg for instrumental delivery compared with weight gain 8–16 kg (95% CI) G1: 1.28 (1.15–1.43) G2: 1.19 (1.14–1.25) G3: 1.14 (1.06–1.23) G4: 1.09 (0.93–1.27) G5: 1.04 (0.77–1.40)	
Chen et al., 2004[73] USA, private practice 3,355 All weights/BMI Fair	Pregravid weight: Weight taken at first prenatal visit if presented before 20 weeks; if after 20 weeks, self report Total weight gain: Last clinically measured weight prior to delivery	Gestational weight gain in lbs	Progression of AOR of cesarean delivery weight gain (for every 5 lbs): 1.094 (1.074–1.115)	BMI, maternal height, maternal age, pregnancy weight gain, gestational age at delivery, and fetal birthweight
DeVader et al., 2007[25]	Pregravid weight: Medical record; if	G1: < 30 lbs G2: 30–35 lbs	AOR for cesarean (95% CI): G1: 0.82 (0.78–0.87)	Maternal age, maternal race or ethnicity,

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Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
USA, birth certificate data 94,696 Normal weight only Fair	missing, obtained from mother during postpartum hospital stay Total weight gain: Obstetrical records	G3: > 35 lbs	G2: 1.0 G3: 1.35 (1.29–1.40) AOR for instrumental (95% CI): G1: 0.97 (0.90–1.04) G2: 1.0 G3: 1.03 (0.97–1.08)	maternal education, Medicaid status, tobacco use, alcohol use, maternal height, prior pregnancy, adequacy of prenatal care, child's sex, and child's birth year
Graves et al., 2006[67] USA, midwifery practices 1,500 All weights/BMI Fair	Pregravid weight: Actual prepregnant weight or early first trimester weight documented in medical records Total weight gain: Last prenatal assessment	≤ 45 lbs vs. > 45 lbs	Greater weight gain in pregnancy was not associated significantly with route of delivery	Prepregnancy BMI category, total prenatal weight gain category, induction of labor, newborn birthweight ≥ 4,000 g, gestational age > 41 weeks, and race/ethnicity
Jain et al. 2007[77] USA, birth certificate records and Pregnancy Risk Assessment Monitoring System 7,661 All weights/BMI Fair	Pregravid weight: Not stated Total weight gain: Birth certificate	G1: WG ≤ 15 lbs G2: WG 15–24 lbs G3: WG 25–35 lbs G4: WG ≥ 35 lbs	AOR for primiparous cesarean delivery (from model including interaction term for overweight/obese + > 25 lbs weight gain) G1: 0.95 (0.59–1.52) G2: 1.0 (ref) G3: 1.10 (0.76–1.60) G4: 1.62 (1.10–2.39) AOR for multiparous cesarean delivery (from model including interaction term for overweight/obese + > 25 lbs weight gain) G1: 1.11 (0.60–2.04) G2: 1.0 (ref) G3: 1.08 (0.63–1.85) G4: 1.95 (1.02–3.72)	Maternal age, pregravid BMI, parity, education, race/ethnicity, US/foreign origin, interaction terms for pregravid BMI and weight gain
Johnson et al., 1992[70] USA, prenatal clinics	Pregravid weight: Self report collected at first antepartal visit Total weight gain:	G1: total weight gain < 16 lbs G2: total weight gain 16–25 lbs G3: total weight gain 26–35 lbs	AOR for unscheduled cesarean (95% CI) G1: 1.0 G2: 0.95 (0.6–1.5) G3: 1.3 (0.86–1.95) G4: 1.95 (1.32– 2.87)	Prepregnancy weight quartile, height (tertile), BMI category, private physician (yes/no), maternal age, parity, birthweight, diabetes,

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Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
3,191 All weights/BMI Fair	Last prenatal visit	G4: total weight gain > 35 lbs		hypertension, and maternal education
Joseph et al., 2003[72] Nova Scotia Atlee Perinatal Database 100,259 All weights/BMI Fair	Pregravid weight: Data taken from standardized forms and hospital medical records - no mention of self report Total weight gain: Not explained by authors - data taken from maternity records	G1: < 5 kg G2: 5–9 kg G3: 10–14 kg G4: 15–19 kg G5: ≥ 20 kg	AOR for cesarean delivery (95% CI) G1: 1.10 (1.00–1.20) G2: 1.04 (0.99–1.10) G3: 1.00 G4: 1.09 (1.05–1.14) G5: 1.41 (1.35,–1.47)	Age, parity, prepregnancy weight, smoking, pregnancy (singleton or multiple), hypertension, diabetes, previous fetal death, induction, epidural, physician type, time
Kiel et al., 2007[4] USA, birth registry 120,170 Obese women only Fair	Pregravid weight: Self report from data on birth certificate Total weight gain: Abstracted from medical chart	All obese women G1: Loss 10 lbs or more G1: Loss 2–9 lbs G1: No change G1: Gain 2–9 lbs G1: Gain 10–14 lbs G1: Gain 15–25 lbs G1: Gain 25–35 lbs	Compared with women who gained 15–25 lbs during their pregnancies, those who gained less weight had significantly lower odds of preeclampsia, cesarean delivery, and LGA births, but higher odds for SGA births Magnitude differed by obesity classification, even after adjusting for known or suspected confounders	Age, race, parity, education, poverty (enrollment in Medicaid, WIC, food stamp programs), tobacco use, chronic hypertension
Murakami et al., 2005[52] Japan, hospital 633 All weights/BMI Fair	Pregravid weight: Self report at first visit to clinic Total weight gain: Based on last clinically measured weight prior to delivery	G1: < 8.5 kg G2: 8.5–12.5 kg G3: > 12.5 kg	AOR for cesarean delivery (95% CI) G1: 1.08 (0.56–2.07) G2: 1.00 G3: 1.23 (0.61–2.48)	Maternal age, parity, smoking, prepregnancy BMI, and gestational age (weeks)
Purfield and Morin, 1995[69] USA, Tertiary care medical	Pregravid weight: Self report as noted in medical chart Total weight gain:	G1: prepregnant weight increased by 25% or less G2: prepregnant weight increased by	Higher rate of vacuum extraction and cesarean delivery and lower rate of spontaneous vaginal delivery with excessive weight gain	NA

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Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
center 104 Normal weight women only Fair	Weight at admission to hospital for birth	more than 25%	No difference in forceps delivery and vaginal delivery by weight gain status Vaginal delivery G1: n = 27 G2: n = 9 Vacuum extraction G1: n = 14 G2: n = 25 low forceps G1: n = 8 G2: n = 8 Cesarean section G1: n = 3 G2: n = 10 χ^2 = 15.87, *P* = 0.001 for all 4 modes of delivery by weight groups	
Rosenberg et al., 2005[71] USA, vital statistics data 329,988 All weights/no BMI Fair	Pregravid weight: Self report on birth certificate Total weight gain: Weight data on birth file	< 41 vs. ≥ 41 pounds	AOR for primary cesarean (95% CI): 1.38 (1.34–1.41)	Age, parity, GDM, pregnancy-induced hypertension, preeclampsia, prepregnancy weight, chronic diabetes, chronic hypertension, marital status, maternal education, mother's birthplace, prenatal care payer, social risk, trimester prenatal care began
Shepard et al., 1998[75] USA, obstetrical practices 2,301 All weights/BMI Fair	Pregravid weight: Self report before 15 weeks of gestation Total weight gain: Self report of weight at delivery	Proportional weight gain (total weight gain/prepregnancy weight) and absolute weight gain	G1: Proportional Gain: Adjusted Relative Risk (95% CI) G2: Absolute Gain: Adjusted RR (95% CI) Underweight (< 19.4), ≤ median G1: 1.00 G2: 1.00 Underweight (< 19.4), >	Preeclampsia, gestational diabetes, placental problems, fetal distress, macrosomia, induction, maternal age and height, parity, ethnicity, and marital status

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Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
			median G1: 2.08 (0.86–5.04) G2: 1.20 (0.56–2.59) Low-Average (19.5–22.4), ≤ median G1: 1.62 (0.90–3.67) G2: 1.00 (0.54–1.84) Low-Average (19.5–22.4), > median G1: 2.35 (1.06–5.21) G2: 1.62 (0.94–3.02) High-Average (22.5–28.4), ≤ median G1: 2.78 (1.26–6.12) G2: 1.80 (1.01–3.21) High-Average (22.5–28.4), > median G1: 3.06 (1.40–6.73) G2: 2.02 (1.14–3.57) Obese (> 28.5), ≤ median G1: 3.25 (1.40–7.54) G2: 2.13 (1.12–4.08) Obese (> 28.5), > median G1: 2.69 (1.18–6.16) G2: 1.65 (0.90–3.03)	
Sherrard et al., 2007[78] Canada, hospital database 63,390 All BMIs Fair	Pregravid weight: Self report Total weight gain: Self report or measured	Rate of weight gain (kg/wk) G1: Low (≤0.17) G2: Normal (0.18–0.50) G3: High (>0.50)	AOR for unlabored cesarean, primary G1: 0.79 (0.59–1.05) G2: 1.00 (ref) G3: 1.03 (0.64–1.64) AOR for labored cesarean, primary G1: 0.77 (0.68–0.86) G2: 1.00 (ref) G3: 1.40 (1.23–1.60) AOR for unlabored cesarean, repeat G1: 0.91 (0.76–1.09) G2: 1.00 (ref) G3: 1.38 (1.04–1.83)	BMI, gestational diabetes, pregnancy-induced hypertension, macrosomia, socioeconomic factors, parity, and maternal age

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Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
			AOR for labored cesarean, repeat G1: 0.79 (0.54–1.15) G2: 1.00 (ref) G3: 1.22 (0.72–2.09)	
Witter et al., 1995[76] USA,obstetric database at major medical center 4,346 All weights/BMI Fair	Pregravid weight: Self report, unclear at what timepoint Total weight gain: Weight recorded at last prenatal visit	Pregnancy weight gain (kg)	AOR for cesarean (95% CI): 1.04 (1.02–1.05)	Age, pregravid BMI, height, at least one previous viable pregnancy, diagnosis of preeclampsia during the current pregnancy, previous cesarean delivery
Bianco et al., 1998[54] USA, major medical center 11,926 BMI OF 27 and 34 are excluded from analysis Poor	Pregravid weight: Unclear Total weight gain: Weight from before 36 weeks gestation or not within 4 weeks of delivery Maternal weight gain outcomes by BMI presented for morbidly obese women only, N: 613	G1: 0 or weight loss G2: 1–15 lbs G3: 16–25 lbs G4: 26–35 lbs G5: > 35 lbs	Cesarean % G1: 25.8% G2: 26.8% G3: 28.8% G4: 35.0% G5: 33.8% (P = NS)	NA
Brennand et al., 2005[49] Canada, medical records 603 All weights/BMI Poor	Pregravid weight: Routine prenatal care medical records, measured within 14 weeks of gestation Total weight gain: Based on last clinically measured weight prior to delivery: within 4 weeks of birth	Primigravid women (maternal weight gain outcomes by BMI presented only for obese women) G1: Obese - low weight gain (< 7 kg) G2: Obese - acceptable weight gain (7–11.5 kg) G3: Obese - excessive weight gain (> 11.5 kg)	Cesarean section (%) G1: 25.3 G2: 23.5 G3: 23.7 χ^2 P = 0.952 G4: 24.1	NA

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Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
	Primigravid women (maternal weight gain outcomes by BMI presented only for obese women)	G4: Total		
Ekblad and Grenman, 1992[68] Finland, hospital 357 Normal weight only Poor	Pregravid weight: Data from records, unclear if self reported Total weight gain: Routine prenatal care or maternity records based on last clinically measured weight prior to delivery	G1: weight gain ≤ 5 kg G2: weight gain ≥ 20 kg G3: reference (normal prepregnancy weight and normal weight gain [undefined])	Normal vaginal delivery (%) G1: 90 $P < 0.05$ compared to reference category G2: 64 G3: 71 Forceps or vacuum delivery (%) G1: 3 G2: 13 G3: 5 Breech (%) G1: 1 G2: 0 G3: 2 Cesarean section - elective% G1: 3 G2: 5 G3: 13 Cesarean section - emergency% G1: 3 G2: 18 G3: 9	NA
Jensen et al., 2005[59] Denmark, university hospitals 481 Obese only Poor	Pregravid weight: Records or self report of pregravid BMI Total weight gain: Last prenatal assessment	G1: GWG 5.0–9.9 kg G2: GWG 10.0–14.9 kg G3: GWG ≥ 15 kg	OR for cesarean delivery (95% CI) G1: 1.0 G2: 2.4 (1.1–5.3) G3: 3.0 (1.4–6.4) G4: 3.6 (1.6–7.8) P for trend=0.002	2-h OGTT result, maternal age, prepregnancy BMI, gestational age (continuous variables), parity, smoking, ethnic background, and clinical center (categorical variables
Kabiru and	Pregravid weight:	Primary cesarean	Operative vaginal delivery	Pregravid BMI, none

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Author, Date Country, Setting Sample Size Baseline BMI Quality	**Pregravid Weight (How Measured) Total Weight Gain (How Measured)**	**Definition of Groups**	**Results**	**Confounders and Effect Modifiers Included in Analysis**
Raynor, 2004[51] USA, hospital 5,131 All BM's > 20I Poor	First prenatal visit Total weight gain: Weight at admission for birth	G1: normal BMI, no change in BMI between first prenatal visit and delivery G2: normal BMI, 1 category increase in BMI between first prenatal visit and delivery G3: normal BMI, > 1 category increase in BMI between first prenatal visit and delivery G4: overweight BMI, no change in BMI between first prenatal visit and delivery G5: overweight BMI, 1 category increase in BMI between first prenatal visit and delivery G6: overweight BMI, > 1 category increase in BMI between first prenatal visit and delivery	G1: 11.4 G2: 12.4 G3: 12.2 $P = 0.837$ G4: 8.4 G5: 11.4 G6: 17.3 $P < 0.001$ Cesarean delivery rate for failure to progress G1: 2.5 G2: 6.5 G3: 10.2 $P = 0.203$ G4: 3.5 G5: 6.9 G6: 10.2 $P = 0.002$	other
Wataba et al., 2006[61] Japan, academic medical center 21,718 All weights/BMI Poor	Pregravid weight: Unreported Total weight gain: From hospital database/register	Rate of weight gain, categorized differently across different BMI groups	For nulliparous, low BMI women: Higher risk of cesarean delivery for women with weight gain ≥ 0.4 kg/week (AOR: 2.30 [95% CI, 1.06–4.98] compared with women gaining 0.25–0.3 kg/week) For nulliparous, medium BMI women: Higher risk of cesarean delivery for women with weight gain ≥ 0.4 kg/week (AOR: 1.61 [95% CI, 1.21–2.14] compared with women gaining 0.25–0.3 kg/week) and for women with weight gain 0.35–0.4 kg/week (AOR: 1.68 [95% CI, 1.22–2.30]	Parity, baseline BMI

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Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
			compared with women gaining 0.25–0.3 kg/week) For nulliparous, high BMI women: No increased risk For parous, medium BMI women: Higher risk of cesarean delivery for women with weight gain 0.25–0.3 kg/week (AOR: 1.49 [95% CI, 1.09–2.04] compared with women gaining 0.20–0.25 kg/week) No data presented on cesarean delivery for other BMI groups for parous women	
Young et al., 2002[74] USA, private practice 3,375 All weights/BMI Poor	Pregravid weight: Self reported Total weight gain: Based on last clinically measured weight prior to delivery	G1: < 30 lbs G2: 30–35 lbs G3: > 35 lbs	Increase in overall cesarean delivery rate with increased weight gain was significant at all BMI levels	BMI

AOR, adjusted odds ratio; BMI, body mass index; G, group; GDM, gestational diabetes mellitus; kg, kilogram; lbs, pounds; LGA, large-for-gestational-age; SGA, small-for-gestational-age.

All 21 studies examined cesarean delivery as an outcome. Five examined instrumental delivery in addition to cesarean delivery.[25,51,58,68,69] Eight studies reported on cesarean delivery without providing further definition.[4,25,49,54,58,59,74,76] The studies that offered some detail varied in their definition; these studies defined cesarean delivery as failure to progress,[51] unscheduled cesarean,[67,70] cesarean including elective and emergency,[52] elective cesarean and emergency cesarean,[61,68] cephalopelvic disproportion/failure to progress, fetal distress, breech, and other indications,[73] cesarean delivery for cephalic presentation,[77] and cesarean delivery for singleton cephalic presentation separately analyzed for primary and repeat cesareans, with and without labor.[78] A key consideration in assessing the risk of cesarean delivery is the route of previous delivery; with the declining prevalence of vaginal birth after cesarean (VBAC), a history of prior

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cesarean delivery is likely to result in cesareans for all subsequent pregnancies. Studies that fail to account for prior route of delivery cannot therefore control for its confounding effect. Eleven studies did not take into account prior route of delivery.[4,25,49,52,54,58,59,61,67,68,70]

Definitions of gestational weight gain also varied greatly. Some studies used categorical definitions designed to identify high weight gain alone,[67,71] weight gain across a spectrum of gain,[4,25,49,52,54,58,59,70,72,74,77] continuous weight gain,[73,76] rate of weight gain,[61,78] and weight gain in relation to pregravid weight.[51,68,69,75]

Overview of results Across the 14 fair[4,25,52,58,67,69–73,75–78] and 7 poor[49,51,54,59,61,68,74] studies that examined gestational weight gain as a predictor of route of delivery, only four (2 poor) failed to show an effect of gestational weight gain on route of delivery.[49,52,54,67] The remainder demonstrated higher risks of cesarean delivery associated with gestational weight gain, with some evidence suggesting more pronounced risks associated with high pregravid BMI status. Notably, only 10 studies controlled for route of previous delivery. Of these, five controlled for co-morbidities that could have been significant confounders for route of delivery.[71,72,75,76,78] One study explicitly examined the interactions between weight gain and pregravid weight; it did not find any significant effect.[77]

Results across BMI categories for categorical measures of weight gain. Fifteen studies considered weight gain across a range of pregravid weight categories. Of these, two fair studies defined gestational weight gain as a categorical variable (≤ 45 pounds vs. > 45 pounds,[67] and < 41 vs. ≥ 41 pounds[71]). One of these two studies, limited to primary cesarean, found a significant association between weight gain and cesarean delivery (AOR, 1.38; 95% CI, 1.34–1.41).[71] This study found pregravid BMI, diabetes, and hypertension to also be strong predictors of cesarean delivery. The other, which did not control for route of previous delivery, did not find any association between gestational weight gain and route of delivery.[67]

Six studies defined gestational weight gain in categories that allowed for the identification of both low and high weight gain, across a spectrum of pregravid weight categories;[52,58,70,72,74] of these, one was rated poor quality[74] and the remainder fair. One study showed no difference in cesarean delivery by weight gain category.[52] All others showed some patterns of association with higher levels of weight gain, although the magnitude of the effect varied.[58,70,72,74] Three studies found similar thresholds for the rise in risk of cesarean delivery, namely, weight gains in excess of 15 kg[72] or greater than 35 pounds.[70,77] One study looked at both relatively low weight gain (< 8 kg) and relatively high weight gain (> 16 kg) in comparison with weight gain of 8 to 16 kg.[58] The study found no statistically significant risk of cesarean delivery for low or normal BMI categories but significantly higher risk with higher weight gain for overweight, obese, and morbidly obese women.[58] One study examined the effects of pregravid weight, gestational weight gain, and the interaction between the two as predictors of cesarean delivery for primiparous and multiparous women (defined in two different ways). The study found that pregravid overweight or obese status as well as weight gain over 35 pounds are associated with the risk of cesarean delivery for primiparous women, but no significant effect of the interaction between weight gain and pregravid weight. The study did not find consistently significant effects of these variables on cesarean delivery for multiparous women; the previous route of delivery, a likely confounder, was not controlled in these analyses.

Results across BMI categories for rate of weight gain. Two studies, one rated fair[78] and the other poor,[61] examined the rate of weight gain across a range of pregravid weight categories. The fair study separately examined the risks of primary and repeat cesarean, with and without labor in models that accounted for gestational diabetes, pregnancy-induced hypertension, macrosomia,

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socioeconomic factors, parity, or maternal age. The study found that a high rate of weight gain (> 0.5 kg/week) significantly increased the risk of a labored primary cesarean, while a low rate of weight gain (≤ 0.17 kg/week) significantly reduced the risk, compared with an average rate of weight gain (0.18–0.50 kg/week). High rate of weight gain significantly increased the risk of unlabored repeat cesareans. The rate of weight gain during pregnancy did not predict the risk of primary unlabored cesarean or repeat labored cesarean. In contrast, pregravid overweight and obese status was a significant risk factor for all types of cesarean delivery. The poor study examined associations between cesarean delivery and rates of weekly weight gain (seven categories), categorized differently across different BMI groups (three groups) and parity (two categories), resulting in 42 comparisons.[61] As with the fair study, a subset of results were significant, suggesting that for nulliparous women with low or medium BMI, high rates of weight gain increased the risks of cesarean delivery. Specifically, the study found:

- among nulliparous, low-BMI women, a higher risk of elective cesarean delivery for women with weight gain ≥ 0.4 kg per week (AOR: 2.30 [1.06–4.98]) than for women gaining between 0.25 and 0.3 kg per week.
- among nulliparous, medium-BMI women, a higher risk of elective cesarean delivery
    - for women with weight gain ≥ 0.4 kg per week (AOR: 1.61 [1.21–2.14]) than for women gaining 0.25 to 0.3 kg per week and
    - for women with weight gain of 0.35 to 0.4 kg per week (AOR: 1.68 [1.22–2.30]) than for women gaining 0.25–0.3 kg per week.
    -

The study examined risk of emergency (rather than elective) cesarean for high BMI nulliparous women and failed to find an association with gestational weight gain rates.

In examining outcomes for parous women, with a single exception—a higher risk of cesarean delivery for women with weight gain 0.25–0.3 kg/wk (AOR, 1.49 [1.09–2.04]) than for women gaining 0.20 to 0.25 kg/week—the poor study did not find statistically significant effects for rate of weight gain on cesarean delivery for parous, medium-BMI women. No data were presented on cesarean delivery (emergency or elective) for low or high BMI groups for parous women.

Results across BMI categories for continuous measures of weight gain Of the 15 studies that considered a range of pregravid weight categories, two fair studies modeled gestational weight gain as a continuous variable.[73,76] Both found significantly higher risks of cesarean delivery with increasing weight. One study identified the progression of AOR of cesarean delivery weight gain for every 5 pounds of gestational weight gain to be 1.094 (95% CI, 1.074–1.115).73 The second study calculated the attributable risk for cesarean delivery of gaining more than 16 kg to be 6.9 percent.[76] Both studies account for route of previous delivery.

Results across BMI categories for other measures of weight gain Of these same 15 studies, three (1 fair,[75] and 2 poor[51,53,68]) defined gestational weight gain as a function of pregravid weight.[51,68,75] Two of three studies controlled for previous route of delivery by limiting their sample to primary cesareans. The fair study used underweight women who gained less than the median for proportional weight gain (total weight gain/prepregnancy weight) as the referent.[75] This study found higher risks of cesarean delivery for all other categories, although risks were statistically significant only for women in the high and obese BMI category in all weight gain categories and women in the average BMI category who gained less than the median proportional weight gain. One poor-quality study characterized weight gain as change in BMI class between prepregnancy weight and at delivery. BMI categories were defined as follows:

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normal, BMI 20 to 24.9; overweight, BMI 25 to 29.9; obese I, BMI 30 to 34.9; obese II, BMI 35 to 39.9; morbid obesity, BMI ≥ 40.51 This study found no statistically significant association between weight gain and cesarean delivery among normal-BMI women but did find a positive association for high-BMI women. The extent to which these results corroborate findings from the fair study is hard to determine given the differences in the reference category, but both studies imply that increased risks of cesarean are pronounced among overweight and obese women. A third study, also of poor quality, examined differences in route of delivery between women with normal prepregnancy weight and weight gain during pregnancy with those with abnormal weight gain (≥ 20 kg or ≤ 5 kg) during pregnancy; the study did not specify the prepregnancy weight status of women in these "abnormal" weight gain categories.[68] Unlike the other two studies in this category, the rates for cesarean delivery were not statistically significantly different across groups. The study did find a statistically significant higher rate of normal vaginal delivery for low weight gain women compared with the reference category of normal prepregnancy weight and weight gain. Notably, this study did not control for route of previous delivery.

Results within BMI categories for other measures of weight gain. Two studies were limited to women of normal BMI.[25,69] Both suggested an increase in the risk of cesarean delivery with increasing weight gain, defined in one study as 25 percent gain over prepregnancy weight,[69] and in the other as a weight gain > 35 pounds as compared with a weight gain of 30 to 35 pounds. Weight gain of < 30 pounds was associated with a lower risk of cesarean delivery, suggesting a linear increase in the risk of cesarean delivery with weight gain for women of normal weight. One of the two studies controlled for previous cesarean delivery by limiting its sample to primigravidas.[69]

Four studies limited their analysis to obese women or morbidly obese women.[4,49,54,59] Of these, two studies (both rated poor quality) suggested no difference in cesarean delivery outcomes by gestational weight gain.[49,54] Neither accounted for route of previous delivery.

The other two studies suggested that the risk of cesarean delivery increased with higher levels of weight gain for obese and morbidly obese women.[4,59] One poor study suggested that risk increases with higher levels of weight gain.[59] Compared with the risk of cesarean delivery for women gaining < 5 kg, the results were as follows: AOR of cesarean delivery for women gaining 5 to 9.9 kg, 2.4 (95% CI, 1.1–5.3); AOR for women gaining 10 to 14.9 kg, 3.0 (95% CI, 1.4–6.4); and AOR for women gaining ≥ 15 kg, 3.6 (95% CI, 1.6–7.8).59 The other study suggested that women who had lower weight gain than women who gained 15 to 25 pounds had lower risks of cesarean delivery, but the magnitude of the association varied by obesity classification.[4] Overall, across a range of outcomes the study suggested that minimal risk may correspond to a weight gain of 10 to 25 pounds for class I obese women (BMI 30–34.9), a weight gain of 0 to 9 pounds for class II obese women (BMI 35–39.9), and a weight loss of 0 to 9 pounds for class III obese women (BMI > 40). Neither of these studies controlled for route of previous delivery.

Results for instrumental delivery Five studies examined instrumental delivery in addition to cesarean delivery.[25,51,58,68,69] Two found no association.[25,68] Of the remaining studies, one found a higher risk of instrumental delivery with increased weight gain only for normal BMI and overweight women,[58] and a second found this only for overweight women.[51] A third study, limited to women of normal weight, examined differences in the rate of vacuum extraction and forceps delivery by amount of weight gain; it found a higher rate of vacuum extraction with excessive weight gain but no difference in rate of forceps delivery.[69]

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Results controlling for confounding Studies varied in their adjustment for confounding factors. Seven studies controlled for route of previous delivery by limiting their sample to primary cesarean[51,71,72,75] or primigravidas.[69,73,74] Three studies included multigravidas but accounted for previous cesarean delivery in the analysis.[76–78] The remaining 11 studies did not control for route of previous delivery.[4,25,49,52,54,58,59,61,67,68,70]

Of the 10 studies that controlled for route of previous delivery, five studies examined underlying health risks (e.g., preeclampsia, pregnancy-induced hypertension) as predictors of cesarean delivery; all five found these health factors to be significantly associated with risks of cesarean delivery.[71,72,75,76,78]

Vaginal birth after cesarean

Study characteristics One U.S. cohort study (rated poor quality) examined the effect of weight gain on the success of vaginal birth after cesarean (VBAC) (Evidence Table 12).[79]

Overview of results A single poor study found that gestational weight gain of 40 pounds or more increased the risk of VBAC failure.

Results Women who gained more than 40 pounds during pregnancy were less likely to have VBAC success than women who gained 40 pounds or less (OR, 0.65; 95% CI, 0.42–0.98). This study controlled for previous normal spontaneous vaginal delivery, previous VBAC, diabetes, induction, birthweight > 4,000 g, recurrent indication, one layer closure, pregnancy complications, and BMI, but it failed to account for age or parity. The study suggested that pregravid BMI was also a predictor of VBAC success, with lower pregravid BMI being predictive of success.

Vaginal lacerations

Study characteristics Two cohort studies examined vaginal lacerations (Evidence Table 13).[51,68] One U.S. study (rated poor quality) examined the incidence of third- or fourth-degree lacerations among women.[51] Weight gain was characterized as change in BMI class between prepregnancy weight and weight at delivery. BMI categories were defined as follows: normal, BMI 20 to 24.9; overweight, BMI 25 to 29.9; obese I, BMI 30 to 34.9; obese II, BMI 35 to 39.9; morbid obesity, BMI ≥ 40. The second study (described earlier, also rated poor quality) was set in Finland.[68] It examined the rate of vaginal repairs for women with normal prepregnancy weight and weight gain during pregnancy and for those with abnormal weight gain (≥ 20 kg, or ≤ 5 kg) during pregnancy.[68]

Overview of results Two studies, both of poor quality, did not report consistent results on the effects of gestational weight gain on vaginal lacerations.

Results The U.S. study found no differences in the incidence of third- and fourth-degree lacerations among women who were overweight before pregnancy.[51] It did find a statistically significant difference among normal weight women; the incidence of lacerations rose from 24 percent for women with no change in BMI category to 29.3 percent for women gaining enough to change weight status by one BMI category and to 31.7 percent for women who gained enough to change weight status by more than one BMI category. The Finnish study found no statistical differences between study and control mothers in the rate of repair of second- or third-degree lacerations.[68] Neither study controlled for any variable other than pregravid BMI.

Shoulder dystocia

Study characteristics Three studies, set in Ireland,[80] the United States,[51] and Finland,[68] examined the effect of gestational weight gain on shoulder dystocia (Table 9, Evidence Table

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14). The Irish study, a case-control investigation (rated poor) comparing cesarean delivery for shoulder dystocia with cephalic vaginal term deliveries, distinguished between two groups of gestational weight gain (< 12 kg and ≥ 12 kg).[80] The Finnish study (described earlier and rated poor quality), stratified its sample by weight gain categories, comparing women with normal prepregnancy weight and weight gain during pregnancy with those with abnormal weight gain (≥ 20 kg or ≤ 5 kg) during pregnancy.[68] The U.S. case-control study (also rated poor quality), stratified its sample between normal and overweight BMI categories and examined the effect of change in BMI class between prepregnancy weight and weight at delivery. The Irish study defined shoulder dystocia to include mild, moderate, and severe cases;[80] the other two studies did not define their outcome variable.[51,68]

TABLE 9. Gestational weight gain and shoulder dystocia

Author, Date Country, Setting Sample Size Baseline BMI Quality	**Pregravid Weight (How Measured) Total Weight Gain (How Measured)**	**Definition of Groups**	**Results**	**Confounders and Effect Modifiers Included in Analysis**
Ekblad and Grenman, 1992[68] Finland, hospital 357 Normal weight only Poor	Pregravid weight: Data from records, unclear if self reported Total weight gain: Last clinically measured weight prior to delivery	G1: weight gain ≤ 5 kg G2: weight gain ≥ 20 kg G3: reference (normal prepregnancy weight and normal weight gain [undefined])	Shoulder dystocia% G1: 3 G2: 2 G3: 0.6	NA
Geary et al., 1995[80] Ireland, hospital 363 All weights/BMI Poor	Pregravid weight: First prenatal visit Total weight gain: Not described	Weight gain < 12 kg and ≥ 12 kg for cases shoulder dystocia and controls G1: Cases with shoulder dystocia G2: Controls	Maternal weight gain <12 kg G1: 59.1% G2: 74.1% OR 2.0 (1.6–2.2)	Parity Previous birth ≥ 4,000 g
Kabiru and Raynor, 2004[51] USA, hospital 5,131 All weights/BMI	Pregravid weight: First prenatal visit Total weight gain: Weight at admission for birth	G1: normal BMI, no change in BMI between first prenatal visit and delivery G2: normal BMI, 1 category increase in BMI between first prenatal visit and delivery G3: normal BMI, > 1 category increase in BMI between first prenatal visit and delivery	Shoulder dystocia% G1: 0.5 G2: 1.4 G3: 1.1 *P* = 0.278 for associations within normal BMI categories G4: 1.0	NA

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Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Poor		G4: overweight BMI, no change in BMI between first prenatal visit and delivery G5: overweight BMI, 1 category increase in BMI between first prenatal visit and delivery G6: overweight BMI, > 1 category increase in BMI between first prenatal visit and delivery	G5: 1.8 G6: 1.9 *P* = 0.357 for associations within overweight BMI categories	

BMI, body mass index; g, gram; G, group; kg, kilogram; N, number; OR, odds ratio.

Overview of results Only one[80] of three poor studies found a positive association between gestational weight gain and shoulder dystocia.

Results The three studies found rates of shoulder dystocia ranging from 0.6 percent to 1.4 percent.[51,68,80] Two studies reported no statistically significant differences in rates of shoulder dystocia between weight gain groups.[51,68] The Irish case-control investigation found that higher gestational weight gain during pregnancy was a significant predictor of shoulder dystocia (OR, 2.0; 95% CI, 1.6–2.2; P = 0.015). The authors calculated positive predictive value percentages from the study group and applied them to the total hospital population of singleton vaginal deliveries without shoulder dystocia over the same time period. These results suggest a positive predictive value of 1 percent for shoulder dystocia when gestational weight gain is 12 kg or greater.

The Irish study accounted for a subset of confounders and effect modifiers other than pregravid BMI.[80] Multiparity and birth of a previous heavy baby were significant and independent determinants for shoulder dystocia, in addition to gestational weight gain. However, the investigators noted that each predictor individually accounted for less than 2 percent of the positive predictive value for shoulder dystocia, and all three put together accounted for less than 3 percent.

Cephalopelvic disproportion

Study characteristics Two U.S. cohort studies examined the association between gestational weight gain and cephalopelvic disproportion (CPD) (Evidence Table 15).[25,74] One study, using Missouri birth certificate data, defined CPD as the condition when the size, presentation, or position of the fetal head to the maternal pelvis prevented cervical dilation or descent of the fetal head.[25] This study controlled for a range of demographic confounders but not for maternal health characteristics.[25] The other study (rated poor quality) defined CPD among primiparous women as little or no progress over a 2- to 4-hour period, with contractions documented to be adequate and cervix dilated to at least 3 cm or preferably 4 cm. However, if the delivering physician defined

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the indication as CPD, the decision was accepted without chart review, despite the definitions listed earlier.[74]

Both studies defined weight gain in categories: < 30 pounds, 30 to 35 pounds, and > 35 pounds. The study using birth certificate data limited inclusion to normal weight women (pregravid BMI 19.8–26.0);[25] the other study examined the association between gestational weight gain and CPD across four pregravid BMI categories: < 20, 20 to 25, 25 to 30, and > 30.

Overview of results Both studies (1 fair[25] and 1 poor[74]) showed that, for normal-weight women, the risk of CPD rose with higher gestational weight gain

Results The fair study reported an AOR of 1.58 (95% CI, 1.44–1.75) for women gaining > 35 pounds compared with women gaining 25 to 30 pounds, after adjusting for maternal age, maternal race or ethnicity, maternal education, Medicaid status, tobacco use, alcohol use, maternal height, prior pregnancy, adequacy of prenatal care, child's sex, and child's birth year.[25] The poor study showed similar results, with an unadjusted OR of CPD of 1.85 (95% CI, 1.63–2.06) for normal-weight women gaining > 35 pounds compared with women gaining < 30 pounds. This study also showed an increased risk of CPD for underweight women gaining > 35 pounds compared with women gaining < 30 pounds (unadjusted OR: 3.8; 95% CI, 3–4.6). The relationship between weight gain and CPD was not statistically significant at higher pregravid BMI levels.[74]

Complications of labor and delivery

Study characteristics Two retrospective cohort studies, one from Iceland[53] and the other from the United States,[81] evaluated the impact of gestational weight gain on complications of labor and delivery (Evidence Table 16).

Overview of results Two studies, of fair[53] and poor[81] quality respectively found conflicting evidence on the risks of complications. One failed to find statistically significant results;[53] the other reported that gestational weight gain of more than 40 pounds increased the risk for the previously listed complications by 40 percent.[81]

Results The fair study from Iceland analyzed the quartiles of total weight gain in women with normal pregravid BMIs (19.5–25.5) to determine the impact of weight gain on labor and delivery processes.[53] After adjusting for age, height, parity, gestational length, and birthweight, they found that weight gain of 11.5 to 16.0 kg was associated with the highest likelihood of a normal vaginal delivery, defined to include no shoulder dystocia and no asphyxia, and the least likelihood of operative procedures including cesarean delivery and forceps- or vacuum-assisted deliveries. The findings of this study, however, were not statistically significant.

The poor U.S. study enrolled 493 women at 37 or more weeks of gestational age to determine the relationship between various lifestyle choices and complications in term pregnancy.[81] Complications included dystocia, postpartum hemorrhage, retained placenta, fetal and neonatal distress, and pregnancy-induced hypertension. All complications were grouped together for the analysis. Smoking had a protective effect against complications, but entering pregnancy with excess weight for height and gaining more than 40 pounds during gestation both predicted complications. A gestational weight gain of more than 40 pounds increased the risk for the previously listed complications by 40 percent.

Birth Outcomes

Preterm birth

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Study characteristics Twelve studies (Table 10, Evidence Table 17) examined the relationship between weight gain and birth outcomes.[23,59,65,71,82–89] These include eight cohort studies,[59,65,82–86,89] two case-control studies,[87,88] and two cross-sectional studies.[23,71] The majority of the studies defined preterm birth as delivery occurring prior to 37 weeks of gestation; the one exception defined it as delivery between 24 and 35 weeks of gestation.[87] Each study defined weight gain differently. Two studies examined associations of weight gain with early and late preterm birth,[23,65] and two studies examined associations across subtypes of preterm delivery.[65,84]

TABLE 10. Gestational weight gain and preterm birth

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Kramer et al., 1995[88] Canada, University Hospitals 396 All weight/BMI Good	Pregravid weight: Self-report Total weight gain: Self-report	Gestational weight gain categories (kg/wk): G1: < 0.27 G2: ≥ 0.27	AOR (95% CI) for cases with preterm delivery versus controls G1: 1.56 (0.94–2.58) G2: 1.00 (reference)	Parity, marital status, language, age, education, matched on smoking history
Siega-Riz et al., 1996[84] USA, Public Health Clinics (California) 7,589 All weight/BMI Good	Pregravid weight: Self-reported Total weight gain: Measured	Categories of 3rd trimester weekly weight gain rates (kg/week): G1: Inadequate (Underweight, < 0.34; Normal weight, < 0.35; Overweight/Obese, < 0.30) G2: Adequate (Underweight, > 0.34; Normal, > 0.35; Overweight/Obese, > 0.30)	AOR (95% CI) for rate of preterm birth: G1: 1.91 (1.40–2.61) G2: 1.00 (reference) AOR (95% CI) for rate of preterm labor: G1:1.75 (1.15–2.64) G2: 1.00 (reference) AOR (95% CI) for rate of PPROM: G1: 2.70 (1.35–5.42) G2: 1.00 (reference)	Iron status, parity combined with maternal age, ethnicity, hypertension (chronic or pregnancy induced), smoking status, week prenatal care began
Carmichael et al.,1997[82] USA, University Hospital (California) 7,259 Nonobese	Pregravid weight: Self-report Total weight gain: Maternity Records	Total gestational weight gain (continuous)	Linear regression analysis of gestational age (days) as dependent variable and gestational weight gain (kg) as independent variable: Regression coefficient = 0.51; t-statistic = 13.1; $P < 0.001$ AOR (95% CI) of	BMI, maternal age, infant sex cigarettes per day maternal height, parity, race, pattern of gain derived from quadratic curves

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Fair			spontaneous preterm birth/kg increase in total weight gain: 0.84 (0.82–0.87)	
Dietz et al., 2006[23] USA, Pregnancy Risk Assessment Monitoring System 113,019 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Birth Certificates	Categories of mean rate of gestational weight gain (kg/wk) during second and third trimesters stratified by pregravid BMI and type of preterm birth (very preterm, 20–31 weeks; moderate preterm, 32–36 weeks): G1: < 0.12 G2: 0.12–0.22 G3: 0.23–0.68 G4: 0.69–0.79 G5: > 0.79	In general, in comparison to women with normal BMI in G3: underweight women in G1– G5 and normal weight women in G1, G2, and G5 were at increased risk of very preterm births (AOR: 1.5–9.8). Underweight women in G1–G3 and G5 and normal women in G1, G2, and G5 were at increased risk moderate preterm births (AOR: 1.4–3.1). Overweight and obese women in G1 and G5 were at increased risk of very preterm birth (AOR: 2.3–2.5) but had no elevated risk of moderate preterm birth. Very obese women with G1, G4, G5 had increased risks of very preterm births (AOR: 2.1–2.8) and with G4 had increased risks of moderate preterm birth (AOR: 1.3)	Race, Medicaid recipient, parity, marital status
Nohr et al., 2007[65] Danish National Birth Cohort 16,167 All weight/BMI Fair	Pregravid weight: Self-reported Total weight gain: Self-reported	Rate of gestational weight gain (g/wk) for women with early preterm birth (22–33 weeks) with PPROM : G1: < 275 G2: 276–675 G3: ≥ 676 Rate of gestational weight gain (g/wk) for women with early preterm birth (22–33 weeks) without PPROM : G4: < 275 G5: 276–675 G6: ≥ 676	HR (95% CI): G1: 2.1 (1.5–3.0) G2: 1.0 (ref) G3: 1.2 (0.8–1.8) HR (95% CI): G4: 1.9 (1.3–2.6) G5: 1.0 (ref) G6: 1.9 (1.3–2.6) HR (95% CI): G7: 1.3 (1.0–1.6) G8: 1.0 (ref) G9: 1.2 (1.0–1.5) HR (95% CI): G10: 1.0(0.9–1.2)	Pregravid BMI, age, height, parity, socio-occupational status, smoking alcohol consumption

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
		Rate of gestational weight gain (g/wk) for women with late preterm birth (34–36 weeks) with PPROM: G7: < 275 G8: 276–675 G9: ≥ 676 Rate of gestational weight gain (g/wk) for women with late preterm birth (34–36 weeks) without PPROM: G10: < 275 G11: 276–675 G12: ≥ 676	G11: 1.0(ref) G12: 1.0 (0.9–1.2)	
Rosenberg et al., 2005[71] USA, New York City birth files 329,988 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Self-report	Categories of total gestational weight gain (lbs): G1: <41 G2: ≥ 41	AOR (95% CI) for Preterm Birth: G1: 1.00 (reference) G2: 0.54 (0.52–0.57)	Pregravid weight, chronic diabetes, GDM, chronic hypertension, PIH preeclampsia, maternal age marital status maternal education maternal birthplace, prenatal care payer, social risk, parity, trimester that prenatal care began
Schieve, et al., 1999[86] USA, Pregnancy Nutrition Surveillance System (PNSS) 266,172 All weight/BMI Fair	Pregravid weight: Self-reported Total weight gain: Self-reported	Rate of weight gain (kg/week) in percentiles stratified by Low, Average, High, and Obese pregravid BMI: G1: 5th,0.10 G2: 10th, 0.16 G3: 25th,0.26 G4: 50th,0.35 G5: 75th, 0.46 G6: 90th, 0.57 G7: 95th, 0.65	Reference category of rate of weight gain: 0.35–<0.46 kg/wk RD of preterm birth varied by prepregnant BMI and gestational weight gain. Overall, women gaining 0.26–0.46 kg/wk had the lowest RD of preterm birth. The highest RD occurred for women gaining the least and most amount of weight, irrespective of prepregnant BMI; however, the highest RD of preterm births were	None

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
			among women of low BMI	
Stotland et al., 2006[85] USA, University Hospital (California) 15,101 Underweight/Normal BMI Fair	Pregravid weight: Medical Charts Total weight gain: Medical Charts	Categories of rate of gestational weight gain (kg/wk): G1: < 0.27 G2: 0.27 to 0.52 G3: > 0.52	AOR (95% CI) for preterm delivery < 37 weeks: G1: 2.6 (2.1–3.2) G2: 1.0 (reference) G3: 1.0 (0.8–1.2) AOR (95% CI) for preterm delivery < 34 weeks: G1: 3.0 (2.0–4.8) G2: 1.0 (ref)	Race, age pregravid BMI, year of delivery, parity, previous preterm birth, number of days between last weighing and delivery, smoking
Wen et al., 1990[89] USA, University Hospital (Alabama) 17,149 All weight/BMI Fair	Pregravid weight: Measured at first prenatal visit Total weight gain: Medical records	Rate of weight gain (kg/wk) after 20 weeks gestation G1: < 0.24 G2: 0.24–0.57 G3: 0.58–0.74 G4: ≥0.75	AOR for preterm birth: G1: 1.52 ($P < 0.05$) G2: 1.11 (NS) G3: 1.00 (ref) G4: 1.71 ($P < 0.05$)	Race, parity, infant sex, marital status, education, age, previous preterm delivery, smoking, alcohol consumption, drug use, height, pregravid weight
Jensen et al., 2005[59] Denmark, University hospital centers 481 Obese Poor	Pregravid weight: Self-report Total weight gain: Hospital records	Total gestational weight gain categories (kg): G1: < 5.0 G2: 5.0–9.9 G3: 10.0–14.9 G4: > 15.0	Percent (%) preterm delivery by weight gain categories: G1: 6.5 G2: 6.0 G3: 4.6 G4: 2.5 P for trend = 0.11	NA
Spinillo et al., 1998[87] Italy, University Hospital 690 All weight/BMI Poor	Pregravid weight: Self-report Total weight gain: Medical records	G1: Prepregnancy BMI ≤ 19.5 and 2nd/3rd trimester weight gain ≤ 0.37 kg/wk G2: Prepregnancy BMI>19.5 and 2nd/3rd trimester weight gain ≥ 0.37 kg/wk G3: Prepregnancy BMI ≤ 48 kg and 2nd/3rd trimester	AOR (95% CI) for cases with spontaneous preterm delivery versus controls: G1: 5.63 (2.35–13.8) G2: 2.45 (1.60–3.75) P = 0.06 for interaction between G1 and G2 G3: 5.29 (1.45–20.90) G4: 2.42 (1.65–3.55)	Pregravid BMI, pregravid weight, height, age, parity, smoking, social class education, infant sex

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
		weight gain ≤ 0.37 kg/wk G4: Prepregnancy BMI > 48 kg and 2nd/3rd trimester weight gain ≤ 0.37 kg/wk	*P* = 0.21 for interaction between G3 and G4	
Velonakis et al.,1997[83] France, Hospital 2040 All weight/BMI Poor	Pregravid weight: Self-reported Total weight gain: Measured	Total gestational weight gain (continuous)	Regression analysis with gestational age (weeks) as the dependent variable and net gestational weight gain as the independent variable: B = 0.191 (SE, 0.06) *P* = 0.001	Age, race, gravidity, previous diseases, parity, abortions, marital status, pathology of index pregnancy, infant sex, height pregravid weight, job classification, alcohol, smoking, APGAR score, duration of pregnancy

AOR, adjusted odds ratio; BMI, body mass index; CI, confidence interval; HR, hazards ratio; kg, kilogram; n, number; OR, odds ratio; PPROM, preterm premature rupture of amniotic membranes; RD, risk difference; USA, United States of America; wk, week.

Overview of results Taken collectively, the results of these two good,[84,88] seven fair,[23,65,71,82,85,86,89] and three poor[53,59,83,87] studies suggest an association between preterm birth and both low and high rates of weight gain and with low total weight gain, with one study reporting a 16 percent decrease in preterm birth associated with a 1 kg increase in maternal weight. The cut points for low and high weight gains and the severity of the risks of preterm birth associated with them differ by pregravid BMI. In general, low rates of weight gain were ≤ 0.37 kg per week and high rates of gain were > 0.52 kg per week throughout gestation, with the greatest risks found among underweight women. However, as pregravid BMI increases, the risk of preterm birth decreases for women gaining in the lower range of the low rate of weight gain and increases for women gaining in the lower range of the high rate of weight gain, such that the range of adequate rates of weight gain is shifted down for heavier women compared to their lighter counterparts. Some evidence also suggests that low rate of weight gain is associated with greater risks of early preterm birth as well as preterm birth due to premature rupture of the amniotic membranes.

Detailed results from categorical measures of total rate of weight gain. Four studies used categorical definitions of rate of weight gain averaged for the entire length of gestation;[85,86,88,89] one study was rated good[88] and the others rated fair.[85,86,89] In the good study,[88] a rate of weight gain of < 0.27 kg per week was not associated with preterm birth (OR, 1.56; 95% CI, 0.94–2.58). Among the fair studies, all three studies found evidence of an association between low rate of

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weight gain and preterm birth, and two studies found evidence of an association between high rate of weight gain and preterm birth.[86,89]

One study used a retrospective, US-hospital-based cohort of deliveries from 1976 to 2001 to examine the association of preterm birth and gestational weight gain by maternal race or ethnicity.[85] Weight gain was categorized into three groups based on rate of weight gain: < 0.27 kg per week, 0.27–0.52 kg per week, and > 0.52 kg per week. Within the entire cohort and across four racial or ethnic groups (white, black, Latina, and Asian), the highest percentages of preterm birth occurred among women gaining < 0.27 kg per week. The adjusted odds of spontaneous preterm birth were 2.5 times higher in women with rates of weight gain < 0.27 kg per week than in women gaining 0.27 to 0.52 kg per week. The adjusted odds ratios for this association were statistically significant across the different racial or ethnic groups, ranging from 2.1 (95% CI, 1.4–3.1) for white women to 3.6 (95% CI, 2.2–6.0) for black women. No association between spontaneous preterm birth and rate of weight gain > 0.52 kg per week (relative to a weight gain of 0.27 to 0.52 kg per week) was seen either within the entire cohort or across the racial or ethnic groups.

Another study, conducted in a population of young, primarily black, disadvantaged women, found statistically significant higher odds of preterm delivery among women gaining < 0.24 kg per week and > 0.74 kg per week than among women gaining 0.58 to 0.74 kg per week.[89]

The final study used data collected from women participating in U.S. federally funded prenatal public health programs via the Pregnancy Nutrition Surveillance System (PNSS).[86] Gestational weight gain was defined as rates of weight gain and net weight gain (kg/week) and categorized by the percentile distributions based on the total sample. Women with rates of weight gain between 0.35 and 0.46 kg per wk (the 50th through the 74th percentiles) were used as the reference for risk difference calculations. In general, the risk of preterm birth was highest among women with the smallest and greatest rates of weight gain, < 0.10 kg per week and ≥ 0.65 kg per week, respectively. The lowest risks of preterm delivery occurred among women gaining between 0.26 and 0.46 kg per week (the 25th through the 74th percentiles). Preterm risk differences did vary by maternal pregravid BMI status. An increased risk of preterm birth was associated with rates of weight gain for the following pregravid BMI categories:

- pregravid BMI < 19.8: < 0.26 kg per week;
- pregravid BMI of 19.8 to 26.0: < 0.26 kg per week and > 0.65 kg per week;
- pregravid BMI of 26.1 to 29.0: < 0.10 kg per week and > 0.65 kg per week; and
- pregravid BMI > 29.0: ≥ 0.57 kg per week.

The results were similar when rates of weight gain per week excluded the first 14 weeks of gestation.

Results from categorical measures of trimester rate of weight gain Four studies used categorical definitions of rate of gestational weight gain measured during specific trimesters of pregnancy.2[3,65,84,87] All of the studies found evidence for an association between preterm birth and low rate of weight gain and two studies found evidence for an association between preterm birth and high rate of weight gain.

One study of good quality used a cohort of mainly Hispanic women recruited from public health clinics to examine the association between preterm birth and rate of weight gain during the third trimester.[84] Women with preterm deliveries had significantly lower rates of third trimester weight gain than women with term deliveries, 0.50 (standard error of mean [SEM]:

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0.02) kg per week versus 0.53 (SEM: 0.004) kg per week, respectively (P < 0.05). The odds of preterm birth were 1.91 (95% CI, 1.40–2.61) times greater among women with inadequate third trimester weight gains (defined as a rate of weight gain less than the 25th percentile of gain in each pregravid weight status: 0.34 kg/week, underweight; 0.35 kg/week, normal weight; 0.30 kg/week, overweight and obese) than among women with adequate rates of weight gain. When data were stratified by the type of preterm delivery, women with inadequate weight gains were 1.75 (95% CI, 1.15–2.64) times more likely to have preterm delivery resulting from preterm labor and 2.70 (95% CI, 1.32–5.42) times more likely to have preterm delivery resulting from preterm premature rupture of the amniotic membranes (PPROM) than women with adequate rates of weight gain.

One study, rated fair quality, used data from the Danish National Birth Cohort to assess the impact of gestational weight gain on early (22–33 weeks), late (34–36 weeks), and all (22–36 weeks) preterm births with PPROM, without PPROM, and with medical inducement.[65] Gestational weight gain was categorized as low (< 275 g/week), medium (275–675 g/week), and high (> 675 g/week) based on two self-reported measurements recorded at least 6 weeks apart between 12 and 37 weeks of gestation. Women with medium rates of weight gain were used as the reference. Overall, low rates of weight gain were significantly associated with an increased risk of early spontaneous preterm birth with and without PPROM and with all spontaneous preterm births with PPROM, adjusted odds ratios ranged from 1.5 to 2.1. High rates of weight gain were significantly associated with an increased risk of early spontaneous preterm births without PPROM (AOR, 1.9; 95% CI, 1.3–2.6) and early, late, and all medically induced early preterm births. However, when women with obesity-related diseases and abruptio placenta were excluded, the associations for medically induced preterm births were no longer significant.

Another fair quality study used information collected for the Pregnancy Risk Assessment Monitoring System (PRAMS) to examine the effect of rate of weight gain during the second and third trimesters on preterm birth.[23] These investigators stratified women by prepregnancy BMI status and examined the risk of preterm birth in two categories: moderate length of gestation (32–36 weeks) and very short length of gestation (20 to 31 weeks). Second and third trimester rate of weight gain was categorized, in kg per week, as follows: < 0.12, 0.12–0.22, 0.23–0.68, 0.69–0.79, and > 0.79; the investigators also used five pregravid BMI groups: underweight (< 19.8), normal weight (19.8–26.0), overweight (26.1–28.9), obese (29.0–34.9), and very obese (≥ 35.0). Women of normal weight with rates of weight gain of 0.23 to 0.68 kg per week were used as the reference for analyses. After adjusting for covariates and excluding women with diabetes, hypertension, or small-for-gestational-age (SGA) infants, significant associations (AOR range, 1.3–3.1) were reported between moderate preterm birth and rates of weight gain as follows: < 0.69 and > 0.79 kg per week among underweight women; < 0.23 and > 0.79 kg per week among normal weight women; and 0.69 to 0.79 kg per week among obese and very obese women. Significant associations (AOR range, 1.5–9.8) were reported between very preterm birth and rates of weight gain as follows: all weight gain categories among underweight women; < 0.23 and > 0.79 kg per week among normal weight women; < 0.12 and > 0.79 kg per week among overweight and obese women; and < 0.12 and > 0.68 kg per week among very obese women. In general, the greatest odds were found among underweight women and in the extreme weight gain categories.

Results from a poor study[87] were consistent with those of the other studies and revealed an overall increased odds of preterm birth (between 24 and 35 weeks' gestation) with gestational

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weight gain ≤ 0.37 kg per week in the second and third trimesters; however, the odds were greater among women with pregravid BMI ≤ 19.5 compared to those with BMI >19.5.

Results from categorical measures of total weight gain Two studies,[59,71] one rated fair and the other poor, used categories of total weight gain. In the fair study, data from the New York City birth file from 1999 through 2001 was used to examine the odds of preterm birth associated with different levels of gestational weight gain.[71] After adjusting for covariates, the investigators determined that the odds of preterm birth were significantly decreased (OR, 0.54; 95% CI, 0.52–0.57) among women who gained at least 41 pounds compared with women who gained less than 41 pounds. Results from the poor study,[59] which used a population of obese women, showed the highest proportion of preterm birth among those with the lowest gestational weight gain (< 5.0 kg).

Results from continuous measures of weight gain The remaining two studies, one rated fair and the other poor, used gestational weight gain as a continuous measure.[82,83] Both studies reported a significant increase in length of gestation for a 1 kg increase in total gestational weight gain.

In the fair study,[82] simple regression techniques were used to develop a variable for pattern of weight gain that reflected the variation between a woman's pattern of weight gain and a linear pattern of weight gain.[82] Deviations in the pattern of weight gain, such as pronounced speeding up or slowing down of weight gain later in gestation, from an average pattern of weight gain were associated with decreased gestational age and increased risk of spontaneous preterm birth. A 1-kg increase in total gestational weight gain was associated with 0.51 day's increase in gestational age ($P < 0.001$). The odds of spontaneous preterm birth were decreased by 16 percent for each 1-kg increase in total gestational weight gain (OR, 0.84; 95% CI, 0.82–0.87; $P < 0.001$).

Birthweight

Study characteristics Twenty-five studies examined the association between gestational weight gain and infant birthweight (Evidence Table 18).[48,54,55,59,68,70,75,83,90–106] These studies consisted of various groups of women, in many different countries. Nine studies were completed outside the United States, in Canada,[105] France,[83,92] Italy,[91,100] Denmark,[59] Norway and Sweden,[99] Finland,[68] and Austria.[93]

One study observed the association for adolescent mothers.[95] The association was also evaluated for mothers with gestational diabetes mellitus (GDM),[100] mothers who had a positive diabetic screen but normal glucose tolerance levels,[91] and obese glucose-tolerant women.[59] Seventeen studies adjusted their analyses for multiple confounders, including maternal age, BMI, smoking, glucose levels, race, marital status, and parity.[48,55,59,70,75,90–93,97–103,105]

Overview of results The results for four good,[48,98,103,106 12] fair,[55,65,70,75,92,93,97,99–102,104,105] and nine poor[54,59,68,83,90,91,94–96] studies consistently demonstrate an association between higher gestational weight gain and birthweight.

Results from categorical measures of weight gain. Eight studies analyzed the relationship between weight gain and birthweight by categorizing gestational weight gain (Table 11).[54,59,68,94,95,99,101,106] One study was rated to be of good quality,[106] two of fair quality,[99,101] and five of poor quality.[54,59,68,94,95] These studies suggest a positive association between gestational weight gain and infant birthweight.

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TABLE 11. Total gestational weight gain (categorical) and infant birthweight

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Hickey et al., 1990[106] United States, prenatal clinics 325 All weight/BMI Good	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	Infant BW for groups defined by maternal weight near term (% of standard weight-for-height) G1: > 135%, Black G2: > 135%, Hispanic G3: 120–135%, Black G4: 120–135%, Hispanic G5: 110–119%, Black G6: 110–119%, Hispanic G7: 100–109%, Black G8: 100–109%, Hispanic G9: < 100%, Black G10: < 100%, Hispanic	G1: 3,325 g ± 460 G2: 3,543 g ± 410 G3: 3,200 g ± 389 G4: 3,381 g ± 385 G5: 3,157 g ± 373 G6: 3,282 g ± 400 G7: 3,025 g ± 494 G8: 3,154 g ± 375 G9: 2,813 g ± 289 G10: 3,205 g ± 472	N/A
Abrams et al., 1995[101] USA, university hospital 4,420 Nonobese Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	G1: Infant BW among nonobese women	3,485.8 g ± 523.1	Maternal age, parity, pregravid BMI, height, smoking, infant sex, difference in weeks between the last measured weight and delivery
Zaren et al., 1997[99] Norway and Sweden, university hospitals 1,099 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	β is estimated change in infant BW (g) G1: GWG ≤ 11 kg: G2: GWG ≥ 17 kg:	G1: β = -131 (*P* = 0.0001) G2: β = 164 (*P* = 0.0001)	Maternal age, height, pregravid weight, smoking
Bianco et al., 1998[54] USA, medical center 613	Pregravid weight: Self-report Total weight	Infant BW for GWG: G1: Weight loss or 0 lbs G2: 1–15 lbs G3: 16–25 lbs	G1: 3,302 G2: 3,192 G3: 3,337 G4: 3,506 G5: 3,453	N/A

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Morbidly obese (BMI > 35) Poor	gain: Routine prenatal care or maternity records	G4: 26–35 lbs G5: >35 lbs	($P < 0.05$)	
Cherry et al., 1993[95] USA, hospital RCT 599 All weights/BMI Poor	Pregravid weight: Measured by study investigators Total weight gain: Routine prenatal care or maternity records	Infant BW by Quartiles of weight gain Quartiles defined as weekly weight gain in g per cm height G1: Quartile 1 (≤ 1.87g) G2: Quartile 2 (1.88–2.68g) G3: Quartile 3 (2.69–3.58g) G4: Quartile 4 (≥ 3.59g)	G1: 2,829 g G2: 2,990 g G3: 3,112 g G4: 3,189 g	N/A
Ekblad and Grenman, 1992[68] Finland, hospital 357 Prepregnancy weight 20% over or under ideal body weight for height and normal weight Poor	Pregravid weight: Medical records Total weight gain: Routine prenatal care or maternity records	Infant BW by group G1: Normal prepregnancy weight and normal weight gain G2: Weight gain ≤5 kg G3: Weight gain ≥20 kg	G1: 3,538 g ± 535 G2: 3,284 g ± 880 G3: 3,803 g ± 538 ($P < 0.005$ compared to G1)	N/A
Jensen et al., 2005[59] Denmark, university hospitals 481 Obese Poor	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	Infant BW for groups defined by GWG G1: GWG < 5.0 kg G2: GWG 5.0–9.9 kg G3: GWG 10–14.9 kg G4: GWG ≥ 15.0 kg	G1: 3,456 g ± 620 G2: 3,624 g ±675 G3: 3,757 g ± 582 G4: 3,784 g ± 597 $P < 0.0001$	N/A
Shapiro et al., 2000[94] USA, community hospital	Pregravid weight: Routine prenatal care	Infant BW for groups defined by BMI and weight gain	G1: 3,363 g G2: 3,636 g G3: 3,565 g G4: 3,774 g	N/A

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
159 All weight/BMI Poor	Total weight gain: Routine prenatal care or maternity records	G1: Low BMI (< 25), Low gain (< 35lbs) G2: Low BMI (< 25), High gain (> 35lbs) G3: High BMI (> 25), Low gain (< 35lbs) G4: High BMI (> 25), High gain (> 35lbs)		

β unstandardized coefficient from multiple regression; BMI, body mass index; BW, birthweight; cm, centimeters; g, grams; GWG,gestational weight gain; kg, kilogram; lbs, pounds; SC, standardized coefficient; N/A, not applicable; NR, not reported; RCT, randomized controlled trial; SD, standard deviation

A U.S. study rated of good quality found that higher values for maternal weight near term, categorized by the percentage of standard weight-for-height, were associated with higher birthweight for black and Hispanic mothers.[106] Specifically, black mothers > 135 percent of standard weight for height gave birth to infants that weighed on average 512 g more than infants born to black mothers < 100 percent of standard weight for height. Hispanic mothers > 135 percent of standard weight for height gave birth to infants that weighed on average 338 g more than infants born to Hispanic mothers < 100 percent of standard weight for height.

In one Scandinavian study (fair quality), estimated birthweights decreased by 131 g for women who gained less than 11 kg and increased by 164 g for women who gained more than 17 kg, as compared with estimated birthweights for women gaining between 11 and 17 kg.[99] A fair-quality U.S. study examined patterns of weight gain and infant birthweight in a population of white nonobese women.[101] Low weight gain by trimester was defined as having weight gain less than the 25th percentile. Infants of mothers with low weight gain in all three trimesters had weighed 248.1 g less, on average, than infants of mothers in other groups. Low weight gain for the first trimester was associated with a decrease in birthweight of 133 g; low weight gain for the second and third trimesters was associated with an 88.5 g decrease in birthweight.

The five poor-quality studies also found that increases in gestational weight gain resulted in larger infant birthweights.[54,59,68,94,95] This trend held among studies of obese glucose-tolerant women,[59] Finnish women,[68] and adolescent mothers.[94,95] One study stratified by maternal BMI and found that among women with low BMI (< 25) those that gained > 35 lbs had infants that were, on average, 273 g heavier than infants born to women gaining < 35 lbs. Among women with high BMI (> 25), women that gained > 35 lbs had infants that were, on average, 209 g heavier than infants born to women who gained < 35 lbs. One study among morbidly obese women (BMI > 35) found a similar trend, although it was inconsistent at the extremes of weight gain. Specifically, the following infant birthweights were found for each of the gestational weight gain categories: weight loss or 0 lbs, 3,302 g; 1–15 lbs, 3,192 g; 16–25 lbs, 3,337 g; 26–35 lbs, 3,506 g; >35 lbs, 3,453 g.

Results for continuous total weight gain Fourteen studies (Table 12) evaluated the relationship between continuous total weight gain and birthweight using linear regression

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techniques to determine the effect of every 1 kg increase in weight gain.[48,55,59,90,92,93,96,98,100–105] Of these studies, three[48,98,103] were rated of good quality, eight[55,92,93,100–102,104,105] of fair quality, and three[59,90,96] of poor quality. Seven studies of good and fair quality reported that birthweight increased between 16.7 and 22.6 g for every 1 kg increase in weight gain.[48,93,98,101–103,105] Three poor-quality studies reported that birthweight increased between 18.4 and 44.3 g for every 1 kg increase in weight gain.[59,90,96]

TABLE 12. Total gestational weight gain (continuous) and infant birthweight

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Brown et al., 2002[98] USA, primary care clinics 389 All weight/BMI Good	Pregravid weight: Measured by study investigators Total weight gain: Collected by study investigators	G1: Increase in birthweight per 1 kg increase in total pregnancy weight gain	G1: β = 20 g (P < 0.0001)	Maternal age, parity, pregravid BMI, height, infant sex, gestational age
Groff et al., 1997[103] USA, multispecialty clinics 341 All weights/BMI Good	Pregravid weight: Self-report 82% First prenatal visit 18% Total weight gain: Routine prenatal care or maternity records	G1: Increase in birthweight per 1 lb increase in total pregnancy weight gain	G1: β = 10.1g ± 1.76 ($P \leq$ 0.001)	Pregravid BMI, infant sex, smoking
Kieffer et al., 2006[48] USA, community health center 1,041 All weights/BMI Good	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	G1: Increase in birthweight per 1 kg increase in total pregnancy weight gain	G1: β = 19.7 g ± 2.8 (P < 0.01)	Parity, pregravid BMI, height, 1-hour glucose value, gestational age
Abrams et al., 1995[101] USA, university hospital 4,420 Nonobese	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	G1: Increase in birthweight per 1 kg increase in total pregnancy weight gain	G1: β = 22.6 g (P < 0.001)	Maternal age, parity, pregravid BMI, height, smoking, infant sex, gestational age

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Fair				
Butte et al., 2003[97] USA, US Agriculture Research Service Children's Nutrition Research Center 63 All weights/BMI Fair	Pregravid weight: Measured by study investigators Total weight gain: Measured by study investigators	G1: Correlation coefficient G2: Variability in birthweight accounted for by gestational age, pregravid weight, and total pregnancy weight gain	G1: 0.28 G2: 37.9%	Maternal race, pregravid BMI, gestational age
Edwards et al., 1996[55] USA, hospital 1,443 Normal and obese BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	G1: Increase in birthweight per 1 kg increase in total pregnancy weight gain for obese women G2: Increase in birthweight per 1 kg increase in total pregnancy weight gain for normal weight women	G1: β = 11 g ± 2 ($P \leq 0.001$) G2: β = 15 g ± 2 ($P \leq 0.001$)	Maternal age, parity, pregravid BMI, pregnancy-induced hypertension, adequacy of prenatal care, alcohol use, drug use, smoking, gestational age
Guihard-Costa et al., 2004[92] France, hospital database 13,972 All weights/BMI Fair	Pregravid weight: Routine prenatal care Total weight gain: Routine prenatal care or maternity records	G1: Standardized coefficient for effect of pregnancy weight gain on infant birthweight. Standardized coefficients are regression coefficients calculated as if all of the independent variables had a variance of 1	G1: SC = 0.199	Maternal age, parity, pregravid BMI, height
Hediger et al., 1994[102] USA, setting not stated 608 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	G1: Increase in birthweight per 1 kg increase in total pregnancy weight gain	G1: β = 16.7 g ± 2.5 ($P = 0.001$)	Maternal age, maternal race/ethnicity, parity, pregravid weight, height, gestational age, prior poor outcome, fat loss, pregravid weight: low weight, fat accretion, smoking, infant sex

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Kirchengast and Hartmann, 2003[93] Austria, university hospital 8,011 All weights/BMI Fair	Pregravid weight: Estimated from measured weight at first prenatal visit Total weight gain: Routine prenatal care or maternity records	G1: Increase in birthweight per 1 kg increase in total pregnancy weight gain	G1: β = 17.32 (14.62, 20.03)	Maternal age, age at menarche, pregravid weight, height, distantia cristarum
Luke et al., 1996[104] USA, clinic 487 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	Increase in birthweight per 1 kg increase in total pregnancy weight gain for BMI categories: G1: Underweight G2: Normal weight G3: Overweight	G1: β = 44.9 g ±6.8 (*P* < 0.01) G2: β = 22.9 g ± 3.9 (*P* < 0.01) G3: β = 11.9 g ± 5.2 (*P* < 0.05)	Maternal age, parity, black ethnicity, smoking, gestational age, infant sex
Muscati et al., 1996[105] Canada, public health department 371 All weight/BMI Fair	Pregravid weight: Medical records Total weight gain: Collected by study investigators	G1: Increase in birthweight per 1 kg increase in total weight gain up to week 20	G1: β = 22 g ± 6 (*P* < 0.01)	Parity, pregravid standard weight, pregravid excess weight, birth length, infant sex
Pezzarossa et al., 1996[100] Italy, not stated 192 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	Increase in birthweight per 1 kg increase in total pregnancy weight gain for: G1: Controls (normal glucose tolerance) G2: GDM	G1: β = 27.8 g (*P* = 0.0001) G2: β = 39.5 (*P* = 0.0001)	Pregravid BMI, fasting plasma glucose
Di Cianni et al., 2004[91]	Pregravid weight: Not reported		F statistic = 3.16, *P*	Pregravid BMI, maternal

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
			= 0.08	triglycerides, plasma glucose
Italy, diabetes clinic 180 All weights/BMI Poor	Total weight gain: Collected by study investigators			
Jensen et al., 2005[59] Denmark, university hospitals 481 Obese Poor	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	G1: Increase in birthweight per 1 kg increase in total pregnancy weight gain	G1: β = 18.4 g (P < 0.001)	Maternal age, pregravid BMI, smoking, gestational age, result of 2-hour oral glucose tolerance test
Paauw et al., 2005[90] USA, hospital 351 All weights/BMI Poor	Pregravid weight: Self-report Total weight gain: Self-report	G1: Increase in birthweight per 1 kg increase in total pregnancy weight gain	G1: β = 21.0 g	Maternal race, pregravid weight, marital status, smoking, gestational age
Springer et al., 1992[96] USA, university hospital 107 All weights/BMI Poor	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	G1: Increase in birthweight per 1 lb increase in total pregnancy weight gain	G1: β = 20.1g	Maternal age, pregravid weight, length of gestation, smoking, weight gain at 20 weeks

AGA, Appropriate for gestational age; β, unstandardized coefficient from multiple regression; BMI, body mass index; g, gram; GDM, gestational diabetes mellitus; kg, kilogram; lb, pound; LGA, large-for-gestational-age; NR, not reported

Two studies of fair quality reported these values by BMI status.[55,104] One found that 1 kg increases in weight gain among normal-weight women were associated with a 15 g increase in

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infant birthweight and, among obese women, an 11 g increase in infant birthweight.[55] The other study reported, for each 1 kg increase in gestational weight gain, a 44.9 g increase in birthweight for underweight women, a 22.9 g increase for women of normal weight, and an 11.9 g increase for overweight women.[104]

In the one fairquality study that stratified by GDM, the association of total weight gain and infant birthweight was stronger among mothers with GDM than among women not diagnosed with GDM.[100] Specifically, 1 kg increases in weight gain raised infant birthweight by 27.8 g among nondiabetic mothers and by 39.5 g among mothers with GDM.

Several studies reported statistically significant correlations between gestational weight gain and infant birthweight. Correlation coefficients between birthweight and total weight gain ranged from 0.22 to 0.28 in two fair-quality studies.[97,105] A poor-quality study among obese, glucose-tolerant women reported a nonsignificant correlation value of r2 = 0.062.[91]

Results for continuous total weight gain by trimester Three studies reported on the effects of gestational weight gain, by trimester, on infant birthweights (Table 13).[98,101,105] One U.S. study (rated good quality) reported that weight gain during the first trimester was associated with a 31 g increase in birthweight per kg of gestational weight gain. Comparable gains in infant birthweight for each kg of gestational weight gain in the second and third trimesters were 26 g and 7 g.[98] This study also found that infant birthweight decreased by 211 g among mothers who lost weight during the first trimester.[98]

TABLE 13. Continuous gestational weight gain by trimester and infant birthweight

Author, Year Country, Setting Sample Size Baseline BMI Quality	**Pregravid Weight (How Measured) Total Weight Gain (How Measured)**	**Definition of Groups**	**Results**	**Confounders and Effect Modifiers Included in Analysis**
Brown et al., 2002[98] USA, primary care clinics 389 All weight/BMI Good	Pregravid weight: Measured by study investigators Total weight gain: Collected by study investigators	G1: Increase in birthweight per 1 kg increase in first trimester weight gain G2: Increase in birthweight per 1 kg increase in second trimester weight gain G3: Increase in birthweight per 1 kg increase in third trimester weight gain	G1: β = 31 g (*P* < 0.0007) G2: β = 26 g (*P* < 0.007) G3: β = 7 g (*P* < 0.40)	Maternal age, parity, pregravid BMI, height, infant sex, gestational age
Abrams et al., 1995[101] USA, university hospital 4,420 Nonobese	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	G1: Increase in birthweight per 1 kg increase in first trimester weight gain G2: Increase in birthweight per 1 kg increase in second trimester weight gain G3: Increase in birthweight per 1 kg increase in third trimester weight gain	G1: β = 18.0 g ± 2.4 (*P* < 0.001) G2: β = 32.8 g ± 2.8 (*P* <	Maternal age, parity, pregravid BMI, height, smoking, infant sex, gestational age

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Fair			0.001) G3: β = 17.0 g ± 2.9 (*P* < 0.001)	
Muscati et al., 1996[105] Canada, public health department 371 All weight/BMI Fair	Pregravid weight: Medical records Total weight gain: Collected by study investigators	G1: Increase in birthweight per 1 kg increase in total weight gain from weeks 21 to 30 G2: Increase in birthweight per kg increase in total weight gain from weeks 31 to term	G1: β = 31 g ± 7 (*P* < 0.001) G2: β = 12 g ± 6 (*P* < 0.05)	Parity, pregravid standard weight, pregravid excess weight, birth length, infant sex

β, unstandardized coefficient from multiple regression; BMI, body mass index; g, gram; kg, kilogram; lbs, pounds.

A Canadian study of fair quality found similar results: for each 1 kg increase in weight gain up to week 20, birthweight increased by 22 g; increases from week 21 to 30 increased birthweight by 31 g; and weight gain from week 31 to term increased birthweight by 12 g.[105] Lastly, another U.S. study of fair quality reported an 18 g increase in birthweight for each kilogram gained by the mother in the first trimester. Corresponding increases in the second and third trimesters were 32.8 g and 17.0 g, respectively.[101]

Results from other measures of weight gain (net weight gain and proportional weight gain) Four studies examined the associations between infant birthweight and various other measures of gestational weight gain. Three studies (1 rated poor quality) of net weight gain (total gestational weight gain minus infant birthweight) showed that infant birthweight increased as net gestational weight gain increased (Table 14).[70,83,104] In one study, for every 1 kg increase in net weight gain, birthweight rose by 15.4 g.[70] In another, which examined differences by BMI status, increases of 1 kg in net weight gain raised infant birthweight as follows: for underweight women, 41.9 g; for women of normal weight, 19.2 g; and for obese women, 9.1 g.[104] Each kilogram of net weight gain associated with an increase of 111.2 g in birthweight in another study.[83]

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TABLE 14. Net and proportional gestational weight gain and infant birthweight

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Johnson et al., 1992[70] USA, prenatal clinics 3,191 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	G1: Increase in birthweight per 1 lb increase in net pregnancy weight gain	G1: β = 15.4 g ± 2.2 (*P* < 0.0001)	Maternal race, parity, pregravid BMI, height, pregravid weight, marital status, education, tobacco/alcohol/drug use, pregnancy-induced hypertension, gestational age, macrosomia, infant sex
Luke et al., 1996[104] USA, clinic 487 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	Increase in birthweight per 1 kg increase in net pregnancy weight gain for BMI categories: G1: Underweight G2: Normal weight G3: Overweight	G1: β = 41.9 g ± 7.5 (*P* < 0.01) G2: β = 19.2 g ± 3.9 (*P* < 0.01) G3: β = 9.1 g ± 5.3	Maternal age, parity, black ethnicity, smoking, gestational age, infant sex
Shepard 1998[75] USA, obstetrical practices in New Haven, CT 2,301 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	Infant birthweight for mothers with: G1: Low average BMI (19.5 to 22.4), proportional weight gained > median G2: Low average BMI (19.5 to 22.4), gained < median G3: High average BMI (22.5 to 28.5), gained > median G4: High average BMI (22.5 to 28.5), gained < median G5: Obese (> 28.5 BMI), gained > median G6: Obese (> 28.5 BMI), gained < median	G1: 3,231 g G2: 3,553 g G3: 3,395 g G4: 3,620 g G5: 3,685 g G6: 3,453 g	N/A
Velonakis et al., 1997[83]	Pregravid weight:	G1: Increase in birthweight for net	G1: β = 111.17 g	Maternal age, parity, pathology of previous/current pregnancy, previous diseases,

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
France, hospital 2,040 All weights/BMI Poor	Self-report Total weight gain: Routine prenatal care or maternity records	pregnancy weight gain	± 12.94 (*P* = 0.000)	reproductive history, marital status, employment, infant sex, height, weight, smoking, alcohol use, APGAR score, gestational age, nationality

β, unstandardized coefficient from multiple regression; BMI, body mass index; C-section, cesarean section; g, gram; kg, kilogram; lbs, pounds; N/A, not applicable.

The fourth study, which considered proportional gestational weight gain (total gestational weight gain divided by pregravid weight) found that for mothers with BMIs of 19.5 to 22.4, those who gained above the median proportional gestational weight gain had infants who were 322 g heavier than the infants of mothers who gained below the median.

Similar results were found for mothers with BMIs of 22.4 to 28.5: those who gained above the median gave birth to infants who were 225 g heavier. Finally, for women with BMIs above 28.5, the increase in birthweight was 232 g.[75]

Low birthweight

Study characteristics Thirteen studies examined the effect of gestational weight gain on low birthweight (LBW) (Evidence Table 19).[2,4,52,54,70,71,75,93,95,106–109] LBW is defined as infant birthweight < 2,500 g. Overall, the risk of LBW decreased as gestational weight gain increased. In general, risks for LBW began to decrease for gestational weight gains above 25 to 30 pounds. In 11 of these studies, the analyses were adjusted for multiple confounders, including maternal age, pregravid BMI, smoking, alcohol use, gestational age, parity, race, marital status, maternal education, pregnancy complications, and infant sex.[2,4,52,70,71,75,93,106–109]

Overview of results Ten studies considered the relationship between LBW and total gestational weight gain (Table 15).[2,4,52,54,71,93,106–109] One of these studies was rated good quality,106 seven of fair quality,[2,4,52,71,93,107,108] and two of poor quality.[54,109] In general, as gestational weight gain increased, LBW decreased.

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TABLE 15. Total gestational weight gain and low birthweight (LBW)

Author, Year Country, Setting Sample Size Baseline BMI Quality	**Pregravid Weight (How Measured) Total Weight Gain (How Measured)**	**Definition of Groups**	**Results**	**Confounders and Effect Modifiers Included in Analysis**
Hickey et al., 1990[106] United States, prenatal clinics 325 All weights/BMI Good	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	G1: Percent BW < 3,000, Low weight gain < 120% of standard G2: Percent BW ≥ 3,000, Low weight gain < 120% of standard G3: Percent BW < 3,000, Acceptable weight gain ≥ 120% of standard G4: Percent BW ≥ 3,000, Acceptable weight gain ≥ 120% of standard	G1: 38.2 G2: 61.8 G3: 22.1 G4: 77.9	N/A
Cogswell et al., 1994[2] USA, Pregnancy Nutrition Surveillance System 53,541 Normal/Overweight/Obese Fair	Pregravid weight: Self-report Total weight gain: Self-report	ORs and 95% CI, for LBW by GWG and prepregnancy BMI G1: Normal BMI, GWG < 15 lbs G2: Normal BMI, GWG ≥ 40 lbs G3: Normal BMI, GWG 25–29 lbs (Reference for normal BMI) G4: Overweight BMI, GWG 30–34 lbs G5: Overweight BMI, GWG 35–39 G6: Overweight BMI, GWG ≥ 40 lbs G7: Overweight BMI, GWG 15–19 lbs (Reference for overweight BMI)	G1: 2.1 (1.6–2.6) G2: 0.5 (0.4–0.6) G3: 1.0 G4: 0.5 (0.3–0.8) G5: 0.6 (0.3–1.1) G6: 0.4 (0.3–0.7) G7: 1.0	Maternal age, maternal race, height, smoking, infant sex, gestational age
Desjardins and Hardwick, 1999[107]	Pregravid weight: Self-report	G1: OR and 95% CI, for LBW and inadequate weight	G1: 1.15 (0.78–1.67)	Gestational age, adolescence, pregravid underweight, number of Healthiest Baby Possible visits

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Canada, Healthiest Babies Possible Program 1,892 All weights/BMI Fair	Total weight gain: Home visitor's scale	gain (defined by dietician)		
Kiel et al., 2007[4] USA, birth certificate registry 120,251 Obese Fair	Pregravid weight: Medical records Total weight gain: Routine prenatal care or maternity records	G1: Odds of LBW for weight gain > 25 lbs G2: OR of LBW for weight gain < 15 lbs G3: Reference Weight gain 15–25 lbs	G1: Odds of LBW are lower for women in this group G2: Odds of LBW are higher for women in this group Numerical value for ORs not reported in study	Maternal age, maternal race, maternal education, poverty, smoking, parity, chronic hypertension
Kirchengast and Hartmann 2003[93] Austria, university hospital 8,011 All weights/BMI Fair	Pregravid weight: Estimated from measured weight at first prenatal visit Total weight gain: Routine prenatal care or maternity records	G1: OR and 95% CI, for LBW	G1: 0.90 (0.85–0.95)	Maternal age, pregravid weight, height, distantia cristarum
Murakami et al., 2004[52] Japan, hospital 633 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	OR and 95% CI, for LBW G1: GWG < 8.5 kg G2: GWG 8.5–12.5 kg G3: GWG > 12.5 kg	G1: 1.26 (0.57–2.75) G2: Reference G3: 0.62 (0.24–1.62)	Maternal age, parity, pregravid BMI, smoking, gestational age

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Rosenberg et al., 2005[71] USA, vital statistics data 329,988 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	OR and 95% CI, for LBW G1: GWG ≥ 41 lbs G2: GWG < 41 lbs	G1: 0.41 (0.39–0.43) G2: Reference	Maternal age, parity, GDM, pregnancy-induced hypertension, preeclampsia, pregravid weight, chronic diabetes, chronic hypertension, marital status, maternal education, mother's birthplace, prenatal care payer, social risk, trimester prenatal care began
Zhou and Olsen, 1997[108] Denmark, two communities 7,122 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	OR and 95% CI, for LBW for GWG categories by BMI G1: GWG < 11 kg, Underweight (Reference) G2: GWG < 11 kg, Normal weight G3: GWG < 11 kg, Overweight G4: GWG 12–15 kg, Underweight G5: GWG 12–15 kg, Normal weight G6: GWG 12–15 kg, Overweight G7: GWG ≥16 kg, Underweight G8: GWG ≥16 kg, Normal weight G9: GWG ≥16 kg, Overweight	G1: 1.0 G2: 0.9 (0.5–1.5) G3: 0.8 (0.3–2.0) G4: 0.5 (0.2–1.0) G5: 0.8 (0.4–1.5) G6: 0.9 (0.2–3.8) G7: 0.3 (0.1–1.0) G8: 0.4 (0.2–0.8) G9: 0.0 (0.0–2,500)	Maternal age, parity, alcohol, no diabetes, term delivery, smoking, infant sex, gestational age
Bianco et al., 1998[54] USA, medical center 613 Morbidly obese (BMI > 35) Poor	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	% LBW for GWG: G1: Weight loss or 0 lbs G2: 1–15 lbs G3: 16–25 lbs G4: 26–35 lbs G5: > 35 lbs	G1: 2 G2: 11.1 G3: 8.3 G4: 5.2 G5: 3.8	N/A
Lasker et al., 2005[109] USA, hospital	Pregravid weight: Not stated	OR and 95% CI, for LBW	G1: 2.43 (1.45–4.05) G2: 0.63	Maternal age, maternal race, marital status, prenatal care, prior term births, prior abortions, prior preterm

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
5,528 All weights/BMI Poor	Total weight gain: Routine prenatal care or maternity records	G1: GWG < 10 lbs G2: GWG > 30 lbs G3: GWG 21–30 lbs (Reference)	(0.47–0.85) G3: 1.00	births, BMI at delivery, preeclampsia, bleeding, smoking, multiple births, premature birth, congenital anomaly, incompetent cervix, smoking

BMI, body mass index; BW, birthweight; CI, confidence interval; GDM, gestational diabetes mellitus; GWG, gestational weight gain; kg, kilogram; lbs, pounds; LBW, low birthweight; N/A, not applicable; OR, odds ratio.

Three studies evaluated measures of gestational weight gain other than total gestational weight gain (Table 16).[70,75,95] Two[70,75] studies were of fair quality and one[95] was of poor quality. These studies suggest reduced risk of LBW in association with increases in net, proportional, or other measures of change in weight gain.

Results for total gestational weight gain and LBW Results taken from a figure from a good-quality study of low-income black and Hispanic women showed the trend of decreasing LBW as maternal weight near term compared to the standard weight-for-height increased.[106] A population-based cohort study in New York City reported a protective effect for LBW (OR, 0.41; 95% CI, 0.39–0.43) for women who gained more than 41 pounds compared with women who gained less than 41 pounds.[71] A study in Denmark found that the risk of LBW was significantly reduced only for underweight women gaining at least 12 kg when compared to underweight women gaining less than 11 kg (OR, 0.5; 95% CI, 0.2–1.0).108 A study in Austria[93] found that the odds ratio of LBW was 0.9 (95% CI, 0.85–0.95) for each 1 kg increase in gestational weight gain. A study among obese women also found that the risk of having a LBW infant was increased for low gestational weight gains.[4]

TABLE 16. Other gestational weight gain measures and LBW

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Johnson et al., 1992[70] USA, prenatal clinics 3,191 All weights/BMI	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity	OR and 95% CI, for LBW G1: Net WG < 14.9 lbs (Reference) G2: Net WG 14.9–23.5 lbs G3: Net WG 24–33 lbs	G1: 1.0 (Reference) G2: 0.51 (0.27–0.98) G3: 0.54 (0.28–1.04) G4: 0.38 (0.2–0.8)	Maternal race, parity, pregravid BMI, height, pregravid weight, marital status, education, tobacco/alcohol/drug use, pregnancy-induced hypertension, gestational age, macrosomia, infant sex

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Fair	records	G4: Net WG > 33 lbs		
Shepard 1998[75] USA, obstetrical practices in New Haven, CT 2,301 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	%LBW G1: Proportional WG < median, underweight (BMI < 19.4) G2: Proportional WG < median, obese (BMI > 28.5) G3: Proportional WG > median, underweight (BMI < 19.4) G4: Proportional WG > median, Low-average BMI (19.5–22.4) G5: Proportional WG > median, High-average BMI (22.5–28.5) G6: Proportional WG > median, obese (BMI > 28.5)	G1: 3.5% G2: 7.4% G3: 2.1% G4: 2.8% G5: 2.1% G6: 4.6%	N/A
Cherry et al., 1993[95] USA, hospital 599 All weights/BMI Poor	Pregravid weight: Measured by study investigators Total weight gain: Routine prenatal care or maternity records	%LBW for each shifting of EW category. Light: < 90% EW Normal: 90 to 110% of EW Heavy: > 110% EW G1: Heavy to normal G2: Normal to light G3: Normal to heavy G4: Light to normal	G1: 5% G2: 32% G3: 3.1% G4: 2.7%	N/A

BMI, body mass index, CI, confidence interval; EW, expected weight; lb, pound; LBW, low birthweight; N/A, not applicable; OR, odds ratio; WG, weight gain

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Among low-income women the effect of weight gain varied by pregravid BMI;[2] only among women of average weight was there a consistent decrease in LBW risk as gestational weight gain increased from < 15 pounds to ≥ 40 pounds. Mothers of average weight who gained less than 15 pounds had an OR for delivering an LBW infant of 2.1 (95% CI, 1.6–2.6). The odds of LBW were substantially lower for women who gained more than 40 pounds (OR, 0.5; 95% CI, 0.4–0.6). There was no reduction in the percentage of LBW infants for weight gains above 30 to 34 pounds for overweight women, and for weight gains above 15 to 19 pounds for obese women. For overweight women gaining 30 to 34 pounds, the OR was 0.5 (95% CI, 0.3–0.8). The poor-quality studies showed results in the same general direction.[54,109]

Two fair-quality studies did not find a statistically significant association between total gestational weight gain and LBW, although the point estimates were in the expected direction.[52,107] Among a cohort of Japanese women,[52] for weight gain < 8.5 kg, the adjusted OR of LBW was 1.26 (95% CI, 0.57–2.75) and for weight gain > 12.5 kg, it was 0.62 (95% CI, 0.24–1.62), when these groups were compared with women gaining between 8.5 and 12.5 kg. Another study found that inadequate weight gain was associated with an OR for LBW of 1.15 (95% CI, 0.78–1.67);[107] in this study, a dietitian determined inadequate weight gain status (exact criteria were not reported).

Results for net, proportional, or other measures of change in weight gain and LBW One study looked at the relationship between net weight gain (total gestational weight gain minus infant birthweight) and the risk of LBW; the risk decreased as net weight gain increased.[70] Odds ratios reported are in comparison with women gaining < 14.9 pounds. For mothers gaining > 33 pounds, the OR was 0.38 (95% CI, 0.2–0.8); for women gaining 24 to 33 pounds, the OR was 0.54 (95% CI, 0.28–1.04); and for women gaining 14.9 to 23.5 pounds, the OR was 0.51 (95% CI, 0.27–0.98). The association between risk of LBW infants and proportional weight gain (total gestational weight gain divided by pregravid weight) above and below the median was also evaluated in relation to BMI status.[75] Obese women had a higher percentage of LBW infants than underweight women. The risk of LBW was even higher for women gaining less than the median.

A study of adolescent mothers (rated poor quality) showed similar effects. Mothers who shifted to lower weight classes during pregnancy were more likely to have LBW babies, and mothers who progressed to higher weight classes had lower percentages of LBW.[95]

Macrosomia

Study characteristics Twelve studies examined the influence of gestational weight gain on macrosomia in their infants (Evidence Table 20).[2,4,49,59,70,77,93,108,110–113] Studies did not define macrosomia consistently. Four studies defined macrosomia as birthweight > 4,500 g.[2,108,110,113] Seven of the remaining eight studies defined macrosomia as birthweight > 4,000 g.[4,59,70,77,93,111,112] One study applied both definitions.[49] One[110] study was rated to be of good quality, nine[2,4,70,77,93,108,111–113] of fair quality, and two[49,59] of poor quality.

Overview of results In four studies (all fair[2,108,110,113]) defining macrosomia as birth > 4,500 g and seven (6 fair[4,70,77,93,111,112] and 1 poor[59]) studies defining macrosomia as birthweight > 4000 g, the highest weight gains were demonstrated to be associated with macrosomia. A single poor study failed to show a significant association, using either definition of macrosomia.[49]

Detailed results In four of the studies in which macrosomia was defined as birthweight > 4,500 g,[2,108,110,113] the highest weight gains were associated with increased risk of macrosomia (Table 17). These four studies adjusted for multiple confounders such as age, BMI, race, parity, glucose levels, placental weight, smoking status, gestational age, and infant sex.[2,108,110,113] A

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nested case-control study (rated good quality), using women gaining 0.22 to 0.31 kg per week as the reference group, found that women with the highest rates of pregnancy weight gain (0.40 to 1.03 kg/week) were at increased risk for macrosomia (OR, 2.23; 95% CI, 1.54–3.22) and that women with the lowest rates (-0.26 to 0.21 kg/week) were at decreased risk (OR, 0.52; 95% CI, 0.34–0.79).[110] Results were similar when considering rates of weight gain only before 24 to 28 weeks of gestation.

TABLE 17. Gestational weight gain and macrosomia > 4,500 g

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Hedderson et al., 2006[110] USA, Kaiser Permanente Medical Care Program 45,245 All weights/BMI Good	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	ORs and 95% CI, for macrosomia based on rate of weight gain Rate of gain kg/wk: G1: -0.26 to 0.21 G2: 0.22 to 0.31 (Reference) G3: 0.32 to 0.39 G4: 0.40 to 1.03	G1: 0.52 (0.34–0.79) G2: 1.00 G3: 0.99 (0.67–1.47) G4: 2.23 (1.54–3.22)	Maternal age, maternal race/ethnicity, parity, pregravid BMI, screening glucose value, gestational age
Clausen et al., 2005[113] Norway, university hospital 2050 All weights/BMI Fair	Pregravid weight: Routine prenatal care Total weight gain: Routine prenatal care of maternity records	ORs and 95% CIs for macrosomia G1: WG, Quartile 1 (Reference) G2: WG, Quartile 2 G3: WG, Quartile 3 G4: WG, Quartile 4	G1: 1.0 G2: 2.1 (0.8–5.1) G3: 3.5 (1.5–8.0) G4: 4.3 (1.9–9.8)	Maternal age, parity, smoking, placental weight, gestational diabetes, first trimester BMI
Cogswell et al., 1994[2] USA, Pregnancy Nutrition Surveillance System 53,541 Normal/Overweight/Obese Fair	Pregravid weight: Self-report Total weight gain: Self-report	ORs and 95% CIs for macrosomia by GWG and prepregnancy BMI G1: Normal BMI, GWG 25–29 lbs (Reference for normal BMI) G2: Normal BMI, GWG 35–39 lbs G3: Normal BMI, GWG ≥ 40 lbs G4: Overweight BMI, GWG 15–19 lbs (Reference for overweight BMI) G5: Overweight BMI, GWG ≥ 40 lbs	G1: 1.0 G2: 1.5 (1.0–2.3) G3: 3.3 (2.3–4.7) G4: 1.0 G5: 4.0 (1.6–10.1) G6: 1.0 G7: 1.9 (1.3–2.9) G8: 2.1 (1.3–3.2) G9: 2.3 (1.6–3.3)	Maternal age, maternal race, height, smoking, infant sex, gestational age

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
		G6: Obese, GWG 15–19 lbs (Reference for obese GMI) G7: Obese BMI, GWG 30–34 lbs G8: Obese BMI, GWG 35–39 lbs G9: Obese BMI, GWG ≥ 40 lbs		
Zhou and Olsen, 1997[108] Denmark, two communities 7,122 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	OR and 95% CI, for macrosomia by GWG categories and BMI G1: GWG < 11 kg, underweight (Reference) G2: GWG < 11 kg, normal weight G3: GWG < 11 kg, overweight G4: GWG 12–15 kg, underweight, G5: GWG 12–15 kg, normal weight G6: GWG 12–15 kg, overweight G7: GWG ≥ 16 kg, underweight G8: GWG ≥ 16 kg, normal weight G9: GWG ≥ 16 kg, overweight	G1: 1.0 G2: 52.8 (0.3–22.9) G3: 9.7 (1.2–81.8) G4: 0.0 (0.0–7×10^5) G5: 6.8 (0.9–51) G6: 27.1 (3.3–220) G7: 6.1 (0.7–52.5) G8: 15.7 (2.2–114) G9: 45.6 (6.0–349)	Maternal age, parity, alcohol, no diabetes, term delivery, smoking, infant sex, gestational age
Brennand et al., 2005[49] Canada, medical records 603 Normal/Overweight/Obese Poor	Pregravid weight: Medical records Total weight gain: Routine prenatal care or maternity records	% Macrosomia among obese women only G1: Low WG, < 7 kg G2: Acceptable WG, 7–11.5 kg G3: Excessive WG, > 11.5 kg	G1: 16.9% G2: 15.3% G3: 18.4% (P = 0.834)	N/A

BMI, body mass index; CI, confidence interval; GWG,gestational weight gain; kg, kilogram; kg/wk, kilogram per week; N/A, not applicable; OR, odds ratio; wk, week.

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A fair-quality study in Denmark also showed increased risk of macrosomia at the highest weight gains, with the highest risks among overweight and obese women.[108] However, the confidence intervals from this study are very imprecise. A fair-quality study in Norway showed similar results, with increasing ORs as weight gain increased. Women with weight gain in the fourth quartile, as compared to weight gain in the first quartile, had the highest OR of 4.3 (95% CI, 1.9–9.8).[113]

Among low-income women enrolled in the Supplemental Food Program for Women, Infants, and Children (WIC), a fair-quality US study reported significant associations between weight gain and macrosomia only for women gaining more than 30 to 34 pounds when compared with women gaining 25 to 29 pounds for women of normal weight or with women gaining 5 to 19 pounds for overweight and obese women.[2] For average-weight women, the OR was 1.5 (95% CI, 1.0–2.3), for those gaining 35 to 39 pounds and 3.3 (95% CI, 2.3–4.7) for women gaining 40 pounds or more. Overweight women also had high risks for macrosomia, but only at weight gains of 40 pounds or more (OR, 4.0; 95% CI, 1.6–10.1). The OR among obese women gaining 30 to 34 pounds was 1.9 (95% CI, 1.3–2.9). Similar results were found for obese women gaining more than 35 pounds with odds ratios ranging from 2.1 to 2.3.

In a U.S. study of Cree women (rated poor quality), weight gain among obese women was not significantly associated with macrosomia.[49]

Of the eight studies that considered macrosomia as > 4,000 g, seven found a significant association between gestational weight gain and macrosomia (Table 18).[4,59,70,77,93,111,112] In general, the highest weight gains were associated with an increased risk of macrosomia. Six[4,70,77,93,111,112] of these studies were rated of fair quality, and one[59] of poor quality. These studies were adjusted for multiple confounders including maternal age, race, education, parity, height, pregravid weight, pregravid BMI, distantia cristarum, length of gestation, glucose levels, smoking status, and infant sex.

TABLE 18. Gestational weight gain and macrosomia > 4,000g

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Bergmann et al., 2003[112] Germany, Berlin Perinatal Registry 206,308 All weights/BMI Fair	Pregravid weight: Not stated Total weight gain: Not stated	ORs and 95% CIs of macrosomia G1: WG < 10 kg (Reference) G2: WG 10–16 kg G3: WG ≥ 16 kg	G1: 1.0 G2: 1.85 (1.77–1.93) G3: 3.37 (3.22–3.53)	Pregravid BMI, height, parity, smoking, diabetes, postterm delivery
Jain et al., 2007[77] USA, birth certificate records and	Pregravid weight: Not stated	ORs and 95% CIs for macrosomia	G1: 0.49 (0.30–0.82) G2: 1.0 G3: 1.17 (0.82–	Maternal age, pregravid BMI, parity, education, race/ethnicity, US/foreign origin

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Pregnancy Risk Assessment Monitoring System 7,661 All weights/BMI Fair	Total weight gain: Birth certificate	G1: WG ≤ 15 lbs G2: WG 15–24 lbs G3: WG 25–35 lbs G4: WG ≥ 35 lbs	1.65) G4: 2.83 (2.04–3.92)	
Johnson et al., 1992[70] USA, prenatal clinics 3,191 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	OR and 95% CI, for macrosomia G1: Net WG < 14.9 lbs (Reference) G2: Net WG 14.9–23.5 lbs G3: Net WG 24–33 lbs G4: Net WG > 33 lbs	G1: 1.0 G2: 1.20 (0.83–1.75) G3: 1.77 (1.24–2.52) G5: 2.86 (2.02–4.02)	Maternal race, parity, pregravid BMI, height, pregravid weight, marital status, education, tobacco/alcohol/drug use, pregnancy-induced hypertension, gestational age, macrosomia, infant sex
Kiel et al., 2007[4] USA, birth certificate registry 120,251 Obese Fair	Pregravid weight: Medical records Total weight gain: Routine prenatal care or maternity records	G1: Odds of macrosomia for WG > 25 lbs G2: OR of macrosomia for WG < 15 lbs G3: Reference WG 15–25 lbs	G1: Odds of Macrosomia are higher for women in this group G2: Odds of Macrosomia are lower for women in this group Numerical value for ORs not reported in study	Maternal age, maternal race, maternal education, poverty, smoking, parity, chronic hypertension
Kirchengast and Hartmann 2003[93] Austria, university hospital 8,011 All weights/BMI Fair	Pregravid weight: Estimated from measured weight at first prenatal visit Total weight gain: Routine prenatal care or maternity records	G1: OR and 95% CI, for Macrosomia	G1: 1.07 (1.05–1.10)	Maternal age, pregravid weight, height, distantia cristarum

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Takimoto et al., 2006[111] Japan, obstetric units 112,257 All weights/BMI Fair	Pregravid weight: Medical records Total weight gain: Routine prenatal care or maternity records	ORs and 95% CI, for macrosomia G1: Total GWG < 25th percentile for GA G2: Total GWG 25–49th percentile for GA G3: Total GWG 50–74th percentile for GA (Reference) G4: Total GWG 75–89th percentile for GA G5: Total GWG ≥90th percentile for GA	G1: 0.31 (0.20,–0.47) G2: 0.49 (0.34–0.70) G3: 1.0 G4: 1.62 (1.24–2.12) G5: 2.41 (1.83–3.17)	Maternal age, parity, pregravid weight, gestational age, infant sex
Brennand et al., 2005[49] Canada, medical records 603 Normal/Over-weight/Obese Poor	Pregravid weight: Medical records Total weight gain: Routine prenatal care or maternity records	% Macrosomia among obese women only G1: Low WG, < 7 kg G2: Acceptable WG, 7–11.5 kg G3: Excessive WG, > 11.5 kg	G1: 47.0% G2: 42.9% G3: 54.4% (P = 0.234)	N/A
Jensen et al., 2005[59] Denmark, university hospitals 481 Obese Poor	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	ORs and 95% CIs for Macrosomia G1: GWG < 5.0 kg (Reference) G2: GWG 5.0–9.9 kg G3: GWG 10.0–14.9 kg G4: GWG ≥ 15.0 kg	G1: 1.0 G2: 1.8 (0.8–3.8) G3: 2.2 (1.0–4.7) G4: 4.0 (1.8–9.0)	Maternal age, pregravid BMI, gestational age, 2-hour OGTT, parity, smoking, ethnicity, clinical center

BMI, body mass index; CI, confidence interval; GA, gestational age; GWG, gestational weight gain; lbs, pounds; OGTT, oral glucose tolerance test; OR, odds ratio; WG, weight gain.

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Among the fair-quality studies of gestational weight gain on macrosomia, ORs for this association were between 2.41 and 3.37 for the highest weight gains when compared to weight gains within the normal range.[70,77,111,112] Among a cohort of Japanese women (fair-quality study), the group with total weight gain above the 90th percentile for gestational age had an OR for macrosomia of 2.41 (95% CI, 1.83–3.17) relative to the group in the 50th to 74th percentile.[111] The effect was reduced for total weight gain based on percentile for gestational age for the lower percentile ranges. A fair-quality U.S. study looked at the association between net weight gain (total gestational weight gain minus infant birthweight) and macrosomia.[70] With women gaining < 14.9 pounds as the reference group, the strongest effect was noted among women gaining > 33 pounds (OR, 2.86; 95% CI, 2.02–4.02), followed by women gaining 24 to 33 pounds (OR, 1.77; 95% CI, 1.24–2.52); no significant effect was observed for women gaining 14.9 to 23.5 pounds. A fair-quality study in Germany found a higher risk of macrosomia for women gaining more than 16 kg as compared to women gaining less than 10 kg (OR, 3.37; 95% CI, 3.22–3.53).[112] Similar results were noted in a fair-quality U.S. study where weight gains above 35 pounds (as compared to weight gains of 15 to 25 pounds) were associated with an OR for macrosomia of 2.83 (95% CI, 2.04–3.92).[77] A fair-quality study in Austria found that for each 1 kg increase in gestational weight gain, the OR for macrosomia was 1.07 (95% CI, 1.05–1.10).[93] Of the poor-quality studies, one found results in a similar direction.[59] One poor-quality study among obese Cree women found that the percent macrosomia did not differ between weight gain groups.[49]

Size based on gestational age

Study characteristics Twenty-five articles from 23 studies examined the association between gestational weight gain and large-for-gestational-age (LGA) and small-for-gestational-age (SGA) infants.[4,51,58,59,61,66,68,89,95,100,105,108,111,114–123] These investigators used various definitions to classify both LGA and SGA infants. Some defined LGA as birthweight greater than the 90th percentile or more than 2 standard deviations (SD) above the mean. Some defined SGA as birthweight less than the 10th (or 15th) percentile or more than 2 (or 1.5) SD below the mean.

Of the 14 articles addressing LGA (Evidence Table 21),[4,54,58,59,61,68,100,105,115,116,118,120–122] two defined LGA using the > 2 SD criterion.[58,120] Ten used the commonly applied 90th percentile definition;[4,54,59,61,100,105,115,116,118,121] one study evaluated multiple percentiles;[68] and one defined LGA as fetal growth ratio (FGR) > 1.15.122 FGR is the ratio of the observed birthweight at a given gestational age to the mean birthweight at a given gestational age for a certain fetal growth distribution.

SGA definitions varied considerably as well: birthweight < 10th percentile; < 2 (or 1.5) SD below the mean; FGR < 0.85; or a combination of birthweight and percentile of placenta weight. If a study used a definition other than birthweight < 10th percentile, the specific criterion used will be noted in the text below. In general, the lowest weight gains were associated with increased risks for SGA.

Overview of results for LGA Among the studies that did not use BMI status (Table 19), six[100,105,115,118,121,122] were rated of fair quality and four[54,59,68,120] of poor quality. All reported lower risks of LGA with lower gestational weight gain. Studies that stratify by BMI status present greater challenges to synthesis. Two studies (1 good[116] and 1 fair[58]) examined a range of BMI categories, and found inconsistent results: one reported that the estimates of LGA did not differ greatly across BMI categories[116] while the other reported that high weight gain (> 16 kg) was strongly associated with LGA, and this association was most pronounced in the lowest BMI

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categories. A fair-quality study of obese women[4] observed lower odds of LGA among women who gained less than the reference group (15–25 pounds) and higher odds of LGA among women who gained more the reference group. A poor-quality study among Japanese women found that nulliparous women in the highest weight gain category (> 0.40 kg/week) had ORs for LGA of 2.25 (95% CI, 1.03–4.94) for low BMI women and 2.58 (95% CI, 1.71–3.89) for medium BMI women.[61]

TABLE 19. Gestational weight gain and LGA

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Bo et al., 2003[115] Italy, university clinic 700 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Not collected	G1: OR and 95% CI, for LGA for each 1 kg increase in GWG	G1: 1.08 (1.03–1.12)	Maternal age, pregravid BMI, smoking, gestational hyperglycaemia
Kitajima et al., 2001[121] Japan, university hospital 146 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	G1: OR and 95% CI, for LGA for each 1 kg increase in GWG	G1: 1.08 (0.81–1.44)	Pregravid BMI, maternal plasma glucose levels, gestational age, infant sex
Kramer et al., 1990[122] Canada, university hospital 8,719 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	G1: OR and 95% CI, for LGA for each 5 kg decrease in net gestational WG	G1: 0.73 (0.68–0.79)	Pregravid weight, infant sex, smoking, parity, maternal diabetes, height, previous LBW infant, severe pregnancy-induced hypertension
Muscati et al., 1996[105] Canada, public health department 371	Pregravid weight: Medical records Total weight gain: Collected by	G1: OR for LGA per 1 kg increase in WG up to week 20 G2: OR for LGA per 1 kg increase in WG from weeks 21 to 30 G3: OR for LGA per 1	G1: 1.17 ($P <$ 0.001) G2: 1.16 ($P < 0.01$) G3: 1.02 (P = NS)	Parity, pregravid standard weight, pregravid excess weight, birth length, infant sex

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
All weight/BMI Fair	study investigators	kg increase in WG from weeks 31 to term		
Parker and Abrams, 1992[118] USA, hospital 6,690 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	ORs and 95% CIs of LGA for high WG G1: Compared to UCSF Cohort 25–75th percentile of WG G2: Compared to UCSF 10–90th percentile of WG	G1: 1.89 (1.51–2.37) G2: 1.87 (1.39–2.52)	Maternal age, maternal race, parity, gestational age, smoking, pregravid BMI, height
Pezzarossa et al., 1996[100] Italy, not stated 192 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	Relative risks for LGA G1: GWG < 9 kg G2: GWG 9–14 kg	G1: Relative risks for LGA similar between non-diabetic and GDM groups G2: GDM group has 2 times higher risk that non-diabetics Numerical results not reported.	Pregravid BMI, fasting plasma glucose
Bianco et al., 1998[54] USA, medical center 613 Morbidly obese (BMI > 35) Poor	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	% LGA for GWG: G1: Weight loss or 0 lbs G2: 1–15 lbs G3: 16–25 lbs G4: 26–35 lbs G5: > 35 lbs	G1: 12.0 G2: 11.8 G3: 18.8 G4: 25.8 G5: 23.8 ($P < 0.01$)	N/A
Ekblad and Grenman, 1992[68] Finland, hospital 357 Prepregnancy weight 20% over or under ideal body weight for height	Pregravid weight: Medical records Total weight gain: Routine prenatal care or maternity records	Infant BW by group Infant weight percentile for mothers with normal pregravid weight and normal weight gain G1: < 2.5% G2: 2.5–10% G3: 10–50%	G1: 1% G2: 6% G3: 35% G4: 43% G5: 13% G6: 2% G7: 3% G8: 14% G9: 32%	N/A

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
and normal weight Poor		G4: 50–90% G5: 90–97.5% G6: > 97.5% Infant weight percentile for mothers with weight gain ≤5 kg G7: < 2.5% G8: 2.5–10% G9: 10–50% G10: 50–90% G11: 90–97.5% G12: > 97.5% Infant weight percentile for mothers with weight gain ≥20 kg G13: < 2.5% G14: 2.5–10% G15: 10–50% G16: 50–90% G17: 90–97.5% G18: > 97.5%	G10: 34% G11: 14% G12: 3% G13: 0% G14: 2% G15: 42% G16: 29% G17: 20% G18: 7%	
Jensen et al., 2005[59] Denmark, university hospitals 481 Obese Poor	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	ORs and 95% CIs for LGA G1: GWG < 5.0 kg (Reference) G2: GWG 5.0–9.9 kg G3: GWG 10.0–14.9 kg G4: GWG ≥ 15.0 kg	G1: 1.0 G2: 2.4 (1.1–5.3) G3: 2.1 (1.1–4.8) G4: 4.7 (2–11)	Maternal age, pregravid BMI, gestational age, 2 hour OGTT, parity, smoking, ethnicity, clinical center
Sunehag et al., 1991[120] Italy, prenatal clinics 133 All weights/BMI Poor	Pregravid weight: Not stated Total weight gain: Not stated	G1: Association between LGA and GWG > 18 kg	G1: $\chi^2 = 8.2$ ($P < 0.005$)	N/A

BMI, body mass index; CI, confidence interval; g, grams; GDM, gestational diabetes mellitus; GWG: gestational weight gain; kg, kilogram; lbs, pounds; LGA, large-for-gestational-age; N/A, not applicable; NS, non-significant;

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis

OR, odds ratio; SD: standard deviation; USCF, University of Southern California at San Francisco; WG, weight gain.

Detailed results Among the studies that did not use BMI status (Table 19), three fair-quality studies that evaluated the impact of a 1 kg increase in weight gain produced similar results.[105,115,121] For Italian women, the OR of having an LGA infant was 1.08 (95% CI, 1.03–1.12).[115] For nondiabetic Japanese women with a positive diabetic screen, the OR was 1.08 (95% CI, 0.81–1.44).[121] The third study evaluated this relationship separately for weight gain by time: up to week 20, from week 21 to week 30, and from week 31 to term. It found ORs of 1.17, 1.16, and 1.02 (non-significant), respectively.[105] The OR for weeks 31 to term was not significant. In other words, the odds of giving birth to an LGA infant tends to increase for each 1 kg increase in gestational weight gain during the first and second trimester.

Two fair-quality studies[100,118] considered the association between categorical weight gain and LGA. In a U.S. study,[118] women with the highest weight gains were at increased risk for LGA (OR, 1.89; 95% CI, 1.51–2.37) relative to women in the 25th to 75th percentile of weight gain and to women in the 10th to 90th percentile (OR. 1.87; 95% CI, 1.39–2.52). In a study involving mothers with GDM,[100] the risks for LGA were similar for weight gains up to 9 kg. However, for weight gains of 9 to 14 kg, the risk of LGA for mothers with GDM was two times that for nondiabetic mothers.

In a study that defined LGA as FGR > 1.15,122 the OR for having an LGA infant given a 5 kg decrease in net gestational weight gain (total gestational weight gain minus infant birthweight) was 0.73 (95% CI, 0.68–0.79). This result is consistent with other studies reporting that the odds of LGA drops with lower gains in maternal weight.

The poor-quality studies showed similar results. LGA was significantly related to the highest weight gains among studies of GDM mothers,[120] obese glucose-tolerant mothers,[59] and morbidly obese mothers.[54] A Finnish study[68] noted that women gaining ≥ 20 kg were more likely to have babies in the higher weight gain percentile categories, but these differences were not significantly different.

Four studies stratified results by BMI status (Table 20).[4,58,61,116] In a good-quality U.S. study, the estimates of LGA did not differ greatly across BMI categories.[116] The ORs of LGA for rate of weight gain of 50 g per week were as follows: among underweight women, 1.25 (95% CI, 1.11–1.41); among women of normal weight, 1.14 (95% CI, 1.08–1.20); and among overweight and obese women, 1.14 (95% CI, 1.07–1.20). In a fair-quality study based on the Swedish birth registry,[58] high weight gain (> 16 kg) was strongly associated with LGA, and this association was most pronounced in the lowest BMI categories. In comparison with the risk of LGA among women with weight gain between 8 and 16 kg (the reference group), adjusted ORs by BMI categories were as follows: BMI < 20, 3.26 (95% CI, 2.76–3.86); BMI ≥ 35, 1.54 (95% CI, 1.24–1.90).

TABLE 20. Gestational weight gain and LGA by BMI status

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Caulfield et al., 1998[116] USA, hospital obstetric database 3,870 All weights/BMI Good	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	ORs and 95% CIs for LGA per 50g/wk increase in rate of weight gain by BMI G1: Underweight G2: Normal weight G3: Overweight	G1: 1.25 (1.11–1.41) G2: 1.14 (1.08,–1.20) G3: 1.13 (1.07–1.20)	Maternal age, race, parity, pregravid BMI, height, hypertension, provider type, smoking, infant sex
Cedergren, 2006[58] Sweden, Medical Birth Registry 245,526 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	ORs and 95% CIs for LGA (> 2 SD above the mean) Weight gain < 8 kg G1: BMI < 20 G2: BMI 20–24.9 G3: BMI 25–29.9 G4: BMI 30–34.9 G5: BMI ≥ 35 Weight gain > 16 kg G6: BMI < 20 G7: BMI 20–24.9 G8: BMI 25–29.9 G9: BMI 30–34.9 G10: BMI ≥ 35 Weight gain 8–16 kg (Reference)	G1: 0.43 (0.24–0.75) G2: 0.53 (0.47–0.61) G3: 0.48 (0.43–0.53) G4: 0.66 (0.59–0.75) G5: 0.54 (0.46–0.63) G6: 3.26 (2.76–3.86) G7: 2.73 (2.60–2.88) G8: 2.14 (2.01–2.28) G9: 2.24 (2.00–2.51) G10: 1.54 (1.24–1.90)	Maternal age, parity, smoking, year of birth
Kiel et al., 2007[4] USA, birth certificate registry 120,251 Obese Fair	Pregravid weight: Medical records Total weight gain: Routine prenatal care or maternity records	G1: Odds of LGA for weight gain > 25lbs G2: OR of LGA for weight gain < 15lbs G3: Reference weight gain 15–25 lbs	G1: Odds of LGA are higher for women in this group G2: Odds of LGA are lower for women in this group Numerical value for ORs not reported in study	Maternal age, maternal race, maternal education, poverty, smoking, parity, chronic hypertension
Wataba et al.,	Pregravid weight:	ORs and 95% CIs for LGA	G1: 2.25 (1.03–	Preeclampsia, C-section, 1-

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
2006[61] Japan, academic medical center 21,718 All weights/BMI Poor	Not stated Total weight gain: Routine prenatal care or maternity records	Nulliparous G1: Low BMI (< 18), WG > 0.40 kg/wk G2: Medium BMI (18–23.9), WG 0.20–0.25 kg/wk G3: WG 0.25–0.30 kg/wk (Reference) G4: Medium BMI, WG 0.30–0.35 kg/wk G5: Medium BMI, WG 0.35–0.40 kg/wk G6: Medium BMI, WG > 0.40 kg/wk Parous G7: Low BMI (< 18), WG > 0.40 kg/wk G8: WG 0.20–0.25 kg/wk (Reference for low/med BMI) G9: Medium BMI (18–23.9), WG 0.25–0.30 kg/wk G10: Medium BMI, WG 0.30–0.35 kg/wk G11: Medium BMI, WG 0.35–0.40 kg/wk G12: Medium BMI, WG > 0.40 kg/wk G13: High BMI (≥24), WG 0.15–0.20 kg/wk G14: WG ≥ 0.30 kg/wk (Reference for high BMI)	4.94) G2: 1.41 (1.31–1.76) G3: 1.0 G4: 1.76 (1.38–2.23) G5: 2.34 (1.77–3.10) G6: 2.58 (1.71–3.89) G7: 2.16 (0.63-/7.44) G8: 1.0 G9: 1.48 (1.15–2.33) G10: 1.64 (1.18–2.27) G11: 2.23 (1.51–3.31) G12: 3.94 (2.56–6.03) G13: 2.27 (1.31–3.95) G14: 1.0	minute Apgar score < 4

BMI, body mass index; CI, confidence interval; g, grams; g/wk, gram per week; kg/wk, kilogram per week; LGA, large for gestational age; OR, odds ratio; SD, standard deviation; WG, weight gain.

In a fair-quality study of obese women,[4] lower odds of LGA were observed among women who gained less than the reference group (15–25 pounds) and higher odds of LGA were observed among women who gained more the reference group. Minimal risk for LGA was observed at weight gains of 10 to 25 pounds for class I obese women (BMI 30–34.9), at gains of 0 to 9 pounds for class II obese women (BMI 35–39.9), and at gains of 0 to 9 pounds for class II obese women (BMI ≥ 40). A poor-quality study among Japanese women found that nulliparous

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women in the highest weight gain category (> 0.40 kg/week) had ORs for LGA of 2.25 (95% CI, 1.03–4.94) for low BMI women and 2.58 (95% CI, 1.71–3.89) for medium BMI women.[61]

Eleven studies[4,58,59,61,100,105,115,116,118,121,122] adjusted for potential confounders including age, pregravid BMI, glucose levels, smoking status, parity, and gestational age.

Overview of results for SGA Twenty studies examined the relationship between gestational weight gain and SGA (Evidence Table 22).[4,51,54,58,59,61,66,68,89,95,105,108,111,114,116,118,119,122–124] One study was of good quality,[116] twelve of fair quality,[19,24,53,69,72,76,79,83,85,89–91] and seven of poor quality.[11,14,20,22,29,31,59] SGA births as a percentage of all births tended to be highest for the lowest weight gains.

Detailed results for SGA As with LGA results, we discuss results relating to the simple association between weight gain and risk for SGA separately (Table 21) from those that also take BMI status into account (Table 22). Among indigent US women (fair-quality study),[89] the percentage of SGA infants was 9.9 among women gaining < 0.24 kg per week, and 5.7 among the group gaining ≥ 0.75 kg per week. Similar results were observed among a cohort of Japanese women (fair-quality study),[111] which defined SGA as birthweight < 1.5 SD below the mean. The percentage of SGA infants ranged from 10.9 percent in the lowest weight gain group (< 25th percentile of weight gain) to 3.1 percent in the highest weight gain group (≥ 90th percentile of weight gain). Another fair-quality U.S. study observed an increased incidence of SGA at low weight gains; the incidence among obese women with low weight gain was two times that among obese women with normal weight gain.[118] Similar results were obtained in a poor-quality study of morbidly obese women.[54]

TABLE 21. Gestational weight gain and SGA

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Parker and Abrams, 1992[118] USA, hospital 6,690 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	ORs and 95% CIs of SGA for low WG G1: Compared to UCSF Cohort 25–75th percentile of WG G2: Compared to UCSF 10–90th percentile of WG	G1: 2.06 (1.62–2.63) G2: 1.82 (1.35–2.47))	Maternal age, maternal race, parity, gestational age, smoking, pregravid BMI, height
Cheng et al., 2004[124] USA, birth certificate registry 14,114 All weights/BMI	Pregravid weight: Self-report Total weight gain: Not stated	OR and 95% CI, for SGA G1: WG < 0.2 kg/wk G2: WG ≥ 0.2 kg/wk (Reference)	G1: 1.9 (1.8–2.2) G2: 1.0	Maternal age, education, Medicaid status, pregravid BMI, smoking, previous SGA, adequacy of prenatal care, maternal cardiac disease, preeclampsia, year of birth of second infant

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Fair				
Cnattingius et al., 1998[123] Sweden, Medical birth register 167,750 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	ORs and 95% CIs for SGA G1: WG < 0.25 kg/wk G2:WG 0.25–0.34 kg/wk G3: WG 0.35–0.44 kg/wk G4: ≥ 0.45 kg/wk (Reference)	G1: 3.0 (2.5–3.5) G2: 1.9 (1.6–2.2) G3: 1.3 (1.1–1.5) G4: 1.0	Maternal age, parity, pregravid BMI, height, education, mother living with father, smoking
Dawes and Grudzinskas, 1991[119] UK, hospital 1,092 All weights/BMI Fair	Pregravid weight: Measured at first prenatal visit Total weight gain: Routine prenatal care or maternity records	Average weekly weight gain < 0.20 kg as a predictor of SGA G1: Sensitivity G2: Specificity	G1: 12.9% G2: 91.3%	Maternal age, parity, pregravid BMI, weight, smoking, gestational age
Kiel et al., 2007[4] USA, birth certificate registry 120,251 Obese Fair	Pregravid weight: Medical records Total weight gain: Routine prenatal care or maternity records	G1: Odds of SGA for weight gain > 25lbs G2: OR of SGA for weight gain < 15 lbs G3: Reference Weight gain 15–25 lbs	G1: Odds of SGA are lower for women in this group G2: Odds of SGA are higher for women in this group Numerical value for ORs not reported in study	Maternal age, maternal race, maternal education, poverty, smoking, parity, chronic hypertension
Kramer et al., 1990[122] Canada, university hospital 8,719 All weights/BMI	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	G1: OR and 95% CI, for SGA for each 5 kg decrease in net gestational WG	G1: 1.32 (1.20–1.44)	Pregravid weight, infant sex, smoking, parity, maternal diabetes, height, previous LBW infant, severe pregnancy-induced hypertension

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Fair				
Muscati et al., 1996[105] Canada, public health department 371 All weight/BMI Fair	Pregravid weight: Medical records Total weight gain: Collected by study investigators	G1: OR for SGA per 1 kg increase in WG up to week 20 G2: OR for SGA per 1 kg increase in WG from weeks 21 to 30 G3: OR for SGA per 1 kg increase in WG from weeks 31 to term	G1: 0.93 (*P* = *NS*) G2: 0.85 (*P* < 0.01) G3: 0.89 (*P* < 0.01)	Parity, pregravid standard weight, pregravid excess weight, birth length, infant sex
Steward and Moser, 2004[114] USA, vital statistics data 2,933 All weights/BMI Fair	Pregravid weight: Not stated Total weight gain: Self-report	G1: OR and 95% CI, for SGA defined as FGR < 0.85	G1: 0.98 (0.97–0.98)	Maternal age, race, education, marital status, pregravid weight, adequacy of prenatal care, smoking, infant sex
Takimoto et al., 2006[111] Japan, obstetric units 112,257 All weights/BMI Fair	Pregravid weight: Medical records Total weight gain: Routine prenatal care or maternity records	ORs and 95% CI, for SGA G1: Total GWG < 25th percentile for GA G2: Total GWG 25–49th percentile for GA G3: Total GWG 50–74th percentile for GA (Reference) G4: Total GWG 75–89th percentile for GA G5: Total GWG ≥90th percentile for GA	G1: 2.87 (2.56–3.21) G2: 1.49 (1.35–1.66) G3: 1.0 G4: 0.55 (0.55–0.72) G5: 0.45 (0.45–0.63)	Maternal age, parity, pregravid weight, gestational age, infant sex
Wen et al., 1990[89] USA, hospital	Pregravid weight: Measured at first prenatal	ORs for SGA G1: GWG < 0.24 kg/wk	G1: 2.24 (*P* < 0.05) G2: 1.55 (*P* < 0.05) G3: 1.0 G4: 1.25 (NS)	Maternal age, race, parity, marital status, education, previous preterm delivery, alcohol use, drug use, maternal height, maternal weight,

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Cohort 17,149 Fair	visit Total weight gain: Routine prenatal care or maternity records	G2: GWG 0.24–0.57 kg/wk G3: GWG 0.58–0.74 kg/wk (Reference) G4: GWG ≥ 0.75 kg/wk		smoking, infant sex
Bianco et al., 1998[54] USA, medical center 613 Morbidly obese (BMI > 35) Poor	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	% SGA for GWG: G1: Weight loss or 0 lbs G2: 1–15 lbs G3: 16–25 lbs G4: 26–35 lbs G5: > 35 lbs	G1: 4 G2: 3.9 G3: 5.6 G4: 3.1 G5: 3.8	N/A
Cherry et al., 1993[95] USA, hospital 599 All weights/BMI Poor	Pregravid weight: Measured by study investigators Total weight gain: Routine prenatal care or maternity records	%SGA for each shifting of EW category. Light: < 90% EW Normal: 90 to 110% of EW Heavy: > 110% EW G1: Normal to Heavy G2: Light to Normal G3: Heavy to Heavy G4: Normal to Normal G5: Light to Light G6: Heavy to Normal G7: Normal to Light	G1: 22% G2: 39% G3: 38% G4: 41% G5: 62% G6: 60% G7: 65%	N/A
Ekblad and Grenman, 1992[68] Finland, hospital 357 Prepregnancy	Pregravid weight: Medical records Total weight gain: Routine	Infant BW by group Infant weight percentile for mothers with normal prepregnancy weight and normal weight gain	G1: 1% G2: 6% G3: 35% G4: 43% G5: 13% G6: 2% G7: 3%	N/A

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
weight 20% over or under ideal body weight for height and normal weight Poor	prenatal care or maternity records	G1: < 2.5% G2: 2.5–10% G3: 10–50% G4: 50–90% G5: 90–97.5% G6: > 97.5% Infant weight percentile for mothers with weight gain ≤5 kg G7: < 2.5% G8: 2.5–10% G9: 10–50% G10: 50–90% G11: 90–97.5% G12: > 97.5% Infant weight percentile for mothers with weight gain ≥20 kg G13: < 2.5% G14: 2.5–10% G15: 10–50% G16: 50–90% G17: 90–97.5% G18: > 97.5%	G8: 14% G9: 32% G10: 34% G11: 14% G12: 3% G13: 0% G14: 2% G15: 42% G16: 29% G17: 20% G18: 7%	
Jensen et al., 2005[59] Denmark, university hospitals 481 Obese Poor	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	Rates of SGA G1: GWG < 5.0 kg (Reference) G2: GWG 5.0–9.9 kg G3: GWG 10.0–14.9 kg G4: GWG ≥ 15.0 kg	No significant difference in rates of SGA by maternal weight gain group. Numerical results not reported in article.	Maternal age, pregravid BMI, gestational age, 2 hour OGTT, parity, smoking, ethnicity, clinical center
Lang et al., 1996[66] USA, hospital 11,505 All weights/BMI Poor	Pregravid weight: Not stated Total weight gain: Not stated	ORs and 95% CIs for SGA G1: WG ≤ 0.40 lbs/wk G2: WG 0.40–0.65 lbs/wk G3: WG 0.65–0.90 lbs/wk (Reference)	G1: 2.8 (2.2–3.6) G2: 1.6 (1.4–1.9) G3: 1.0 (Reference) G4: 0.6 (0.5–0.7)	Maternal age, race, parity, height, pregravid weight, maternal education, health insurance, planned pregnancy, previous induced abortion, previous spontaneous abortion, previous still birth, maternal morbidity, caffeine intake, marijuana, prenatal care, smoking, infant sex

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
		G4: WG > 0.90 lbs/wk		

BMI, body mass index; CI, confidence interval; EW, expected weight; FGR, fetal growth ratio; G, group; GWG, gestational weight gain; kg, kilogram; kg/wk, kilogram per week; lb, pound; NS, non-significant; OR, odds ratio; SGA, small-for-gestational-age; WG, weight gain.

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TABLE 22. Gestational weight gain and SGA by BMI status

Author, Year Country, Setting Sample Size Baseline BMI Quality	**Pregravid Weight (How Measured) Total Weight Gain (How Measured)**	**Definition of Groups**	**Results**	**Confounders and Effect Modifiers Included in Analysis**
Caulfield et al., 1998[116] USA, hospital obstetric database 3,870 All weights/BMI Good	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	ORs and 95% CIs for SGA per 50g/wk increase in rate of weight gain by BMI G1: Underweight G2: Normal weight G3: Overweight	G1: 0.87 (0.78–0.97) G2: 0.90 (0.84–0.96) G3: 0.93 (0.86–1.01)	Maternal age, race, parity, pregravid BMI, height, hypertension, provider type, smoking, infant sex
Cedergren, 2006[58] Sweden, Medical Birth Registry 245,526 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	ORs and 95% CIs for SGA (< 2 SD below the mean) Weight gain < 8 kg G1: BMI < 20 G2: BMI 20–24.9 G3: BMI 25–29.9 G4: BMI 30–34.9 G5: BMI ≥ 35 Weight gain > 16 kg G6: BMI < 20 G7: BMI 20–24.9 G8: BMI 25–29.9 G9: BMI 30–34.9 G10: BMI ≥ 35 Weight gain 8–16 kg (Reference)	G1: 2.35 (1.92–2.88) G2: 1.99 (1.77–2.23) G3: 1.75 (1.48–2.07) G4: 1.68 (1.26–2.25) G5: 1.71 (1.03–2.85) G6: 0.50 (0.41–0.61) G7: 0.50 (0.45–0.56) G8: 0.57 (0.47–0.68) G9: 0.61 (0.40–0.93) G10: 0.50 (0.20–1.24)	Maternal age, parity, smoking, year of birth
Cheng et al.,	Pregravid	95% CIs of SGA for low	G1: (1.2–	Maternal age, education, Medicaid status,

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
2004[124] USA, birth certificate registry 14,114 All weights/BMI Fair	weight: Self-report Total weight gain: Not stated	weight gain (< 0.2 kg/wk) by BMI G1: Underweight G2: Normal weight G3: Overweight G4: Obese	2.4) G2: (1.9–2.7) G3: (1.6–2.9) G4: (1.4–2.1)	pregravid BMI, smoking, previous SGA, adequacy of prenatal care, maternal cardiac disease, preeclampsia, year of birth of second infant
Zhou and Olsen, 1997[108] Denmark, two communities Cohort 7,122 Fair	Pregravid weight: Self-report Total weight gain: Routine prenatal care or maternity records	% Growth retardation (birthweight < 3,000g and placental weight > 490g) by weight gain category and BMI Weight gain < 11 kg G1: Underweight (Reference) G2: Normal G3: Overweight Weight gain 12–15 kg G4: Underweight G5: Normal G6: Overweight Weight gain > 16 kg G7: Underweight G8: Normal G9: Overweight	G1: 1.0 G2: 0.6 (0.4–0.8) G3: 0.6 (0.4–1.1) G4: 0.3 (0.2–0.5) G5: 0.4 (0.3–0.6) G6: 0.4 (0.1–1.0) G7: 0.3 (0.2–0.5) G8: 0.2 (0.1–0.3) G9: 0.2 (0.1–0.6)	Maternal age, parity, alcohol, diabetes, term delivery, smoking, gestational age, infant sex
Kabiru and Raynor, 2004[51] USA, hospital 5,131 All weights/BMI Poor	Pregravid weight: Measured at first prenatal visit Total weight gain: Routine prenatal care or maternity records	% SGA G1: No change in BMI category G2: 1 category increase in BMI G3: > 1 category increase in BMI % SGA among overweight G4: No change in BMI category G5: 1 category increase in BMI G6: > 1 category increase in	G1: 19.5% G2: 13.5% G3: 9.5% G4: 14.2% G5: 9.9% G6: 11.5%	N/A

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
		BMI		
Wataba et al., 2006[61] Japan, academic medical center 21,718 All weights/BMI Poor	Pregravid weight: Not stated Total weight gain: Routine prenatal care or maternity records	ORs and 95% CIs for SGA Parous, Low BMI (< 18) G1: WG < 0.15 kg/wk G2: WG 0.15–0.20 kg/wk G3: WG 0.20–0.25 kg/wk G4: WG 0.25–0.30 kg/wk (Reference Parous, Medium BMI (18–23.9) G5: WG < 0.15 kg/wk G6: WG 0.15–0.20 kg/wk G7: WG 0.20–0.25 kg/wk (Reference) Parous, High BMI (> 24) G8: WG < 0.15 kg/wk G9: WG 0.15–0.20 kg/wk (Reference) Nulliparous, Low BMI (< 18) G10: WG < 0.15 kg/wk G11: WG 0.15–0.20 kg/wk G12: WG 0.20–0.25 kg/wk G13: WG 0.25–0.30 kg/wk (Reference Nulliparous, Medium BMI (18–23.9) G14: WG < 0.15 kg/wk G15: WG 0.15–0.20 kg/wk G16: WG 0.20–0.25 kg/wk G17: WG 0.25–0.30 (Reference) Nulliparous, High BMI (> 24) G18: WG < 0.05 kg/wk G19: WG 0.15–0.20 kg/wk (Reference)	G1: 5.42 (2.86–10.27) G2: 2.78 (1.53–5.06) G3: 1.39 (0.82–2.42) G4: 1.0 G5: 2.21 (1.67–2.93) G6: 1.68 (1.23–2.07) G7: 1.0 G8: 2.82 (1.17–6.78) G9: 1.0 G10: 6.20 (2.72–14.09) G11: 2.58 (1.14–5.87) G12: 2.46 (1.19–5.08) G13: 1.0 G14: 2.64 (1.88–3.71) G15: 1.60 (1.15–2.23) G16: 1.39 (1.03–1.87) G17: 1.0 G18: 7.06	Preeclampsia, C-section, 1-minute Apgar score < 4

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
			(2.11–23.61) G19: 1.0	

BMI, body mass index; cat, category; CI, confidence interval; G, group; g, gram; kg, kilogram; kg/wk, kilogram per week; lbs, pounds; med, medium; OR, odds ratio; SD, standard deviation; SGA, small-for-gestational-age.

Six fair-quality studies[24,53,76,83,90,91] evaluated the ORs for SGA and found that the lowest weight gains (as compared to normal weight gains) were associated with ORs between 1.82 and 3.0. Among indigent US women,[89] the risk of SGA was highest for mothers in the lowest weight gain category (< 0.24 kg/week) when compared with women gaining 0.58 to 0.74 kg per week (OR2.24; P < 0.05). A weaker association was observed for women gaining 0.24 to 0.57 kg per week (OR1.55; P < 0.05). A U.S. study noted earlier also found that women with the lowest weight gains had an OR for an SGA infant of 2.06 (95% CI, 1.62–2.63) when compared with women gaining between the 25th and 75th percentile, and an OR of 1.82 (95% CI, 1.35–2.47) when compared with women gaining between the 10th and 90th percentiles.[118] A U.S. study found that the OR of SGA in a second pregnancy was 1.9 (95% CI, 1.8–2.2) for weight gains less than 0.2 kg/wk as compared to weight gains greater than 0.2 kg/wk.[124] The study of Japanese women noted earlier defined SGA as birthweight < 1.5 SD below the mean and gestational weight gain according to percentiles.[111] Among women in the two lowest weight gain categories (very low and low) the ORs of SGA were 2.87 (95% CI, 2.56–3.21) and 1.49 (95% CI, 1.35–1.66), respectively, when compared with women in the moderate weight gain category. In addition, a significant protective effect was observed for the two highest weight gain categories. One study, using data from the Swedish Medical Birth Registry, observed higher rates of SGA (here defined as < 2 SD below the mean) among the lowest weight gain groups.[123] Specifically, women gaining < 0.25 kg per week had an OR of 3.0 (95% CI, 2.5–3.5) when compared with women gaining ≥ 0.45 kg per week. The ORs decreased as gestational weight gain category dropped. Similar results were found in a study of obese women.[4]

Among white nonsmokers in Canada (fair-quality study),105 for each 1 kg increase in weight gain up to week 20, the OR of an SGA infant was 0.93 (not significant); for weight gain from weeks 21 to 30, it was 0.85 (P < 0.01); and for weight gain from week 31 to term, it was 0.89 (P < 0.01). In other words, increases in weight gain from weeks 21 to term lowered a woman's risk of an SGA infant. A fair-quality study of the predictors of SGA found that average weekly weight gain < 0.20 kg had 12.9 percent sensitivity and 91.3 percent specificity.[119]

Two fair-quality studies defined growth restriction using FGR, with SGA specified as an FGR < 0.85.79,[89] In general, increases in weight gain were associated with lower risks of SGA. Specifically, one study found an OR of 0.98 (95% CI, 0.97–0.98) for each 1 kg increase in total gestational weight gain.[114] Another study found an OR of 1.32 (95% CI, 1.20–1.44) for each 5 kg decrease in net gestational weight gain (total gestational weight gain minus infant birthweight).[122]

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In a poor U.S. study,[66] using women gaining 0.65 to 0.9 pounds per week as the reference group, women gaining ≤ 0.40 pounds per week had an OR for an SGA infant of 2.8 (95% CI, 2.2–3.6), and women gaining 0.4 to 0.65 pounds per week an OR of 1.6 (95% CI, 1.4–1.9). In this study, however, women gaining > 0.9 pounds per week also experienced a significant protective effect against SGA (OR, 0.6; 95% CI, 0.5–0.7).

The results from three[14,20,31] poor-quality studies did not find statistically significant results. One study was among Finnish women,[68] one defined SGA as birthweight < 2 SD below the mean,[59] and one study was among morbidly obese women.[54] A study among adolescents (also rated poor) looked at the proportion of infants who gained less than the median weight (instead of the 10th percentile).[95] Mothers who shifted to higher weight classes had fewer infants who fell below the median for intrauterine growth; women who did not maintain their weight and shifted to lower weight classes were more likely to have infants below the median for intrauterine growth.

Six studies presented stratified analyses by BMI (Table 22).[11,19,22,72,81,91] In general, the risk of SGA among women with low weight gain decreased as BMI increased.

A U.S. database study (rated good quality) found that increasing rates of weight gain were associated with reduced risk of an SGA infant, with the risk decreasing with increasing BMI.[116] Specifically, the ORs of SGA for each 50 g per week increase in maternal weight were as follows: 0.87 (95% CI, 0.78–0.97) for underweight mothers; 0.90 (95% CI, 0.84–0.96) for mothers of normal weight; and 0.93 (95% CI, 0.86–1.01) for overweight and obese women. In the Swedish birth registry study (rated fair quality), the risk of SGA was higher in the low weight gain group (< 8 kg), but the risk decreased with increasing BMI.[58] Using women gaining between 8 and 16 kg as the reference group, these researchers reported that the OR for delivering an SGA infant for women with low weight gain (< 8 kg) was 1.71 (95% CI, 1.03–2.85) among women with a BMI ≥ 35; it was 2.35 (95% CI, 1.92–2.88) among women with a BMI < 20. Women gaining > 16 kg were at decreased risk for delivering an SGA infant, with the risk being similar between all BMI categories.

Among nondiabetic women in Denmark (fair-quality study) for whom SGA was defined as birthweight < 3,000 g despite placenta weight being above the 66th percentile (491 g), women who gained more than 16 kg were at lower risk of delivering an SGA infant; this risk was the same regardless of BMI status.[108] The risk of SGA decreased with increasing weight gain, and it also tended to decrease as BMI increased. In a U.S. study, 95% CIs of the OR of SGA for low weight gain (< 0.2 kg/wk) compared to weight gain >0.2 kg/wk, were similar across BMI categories: underweight (95% CI, 1.2–2.4), normal weight (95% CI, 1.9–2.7), overweight (95% CI, 1.6–2.9), obese (95% CI, 1.4–2.1).[124]

A poor-quality study of the effect of changing BMI categories found that excessive weight gain (defined in various ways depending on BMI) was associated with lower rates of SGA for two groups of women: normal weight (excessive gain, > 35 pounds; P = 0.016) and overweight (excessive gain, > 25 pounds; P = 0.003); this association did not hold for obese women.[51] A study among Japanese women (also poor quality) found high risks for SGA among nulliparous women with low BMI (< 18) and low rates of weight gain (< 0.15 kg/week).[61]

Sixteen of these studies adjusted for multiple confounding factors such as age, pregravid BMI, smoking, glucose levels, parity, race, gestational age, marital status, height, education, and sex of infant.[19,20,22,24,29,53,69,72,76,79,81,83,85,89–91]

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Apgar scores

Study characteristics Four studies, set in Sweden,[58] the United States,[33,92] and Japan,[61] examined the effect of gestational weight gain on Apgar scores (Evidence Table 23, Table 23). Apgar scores, calculated on the basis of five criteria (appearance, pulse, grimace, activity, respiration), range from 0 to 10. Three were cohort studies; the fourth was a case-control study examining outcomes of macrosomic infants (≥ 4,000 g) and normal-weight babies (2,500–3,999 g).[125]

TABLE 23. Gestational weight gain and Apgar scores

Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Cedergren, 2006[58] Sweden, Medical Birth Registry 245,526 All weights/BMI Fair	Pregravid weight: Self report, if unknown, standardized measurement is made during first visit to maternity health care center Total weight gain: Measured when woman entered delivery unit	Weight gain < 8 kg, 8–16 kg, and >16 kg for each BMI class below G1: BMI < 20 G2: BMI 20–24.9 G3: BMI 25–29.9 G4: BMI 30–34.9 G5: BMI ≥35	No association between low weight gain and Apgar score (< 7), despite BMI of mother	BMI, maternal age, parity, smoking in early pregnancy, year of birth
Johnson et al., 1992[70] USA, prenatal clinics 3,191 All weights/BMI Fair	Pregravid weight: Self report collected at first antepartal visit Total weight gain: Last prenatal visit	G1: total weight gain < 16 lb G2: total weight gain 16–25 lb G3: total weight gain 26–35 lb G4: total weight gain > 35 lb	Increased OR for gestational weight gain on 1-minute and 5-minute Apgar score ≤ 7, persists after adjusting (no further details provided)	Prepregnancy weight quartile, height (tertile), BMI category, race, parity, hypertension, other variables entered by stepwise regression model
Nixon et al., 1998[125] USA, county nurse-midwifery services 2,228 All weights	Pregravid weight: Routine data forms, self report collected at first prenatal visit Total weight gain: Routine data forms, prenatal care or maternity records prior to delivery	Continuous weight gain measure	Gestational weight gain was not a predictor of Apgar scores < 7	Age, parity, BMI

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Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
(IOM) Fair				
Wataba et al., 2006[61] Japan, academic medical center 21,718 All weights/BMI Poor	Pregravid weight: Hospital database/register Total weight gain: Hospital database/record	Rate of weight gain, categorized differently across different BMI groups	AOR for 1 min Apgar scores < 4 for nulliparous women with low BMI, weekly weight gain < 15 kg/wk, compared with women gaining 0.25–0.3 kg/wk: 12.24 (2.04–73.43) AOR for 1 min Apgar scores < 4 for parous women with medium BMI, weekly weight gain 0.35–0.4 kg/wk compared with women gaining 0.2–0.25 kg/wk: 2.21 (1.08–4.53) No other relationships were significant	Parity, baseline BMI

AOR, adjusted odds ratio; BMI, body mass index; kg/wk, kilogram per week.

Overview of results These four studies, three rated fair[19,33,92] and one poor,[61] did not provide consistent evidence on the direction or trend of effect. These studies inconsistently controlled for confounders. None controlled for a range of maternal pregnancy complications that could account for low Apgar scores.

Detailed results Three studies examined 1-minute or 5-minute Apgar scores at two levels: > 7 or ≥ 7. Two found no association between gestational weight gain and Apgar scores.[19,92] The third found increased ORs for gestational weight gain, after adjusting for prepregnancy weight quartile, height (tertile), BMI category, race, parity, hypertension, and other variables entered by stepwise regression model, but the authors provided no further details on the magnitude of the effect.[70]

One poor-quality study examined associations between 1-minute Apgar scores > 4 and rates of weekly weight gain (7 categories), categorized differently across different BMI groups (3 groups) and parity (2 categories), resulting in 42 comparisons.[61] Two comparisons were statistically significant: (1) higher risk for low Apgar scores for nulliparous women with low BMI and lower-than-median weight gain for their peer group; and (2) higher risk for parous women with medium BMI with higher-than-median weight gain for their peer group.

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Infant Outcomes

Perinatal mortality

Study characteristics Three studies, two set in the United States[93,94] and one in Denmark,[126] looked at the association between maternal weight gain and mortality, defined in one study as stillbirth[126] and in two others as perinatal mortality (neonatal plus fetal deaths)[93,94] (Table 24, Evidence Table 24). All three studies used different definitions of maternal weight gain:

- weight gain per week;[126]
- optimal weight gain[127] defined as 36 to 40 pounds for underweight women, 31 to 40 pounds for women of ideal prepregnancy weight, and 26 to 30 pounds for overweight women, based on associations between maternal prepregnancy weight, height, weight gain, and adverse perinatal outcomes; and
- low weight gain (< 0.8 kg per week).[128]

TABLE 24. Gestational weight gain and perinatal mortality

Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Bracero and Byrne, 1997[127] USA, hospital 20,971 All weights/BMI Fair	Pregravid weight: Self-report at first prenatal visit Total weight gain: Last prenatal assessment	G1: Suboptimal weight gain G2: Optimal weight gain	Perinatal mortality% G1: 0.6 G2: 0.2 *P* < 0.0001	NA
Nohr et al., 2005[126] Denmark, National Birth Cohort 54,505 All weights/BMI Fair	Pregravid weight: Self-report of pre-pregnancy weight at first telephone interview between 9 and 24 weeks weeks Total weight gain: Average weekly increase between self reported weights in first and second pregnancy interviews for women who provided a first interview between 9–24 weeks, those who provided a second interview between 26 and 38 completed weeks of gestation, and those who had at least 6 weeks between 2 interviews	Weight per week for BMI groups (underweight < 18.5; 18.5 ≤ normal weight < 25; 25 ≤ overweight < 30; and obese ≥ 30)	Weight gain in pregnancy was not significantly associated with the risk of stillbirth for any BMI groups.	AOR adjusted for age, height, parity, socio-occupational status, physical exercise, smoking, alcohol and coffee consumption

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Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Naeye, 1990[128] USA, hospitals affiliated with medical schools 56,857 All weights/BMI Poor	Pregravid weight: Self-report at first antenatal care visit Total weight gain: Data from medical records after first trimester	Low weight gain defined as < 0.8 kg/week after the first trimester for pregravid BMI groups below: G1: BMI < 20 G2: BMI 20–24 G3: BMI 25–30 G4: BMI > 30	Attributable risk estimates for perinatal death for low pregnancy weight gain G1: 0.03 (95% CI, 0.02–0.05) G2: 0.02 (95% CI, 0.01–0.03) G3: 0.01 (95% CI, 0.00–0.02) G4: 0.00	Age 35–40, diabetes mellitus, hypertensive disorders, black, preterm birth, major congenital malformations, twins, neonatal respiratory distress syndrome

Overview of results One of these studies was rated poor quality[128] and the others were rated fair. These studies suggest a protective effect of gestational weight gain on perinatal mortality but not on stillbirth.

Results for categorical measures of weight gain Both studies that focused on optimal or low weight gain found a protective effect of weight gain on infant mortality, but variations in the definition of maternal weight gain and the outcome do not allow quantification of the magnitude of the effect.[93,94]

Results for rate of weight gain The study that examined associations between weight gained per week and stillbirth found no effect of weight gain on stillbirth within groups defined by BMI.[126] It found increased risks of stillbirth with pregravid obesity and overweight status. This association between higher pregravid weight and stillbirth persisted after the investigators excluded women with obesity-related diseases (diabetes, preeclampsia, and other hypertensive disorders). Within this subset of women without obesity-related diseases (n = 39,187), the AOR for stillbirth related to an increased weight of 100 g per week was 0.94 (95% CI, 0.87–1.03).

Neonatal distress

Study characteristics A Swedish study examined the effects of gestational weight gain on fetal distress (equivalent to International Classification of Diseases [ICD] 9-codes 768.[2–4]; and ICD 10-codes P20.0, P20.1, and P20.9) using medical birth registry data from 245,526 singleton, term pregnancies over a 9-year period. (Evidence Table 25).[58] Women were grouped by BMI status into three gestational weight gain categories: < 8 kg (low), 8 to 16 kg, and >16 kg (high).

Overview of results The results of this fair study show that after adjusting for maternal age, parity, smoking in early pregnancy, and year of birth, the authors reported that fetal distress was not significantly associated with low weight gain despite the BMI of the mother. Overweight and morbidly obese women with excessive weight gain did have an increased risk for fetal distress.

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Detailed results Compared with women with gestational weight gain of 8–16 kg, the OR for fetal distress among women gaining 16 kg or more was 2.15 (95% CI, 1.10–4.20) for women with BMI ≥ 35 and 1.31 (95% CI, 1.05–1.53) for women with BMI 25–29.9.

Neonatal hypoglycemia

Study characteristics Two studies examined the effect of gestational weight gain on neonatal hypoglycemia (Evidence Table 26).[75,96] One was a retrospective cohort study of 20,465 women;[129] the other[110] was a retrospective case-control study using data from 45,245 singleton, live births from a U.S. prepaid group practice health plan. The studies categorized gestational weight gain differently; one examined gestational weight gain as a dichotomous variable based on extremes of weight gain (< 7 kg and >18 kg),[129] and the other used maternal rate of weight gain (total pregnancy weight gain minus infant birthweight divided by weeks of gestation when the last weight was measured) in kg per week.[110] Hypoglycemia was defined by ICD codes[129] or as at least one plasma glucose test result < 40 mg/dL.[110]

In the case-control study,[110] babies were identified as cases if they had the following complications: macrosomia (birthweight > 4,500 g), hypoglycemia (at least one plasma glucose < 40 mg/dL), or hyperbilirubinemia (at least one total serum bilirubin of 20 mg/dL or more). In general, hypoglycemic cases tended to be infants whose mothers were younger, nonwhite, and less educated than mothers of controls. More women with a prepregnancy BMI >29.0 appeared among the cases (22.9 percent) than the controls (17.6 percent).

Overview of results The results of these studies (1 good[110] and 1 fair[129]) suggest that gestational weight gain is associated with the risk of infant hypoglycemia.

Results In the case-control study (rated good quality),[110] after adjusting for age, race-ethnicity, parity, plasma screening value, and gestational age at last weight measured, the authors found that women who gained in the highest bracket of weight gain per week (more than 0.40 kg/week) had a increased risk of delivering an infant with hypoglycemia (AOR, 1.94; 95% CI, 1.33–2.82) than women gaining 0.22 to 0.31 kg per week.

Findings from the retrospective cohort study were similar.[129] After controlling for several confounders, the authors found that weight gain of more than 18 kg was associated with hypoglycemia (AOR, 1.67; 95% CI, 1.13–2.46) when compared with weight gain of 11.5 to 16.0 kg.

Hyperbilirubinemia

Study characteristics The retrospective case-control study described above also examined the effect of gestational weight gain on infant hyperbilirubinemia (Evidence Table 27).[110]

Overview of results One good study[110] suggested that increased gestational weight gain is associated with a higher risk of hyperbilirubinemia.

Detailed results Compared with controls, the hyperbilirubinemia case group had more Asians (20.1 percent vs. 8.1 percent) and tended to be born at a gestational age < 37 weeks. Compared with women gaining 0.22 to 0.31 kg per week, women who gained in the highest bracket of weight gain/week (more than 0.40 kg/week) had an increased risk of delivering an infant with hyperbilirubinemia (AOR, 1.94; 95% CI, 1.33–2.82).

Neonatal hospitalization

Study characteristics One study investigated the influence of gestational weight gain on perinatal outcomes, including hospitalization of infant (Evidence Table 28).[12] Using a hospital-

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based, retrospective cohort study design, the authors studied 633 women who delivered live, singleton babies in Japan between 24 and 42 weeks' gestation. Mean age of the women was 29.1 and most were nulliparas. Most of the women gained between 8.5 and 12.5 kg (mean, 10.5 kg) during their pregnancy. Gestational weight gain was collected from maternity records and was based on last weight taken at the hospital prior to delivery.

Overview of results One fair study suggested that infants of women who gained less than 8.5 kg during their pregnancy were 60 percent more likely to require hospitalization.[12]

Detailed results Overall, 13.3 percent had babies with complications requiring hospitalization, excluding admissions for phototherapy necessitated by neonatal jaundice. After adjusting for maternal age, parity, smoking, prepregnancy BMI, and gestational age, the authors did not find a significant relationship between gestational weight gain of less than 8.5 kg (AOR, 1.60; 95% CI, 0.88–2.88) or weight gain greater than 12.5 kg (AOR, 0.93; 95% CI, 0.46–1.88) and hospitalization of infant.

Other infant morbidity

Study characteristics Two studies addressed other neonatal morbidity in association with gestational weight gain (Evidence Table 29); one was the large cohort study noted above,[129] and the other used a case-control design.[130] Both studies relied on self-reported prepregnancy weights. Total weight gained during pregnancy was ascertained from prenatal records[129] and women's self-report.[130] The studies differed on how gestational weight gain was categorized: the cohort study categorized gestational weight gain according to both the IOM recommendations (i.e., the woman was below, within, or above the IOM thresholds) and by extremes of weight gain (< 7 kg, > 15 kg); the case-control study defined gestational weight gain as a continuous variable.

Overview of results One fair study reported that gestational weight gain less than 7 kg was associated with neonatal seizure.[129] Another fair study reported no significant association between infant leukemia and weight gain during pregnancy.[130]

Detailed results The cohort study looked at the relationship between gestational weight gain and several adverse neonatal outcomes (birth trauma, 5-minute Apgar score < 7, need for assisted ventilation, SGA, LGA, umbilical cord arterial pH < 7.1, umbilical cord arterial base excess < 10, admission to the neonatal intensive care unit [NICU], admission to the special-care nursery [a step-down unit], neonatal infection, seizure, hypoglycemia, polycythemia, jaundice, meconium aspiration syndrome, respiratory distress or tachypnea, anemia, birth asphyxia, and perinatal death).[129] The authors controlled for maternal age, race, parity, smoking, pregravid BMI, date of delivery, pregnancy-induced hypertension, mode of delivery, length of first stage of labor, length of second stage of labor, gestational age, and birthweight. Using weight gain of 11.5 to 16 kg as a reference, the authors reported that gestational weight gain less than 7 kg was associated with neonatal seizure (AOR, 10.66; 95% CI, 2.17–52.36). Gestational weight gain >18 kg was associated with assisted ventilation (AOR, 1.52; 95% CI, 1.16–2.00), seizure (AOR, 6.19; 95% CI, 1.32–28.96), polycythemia (AOR, 1.59; 95% CI, 1.13–2.22), and meconium aspiration syndrome (AOR, 1.86; 95% CI, 1.13–3.05).

The case-control study[130] examined the association between maternal reproductive history, including gestational weight gain, and the risk of infant leukemia in 240 cases, defined as infant leukemia diagnosed at < 1 year of age, and 255 controls matched to cases by year of birth. Infants with infant leukemia were significantly ($P < 0.003$) less likely to be white (79.5 percent vs. 85.5 percent) and more likely to be Hispanic (10.5 percent vs. 3.5 percent) than controls.

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After adjusting for sex, race or ethnicity, maternal education, and prepregnancy BMI, the authors found no significant association between infant leukemia and weight gain during pregnancy.

Infant BMI

Study characteristics Two older studies examined the influence of gestational weight gain on the offspring's BMI (Evidence Table 30). One cohort study comprised 8,719 singleton, live-born infants from a hospital in Montreal, Canada, from 1980 to 1986.[122] Of these mothers, 48 percent were primiparas, 90 percent were married, and 87 percent had started prenatal care in the first trimester. The infant's weight and length at birth was used to calculate BMI. Weight gain was expressed as total weight gain minus the weight of the infant at birth. The second study enrolled 119 term GDM and 143 term control mother-infant dyads from a hospital in Rhode Island in 1982.[131] The mothers were all screened for gestational diabetes using a universal screen approach between 24 and 28 weeks' gestation. Anthropometric measurements on the infants were done by study staff on the second day of life; weight and height was used to calculate infants' BMI. Total gestational weight gain was defined as measured weight at last prenatal visit (within one week of delivery) minus self-reported pregravid weight.

Results The Canadian study reported that net gestational weight gain was weakly but significantly correlated with infant's BMI ($r = .04$, $P < 0.01$).[122] In multivariate analysis, net gestational weight gain did not meet the criterion threshold for remaining in the stepwise regression.[122]

In the U.S. study, total gestational weight gain was significantly correlated with infant's BMI ($r = .22$, $P = 0.01$).[131] In multivariable regression analysis done separately for mothers with GDM and controls, total gestational weight gain significantly predicted infant's BMI such that a 1 kg increase in weight gain was associated with a 0.06 and 0.05 increase in BMI for GDM and control infants, respectively, after controlling for pregravid BMI and glucose values. The difference between the results of these two studies lies in the fact that once the weight of the infant is removed from total weight gain, an important product of conception is missing from the measure of weight gain and thus the strength of the association is reduced.[131]

Other infant growth characteristics

Study characteristics Six studies examined the association between gestational weight gain and various other infant growth characteristics (Evidence Table 31, Table 25).[31,56,57,62,82,89]

Overview of results The evidence from one good,[98] three fair,[14,56,57,82] and one poor study[68] suggest that gestational weight gain is associated with various measures of infant growth characteristics. A single fair study failed to find an association between gestational weight gain and infant proportionality.[122]

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TABLE 25. Gestational Weight Gain and Other Infant Growth Measures

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Brown et al., 2002[98] USA, primary care clinics 389 All weight/BMI Good	Pregravid weight: Measured by study investigators Total weight gain: Collected by study investigators	G1: Increase in ponderal index per 1 kg increase in first trimester weight gain G2: Increase in ponderal index per 1 kg increase in second trimester weight gain G3: Increase in Ponderal Index per 1 kg increase in third trimester weight gain	G1: β = 0.21 (*P* < 0.0003) G2: β = 0.05 PI (*P* < 0.4) G3: β = 0.12 (*P* < 0.03)	Maternal age, parity, pregravid BMI, height, infant sex, gestational age
Guihard-Costa et al., 2004[92] France, hospital database 13,972 All weights/BMI Fair	Pregravid weight: Routine prenatal care Total weight gain: Routine prenatal care or maternity records	G1: SC for effect of GWG on crown-heel length G2: SC for effect of GWG on head circumference G3: SC for effect of GWG on subscapular skinfold thickness SCs are regression coefficients calculated as if all of the independent variables had a variance of 1	G1: SC 0.142 G2: SC 0.120 G3: SC 0.146	Maternal age, parity, pregravid BMI, height
Kirchengast and Hartmann 2003[93] Austria, university hospital 8,011 All weights/BMI Fair	Pregravid weight: Estimated from measured weight at first prenatal visit Total weight gain: Routine prenatal care or maternity records	Change in infant size characteristics per 1 kg increase in GWG G1: Birth length (cm) G2: Head circumference (cm) G3: Acromial circumference (cm) G4: Diameter frontoccipitalis (cm)	G1: β = 0.55 (0.43–0.68) G2: β = 0.33 (0.23–0.42) G3: β = 0.47 (0.39–0.55) G4: β = 0.12 (0.07–0.18)	Maternal age, age at menarche, pregravid weight, height, distantia cristarum
Kramer et al., 1990[122] Canada, university	Pregravid weight: Self-report	Correlation coefficients between GWG and:	G1: -0.04 (*P* < 0.01)	Pregravid weight, infant sex, smoking, parity, maternal diabetes, height,

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
hospital 8,719 All weights/BMI Fair	Total weight gain: Routine prenatal care or maternity records	G1: Length G2: Head circumference G3: BMI G4: Ponderal Index G5: Weight/Head circumference Net gestational weight gain was associated with correlation coefficients of -0.04 for length, -0.01 for head circumference, 0.04 for BMI, 0.04 for Ponderal Index, and 0.01 for weight/head circumference. Results were significant ($P < 0.01$) for length, BMI, and Ponderal Index	G2: -0.01 G3: 0.04 ($P < 0.01$) G4: 0.04 ($P < 0.01$) G5: 0.01	previous LBW infant, severe pregnancy-induced hypertension
Shepard et al., 1996[117] Norway and Sweden, multicenter study 369 All weights/BMI Fair	Pregravid weight: Medical records Total weight gain: Measured at 3 study time periods	Increase in mean abdominal fetal growth rate (mm/day) per 5% increase in proportional weight gain in this period: G1: Weeks 17–25 G2: Weeks 25–33 G3: Weeks 33–37	G1: β = 0.35 (P = 0.49) G2: β = 0.88 (P = 0.02) G3: β = 1.53 (P = 0.02)	Maternal age, pregravid BMI, previous SGA, infant sex
Ekblad and Grenman, 1992[68] Finland, hospital 357 Prepregnancy weight 20% over or under ideal body weight for height and normal weight Poor	Pregravid weight: Medical records Total weight gain: Routine prenatal care or maternity records	Mean symphysis-fundus height: G1: Weight gain ≤ 5 kg G2: Weight gain 5–20 kg G3: Weight gain ≥ 20 kg	G1: 30.8 cm ± 4.0 G2: 32.8 cm ± 3.4 G3: 35.0 cm ± 3.9	N/A

β, unstandardized coefficient from multiple regression; BMI, body mass index; cm, centimeters; g, gram; GWG, gestational weight gain; kg, kilogram; SC, standardized coefficient; SGA, small-for-gestational-age.

Detailed results One good-quality study analyzed the relationship between weight gain (total and by trimester) and ponderal index (PI, a way of characterizing the relationship of height to

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mass for an individual).[98] Each kilogram of weight gained in the first and third trimesters significantly increased the PI: first trimester, an estimated 0.21 units; third trimester, by 0.12 units. Second trimester weight gain was not associated with newborn PI. The authors adjusted their models for gestational age, sex, parity, maternal height, maternal age, and pregravid BMI.

A retrospective cohort study (rated fair quality) conducted in France examined predictors of various infant growth measures,[92] using standardized coefficients (SC) from stepwise regression models. SCs are regression coefficients calculated as if all of the independent variables had a variance of 1. Pregnancy weight gain had a significant influence on birthweight (SC 0.199), crown-heel length (SC 0.142), head circumference (SC 0.120), and subscapular skinfold thickness (SC 0.146).

One fair-quality study examined proportional weight gain in relation to fetal growth rate in millimeters (mm) per day, calculated by averaging three ultrasound measurements of the sagital and transverse diameters of the fetal abdomen in three study time periods.[117] Increases in proportional weight gain during the second period (weeks 25 to 33) and third period (weeks 33 to 37), but not the first period (weeks 17 to 25) were significantly associated with significant increases in fetal growth. These results were adjusted for age, BMI, smoking, history of delivering an SGA infant, and infant sex.

Infant body proportionality was studied in a Canadian population (rated fair quality) with validated gestational ages.[122] Proportionality was evaluated using z transformations of crown-heel length, head circumference, BMI, PI, and birthweight/head circumference. Net gestational weight gain was associated with correlation coefficients of -0.04 for length, 0.04 for BMI, and 0.04 for PI (all $P < 0.01$). ORs of low and high PI for each 5 kg decrease in net gestational weight gain were not significant.

A fair-quality study conducted in Austria found that for each 1 kg increase in total gestational weight gain, birth length increased by 0.55 cm (95% CI, 0.43–0.68), head circumference increased by 0.33 cm (95% CI, 0.23–0.42), acromial circumference increased by 0.47 cm (95% CI, 0.39–0.55), and diameter frontoccipitalis increased by 0.12 cm (95% CI, 0.07–0.18).93 This study adjusted for maternal age, age at menarche, pregravid weight, height, and distantia cristarum.

Finally, a poor-quality retrospective cohort study conducted in Finland examined the relationship between weight gain and symphysis-fundus (SF) height.[68] SF height did not differ significantly between weight gain groups at 24 weeks, but higher gestational weight gains were associated with longer SF height.

Child Outcomes

Childhood weight status

Study characteristics Four studies, using different definitions of outcomes, examined the long-term effect of gestational weight gain on children's weight status (Evidence Table 32).[99–102] Three studies enrolled the subjects at birth and then followed them through various end points; up to 15 months postpartum,[132] 3 years of age,[24] and 2 and 5 years for the Avon longitudinal study of pregnancy and childhood (ALSPAC) in England.[133] All three included only singleton births. One was conducted using a national representative sample from 1979 that followed the children of mothers who were born in 1984, 1986, 1988, and 1990 for up to 12 years.[134]

Overview of results Due to the different definitions of the outcomes, the results from three fair[100–102] and one poor[99,102] studies are mixed for an association between gestational weight gain and childhood weight status.

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Detailed results In the ALSPAC study (rated fair), which used as its outcome "catch up growth" from birth to 2 years of age (for definition see Table 26), bivariate analysis suggested that children who showed catch-up growth were no different in the amount of weight that their mothers gained during pregnancy than children who showed no change or those who had catch-down growth. No adjustments were made for confounding.

In another fair study that reported on the effect of total weight gain and net weight gain (excluding infant birthweight),[24] child BMI percentiles at age 3 were grouped as follows: below 50th (referent category), 50th to 84th, 85th to 94th, and 95th or higher. Gestational weight gain was associated with a BMI of ≥ 95 percentile in both bivariate and multivariate analysis; a 5 kg increase in weight gain was associated with a 52 percent increase in risk of obesity in the offspring. Gestational weight gain was also associated with BMI z score. Similar associations were found when using net weight gain as the exposure.

TABLE 26. Gestational weight gain and childhood weight status

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Li et al., 2007[134] USA, National Longitudinal Survey of youth 1979 Child and Young Adult file 1,739 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Self-report	Maternal weight gain categories (kg): G1: < 15 lbs G2: 15–24 lbs G3: 25–34 lbs G4: 35–44 lbs G5: > 45 lbs	AOR (95% CI) for early onset overweight (early onset of overweight that persisted throughout childhood) compared with normal (low probability of overweight throughout childhood and was characterized as the never overweight class) G5: 1.7 (1.0–2.9) G3: 1.0 (reference) Other AOR for weight gain categories for early onset overweight not significant compared with weight gain 25–34 lbs No association between maternal weight gain and risk of late onset overweight (moderately high probability of overweight at age 2 years, low probability of overweight at age 4 and 6 years, but growing probability of overweight after age 8 years)	Infant sex, race, birth order, gestational age, birthweight, breastfeeding, pregravid BMI, maternal age, maternal education, family income
Oken et al., 2007[24] USA, HMO	Pregravid weight: Self-report	Maternal weight gain, 5 kg	AOR (95%CI) BMI≥95th percentile vs BMI<50th percentile associated with a 5 kg increase in gestational weight gain:	Smoking, race, household income, marital status, glucose tolerance, gestation length,

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
1,044 All weight/BMI Fair	Total weight gain: Measured	increments	1.52 (1.19–1.94) Child BMI z-score at age 3 years for AOR listed above (95% CI): 0.11 (0.05, 0.17)	breastfeeding duration, child's sex
Ong et al., 2000[133] UK, Avon longitudinal study of pregnancy and childhood 848 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Obstetric records	Maternal weight gain, continuous measure	Children were grouped into three growth categories (catch-up, no change, and catch-down) based on a gain in weight (SD score > 0.67 for catch-up; SD score < 0.67 for catch-down. Maternal weight gain was not a significant predictor of catch-up growth between 0 and 2 years	NA
Sowan et al., 2000[132] USA, NIH-funded Infant Growth Study 630 All weight/BMI Poor	Pregravid weight: Self-report Total weight gain: Self-report	Maternal weight gain, 5 lb. increments	AOR (95%CI) for infant obesity (BMI> gender and age specific 84th percentile based on Infant Growth Study population norms) at 1, 4, 7, and 10 months: NS AOR (95%CI) for obesity at 14 months: .8 (0.7–1.0)	Maternal age, nonpregnant weight, smoking, marital status, father living in home, family stress, grandmother living in home, socioeconomic status, gender, race, infant BMI at birth, infant BMI from previous study month

AOR, adjusted odds ratio; BMI, body mass index; CI, confidence interval; G, group; HMO, health maintenance organization; kg, kilogram; lbs, pounds; USA, United States of America; vs, versus.

The one poor study that examined BMI ≥ 85th percentile at ages less than 14 months did not find any association with gestational weight gain.[132] However, the nationally representative study did find an association for early onset of overweight associated with weight gains ≥ 20.43 kg (≥ 45 lbs) but not later on in life.[134]

The U.S. study (rated poor quality) determined, using multivariable logistic regression models, that gestational weight gain was a significant predictor of infant obesity at 1 and 14 months of age.[132] The odds of obesity rose 10 percent at 1 month for every 5-pound increase in weight gain adjusting for parental and household variables, sex of the infant, and ethnicity (OR, 1.1; 95% CI, 1.0–1.2). At 14 months the association was reversed; the odds of obesity was

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decreased by 20 percent for every 5-pound increase in gestational weight gain (OR, 0.8; 95% CI, 0.7–1.0), adjusting for several variables include birth BMI and BMI from the previous study month.

Childhood hospitalization

Study characteristics One study, a cohort of children (N = 11,980) born to mothers attending midwifery centers in Denmark from April 1984 to 1987, examined the effect of maternal prenatal lifestyle factors on children's hospitalizations with infectious diseases (Evidence Table 33).[135] After excluding stillbirths, multiple births, and children with congenital malformations, the authors followed 10,440 newborns from 6 months to 12 years. Information on prenatal factors was self-reported by the mother via a questionnaire. Weight gain, calculated as the difference between the self-reported pregravid weight and the weight measured at the time of delivery obtained from the medical records, was categorized as < 10, 10 to 12, 13 to 15, and ≥ 16 kg. Outcome data on hospitalizations related to infections were obtained from registry information based on ICD codes.

Overview of results One fair study suggested that weight gain > 13 kg only for women who were underweight before pregnancy (BMI < 18) was associated with an increased risk of childhood hospitalization for infectious diseases.[135]

Detailed results The crude incidence rate ratios (IRR) for the effect of weight gain on hospitalizations were nonsignificant compared with weight gains of 13 to 15 kg: < 10 kg, 0.99; 10 to 12 kg, 0.93; and > 16 kg, 1.01). When maternal pregravid weight status was stratified as BMI < 18 and BMI ≥ 18, weight gain greater than 13 kg among women with a pregravid BMI < 18 increased the risk of hospitalizations compared with women with higher BMI and gaining similar weight (IRR, 1.42; 95% CI, 1.09–1.86). This model adjusted for maternal and paternal age, social group, marital status, number of siblings, and maternal smoking during pregnancy.

Short- and Long-term Maternal Outcomes

Lactation We found no evidence on the effect of gestational weight gain (not defined by IOM definitions) on lactation that accounted for pregravid weight. We present results for studies relying on IOM definitions of weight gain under KQ 3.

Postpartum weight retention

Study characteristics Twelve articles from 10 study populations examine the relationship between gestational weight gain and postpartum weight retention (Evidence Table 34, Table 27).[105,136–146] Six articles used data collected within 1-year postpartum;[105,140–143,145] four used long-term follow-up data of greater than 1 year postpartum;[136,142,144,146] and three used interpregnancy interval data.[137–139]

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TABLE 27. Gestational weight gain and postpartum weight retention

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Callaway et al., 2007[146] Australia, University Hospital 3,572 All weight/BMI Good	Pregravid weight: Self report Total weight gain: Obstetric records/maternal questionnaires	G1: Gestational weight gain ≤ 15 kg G2: Gestational weight gain >15 kg	G1: Mean change (95% CI) in BMI at 21 years postpartum: 5.06 kg/m^2 (4.85–5.27) G2: Mean change (95% CI) in BMI at 21 years postpartum: 6.40 kg/m^2 (6.19–6.61) $P < 0.001$ G2 was associated with a mean change in BMI over 21 years of 0.19 kg/m^2 (95%CI: 0.16–0.22)	Baseline income, secondary school completion, ethnicity, maternal age at birth, parity, birthweight, gestational age, infant sex, maternal smoking during pregnancy, smoking at 21 years, sedentary lifestyle at 21 years, baseline maternal BMI, hypertensive disorders during pregnancy
Harris et al., 1999[144] UK, Antenatal Care Project 74 All weight/BMI Good	Pregravid weight: Measured at first trimester prenatal visit Total weight gain: Self report	Continuous maternal weight gain, kg	ANCOVA model with weight (kg) at 2.5 years postpartum as dependent variable and maternal weight gain (kg) as independent variable: B = -0.031 β = -0.029 SEM = 0.120 $P = 0.796$	Marital status, increased dissatisfaction with body, increased access to food, increased energy intake, decreased activity, smoking status, maternal age, duration of followup, pregravid BMI, parity, gestational age at booking, parental obesity, social support
Harris et al., 1997[137] UK, Hospital 523 All weight/BMI Fair	Pregravid weight: Measured within 13 weeks' gestation Total weight gain: Measured	Gestational weight gain during previous pregnancy (kg), continuous	ANCOVA model for interpregnancy weight change (kg), defined as the difference between weight at start of index pregnancy and weight at start of previous pregnancy: B = 0.262 β = 0.227 SEM = 0.52 $P < 0.001$	Marital status, smoking status, alcohol, parity, age, socioeconomic status, nulliparous BMI, birthweight, gestational age at start of previous pregnancy, gestational age at start of index pregnancy, gestational age at start of first pregnancy, interpregnancy interval, gestational age at delivery
Harris et al., 1997[138] UK, Hospital 243 All weight/BMI	Pregravid weight: Measured within 13 weeks' gestation Total weight gain: Measured	Gestational weight gain during first pregnancy as a continuous measure (kg)	ANCOVA model for interpregnancy weight change (kg), defined as the difference between weight at start of first pregnancy and weight at start of the second pregnancy: B = 0.176 β = 0.169	Marital status, lactation, smoking status, alcohol, height, nulliparous BMI, birthweight, gestational age at start of previous pregnancy, terminations between pregnancy, interpregnancy interval

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Fair			SEM = 0.070 *P* < 0.013	
Hunt et al., 1995[139] USA, population-based family history database (Utah) and participants of an obesity study 221 All weight/BMI Morbidly obese Fair	Pregravid weight: Self-report (validated by hospital records if available) Total weight gain: Self-report (validated by hospital records if available)	G1: Population-based sample G2: Morbidly obese women who were normal weight at age 20–24 years or prior to first pregnancy	Regression of current weight on total number of pregnancies showed a 1.3 kg/pregnancy increase in current weight (*P* = 0.03) with no difference between G1 and G2 (*P* = 0.60) Gestational weight gain was significantly greater in G2 than G1 for the first pregnancy only (*P* < 0.05) G2 had a net weight retention after the first pregnancy of 4.0 kg greater than G1 at 6 weeks postpartum G2 averaged 1.6 kg/pregnancy greater weight retention than G1 for additional pregnancies	Weight at ages 20 to 24, current age
Linne et al., 2004[142] Sweden, Stockholm Pregnancy and Weight Development Study 563 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Medical Records	Gestational weight gain as a continuous variable (kg): G1: Pregravid BMI ≤ 25 G2: Pregravid BMI > 25	G2 had significantly greater weights at prepregnancy, delivery, 1 year postpartum, and 15 years postpartum compared to G1 (*P* < 0.001); however, G2 did not have a higher risk of postpartum retention than G1	Alcohol use, smoking, number of pregnancies since index child, employment area
Linne et al., 2003[136] Sweden, Stockholm Pregnancy and Women's Nutrition Study 563 Normal weight/overweight	Pregravid weight: Self-report Total weight gain: Medical records	G1: Women with normal BMI (20–25) at prepregnancy and 15 years postpartum G2: Women with normal BMI at prepregnancy who had	G1: Mean (SD) maternal weight gain, 13.6 (3.7) kg G2: Mean (SD) maternal weight gain, 15.4 (4.4) kg t-Test: *P*<0.001	None

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Fair		overweight BMI (> 25) at 15 years postpartum		
Muscati et al., 1996[105] Canada, Prenatal Nutrition Counseling Program 371 All weight/BMI Fair	Pregravid weight: Physicians' records Total weight gain: Measured	G1: Weight gain ≤ week 20 (kg) G2: Weight gain weeks 21–30 (kg) G3: Weight gain weeks 31- term G4: Total weight gain ≤ 12 kg G5: Total weight gain > 12 kg	Regression model of weight retention (kg) at 6 weeks postpartum as the dependent variable and G1–G5 as independent variables: G1: β = 0.86 (SE: 0.05) *P*< 0.001 G2: β = 0.68 (SE: 0.07) *P*< 0.001 G3: β = 0.49 (SE: 0.07) *P* < 0.001 G4: β = 0.58 (0.13) *P*=NR G5: β = 0.77 (0.04) *P*=NR Among women with AGA infants, women with 6 week postpartum weights greater than the median value (6.2kg, underweight; 5.7kg, normal weight; 3.1kg, overweight) had significantly greater total weight gains and weight gains during the first 20 weeks' gestation compared to women with 6 week postpartum weights of the median value or lower	Standard weight for height (based on 1983 Metropolitan Life Insurance Tables), pregravid weight above standard (difference between actual weight and standard weight), parity, gestational age, infant sex
Ohlin et al., 1990[145] Sweden, maternity clinics 1423 All weight/BMI	Pregravid weight: Self-report Total weight gain: Maternity records	Gestational weight gain as a continuous variable (kg)	Regression model for weight change (kg), defined as the difference between prepregnancy and 1 year postpartum weights: B = 0.32 *P*< 0.001	Lactation score, age, prepregnancy BMI, parity

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Fair				
Soltani et al., 2000[143] UK, Hospital 77 All weight/BMI Fair	Pregravid weight: Measured at 13 weeks' gestation Total weight gain: Measured	Pregravid weight G1: Normal weight G2: Underweight G3: Overweight G4: Obese	G1: Patterns of changes in body weight (kg) and fat mass follow a monotonous trend; body weight and fatness increased during gestation, decreased substantially at 6 weeks postpartum, and then stayed the same or slightly decreased until 6 months postpartum G2: Showed similar pattern to G1. G3: Divergent pattern of weight gains and losses; body fat mass changes show a very scattered pattern G4: Divergent pattern of both weight and fat mass gains and losses; heavier and greater fat masses at 6 months postpartum compared to 13 weeks gestation; significantly lower fat mass loss and greater skinfold thickness gain between 36 weeks gestation and 6 months postpartum compared to normal weight women ($P < 0.05$)	None
Walker et al., 2004[141] USA, Austin New Mothers Study 382 All weight/BMI Fair	Pregravid weight: Self report Total weight gain: Self report	Continuous gestational weight gain (kg)	Each kg of gestational weight gain was associated with 0.314 kg/m^2 of postpartum BMI ($P < 0.001$)	Ethnicity, time, interaction of ethnicity and time, pregravid BMI, weight-related distress, energy intake
Parham et al., 1990[140]	Pregravid weight: Self report	Gestational weight gain for	Change in BMI category between prepregnancy and 1–	None

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
USA, prenatal clinics serving low income women 158 All weight/BMI Poor	Total weight gain: Measured	population in tertiles, mean (se): G1: 3.7 (2.9) G2: 9.4 (1.3) G3: 16.0 (3.7)	3months postpartum: G1, G2: 83% No change; 7% Desirable change (i.e. underweight women becoming normal weight); 10% Undesirable change (~5% had an increase in BMI category and ~5% had a decrease in BMI category) G3: 42% no change; 19% desirable change; 39% undesirable change (all increases in BMI category)	

AGA, average gestational age; ANCOVA, analyses of covariances; β, unstandardized regression coefficient; B, standardized regression coefficient; kg, kilogram; SD, standard deviation; SE, standard error; SEM, standard error of the mean; UK, United Kingdom.

Overview of results The results of the two good[144,147] and eight[105,136–143,145] fair studies reviewed in this section suggest that gestational weight gain is positively associated with weight retention within 1 year postpartum[105,141,145] and with interpregnancy weight gains.[137–139] There is evidence to suggest that pattern of weight gain influences weight retention; a higher percentage of weight gained within the first 20 weeks of gestation is retained at 6 weeks postpartum compared to weight gains later in pregnancy.[105] Additionally, weight retention differs across pregravid BMI strata,[138,143] with overweight and obese women retaining more weight compared to normal weight women. Postpartum weight retention seems to be especially problematic for obese women, who may be at risk for increases in fat mass and central adiposity in the postpartum period.[143] In the long term, the effect of gestational weight gain on weight retention is less conclusive; two studies[144,146] found little to no association between gestational weight gain and weight at 2.5 and 21 years after the index pregnancy and one study[136] found that women who became overweight at 15 years follow-up had higher gestational weight gains compared to women who remained normal weight.

Results for less than 1-year postpartum Three cohort studies, two rated[105,143] and the other rated poor,[140] examined the association between weight gain and weight retention prior to 1-year postpartum.

One study used a population of low-income white women to examine the influence of total gestational weight gain and partial weight gains, categorized as weight gain ≤ 20 weeks, 21–30 weeks, and 31 weeks to term, on postpartum weight retention at 6 weeks.[105] Each kilogram of gestational weight gain at ≤ 20 weeks, 21–30 weeks, and 31 weeks to term was significantly ($P < 0.001$) associated with an increase of 0.86 (±0.05), 0.68 (±0.07), and 0.49 (±0.07) kg at 6 weeks postpartum, respectively. Pregravid weight status, defined as underweight, normal weight, and overweight, was based on 1983 Metropolitan Life Insurance Table weight-for-height values. The

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mean gestational weight gains for women with < median postpartum weight retention (median values of postpartum weight retention were 5.7 kg for underweight, 6.2 kg for normal weight, and 3.1 kg for overweight women) were 13.3, 13.2, and 9.6 kg for underweight, normal weight, and overweight women, respectively. In contrast, the mean weight gains for women ≥ median postpartum weight retention were 19.6, 20.2, and 19.1 kg, respectively ($P < 0.001$). Similar significant differences were seen for mean partial weight gains between women with postpartum weight retention < median and ≥ median values ($P < 0.05$- $P < 0.001$), with the greatest weight gain differences seen within 20 weeks of gestation. Gestational weight gain of 12 kg was associated with 2.5 kg of postpartum weight retention; regression analyses for weight gains of ≤12 kg and >12 kg were associated with 0.58 (SE: 0.13) and 0.77 (SE: 0.04) kg of postpartum weight retention per kg of weight gain, respectively.

Another study measured body weight, body fat mass (kg), and skinfold thickness (sum of five skinfold thicknesses) from 13 weeks of gestation through 6 months postpartum.[143] BMI categories at 13 weeks' gestation were defined using the IOM BMI classifications. Patterns in changes of body weight and fat mass across the study period were described for each BMI category. Among normal-weight women, the patterns of changes in both body weight and fat mass follow a monotonic trend; body weight and fatness increased during gestation, decreased substantially at 6 weeks postpartum, and then stayed the same or slightly decreased until 6 months postpartum. Overweight women show a divergent pattern of weight gains and losses; women with the highest weight gains and losses at 6 months postpartum were in this group. Body fat mass changes showed a very scattered pattern. Obese women also show a divergent pattern of both weight and fat mass gains and losses; however, the majority of obese women are heavier and have greater fat masses at 6 months postpartum compared to 13 weeks' gestation. Compared with normal-weight women, obese women have significant ($P < 0.05$) increases in total skinfold thickness between 36 weeks' gestation and 6 months postpartum and in waist to hip ratio between 6 weeks' and 6 months postpartum.

Results from the poor study were consistent[140] among the women within the upper tertile for gestational weight gains (mean 16.0 ± 3.7 kg), approximately 39 percent had an increase in BMI category at 1 to 3 months postpartum compared to only 5 percent among women within the lower and middle tertiles for gestational weight gains (mean 3.7 ± 2.9 kg and 9.4 ± 1.3 kg, respectively).

Postpartum weight retention at 1 year Three publications (2 studies), all rated fair quality, measured weight retention at 1 year postpartum.[141,142,145] One study using data from a low income, racially/ethnically diverse population reported that a 1 kg increase in gestational weight gain was associated with an increase of 0.314 kg/m2 in BMI at 1 year postpartum.[141] Two articles based on data from the Stockholm Pregnancy and Weight Development Study examined the association between gestational weight gain and weight retention at 1 year postpartum.[142,145] In one article, a 1 kg increase in total gestational weight gain was associated with a 0.32 kg increase in weight at 1 year postpartum ($P < 0.001$), which explained 12.7 percent of the variation in the change in weight from prepregnancy to 1 year postpartum ($P < 0.001$).[145] The other article examined body weight at prepregnancy, delivery, 1 year followup, and 15 years followup in women with normal (BMI 20–25) and overweight (BMI > 25) pregravid BMI.[142] Women who were overweight before pregnancy were significantly heavier at each time point ($P < 0.001$); however, there were no significant differences between normal-weight women and overweight women in the amount of weight retained from prepregnancy to 6 months and 1 year postpartum.

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Postpartum weight retention in the medium term One good-quality study[144] found no association between gestational weight gain and weight retention at two and half years postpartum in a small cohort of women with low antenatal risks enrolled in the Antenatal Care Project (United Kingdom).

Long-term postpartum weight retention Three publications (2 studies) measured long-term weight retention. One good-quality study in a cohort of Australian women examined the association between gestational weight gain, dichotomized as ≤ 15 kg and > 15 kg, and weight retention at 21 years after the index pregnancy.[146] Excessive weight gain during pregnancy (> 15 kg) was associated with a mean change in BMI of 0.19 kg/m2 (95% CI, 0.16–0.22).

Two articles, both rated fair, from the Stockholm Pregnancy and Weight Development Study examined the effects of gestational weight gain on weight retention at 15 years postpartum.[136,142] At 15 years follow-up, women who had been overweight (BMI > 25) before pregnancy were heavier than women who had been of normal weight (BMI 20–25) before pregnancy.[142] The difference in the weight increases from prepregnancy to 15 years follow-up between overweight and normal-weight women were not significant (7.7 ± 7.0 kg and 6.2 ± 12.1 kg, respectively; P = 0.36).[142] Among women with normal pregravid weight, those who remained at a normal weight at 15 years follow-up had significantly lower gestational weight gains than women who were overweight at 15 years follow-up (13.6 ± 3.7 kg and 15.4 ± 4.4 kg, respectively; P < 0.001).[136]

Interpregnancy weight retention Three studies, all rated fair quality, examined the association between gestational weight gain and interpregnancy weight retention.[137–139] Two cohort studies used data collected from women attending a city hospital in England..[137,138] In one, gestational weight gain during a previous pregnancy was associated with a 0.262 kg increase (standard error of the mean [SEM], 0.052; P < 0.001) in weight between the index pregnancy and the previous pregnancy.[137] In the other, gestational weight gain was associated with a 0.176 kg increase (SEM, 0.074; P = 0.001) in weight from the beginning of the index pregnancy to the beginning of the second pregnancy.[138] Prepregnancy BMI and interpregnancy weight gain were independently associated, suggesting that women who had gained the most weight between pregnancies were more likely to have been overweight before their first pregnancy than women who gained less between pregnancies.

A cross-sectional study examined the effect of weight gain (self-reported) from multiple pregnancies on the development of morbid obesity in a group of morbidly obese women, who were not morbidly obese prior to their first pregnancy, and population-based controls.[139] The mean gestational weight gain and net weight retention for all pregnancies was 14.2 kg and 5.7 kg, respectively, for women who became morbidly obese, and 12.5 kg and 3.4 kg, respectively, for the controls. Women who became morbidly obese gained significantly more weight during their first pregnancy than controls (16.4 kg vs. 12.6 kg, respectively; P < 0.05), and they retained significantly more weight after their first and second pregnancies than controls (7.1 kg and 5.9 kg vs. 3.1 kg and 2.9 kg, respectively; P < 0.05). After adjusting for pregravid weight at ages 20 to 24 years, the authors determined that each pregnancy was associated with a 1.3 kg increase in current weight (P = 0.03), with no significant difference between the slopes of women who became obese and controls (1.6 kg/pregnancy and 1.0 kg/pregnancy, respectively; P = 0.6).

Premenopausal breast cancer

Study characteristics One study examined the effect of pregnancy weight gain on a woman's risk of developing premenopausal breast cancer (Evidence Table 35).[148] The study was a nested case-control study within a cohort of 22,610 Finnish women with a mean age of 40 during 1990

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and 1993. Women self-reported their breast cancer status, and their current weight, highest nonpregnancy weight, weight at age 20, and weight gain during any pregnancy in one of four categories (< 10, 10–15, 16–20, and > 20 kg). A total of 114 women had identified themselves as having premenopausal breast cancer; of these, 98 women had provided information on year of birth, had been pregnant, and had their cancer diagnosed after a pregnancy. Four controls for each case from the cohort were selected matched by age and type of intrauterine device.

Overview of results The fair study suggested that gestational weight gain and premenopausal breast cancer are not associated.[148]

Detailed results ORs for breast cancer by gestational weight gain category were close to null and nonsignificant in both crude and adjusted models (age, education, family history of breast cancer, and change in BMI) using the < 10 kg category as the reference: ORs were 0.8 (0.44, 1.47), 1.0 (0.47, 2.04), and 0.8 (0.27, 2.13) for weight categories 10–15 kg, 16–20 kg, and > 20 kg, respectively.

KQ 3: Outcomes of Weight Gain Within or Outside IOM Recommendations

Although the KQ 3 issues are similar to those addressed in KQ 1, the focus here is on analyses that directly apply the categories of weight gain during pregnancy that the IOM laid out in its 1990 document.[1] The recommendations specific to BMI weight status groups and certain sociodemographic or physical characteristics are as follows:

- 28 to 40 pounds for women with low BMI (<19.8);
- 25 to 35 pounds for women with normal BMI (19.8–26);
- 15 to 25 pounds for women high BMI (> 26.0–29.0);
- weight gain of at least 15 pounds for obese women (BMI > 29);
- weight gain in the upper end of the recommended range for adolescents and black women; and
- weight gain in the lower end of the recommended range for short women (< 157 centimeters, or approximately 62 inches).

We present KQ 3 results similar to the presentation for KQ 1. We examine, first, maternal antenatal outcomes and then intrapartum outcomes; we then consider birth outcomes, infant outcomes, and child outcomes; and, finally, we cover maternal short- and long-term outcomes. When we have three or more studies dealing with the same topic (i.e., outcome), we present information in summary tables; otherwise, detailed information on these articles will be found in the relevant evidence tables in Appendix E.† For all outcomes, we first describe the studies (main study characteristics only); we then provide an overview of the results (for topics with more than one study), followed by a more detailed discussion of relevant studies.

We rated studies for quality as good, fair, or poor (as explained in Chapter 2). All studies are reported in summary tables (including quality grades), and they are presented in order by quality rating. The text focuses on studies of good and fair quality, in that order; the vast majority are fair quality, so studies for which no quality grade is specified can be assumed to be of fair quality. We only briefly summarize poor studies. Generally, if studies deal with more than one outcome, we describe the study once and refer back as needed.

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Maternal Antepartum Outcomes

Gestational diabetes mellitus

Study characteristics Four studies examined the relationship between weight gain according to the IOM guidelines and GDM (Evidence Table 36, Table 28).[3,53–55] Two studies were done specifically among obese women;[54,55] two included women of normal weight;[53,55] and one included women of various pregravid weight categories.[3]

TABLE 28. Weight change relative to IOM thresholds and gestational diabetes mellitus

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Saldana et al., 2006[3] USA, hospital 952 All weight/BMI Good	Pregravid weight: Self-report Total weight gain: Measured	G1: Normal glucose tolerance G2: Impaired glucose tolerance G3: Gestational diabetes mellitus BMI IOM	Mean (SE) weight gain ratio (defined as obeserved weight gain/IOM recommended weight gain): G1: 1.43 (0.04) G2: 1.48 (0.21) G3: 1.88 (0.15) $P < 0.05$ AOR (95% CI) for weight gain ratio: G1: 1.0 (reference) G3: 1.2 (0.9–1.4)	Race, age, gestational age
Edwards et al., 1996[55] USA, Hospital 1,443 Normal/obese weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Measured	Obese BMI > 29 (kg): G1: Lost weight/no change G2: 0.5–6.5 G3: 7–11.5 G4: 12–16 G5: > 16 Normal weight BMI 19.8–26 G6: < 11.5 G7: 11.5–16 G8: > 16	Gestational diabetes, % G1: 13.3 G2: 24.3 G3: 11.9 G4: 16.7 G5: 17.3 *P* for linear trend (G1–G5) = 0.554 G6: 2.3 G7: 3.3 G8: 2.9 *P* for linear trend (G6–G8) = 0.759	None
Thorsdottir 2002[53] Iceland, hospital records 614 Normal weight/BMI 19.5–25.5	Pregravid weight: Self-report Total weight gain: Maternity records	Maternal weight gain categories (kg): G1: < 11.5 G2: 11.5–16.0 G3: 16.1–20.0 G4: > 20.0	Gestational diabetes, % G1: 2.9 G2: 0 G3: 0 G4: 0 *P* for trend < 0.015	None

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Fair				
Bianco et al., 1998[54] USA, medical center 11,926 Nonobese (BMI 19–27) and morbidly obese (BMI > 35) Poor	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories among morbidly obese (BMI > 35): G1: Weight loss/no change G2: 1–15 lbs G3: 16–25 lbs G4: 26–35 lbs G5: > 35 lbs	Distribution of GDM, %: G1: 15.7 G2: 15.0 G3: 14.4 G4: 13.4 G5: 12.5 *P* = NS	Race, parity, clinic service, substance abuse, preexisting medical condition

AOR, adjusted odds ratio; BMI, body mass index; CI, confidence interval; g, gram; GDM, gestational diabetes mellitus; IOM, Institute of Medicine; kg, kilogram; NS, not sufficient; SE, standard error; USA, United States of America.

Overview of results No definitive evidence from four studies (1 good,[3] 2 fair,[53,55] 1 poor[54]) exists of an association between high weight gain and risk of developing GDM because of methodological problems with most studies addressing this topic.

Detailed results Obese women, independent of weight gain, had increased risks of developing GDM in three studies (1 of good quality,[3] 1 of poor quality[54]). Overweight women in the one good study that included them also had an increased risk for GDM.[3] All studies used weight gain at the time of delivery, which included the weight gained after the diagnosis of GDM. This measure of weight gain is biased since, once the diagnosis of GDM is made, weight gain is closely monitored and controlled through treatment.

One good study evaluated weight gain up to the time of GDM diagnosis in both white and black women.[3] The authors calculated the ratio of weight gain that expressed the amount of weight a woman gained to the amount she was expected to gain according to the IOM guidelines until the time of diabetes testing (that is, accounting for gestational length). Women who developed GDM had higher weight gain ratios than did women with normal glucose tolerance. In multivariable analysis, weight gain ratio was not significantly associated with developing GDM. However, among overweight women, a higher weight gain ratio was predictive of impaired glucose tolerance and this effect was stronger for white women (data not shown in table).

Two studies (1 poor-quality[54]) examined total weight gain and GDM risk in obese women; neither found any association (using bivariate analyses) with weight gains either above or below the IOM guidelines.[54,55]

Two studies reported findings for women of normal weight.[53,55] One had too few women who developed GDM across the weight gain groups to permit analyses,[53] and the other found no association.[55]

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Hypertension

Study characteristics One poor study compared the effect of total weight gain on the risk of developing pregnancy-induced hypertension among morbidly obese women and nonobese women using data from Mount Sinai Medical Center from 1988 to 1995 (Evidence Table 37).[54]

Results In bivariate analysis, this study found no association between weight gains below or above the IOM guidelines and pregnancy-induced hypertension.

Preeclampsia

Study characteristics The association between gestational weight gain and preeclampsia was examined in four articles (2 from the same database) of fair quality (Evidence Table 38, Table 29).[4,25,53,55] Two studies included obese women;[4,55] three included women of normal weight.[25,53,55]

TABLE 29. Weight change relative to IOM thresholds and preeclampsia

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Devader et al., 2007[25] USA, birth certificate data 94,696 Normal weight/BMI 19.8–26 Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories (lbs): G1: < 25 G2: 25–35 G3: > 35	AOR (95% CI) for preeclampsia: G1: 0.56 (0.49–0.64) G2: 1.00 (reference) G3: 1.88 (1.74–2.04)	Age, race, education, income, alcohol use, height, prior pregnancy, inadequate prenatal care use, smoking, child's gender, birth year
Edwards et al., 1996[55] USA, Hospital 1,443 Normal/Obese weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Measured	Obese BMI > 29 (kg): G1: Lost weight/no change G2: 0.5–6.5 G3: 7–11.5 G4: 12–16 G5: > 16 Normal weight BMI 19.8–26: G6: < 11.5 G7: 11.5–16.0 G8: > 16.0	Preeclampsia G1: 10.7 G2: 7.7 G3: 8.3 G4: 7.9 G5: 16.5 *P* for linear trend (for G1–G5) = 0.076 G6: 2.8 G7: 2.9 G8: 6.6 *P* for linear trend (for G6–G8) = 0.048	None
Kiel et al., 2007[4]	Pregravid weight: Self-report	Maternal weight gain categories stratified by prepregnancy obesity status,	For Obese Class I: OR (95% CI) for preeclampsia were significantly lower (< 1.00,	Age, race, parity, education, poverty (enrollment in Medicaid,

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
USA, Hospital 120,170 Obese BMI Fair	Total weight gain: Medical record	Obese Class I (BMI 30–34.9), Obese Class II (BMI 35–39.9), Obese Class III (BMI ≥ 40): G1: ≤ -10lbs G2: -2 to -9 lbs G3: No change G4: 2–9 lbs G5: 10–14 lbs G6: 15–25 lbs G7: 26–35 lbs G8: > 35 lbs	G6 was reference) for G2–G5 and significantly higher for G7–G8. For Obese Class II: OR (95% CI) for preeclampsia were significantly greater (> 1.00, G6 was reference) for G1 and G3– G5 and significantly lower for G8. For Obese Class III: OR (95% CI) for preeclampsia were significantly greater (> 1.00, G6 was reference) for G1–G3 and G5 and significantly lower for G7–G8	WIC, food stamp programs), tobacco use, chronic hypertension
Thorsdottir et al., 2002[53] Iceland, hospital records 614 Normal weight/BMI 19.5–25.5 Fair	Pregravid weight: Self-report Total weight gain: Maternity record	Maternal weight gain categories (kg): G1: < 11.5 G2: 11.5–16.0 G3: 16.1–20.0 G4: > 20.0	Preeclampsia,% G1: 1.4 G2: 2.3 G3: 5.4 G4: 4.4 *P* for trend = 0.262	None

AOR, adjusted odds ratio; BMI, body mass index; CI, confidence interval; g, gram; kg, kilogram; lbs, pounds; USA, UnitedStates of America.

Overview of results The evidence of an association between high weight gains and increased risk of preeclampsia is inconclusive.

Detailed results Among obese women, preeclampsia risk increased with gains greater than 25 pounds and decreased with gains lower than 15 pounds in one study.[4] Another study reported no association, but it had not conducted multivariate analyses for this outcome.[55]

Among women of normal weight, one study found no association between preeclampsia and gains either below or above the IOM levels.[53] In two other studies, the risk of preeclampsia rose as weight gains above the IOM recommendations increased;[25,55] it dropped with weight gains below IOM thresholds in one of these studies.[25]

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Maternal Intrapartum Outcomes

Cesarean delivery

Study characteristics Nine articles examined the effect on cesarean delivery of weight gain classified according to the IOM guidelines (Evidence Table 39, Table 30).4[,25,53–55,77,118,149,150] These studies were all rated fair except for one poor study.[54] Two articles were based on the same birth certificate data from Missouri;[4,25] three used US hospital databases;[55,118,149] one used data from a U.S. midwifery practice;[150] one used a random selection of normal-weight pregnant women in Iceland;5[3] and one used data from the U.S. Pregnancy Risk Assessment Monitoring System (PRAMS).[77]

TABLE 30. Weight change relative to IOM thresholds and cesarean delivery

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Devader et al., 2007[25] USA-Missouri, birth certificate data 94,696 Normal weight BMI 19.8–26 Fair	Pregravid weight: Self-Report Total weight gain: As reported on birth certificate	G1: Gained less than 25 lbs G2: Gained 25–35 lbs G3: Gained more than 35 lbs	AOR for cesarean delivery (additionally controlled for LGA and cephalopelvic disproportion) G1: 0.82 (0.78–0.87) G2: 1.0 G3: 1.35 (1.29–1.40)	Age, race, education, income, alcohol use, height, prior pregnancy, inadequate prenatal care use, smoking, child's gender, birth year
Edwards et al., 1996[55] USA, hospital 1,443 Normal BMI 19.8–26 Obese BMI > 29 Fair	Pregravid weight: Self-reported Total weight gain: Prenatal records	Obese G1: wt loss or 0 lbs G2: 1–14 lbs G3: 15–25 lbs G4: 26–35 lbs G5: > 35 lbs Normal weight G1: < 25 lbs G2: 25–35 lbs G3: > 35 lbs	Obese G1: 30.7% G2: 21.6% G3: 23.8% G4: 26.2% G5: 30.1% Normal wt G1: 5.7% G2: 12.1% G3: 8.6% No significant difference in rates of cesarean delivery by IOM weight gain categories for normal weight or obese women Obese women AOR = 3.2 (2.3–4.4) for cesarean delivery	Age, parity, pregravid BMI, GDM, pregnancy-induced hypertension, prenatal adequacy, alcohol use, drug use, smoking, gestational age

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Jain et al., 2007[77] USA, PRAMS 7,661 All weight/BMIs (using IOM definitions) Fair	Pregravid weight: Self-reported Total weight gain: Self-reported	G1: ≤ 15 lbs G2: 16 – < 25 lbs G3: 25 – < 35 lbs G4: ≥ 35 lbs G5: interaction term overwt/obese and gaining 25–35	Primipara (AOR, 95% CI) G5: 0.71 (0.43–1.19) Multipara G5: 0.77 (1.37–1.59)	Pregravid BMI, parity
Kaiser and Kirby, 2001[150] USA, university nurse-midwifery system 1,881 All wt/BMI (using IOM definitions) Fair	Pregravid BMI: Self-reported Total weight gain: Measured at last prenatal visit	G1: Below IOM G2: Within IOM G3: Above IOM	Crude OR 95% CI G1: 0.82 (0.49–1.36) G3:1.0 (0.62–1.63) AOR for weight gain above IOM recommendations: 2.04 (95% CI 1.02–4.05)	Age, race, pregravid BMI, preeclampsia, height, previous live births, failure to progress, breech presentation, placental abruption, fetal bradycardia, primigravidity, birthweight
Kiel et al., 2007[4] USA-Missouri, birth certificate 120, 170 Obese BMI > 30 Fair	Pregravid BMI: Self-reported Total weight gain: Birth certificate	G1: Wt loss > 10 lbs G2: Wt loss 2–9 lbs G3: No change G4: 2–9 lbs G5: 10–14 lbs G6: 15–25 lbs G7: 26–35 lbs G8: > 35 lbs	For all three classes of obese women, risks of cesarean delivery rise above an OR of 1 when weight gain exceeds 25 pounds	Age, race, parity, education, poverty (enrollment in medicaid, WIC, food stamp programs), tobacco use, chronic hypertension
Parker and Abrams, 1992[118] USA, hospital data base 6,690 All wt/BMI (using IOM definitions)	Pregravid weight: Self reported Total weight gain: Measured weight in prenatal record	G1: Below IOM G2: Above IOM	AOR for all women weight gain > IOM (G2) = 1.48 (1.25–1.76) For overweight women, there was no significant association between cesarean delivery and weight gain (AOR = 0.71 (0.40–1.26) For nonoverweight women,	Age, race, parity, pregravid BMI, height, maternal high and low weight gain, smoking, gestational age, birthweight

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Fair			the association between cesarean delivery and weight gain was 1.45 (1.21–1.73)	
Stotland et al., 2004[149] USA, university hospital 9,788 All wt/BMI Fair	Pregravid weight: No details reported Total weight gain: No details reported, possibly measured weight in prenatal records	G1: Below IOM G2: Above IOM	AOR with birthweight in model G1: 0.99 (0.82–1.19) G2 :1.40 (1.22–1.59) BMI < 19.8 G1 = 0.96 (0.67–1.37); G2 = 1.93 (1.45–2.53) BMI 19.8–26 G1 = 1.04 (0.81–1.33); G2 = 1.26 (1.06–1.50) BMI > 26 G1 = 0.74 (0.38–1.44); G2 = 1.21 (0.83–1.78)	Age, race, pregravid BMI, year of delivery, smoking, gestational age, birthweight, infant sex
Thorsdottir et al., 2002[53] Iceland, hospital records 614 Normal wt/BMI 19.5–25.5 Fair	Pregravid weight: Self-reported Total weight gain: Measured weight in prenatal records	G1 < 11.5 kg G2 11.5–16 kg G3 16.1–20 kg G4 > 20 kg	G1: 17.4% G2: 9.5% G3: 12.9% G4: 13.1% No significant differences in cesarean delivery rates by IOM weight gain categories in normal weight women	Age, parity, height, gestational age, birthweight
Bianco et al., 1998[54] USA, hospital 11,926 Nonobese BMI 19 to 27 /morbidly obese BMI > 35 Poor	Pregravid weight: Perinatal data base Total weight gain: Perinatal data base	G1: wt loss or 0 lbs G2: 1–15 lbs G3: 16–25 lbs G4: 26–35 lbs G5: > 35 lbs	G1: 25.5% G2: 26.8% G3: 28.8% G4: 35.0% G5: 33.8% No significant difference among morbidly obese women by weight gain categories OR for cesarean comparing morbidly to nonobese = 2.3 (1.9–2.8)	Macrosomia

Overview of results For underweight and normal-weight women, some evidence may suggest an increased risk of cesarean delivery for weight gains above IOM recommendations; evidence for obese or morbidly obese women is inconsistent.

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Detailed results Two studies that examined women across a range of BMI categories found increased risks of cesarean delivery for weight gains exceeding IOM guidelines and these results were consistent in all pregravid weight categories (AORs of 1.6 and 2.0).[149,150]

The six studies stratified by pregravid weight status produced mixed results. Of the articles in this category, five considered women of normal weight.[25,53,55,118,149] Of these five studies, two[53,55] reported no association with weight gains above the IOM guidelines and three found a moderate association between cesarean delivery and weight gain above IOM recommendations[25,118,149] Four studies (1 of poor quality[54]) examined these issues among overweight and obese women.[54,55,118,149] They reported no association between weight gain and risk of cesarean delivery. For underweight women, two studies reported a moderate to strong association between weight gain above IOM recommendations and risk for cesarean delivery;[118,149] for nonobese women, one of these studies reported a moderate association.[118] Three studies reported that the risk of cesarean delivery was higher for obese or morbidly obese women than for nonobese women.[54,55,150] One study suggested that these risks increase within classes of obesity with gains greater than 25 pounds.[4]

The one study that examined the interaction between weight gain of 25–34 pounds and pregravid overweight or obese status did find a significant effect for multiparous women but not primiparous.[77]

Birth Outcomes

Preterm birth

Study characteristics Four studies, all rated fair, reported on the association between weight gain according to the IOM guidelines and preterm birth defined as < 37 completed weeks of gestation (Evidence Table 40, Table 31).[22,85,151,152] One study reported on total weight gain.[22] All four reported on the rate of weight gain or pattern.[22,85,151,152]

TABLE 31. Weight change relative to IOM thresholds and preterm birth (< 37 weeks)

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Hickey et al., 1995[151] USA, university prenatal clinics 1,518 Under/normal wt Fair	Pregravid weight: Self-reported Total weight gain: Prenatal records	G1: Low rate of weight gain in first trimester-underweight (BMI < 19.8) & < 2.3 kg and normal weight (BMI 19.8–26) & < 1.6 kg G2: Low rate of weight gain in second trimester (Underwt & < 0.38 kg/wk or normal wt & < 0.37 kg/wk) G3: Low rate of weight gain in third trimester	OR (95% CI) for spontaneous preterm G1: 1.27 (0.7–2.3) G2: 1.23 (0.7–2.18) G3: 2.46 (1.53–3.92) Pattern of weight gain G1 only: 2.94 (0.73–11.98) G2 only: 1.08 (0.1–11.23) G3 only: 11.54 (2.93–45.28) G1 & G2: 4.89 (0.85–28.14) G1 & G3: 4.49 (0.96–20.96) G2 & G3: 7.37 (1.66–32.76) All trimesters: 4.18 (0.75–23.35)	Age, race, pregravid BMI, height, alcohol use, history of previous infant less than 2,750 g, number of days between last weight observation and delivery, smoking, infant sex

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
		(Underwt & < 0.38 kg/wk or normal wt & < 0.37 kg/wk)		
Schieve et al., 2000[152] USA, nationally representative 1988 births 3,511 All wt/BMI Low BMI < 19.8 Average BMI 19.8–26 High BMI > 26 Fair	Pregravid weight: Self-reported baseline Weight gain: Rate of measured weight between 14 and 28 wks gestation	G1: Low < 0.5 less/week; G2: Average 0.5–1.5 lb/week G3: High > 1.5 lb/week	AOR for preterm birth Low BMI: G1 = 6.7 (1.1–40.6) Low BMI: G2 = 0.8 (0.4–1.4) Low BMI: G3 = 1.0 (0.4–2.6) Average BMI: G1 = 3.6 (1.6–8.0) Average BMI: G2 (Reference) Average BMI: G3 = 1.0 (0.6–1.9) High BMI: G1 = 1.6 (0.7–3.5) High BMI: G2 = 1.1 (0.6–2.1) High BMI: G3 = 0.1 (0.03–0.6)	Age, race, parity, marital status, education, smoking
Siega-Riz et al., 199422 USA, public health clinics 5,854 All wt/BMI (using IOM definitions) Fair	Pregravid weight: self reported Total weight Gain: Prenatal records	Total weight gain expressed as a ratio of observed: expected based on the IOM recommendation for a given gestational age.	Adequacy of weight gain in the third trimester was predictive of preterm birth - the data suggested a threshold effect for all weight status groups with a marked decrease in risk at 90–110% of the IOM recommendation With the rate of weight gain less than 60% of the IOM value, women in all four groups had more than double the risk of delivering preterm, which was statistically significant for all but the obese category. Excessive rate of weight gain was signficantly associated with a preterm birth only for women of normal prepregnancy weight status at a value greater than 200% of the IOM value	Pregravid BMI, gestational age
Stotland et al., 2006[85] USA, academic medical center	Pregravid weight: Self-reported Total weight	G1: low rate of weight gain < .27 kg/wk G2: ref 0.27–0.52 kg/wk G3: high rate of weight gain > 0.52 kg/wk	Crude OR of spontaneous preterm birth G1:2.6 (95% CI 2.1–3.2) G3:1.0 (95% CI 0.8–1.2)	Age, race, parity, pregravid BMI, history of previous PTB, year of delivery, number of days between last weighing

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
15,101 Underweight BMI < 19.8 and normal weight BMI 19.8–26 Fair	gain: prenatal records		AOR of spontaneous preterm birth G1: 2.5 (95% CI 2.0–3.1) G3:1.0 (95% CI 0.8–1.3) No differences in results by parity combined with history of preterm birth Slightly higher risks for Af Am and high wt gain close to sign for Af Am	and DOB, smoking

Af Am, African American; AOR, adjusted odds ratio; BMI, body mass index; CI, confidence interval; DOB, date of birth; G, group; IOM, Institute of Medicine; kg/wk, kilograms per week; OR, odds ratio; wt: weight; PTB, pre-term birth; USA, United States of America; wk, week; wt, weight.

Overview of results Despite inconsistencies in the definitions of rate of weight gain and the timing of its calculation, the four studies are consistent in showing increased risks of preterm birth for underweight and normal-weight women, thereby providing evidence of some association between weight gain below IOM recommendations and preterm birth. Evidence about any association between weight gain above IOM recommendations and preterm birth is inconclusive.

Detailed results on total weight gain The single study on total weight gain, set in the United States, included only singleton live births with no pregnancy complications among predominantly Hispanic women (80 percent) using information reported on the medical record.22 Total weight gain was defined as weight at last prenatal visit minus self-reported pregravid weight (which was checked for biological plausibility). To analyze observed weight gains in light of the IOM recommendations, the authors created an "expected total weight gain" variable using the amount of weight gain a woman was supposed to gain according to the IOM guidelines when her last weight was measured and then calculated a ratio of observed to expected weight gain. Ratios greater than 1 indicate that the women gained more weight than expected; ratios less than 1 indicate that they gained less weight than expected. For all but obese women, the pattern of risk of preterm birth was U-shaped. The lowest risk of preterm birth was observed for all women with weight gain ratios between 1.10 and 1.40.

Results on rate of weight gain for all women In the two studies that examined rate of weight gain among women in all BMI groupings,[22,152] the U.S. study described above found that inadequate or excessive weight gain in the first or second trimester using the IOM definitions was not associated with preterm birth.[22] By contrast, adequacy in the third trimester was predictive of risk of preterm birth; ratios of observed/expected between .90 and 1.10 were associated with decreased risk. A ratio of < 0.60 was significantly associated with a doubling of the risk of preterm birth for women of all but the obese weight groups. Among normal-weight women, a ratio > 2.0 was significantly associated with a preterm birth.

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The other study was conducted in a nationally represented sample of all singleton live births in the U.S. from 1988.[152] This study used common definitions of rate of weight gain (mapping to IOM categories for underweight women): low (< 0.5 lb/week), average (0.5 to 1.5 lb/week), and high > 1.5 lb/week) for all BMI groups. The authors calculated rate of weight gain in a regression model using measured prenatal weights from 14 to 28 weeks of gestation. Among women of normal weight, low weight gain was statistically significantly associated with an increased risk (approximately fourfold) of preterm delivery compared with women in this same category who had average weight gain. This finding held true when medically indicated preterm deliveries were excluded, when women with pregnancy complications were excluded, and when models were adjusted for confounders listed in Table 31. Among underweight women, a low rate of weight gain was statistically significantly associated with the risk of preterm birth when the same exclusions and model adjustments were made. In models with these same exclusions and adjustments, however, for women with a BMI ≤ 26, high weight gain was not associated with significant changes in the risk of preterm birth and for women with a BMI > 26, high weight gain was associated with lower risk of preterm birth.

Detailed results on rate of weight gain for normal or underweight women Two studies examined the effect of rate of weight gain on spontaneous preterm birth among only underweight and normal-weight women.[85,151] In one U.S. study, the authors calculated the rate of weight gain over the entire pregnancy using weight at time of delivery minus self-reported pregravid weight divided by gestational age at delivery (minus 2 weeks because gestational age was based on last menstrual period).[85] Low rate of weight gain during pregnancy, defined as < 0.27 kg per week, was statistically significantly associated with spontaneous preterm birth in both crude and adjusted analyses. High rate of weight gain, defined as > 0.52 kg per week, was not associated with risk of preterm birth. These findings were similar when the models were stratified by ethnicity, parity, and history of preterm birth, and adjusted for the confounders listed.

In another U.S. study, total weight gain in the first trimester was defined as measured weight at 10 to 13 weeks minus self-reported pregravid weight; second and third trimester rates of weight gain were based on measured weights during the trimester.[151] Low weight gain in the first or second trimester alone was not associated with spontaneous preterm birth. By contrast, low third-trimester weight gain was statistically significantly associated with spontaneous preterm birth. The combination of low second- and third-trimester rate of weight gain was also statistically significantly associated with spontaneous preterm birth. All analyses controlled for several confounders listed in Table 31.

Birthweight

Study characteristics Ten studies from nine databases examined the association between weight gain defined by IOM guidelines and birthweight (Evidence Table 41, Table 32).[20,54,60,104,153–158] Three studies were done in only black women;[60,153,154] two stratified by race;[20,155,156] two were done in adolescents;[153,154] one came from a cohort of 233 women enrolled in the WIC program in Iowa;[157] and one used a perinatal database from a medical center in New York.[54]

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TABLE 32. Weight change relative to IOM thresholds and birthweight

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Nielsen 2006[153] USA, prenatal clinics African-Americans only 815 All weight/BMIs Good	Pregravid weight: Self-reported Total weight gain: Prenatal records, measured	G1: < IOM G2: lower half IOM range G3: Upper half IOM range G4: > IOM	Adjusted birthweight BMI < 19.8 G1: 2,986 g G2: 3,167 g G3: 3,198 g G4: 3,277 g All significantly different from each other except G2 & G3 BMI 19.8–26 G1: 3,018 g G2: 3,166 g G3: 3,255 g G4: 3,318 g All significantly different from each other BMI > 26 G1: 3,127 g G2: 3,351 g G3: 3,384 g G4: 3,434 g G1 significantly different from the others, G2,G3 & G4 not significantly different from each other	Parity, pregravid BMI, preeclampsia, time between last weight measure and delivery, height, smoking, infant sex
Hickey et al., 1997[20] USA, public health programs 5,198 All wt/BMI (using IOM definitions) Fair	Pregravid weight: Self-reported Total weight gain: Measured weights-prenatal records	G1: Below IOM range G2: Lower end of IOM range G3: Upper end of IOM range G4: Gain above IOM range	BMI < 19.8 G1: Black: 2,840 White: 3,002 G2: Black: 2,995 White: 3,151 G3: Black: 3,017 White: 3,200 G4: Black: 3,163 White: 3,353	Age, education, height, street drugs, alcohol use, time between last prenatal weight observation and delivery, smoking, gestational age, infant sex

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
			BMI 19.8–26.0 G1: Black: 3,052 White: 3,176 G2: Black: 3,105 White: 3,199 G3: Black: 3,180 White: 3,307 G4: Black: 3,228 White: 3,389 BMI > 26.0 G1: Black: 3,126 White: 3,385 G2: Black: 3,192 White: 3,376 G3: Black: 3,312 White: 3,402 G4: Black: 3,300 White: 3,504	
Hickey et al., 1996[155] USA, prenatal clinics 415 Under & Normal weight Fair	Pregravid BMI: Self-reported Total weight gain: Prenatal records	G1: First trimester < 2.6 kg for underwt (BMI < 19.8) & < 1.6 kg for normal wt (BMI 19.8–26) G2: Second trimester < 0.38 kg/wk for underwt & < 0.37 kg/wk for normal wt G3: < 0.38 kg/wk for underwt & < 0.37 kg/wk for normal wt	Association of low trimester gain with birthwt G1: all women -18 g *P* = .65 Black -15 g *P* = .76 White -42 g *P* = .53 G2: All women -166g *P* = < .001 Black -164 g *P* = .005 White -158 g *P* = .05 G3: All women -111g	Age, race, pregravid BMI, height, alcohol use, third trimester number of weeks between last weight observation and delivery, history of previous infant < 2,750 g, smoking, gestational age, infant sex

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
			P = .008 Black -77 g *P* = .14 White -194 g *P* = .004 No association with low weight gain in only the first or second trimester. G3: All -164 g *P* = .01 Black -80 g *P* = .38 White -300 g *P* = .005 Association with low weight gain during more than one trimester G1 & G2: All -236 g *P* = .01 Black -265 g *P* = .04 White -169 g *P* = .25 G1 & G3: No significant diff G2 & G3: All -206 g *P* = .01 Black -178 g *P* = .08 White -268 g *P* = .06 G1, G2 & G3: All -284 g *P* = .002 Black -252 g *P* = .03 White -379 g *P* = .008	
Hickey et al., 1993[156] USA, prenatal clinics 1,168 All weight/BMIs Fair	Pregravid BMI: Self-reported Total weight gain: Prenatal records	BMI ≤ 29 G1: gain < range G2: gain in the range G3: gain > range BMI > 29 G4: gain < 6.0 kg G5: gain > 6.0 kg	Adjusted Birthwt BMI ≤ 29 G1: Black: 3,027 White: 3,246 G2: Black: 3,177 White: 3,233 G3: Black: 3,293 White: 3,523	Maternal height, education, parity, marital status, smoking, alcohol use, hypertension, GDM, gestational age at delivery, socioeconomic status, time between last weight and delivery

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
			BMI > 29 G4: Black: 3,214 White: 3,500 G5: Black: 3,553 White: 3,596	
Luke et al., 1996[104] USA, clinic 487 All weight/BMIs Fair	Pregravid weight: Self-reported Total weight gain: Prenatal records, measured	G1: Gain < IOM G2: gain equal to IOM G3: gain > IOM	Adjusted birthweight G1: BMI < 19.8 2,873 g* BMI 19.8–26.0 3,157 g* BMI > 26 3,138 g G2: BMI < 19.8 3,190 g BMI 19.8–26 3,298 g BMI > 26 3,338 g G3: BMI < 19.8 3,489 g* BMI 19.8–26 3,494 g* BMI > 26 3,347 g * significantly different from gains within range within each BMI grouping	Maternal age, parity, black ethnicity, smoking, gestational duration, fetal sex
May, 2007[157] USA, WIC clinic 233 All weight/BMI Fair	Pregravid weight: Self reported Total gestational weight gain: Self-reported	G1: Below IOM G2: Greater IOM	Betas from multiple linear regression G1: -162 g G2: -153 g	Maternal BMI, smoking, gestational age at delivery
Ogunyemi et al., 1998[60] USA, Hospital 582 All weight/BMIs	Pregravid weight: Self-reported Total gestational weight gain: Prenatal	G1: Low < IOM G2: Normal = IOM G3: High > IOM	Birthweight G1: 3,029 G2: 3,210 G3: 3,283 ($P < 0.01$)	NR

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
(using IOM definitions) Fair	records, measured			
Scholl et al., 1995[158] USA Camden Study 274 Normal weight BMI 19.8–26 Fair	Pregravid weight: Self-reported Total weight gain: Prenatal records, measured	Rate between 20–36 wks G1: low rate < 0.34 kg/wk G2: moderate rate 0.34–0.68 kg/wk G3: Excessive rate > 0.68 kg/wk	Birthweight (g): G1: 3,049 (56.94) $P <$ 0.05, low vs. moderate plus excessive weight gain G2: 3,208 (36.33) G3: 3,191 (49.46)	NR
Stevens-Simon and McAnarney, 1992[154] USA African-American adolescent maternity program 141 All BMI Fair	Pregravid weight: Self-reported Total weight gain: Prenatal records, measured	G1: slow < 0.23 kg/wk G2: average 0.23–4 kg/wk G3: rapid > 0.4 kg/wk	Birthweight (g): G1: 2,745 (694) G2: 3,097 (457) G3: 3,351 (482) $P <$ 0.0001 No difference in pregravid by weight gain groups	NR
Bianco et al., 1998[54] USA, medical center 11,926 Nonobese (BMI 19–27) and morbidly obese (BMI > 35) Poor	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories among morbidly obese : G1: Weight loss/no change G2: 1–15 lbs G3: 16–25 lbs G4: 26–35 lbs G5: > 35 lbs	G1: 3,302 g G2: 3,192 g G3: 3,337 g G4: 3,506 g G5: 3,453 g $P =$ < 0.05	NR

&, and; AOR, adjusted odds ratio; birthwt, birthweight; BMI, body mass index; G, group; g, gram; GDM,

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
gestational diabetes mellitus; IOM, Institute of Medicine; kg/wk, kilogram per week; NR, not/none reported; OR, odds ratio; underwt, underweight; USA, United States of America; WIC, The Special Supplemental Nutrition Program for Women, Infants, and Children.				

Overview of results Overall, these studies (1 good,[153] 8 fair,[20,60,104,154–157] and 1 poor[54]) support an association between weight gains less than the IOM guidelines and lower birthweight; such an association appears to be stronger when the rate of weight gain is the relevant factor. There is also evidence of an association for gains above the guidelines and higher birthweight but less so when rate of weight gain is the relevant factor.

Detailed results for total weight gain Seven articles examined total weight gain; one was good,[153] one was poor quality,[54] and the remainder were fair.[20,60,104,156,157] The study of women in the WIC program found that weight gains both below and above the IOM guidelines were associated with lower birthweights (162 g and 153 g, respectively).[157] One study found that women who were underweight or normal weight and who gained above the IOM guidelines had higher birthweights; women who gained below the guidelines had lower birthweights than those who gained within them.[104] The association of higher birthweight with higher weight gain was also found among morbidly obese women in one poor study that failed to adjust for any confounders.[54]

Studies that stratified by race[20,156] or that included only one race[60,153] found, overall, that black women gaining above the IOM guidelines experienced significantly higher birthweights (a range of 73 g to 330 g) than those who gained less weight.[20,60,153,156] Among white women,[20,156] weight gain above the IOM guidelines was also associated with higher birthweights for those with a BMI ≤ 2920,156 but not > 29 in one study.[156] This increase in birthweight was close to 200 g.[20,156] In three of these studies,[20,153,156] the analyses were adjusted for multiple confounders listed in Table 32.

One good study conducted among black adolescents that examined total weight gain found infant birthweights to be lower among those who gained less than the IOM recommendations than among those who gained within or above the guidelines;[153] infant birthweights did not differ between those who gained within and those who gained above the thresholds.

Detailed results for rate of weight gain Three fair-quality studies examined rate of weight gain as the exposure of interest with respect to birthweight.[154,155,158] The one including only adolescents found that mothers who gained < 0.23 kg per week had infants with a mean birthweight of 2,745 g; this birthweight was lower than for infants of mothers who gained 0.23 to 0.4 kg per week (3,097 g) and for those who gained > 0.4 kg per week (3,351 g).[154]

One study examined rate of weight gain only among normal-weight women from the ages of 12 to 29 from black, white, and Hispanic groups.[158] The authors used a rate of weight gain between 20 to 36 weeks and defined low as < 0.34 kg per week, moderate as 0.34 to 0.68 kg per week, and excessive as > 0.68 kg per week. Controlling for several confounders, the investigators found that women with low rates of weight gain had infants of statistically

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significantly lower birthweights than did women with higher rates of weight gain. Birthweights did not differ between those who gained at excessive and moderate rates.

The other rate of weight gain study involved both white and black women with a BMI ≤ 26 and a mean age of 25.[155] Their analyses used mothers who gained more than the IOM guidelines as the reference group. Mothers who gained low levels of weight (< 0.38 kg per week for underweight or < 0.37 kg per week for normal weight) in the second trimester had infants who weighed 166 g less than infants from the reference group; mothers who gained low levels of weight in the third trimester had infants who weighed 111 g less than those in the reference group. When all women were included in the analyses, the effect seen in the third trimester was statistically significant; however, when analyses were stratified by race, it was significant only for white women.

In addition, this study showed that pattern of weight gain was important. Low total weight gain in the first trimester combined with low rate of gain in the second was associated with an infant who weighed 236 g less than those whose mothers gained more weight. This finding appeared to be statistically significant for all women and for black women when analyses were stratified by race. Low rate of weight gain in the second and third trimesters was associated with a 206 g deficit in weight of the infant. Low rate of weight gain in all three trimesters was associated with the greatest deficit, 284 g.

Low birthweight

Study characteristics Twelve articles (from 10 databases) examined low birthweight (LBW, defined as < 2,500 g) (Evidence Table 42, Table 33).[2,20,54,55,60,127,154,159–163] Two articles reported on data from the Pregnancy Nutrition Surveillance System (PNSS) from either eight[2] or nine states;[160] two used a single hospital database.[55,159] Two studies used PRAMS data.[162,163]

Overview of results Evidence from twelve articles (2 good,[159,160] 7 fair,[2,20,55,60,77,127,154,161] and 3 poor[54,162,163]) supports an association between weight gain less than the IOM guidelines and LBW for both underweight and normal-weight women; evidence is less conclusive about any association for women with higher body weight.

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TABLE 33. Weight change relative to IOM thresholds and low birthweight (< 2,500 g)

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Hellerstedt et al., 1997[159] USA, hospital 1,343 Normal/obese BMI Good	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories stratified by pregravid BMI and smoking status: Obese (BMI > 29.0): G1: Smokers, < IOM G2: Smokers, within IOM G3: Smokers, > IOM G4: Nonsmokers, < IOM G5: Nonsmokers, within IOM G6: Nonsmokers, > IOM Normal weight (BMI 19.8–26.0): G7: Smokers, < IOM G8: Smokers, within IOM G9: Smokers, > IOM G10: Nonsmokers, < IOM G11: Nonsmokers, within IOM G12: Nonsmokers, > IOM Obese: G13: Lost/no gain G14: 0.5–6.5 kg G15: 7–11.5 kg G16: 12–16 kg G17: > 16 kg Normal weight: G18: < 11.5 kg G19: 11.5–16 kg G20: > 16 kg	G1: 17.3% G2: 10.0% G3: 12.3% G4: 10.5% G5: 7.8% G6: 2.6% G7: 17.5% G8: 3.5% G9: 3.6% G10: 12.4% G11: 6.0% G12: 5.3% G13: 16.0% G14: 11.1% G15: 8.3% G16: 4.0% G17: 6.0% *P* = 0.003 for G13–G17 G18: 14.2% G19: 5.4% G20: 4.9% *P* = 0.001 for G18–G20 For obese women, compared to nonsmokers who gained 7–11.5 kg, smokers who gained < 7 kg were at significantly higher risk of LBW: AOR: 7.7 (95% CI, 1.5–40.0)	Maternal age, pregravid BMI, infant sex, race, parity, prenatal alcohol use, prenatal illicit drug use, adequacy of prenatal care, gestational hypertension, GDM, gestational age
Schieve et al., 1998[160]	Pregravid	Maternal weight gain	Within every BMI-race	Age, height,

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
USA, Pregnancy Nutrition Surveillance System (WIC clinics) 173,006 All weight/BMI Good	weight: Self-report Total weight gain: Self report	categories stratified by pregravid BMI (IOM underweight, normal weight, overweight, and obese) and race (non-Hispanic white, non-Hispanic black, and Hispanic) G1: ≥ 10 lbs below IOM G2: 1–9 lbs below IOM G3: Lower half of IOM G4: Upper half of IOM G5: 1–9 lbs above IOM G6: ≥ 10 lbs above IOM	ethnicity stratum, the odds of delivering a LBW infant tended to decrease as weight gain increased. This trend was statistically significant for all strata; however, the trend diminished with increasing BMI. Women with underweight and normal weight BMI in G2 were 1.1–2.8 times more likely to deliver a LBW infant than women in G3; women in G1 were 1.8 –3.2 times more likely to deliver a LBW infant compared to G3.	education, trimester of the special supplemental nutrition program for WIC
Bracero and Byrne, 1998[127] USA, hospital, Brooklyn, NY 20,971 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories: G1: Maternal weight gain under the IOM guidelines G2: Maternal weight gain within the IOM guidelines G3: Maternal weight gain over the IOM guidelines G4: Optimal weight gain (36–40 lbs for BMI < 19.8; 31–40 lbs for BMI 19.8–26.0; 26–30 lbs for BMI > 26.0) G5: Suboptimal weight gain (< 36 lbs for BMI < 19.8; < 31 lbs for BMI 19.8–26.0; < 26 lbs for BMI > 26.0)	G1: 10.1% G2: 3.3% G3: 2.5% ($P < 0.001$ comparing G1–G3) G4: 4.9% G5: 1.8% ($P < 0.001$ vs. G4)	Not applicable

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Cogswell et al., 1995[2] USA, Pregnancy Nutrition Surveillance System 53,541 Normal/Overweight/Obese Fair	Pregravid weight: Self-report Total weight gain: Self-report	Maternal weight gain categories (lbs) stratified by pregravid BMI: Normal weight (BMI 19.8–26.0): G1: < 15 G2: 15–19 G3: 20–24 G4: 25–29 G5: 30–34 G6: 35–39 G7: ≥ 40 Overweight (BMI > 26.0–29.0): G8: < 15 G9: 15–19 G10: 20–24 G11: 25–29 G12: 30–34 G13: 35–39 G14: ≥ 40 Obese (BMI > 29.0): G15: < 15 G16: 15–19 G17: 20–24 G18: 25–29 G19: 30–34 G20: 35–39 G21: ≥ 40	AOR (95% CI) for low birthweight: G1: 2.1 (1.6–2.6) G2: 1.4 (1.1–1.8) G3: 1.0 (0.8–1.3) G4: 1.0 (reference) G5: 0.8 (0.6–1.0) G6: 0.6 (0.5–0.8) G7: 0.5 (0.4–0.6) G8: 1.1 (0.7–1.9) G9: 1.0 (reference) G10: 0.7 (0.4–1.2) G11: 0.8 (0.5–1.4) G12: 0.5 (0.3–0.8) G13: 0.6 (0.3–1.1) G14: 0.4 (0.3–0.7) G15: 1.5 (0.9–2.4) G16: 1.0 (reference) G17: 0.9 (0.5–1.6) G18: 1.3 (0.8–2.3) G19: 0.9 (0.5–1.7) G20: 1.0 (0.5–1.8) G21: 0.9 (0.5–1.5)	Age, race, height, smoking, gestational age, infant sex
Edwards et al., 1996[55] USA, Hospital 1,443 Normal/Obese weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories (kg) Obese BMI > 29: G1: Lost weight/no change G2: 0.5–6.5 G3: 7–11.5 G4: 12–16 G5: > 16 Normal BMI 19.8–26: G6: < 11.5 kg	G1:12.8% G2: 8.9% G3: 7.9% G4: 6.8% G5: 8.7% *P* (for G1–G5) = 0.405 G6: 8.5% G7: 5.6% G8: 8.9% *P* (for G6–G8) = 0.183 AOR (95% CI) for	Age, parity, pregravid BMI, GDM, pregnancy-induced hypertension, prenatal adequacy, alcohol use, drug use, smoking, gestational age

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
		G7: 11.5–16 G8: > 16 kg	birthweight < 2,500 g among obese women (BMI > 29.0): G3: 1.0 (reference) G1: 4.2 (0.9–19.6)	
Hickey et al., 1997[20] USA, public health programs 5,198 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories stratified by race: Black Women: G1: Below range (< 12.5kg for BMI< 19.8; < 13.9kg for BMI 19.8–26.0; < 7.0kg for BMI > 26.0) G2: In lower range (12.5–15.2kg for BMI< 19.8; 11.5–13.8kg for BMI 19.8–26.0; 7.0–9.2kg for BMI > 26.0) G3: In upper range (15.3–18kg for BMI< 19.8; 13.9–16.0kg for BMI 19.8–26.0; 9.3–11.5kg for BMI > 26.0) G4: Above range (> 18kg for BMI < 19.8; > 16.0kg for BMI 19.8–26.0; > 11.5kg for BMI > 26.0) White Women: G5: Below range G6: In lower range G7: In upper range G8: Above range	AOR (95% CI) G1: 2.6 (1.2–5.6) G2: 1.0 (reference) G3: 1.2 (0.4–3.3) G4: 1.4 (0.6–3.6) G5: 1.5 (0.8–2.6) G6: 1.0 (reference) G7: 0.4 (0.2–0.9) G8: 0.7 (0.3–1.2)	Age, education, height, drug use, alcohol use, time between last prenatal weight observation and delivery, smoking, gestational age, infant sex
Ogunyemi et al., 1999[60] USA, Hospital 582	Pregravid weight: Self-report Total weight	Maternal weight gain categories: G1: < IOM G2: Within IOM	AOR (95% CI) for very low birthweight: G1: 1.8 (0.6–4.7) G2: 1.1 (0.4–4.7) G3: 1.0 (Reference)	Age, parity, pregravid BMI, preeclampsia, cesarean delivery, previous cesarean,

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
All weight/BMI Fair	gain: Measured	G3: > IOM BMI IOM		tobacco use, previous fetal death, hypertension, asthma, previous LBW, vomiting, NICU
Stevens-Simon and McAnarney, 1992[154] USA, adolescent maternity program 141 Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories (kg/wk): G1: < 0.23 G2: 0.23–0.40 G3: > 0.40	Distribution of LBW, %: G1: 21.4 G2: 10.6 G3: 4.3 *P* = NS	Not applicable
Strauss and Dietz, 1999[161] USA, National Collaborative Perinatal Project and the Child Health and Development Study 10,756 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories stratified by pregravid BMI: BMI < 20.0: G1: Low 1st trimester gain (< 0.1kg/wk) G2: Low 2nd trimester gain (< 0.3kg/wk) G3: Low 3rd trimester gain (< 0.3kg/wk) BMI 20.0–25.0: G4: Low 1st trimester gain G5: Low 2nd trimester gain G6: Low 3rd trimester gain BMI > 25.0: G7: Low 1st trimester gain G8: Low 2nd trimester gain G9: Low 3rd trimester gain	AOR (95% CI) for < 2,500g: G1: 0.88 (0.50–1.57) G2: 2.68 (1.46–4.94) G3: 2.07 (1.22–3.51) G4: 1.31 (0.88–1.95) G5: 1.92 (1.29–2.87) G6: 2.12 (1.48–3.04) G7: 1.02 (0.50–2.08) G8: 1.88 (1.03–3.43) G9: 1.53 (0.86–2.74) Reference group-normal rate of weight gain in the trimester	Race, GDM, toxemia, smoking

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Bianco et al., 1998[54] USA, medical center 11,926 Nonobese (BMI 19–27) and morbidly obese (BMI > 35) Poor	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories among morbidly obese (BMI> 35): G1: Weight loss/no change G2: 1–15 lbs G3: 16–25 lbs G4: 26–35 lbs G5: > 35 lbs	Distribution of LBW,%: G1: 2.0 G2: 11.1 G3: 8.3 G4: 5.2 G5: 3.8 *P* = NS	Race, parity, clinic service, substance abuse, preexisting medical condition
Hulsey et al., 2005[162] USA, birth certificates linked to PRAMS data 87,293 All weight/BMI Poor	Pregravid weight: Self-report Total weight gain: Birth certificate	Pregravid BMI and gestational weight gain categories: G1: BMI < 19.8 and < IOM G2: BMI 19.8–26.0 and < IOM G3: BMI 19.8–26.0 and within IOM G4: BMI 26.1–29.0 and < IOM G5: BMI > 29.0 and < IOM G6: BMI > 29.0 and within IOM	AOR (95% CI) for very low birthweight (500–1,499g): G1: 2.06 (1.26–2.87) G2: 1.82 (1.22–2.29) G3: 1.00 (reference) G4: 2.05 (0.90–4.44) G5: 1.25 (0.61–1.61) G6: 1.74 (1.23–2.42) AOR (95% CI) for moderately low birthweight (1500–2499 g): G1: 4.83 (2.98–7.83) G2: 1.77 (1.23–2.60) G3: 1.00 (reference) G4: 0.28 (0.11–1.83) G5: 1.09 (0.67–2.13)	Ethnicity, intendedness of pregnancy, Medicaid status, WIC status, prenatal care, diabetes, hypertension
Nida et al., 1996[163] USA PRAMS No sample size All weight/BMI Poor	Pregravid weight: Self-report Total weight gain: Self-report	G1: < IOM G2: within IOM G3: > IOM	BMI < 19.8 G1: 10.2% G2: 6% G3: 4.7 BMI 19.8–25 G1: 8.4% G2: 3.9% G3: 4.5% BMI > 26 G1: 6.1% G2: 3.8% G3: 5.1% No statistical testing was performed	Pregravid BMI

AOR, adjusted odds ratio; BMI, body mass index; CI, confidence interval; G, group; g, gram; GDM, gestational

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
diabetes; IOM, Institute of Medicine; kg, kilogram; lbs, pounds; LBW, low birthweight; MLBW, moderately low birthweight; NICU, neonatal intensive care unit; USA, United States of America; VLWB, very low birthweight; WIC, The Special Supplemental Nutrition Program for Women, Infants, and Children.				

Detailed results for total weight gain In the nine-state PNSS study,[160] analyses for normal and overweight women stratified by race showed a statistically significant decreased risk of LBW with higher gains. Among underweight women, a protective effect against LBW was seen with higher gains in whites and Hispanic and an increased risk was associated with low weight gains (>10 lbs <IOM threshold) across all the race groups. Similarly, among obese women of all race groups, low weight gains (> 10 pounds below the IOM threshold) were associated with higher risk of LBW.[160]

In the eight-state PNNS study,[2] for women of normal weight, the odds for LBW were elevated and statistically significant when their weight gains were below 19 pounds compared with women whose weight gains were in the recommended range. For overweight and obese women, weight gains below the IOM guidelines were not associated with LBW infants. This was also shown in the study by Edwards et al.[55]

Weight gains above the IOM guidelines starting at > 35 pounds were protective against having a LBW infant for normal-weight women,[2] and starting at ≥ 40 pounds for overweight women, but higher weight gains were not protective for obese women.

Two studies showed almost double the odds of LBW among black women who delivered at term but had weight gain below the IOM range;[20,60] this finding was statistically significant in only one (good) study.[20] The OR among white women was 1.5 (not significant).[20]

The only association seen among obese women was among smokers who gained less than the IOM guidelines.[159] These women had an eightfold increased risk of having an LBW infant compared with obese nonsmokers who gained adequately.[159]

One study performed bivariate analysis between the IOM categories of weight gain and LBW infants.[127] It demonstrated a statistically higher prevalence of LBW among mothers who gained less than the IOM guidelines than among mothers who gained within or more than the guidelines.

Detailed results for rate of weight gain Two studies examined the effect of the rate of weight gain on LBW.[154,161] One among black adolescents found no differences in the prevalence of LBW by rate-of-weight-gain group.[154] The other included only term births, used data from the National Collaborative Perinatal Project and the Child Health and Development Study, and examined total weight gain in the first trimester and rates in the second and third trimesters.[161] Low rate of weight gain in the second and third trimesters was associated with an increased risk of term LBW or intrauterine growth restriction (IUGR) in both data sets. This association held for all weight status groups except women with a BMI > 25 when the analysis was stratified by pregravid BMI and adjusted for multiple confounders.

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Fetal growth (large for gestational age or macrosomia)

Study characteristics We identified 15 studies that examined the association between weight gain categorized according to the IOM guidelines on LGA[4,25,54,116,118,129,154,159] or macrosomia[2,53,55,110,160,164,165] (Evidence Tables 43 and 44, Table 34). Five studies used data from a hospital database;[54,55,116,118,129,159] three were cohort studies.[53,154,164] One study used data from a health maintenance organization;[110] one used a prenatal clinic database;[153] one used state birth certificate data;[4,25] one used the Pregnancy Nutrition Surveillance System;[2,160] and one used controls from a multicenter study of birth defects.[165]

Overview of results for LGA infant weight Eight studies defined LGA as > 90 percentile of birthweight for gestational age (Table 34).[4,25,54,116,118,129,154,159] The majority of these studies, of which two were rated good,[116,159] one poor[54] and the remainder fair,[4,25,118,129,154] showed a consistent association between weight gains above the IOM guidelines and LGA for women of all weight status groups. Four articles examined LGA defined as > 4,500 g;[2,53,110,160] two were good quality,[110,160] two were fair.[2,53] They also showed a consistent association. When macrosomia or high birthweight was the outcome, results were less consistent (1 poor quality,[165] 2 fair-rated studies[55,164]).

TABLE 34. Weight change relative to IOM thresholds and large-for-gestational-age infant weight

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
LGA as > 90th percentile of birthweight for gestational age				
Caulfield et al., 1998[116] USA, university hospital 3,870 All weight/BMI Good	Pregravid weight: Self report Total weight gain: Measured	G1: Underweight, BMI < 19.8 G2: Normal weight, BMI 19.8–26.0 G3: Overweight, BMI > 26.0 Black women: G4: No weight gain < IOM G5: No weight gain > IOM White women: G6: No weight gain < IOM G7: No weight gain > IOM	AOR (95% CI) for LGA and rate of weight gain (per 50 g/wk): G1: 1.25 (1.11–1.41) G2: 1.14 (1.08–1.20) G3: 1.13 (1.07–1.20) Expected absolute change (as % of baseline) in incidence of LGA associated with modifiable risk factor (G4–G7): G4: +1.28 (+26) G5: -0.77 (-16) G6: +2.58 (+17) G7: -2.87 (-19)	Age, race, parity, pregravid BMI, height, hypertension, provider type, smoking, female infant
Hellerstedt et al., 1997[159] USA, medical center	Pregravid weight: Self-report	Maternal weight gain categories stratified by pregravid BMI	Frequencies of LGA,%: G1: 5.3 G2: 10.0	Maternal age, pregravid BMI, infant sex, race,

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
		and smoking status:	G3: 12.3	parity, prenatal
1,343	Total		G4: 12.2	alcohol use, prenatal
	weight gain:	Obese (BMI > 29.0):	G5: 11.7	illicit drug use,
Normal/obese BMI	Measured	G1: Smokers, < IOM	G6: 22.2	adequacy of prenatal
		G2: Smokers, within	G7: 0	care, gestational
Good		IOM	G8: 1.8	hypertension, GDM,
		G3: Smokers, > IOM	G9: 9.1	gestational age
			G10: 4.4	
		G4: Nonsmokers, <	G11: 8.1	
		IOM	G12: 14.3	
		G5: Nonsmokers,		
		within IOM	G13: 9.3	
		G6: Nonsmokers, >	G14: 10.5	
		IOM	G15: 11.3	
			G16: 17.5	
		Normal weight (BMI	G17: 21.8	
		19.8–26.0):	*P* = 0.001 for G13–G17	
		G7:Smokers, < IOM		
		G8: Smokers within	G18: 2.8	
		IOM	G19: 6.7	
		G9:Smokers,> IOM	G20: 13.1	
			P < 0.001 for G18–G20	
		G10: Nonsmokers <		
		IOM	Compared with infants of obese	
		G11: Nonsmokers,	nonsmokers who gained 7–	
		within IOM	11.5kg, the only group at	
		G12: Nonsmokers, >	significantly higher risk of LGA	
		IOM	was non smokers who gained >	
			11.5kg: AOR: 2.3 (95% CI,	
		Obese:	1.2–4.5)	
		G13: Lost/no gain		
		G14: 0.5–6.5 kg		
		G15: 7–11.5 kg		
		G16: 12–16 kg		
		G17: > 16 kg		
		Normal weight:		
		G18: < 11.5 kg		
		G19: 11.5–16 kg		
		G20: > 16 kg		
Devader et al., 2007[25]	Pregravid	Maternal weight gain	AOR (95% CI) for LGA:	Age, race, education,
	weight:	categories:	G1: 0.40 (0.37–0.44)	income, alcohol use,
USA, birth certificate data	Self-report	G1: < 25 lbs	G2: 1.00 (reference)	height, prior
		G2: 25–35 lbs	G3: 2.43 (2.30–2.56)	pregnancy,
94,696	Total	G3: > 35 lbs		inadequate prenatal
	weight gain:			care use, smoking,

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Normal weight/BMI 19.8–26 Fair	Measured			child's gender, birth year
Kiel et al., 2007[4] USA, birth registry 120,170 Obese BMI > 30 Fair	Pregravid weight: Self-report Total weight gain: Medical record	Maternal weight gain categories stratified by prepregnancy obesity status, Obese Class I (BMI 30–34.9), Obese Class II (BMI 35–39.9), Obese Class III (> = BMI 40): G1: ≤ -10 lbs G2: -2 to -9 lbs G3: No change G4: 2–9 lbs G5: 10–14 lbs G6: 15–25 lbs G7: 26–35 lbs G8: > 35 lbs	For Obese Class I: OR (95% CI) for LGA were significantly lower (< 1.00, G6 was reference) for G1– G5 and significantly higher for G7–G8. For Obese Class II: OR (95% CI) for LGA were significantly lower (< 1.00, G6 was reference) for G1– G5 and significantly higher for G7–G8. For Obese Class III:OR (95% CI) for LGA were significantly lower (< 1.00, G6 was reference) for G1– G4 and significantly higher for G7–G8	Age, race, parity, education, poverty (enrollment in Medicaid, WIC, food stamp programs), tobacco use, chronic hypertension
Parker and Abrams, 1992[118] USA, hospital USA, Hospital database (California) 6,690 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories: G1: < IOM range G2: Within IOM range G3: > IOM BMI IOM	AOR (95% CI) for LGA: G3: 1.92 (1.52–2.43) G2: 1.00 (reference) Incidence of LGA in nonobese women,%: G1: 3.25 G2: 6.14 G3: 13.11 Incidence of LGA in obese women,%: G1: 5.88 G2: 17.53	Age, race, parity, pregravid BMI, height, maternal high and low weight gain, smoking, gestational age, birthweight
Stevens-Simon and McAnarney, 1992[154] USA, adolescent maternity program 141 Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories (kg/wk): G1:< 0.23 G2: 0.23–0.40 G3: > 0.40	Distribution of LGA,%: G1: 3.6 G2: 4.5 G3: 12.8 *P* = NS	NA

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Stotland et al., 2006[129] USA, university hospital 20,465 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Prenatal record	Maternal weight gain categories: G1: < IOM G2: Within IOM G3: > IOM G4: weight gain < 7kg G5: weight gain > 18kg BMI IOM	Unadjusted Rates of LGA: G1: 3.85 *P*< 0.001 vs. G2 G2: 6.62 G3:13.76 *P*< 0.001 vs. G2 G4: 5.26 G5: 14.60 *P*< 0.05 vs. G2 AOR (95% CI) for LGA: G1: 0.58 (0.47–0.72) G2: 1.00 (reference) G3: 1.98 (1.74–2.25) G4: 0.50 (0.33–0.78) G5: 2.28 (2.00–2.62)	Age, race, parity, pregravid BMI, pregnancy-induced hypertension, date of delivery, mode of delivery, length of first and second stage of labor, smoking, gestational age, birthweight
Bianco et al., 1998[54] USA, Medical Center 11,926 Nonobese (BMI 19–27) and morbidly obese (BMI > 35) Poor	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories among morbidly obese (BMI > 35): G1: Weight loss/no change G2: 1–15 lbs G3: 16–25 lbs G4: 26–35 lbs G5: > 35 lbs	Distribution of LGA,%: G1: 12.0 G2: 11.8 G3: 18.8 G4: 25.8 G5: 23.8 *P* < 0.01	Race, parity, clinic service, substance abuse, preexisting medical condition
LGA as birthweight > 4,500 gm 275				
Hedderson et al., 2006[110] USA, Kaiser Permanente Medical Care Program 45,245 All weight/BMI Good	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories: G1: < IOM G2: Within IOM G3: > IOM BMI IOM	% Distribution of maternal weight gain categories among women with macrosomia: G1: 4.0 G2: 16.3 G3: 79.7 *P* < 0.05 (compared to controls) AOR (95% CI) for macrosomia: G1: 0.38 (0.20–0.70) G2: 1.00 reference G3: 3.05 (2.19–4.26) OR (95% CI) for macrosomia: Underweight women (BMI < 19.8) G2: 1.00 (reference) G3: 2.70 (0.83–8.61) Normal weight women(BMI	Age, race, parity, pregravid BMI, screening glucose value from 1 hour after the 50g oral glucose challenge test, difference between age at delivery and gestational age at last weight measured

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
			19.8–26.0) G2: 1.00 (reference) G3: 3.60 (2.27–5.83) Overweight/obese women (BMI > 26.0) G2: 1.00 (reference) G3: 2.00 (1.14–3.47)	
Schieve et al., 1998[160] USA Pregnancy Nutrition Surveillance System - data from WIC clinics 173,006 All weight/BMI Good	Pregravid weight: Self-report Total weight gain: Self report	Maternal weight gain categories stratified by pregravid BMI (IOM-underweight, normal weight, overweight, and obese) and race (non-Hispanic white, non-Hispanic black, and Hispanic): G1: ≥ 10 lbs below IOM G2: 1–9 lbs below IOM G3: Lower half of IOM G4: Upper half of IOM G5: 1–9 lbs above IOM G6 ≥ 10 lbs above IOM	Within every BMI-race ethnicity stratum, the odds of delivering a > 4,500g infant tended to increase as weight gain increased. This trend was statistically significant for all strata; however, the trend diminished with decreasing BMI. Women in G6 were 2.2–10.8 times more likely to deliver a > 4,500 g infant compared to women in G3, irrespective of BMI status.	Age, height, education, trimester of the Special Supplemental Nutrition Program for WIC
Cogswell et al., 1995[2] USA, Pregnancy Nutrition Surveillance System 53,541 Normal/Overweight/Obese Fair	Pregravid weight: Self-report Total weight gain: Self-report	Maternal weight gain categories (lbs) stratified by pregravid BMI: Normal weight (BMI 19.8–26.0): G1: < 15 G2: 15–19 G3: 20–24 G4: 25–29 G5: 30–34 G6: 35–39 G7: ≥= 40	AOR (95% CI) for high birthweight: G1: 1.0 (0.5–2.0) G2: 0.4 (0.2–1.0) G3: 0.6 (0.3–1.1) G4: 1.0 (reference) G5: 1.1 (0.7–1.8) G6: 1.5 (1.0–2.3) G7: 3.3 (2.3–4.7) G8: 0.8 (0.2–2.6) G9: 1.0 (reference) G10: 1.1 (0.4–3.5) G11: 2.1 (0.8–5.7)	Age, race, height, smoking, gestational age, sex of infant

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
			G12: 2.4 (0.9–6.4)	
		Overweight (BMI > 26.0–29.0):	G13: 1.6 (0.6–4.6) G14: 4.0 (1.6–10.1)	
		G8: < 15		
		G9: 15–19	G15:0.7 (0.5–1.1)	
		G10: 20–24	G16: 1.0 (reference)	
		G11: 25–29	G17: 1.1 (0.7–1.7)	
		G12: 30–34	G18: 1.3 (0.8–2.0)	
		G13: 35–39	G19: 1.9 (1.3–2.9)	
		G14: ≥ 40	G20: 2.1 (1.3–3.2)	
			G21: 2.3 (1.6–3.3)	
		Obese (BMI > 29.0):		
		G15: < 15		
		G16: 15–19		
		G17: 20–24		
		G18: 25–29		
		G19: 30–34		
		G20: 35–39		
		G21: ≥ 40		
Thorsdottir 2002[53] Iceland, Hospital records 614 Normal weight/BMI 19.5–25.5 Fair	Pregravid weight: Self-report Total weight gain: Maternity records	Maternal weight gain categories: G1: < 11.5 kg G2: 11.5–16.0 kg G3: 16.1–20.0 kg G4: > 20.0 kg G5: 12.5–15.5 kg G6: > 17.8–20.8 kg	Birthweight > 4,500g,% G1: 4.3 G2: 4.1 (*P*< 0.05 between groups) G3: 9.1 (*P*< 0.05 between groups) G4: 10.2 (*P*< 0.05 between groups) *P* for trend< 0.015 RR (95% CI) for > 4,500g: G5: 1.00 (reference) G6: 3.54 (1.26–9.97)	Age, parity, height, gestational age, birthweight
LGA as birthweight > 4,000 gm				
Edwards et al., 1996[55] USA, Hospital 1,443 Normal/Obese weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories (kg) Obese > 29: G1: Lost weight/no change G2: 0.5–6.5 G3: 7–11.5 G4: 12–16 G5: > 16 Normal BMI 19.8–26	Birthweight ≥ 4,000g,%: G1:12.0 G2: 12.5 G3: 13.3 G4: 15.4 G5: 24.4 *P* (for G1–G5) = 0.026 G6: 5.7 G7: 6.6 G8: 16.9	Age, parity, pregravid BMI, GDM, pregnancy-induced hypertension, prenatal adequacy, alcohol use, drug use, smoking, gestational age

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
		G6: < 11.5kg G7: 11.5–16 G8: > 16kg	*P* (for G6–G8) < 0.001 AOR (95% CI) for birthweight ≥ 4,000g among obese women (BMI > 29.0): G3: 1.0 (reference) G8: 2.8 (1.4–5.6) AOR (95% CI) for birthweight ≥ 4,000g among normal weight women (BMI 19.8–26.0): G7: 1.0 (reference) G8: 2.4 (1.3–4.7)	
Rode et al., 2007[164] Denmark Smoke-free Newborn Study, University Hospital 2,248 All weight/BMI Fair	Pregravid weight: Self report Total weight gain: Self report	Maternal weight gain categories stratified by pregravid BMI status: BMI less than 19.8 G1: < IOM G2: Within IOM G3: > IOM BMI 19.8–26.0 G4: < IOM G5: Within IOM G6: > IOM BMI 26.1–29.0 G7: < IOM G8: Within IOM G9: > IOM BMI greater than 29.0 G10: < IOM G11: Within IOM G12: > IOM	AOR (95% CI) for birthweight ≥ 4,000g: G1: 0.8 (0.4–1.6) G2: 1.0 (reference) G3: 1.7 (0.8–3.6) G4: 0.7 (0.5–0.999) G5: 1.0 (reference) G6: 1.9 (1.5–2.5) G7: 0.6 (0.1–3.1) G8: 1.0 (reference) G9: 1.8 (0.8–3.9) G10: 0.8 (0.4–1.7) G11: 1.0 (reference) G12: 0.9 (0.4–2.0)	Preeclampsia, caffeine consumption, gestational age, smoking
Kabali et al., 2007[165] USA,/Canada, Pediatric practice 815	Pregravid weight: Self-report Total weight: Self-report	G1: < IOM G2: within IOM G3: > IOM BMI IOM	AOR G1: 1.0 (0.4, 1.9) G2: ref G3: 1.5 (0.7, 2.5) Combined effect with BMI OR (AOR similar but not all could	Maternal age, marital status, race/ethnicity, family income, years of education, smoking, alcohol, sex of child, parity, gestational age

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
All weight/BMI Poor			be calculated) Underweight/G1: 0.7 (0.2, 3.3) Underweight/G2: 1.0 (0.3, 3.5) Underweight/ G3: 1.7 (0.4, 6.4) Normal/G1: 0.7 (0.3, 1.8) Normal/G2 Ref Normal/ G3: 1.1 (0.5, 2.3) Overweight/G1: 1.2 (0.4, 3.8) Overweight/G2: 0.8 (0,2, 2.7) Overweight/G3: 2.4 (1.2, 4.8)	

AOR: adjusted odds ratio; BMI, body mass index; CI, confidence interval; G, group; g, gram; GDM: gestational diabetes; IOM, Institute of Medicine; kg/wk, kilogram per week; lbs, pounds; LBW, low birthweight; NICU, neonatal intensive care unit; PRAMS, Pregnancy Risk Assessment Monitoring System; USA, United States of America; WIC, The Special Supplemental Nutrition Program for Women, Infants, and Children.

Detailed results for LGA infant weight One study reported the risk of LGA among women of all weight status groups[129] and another among nonobese women (BMI < 30).[118] In both studies, the risk for LGA was nearly doubled for women who gained above the IOM guidelines. For women who gained below the IOM guidelines, the risk for LGA was decreased by close to 40 percent.[129]

For women of normal pregravid weight, the odds of LGA estimated from an adjusted model found a nonsignificant increased risk of having an LGA infant.[55] In another study, the risk was twofold higher and statistically significant for women gaining more than IOM recommendations.[25] This same study found that the odds of LGA was decreased by more than 60 percent with gains below the IOM guidelines for normal-weight women.

Among obese women, the risk of LGA was 2.3 times greater for nonsmokers gaining in excess of the IOM guidelines, but this was not true among obese smokers.[55,159] In a study that grouped women into classes of obesity,[4] the odds of LGA increased with weight gains above 25 pounds for all classes of obesity.

Two studies examined the impact of rate of weight gain according to the IOM guidelines on having an LGA infant.[116,154] One good study defined the rate of weight gain in increments of 50 g per week.[116] The AORs associated with having an LGA infant for each increment were as follows: 1.25 for normal-weight women, 1.14 for overweight women, and 1.13 for obese women. Using these AORs, the authors calculated the expected change in the incidence of LGA if weight gains remained within the IOM guidelines. These changes were -0.77 percent for black women and -2.87 percent for white women; baseline LGA incidence rates were 4.8 percent and 14.8 percent, respectively. The other study investigated rate of weight gain among black adolescents with no difference in pregravid weight status.[154] In bivariate analysis the prevalence of LGA did

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not differ between mothers who were slow weight gainers (< 0.23 kg/week) or rapid weight gainers (> 0.4 kg/week) and mothers who were average weight gainers (0.23 to 0.4 kg/week).

With respect to LGA defined as > 4,500 g, the one study reporting risk estimates for women of all weight groups found that weight gain above the IOM guidelines was associated with a threefold increased risk of LGA after adjustment for various confounders.[110] Women who gained less than the recommendation were 62 percent less likely to have an LGA infant than women who gained within the recommended range.

Analyses for normal-weight women showed a threefold increased risk of LGA with weight gains above the IOM guidelines[110] or at > 40 pounds[2,53] after adjusting for multiple confounders. Overweight and obese women who gained more than the IOM guidelines had twice the risk of having an LGA infant in one study,[110] and in another study,[2] they did not have a significantly increased risk until weight gains exceeded 40 pounds for overweight women (AOR, 4.0; 95% CI, 1.6–10.1) and 30 pounds for obese women (AOR ranged from 1.9 to 2.3).

Low weight gains were not significantly associated with LGA risk in any of these studies.[2,53,110,160] In one study that stratified results by weight status and race across all BMI and racial groups,[160] the risk of LGA was significantly higher with total weight gains 10 pounds more than the IOM recommendation. Weight gains below the IOM guidelines were protective only among white women across all BMI weight status groups.

Detailed results for high birthweight or macrosomia Three studies (1 poor-quality[165]) defined high birthweight or macrosomia as > 4,000 g.[55,164] All stratified results by pregravid weight status. For normal-weight women, those who gained more than the IOM guidelines were at a statistically significant increased risk in the two studies of fair quality.[55,164] Normal-weight women who gained below the guidelines were at decreased risk in one study.[164] For obese women, one study found no difference in the risk of macrosomia with weight gains either above or below the IOM guidelines;[164] the other found that those who gained above the IOM guidelines had 2.8 times the risk for a macrosomic infant relative to those who gained within the recommended range.[55] For underweight and overweight women, weight gains above or below the IOM guidelines were not associated with delivering a macrosomic infant,[164] although women with weight gains above the guidelines appeared to have a slightly increased risk.

Fetal growth (small for gestational age)

Study characteristics Ten articles examined the association of gaining weight according to the IOM guidelines and having an SGA infant (Evidence Table 45, Table 35).[4,25,54,55,116,118,129,153,154,159] Two studies were conducted among black adolescents.[153,154] The majority used hospital databases[54,55,116,118,129,153,159] or clinic databases[153] as the source of their information; one study (2 articles) used birth certificate information;[4,25] and one was a cohort study.[154] All studies used a definition of < 10th percentile to define SGA, but reference populations differed across these studies.

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TABLE 35. Weight change relative to IOM thresholds and small-for-gestational-age

Author, Year Country, Setting Sample Size Baseline BMI Quality	**Pregravid Weight (How Measured) Total Weight Gain (How Measured)**	**Definition of Groups**	**Results**	**Confounders and Effect Modifiers Included in Analysis**
Caulfield et al., 1998[116] USA, University Hospital 3,870 All weight/BMI Good	Pregravid weight: Self report Total weight gain: Measured	G1: Underweight, BMI < 19.8 G2: Normal weight, BMI 19.8–26.0 G3: Overweight, BMI > 26.0 Black women: G4: No weight gain < IOM G5: No weight gain > IOM White women: G6: No weight gain < IOM G7: No weight gain > IOM	AOR (95% CI) for SGA and rate of weight gain (per 50 g/wk): G1: 0.87 (0.78–0.97) G2: 0.90 (0.84–0.96) G3: 0.93 (0.86–1.01) Expected absolute change (as % of baseline) in Incidence of SGA associated with modifiable risk factors (G4–G7): G4: -1.17 (-16) G5: +0.97 (+13) G6: -0.44 (-11) G7: +0.60 (+15)	Age, race, parity, pregravid BMI, height, hypertension, provider type, smoking, female infant
Hellerstedt et al., 1997[159] USA, medical center 1,343 Normal/obese BMI Good	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories stratified by pregravid BMI and smoking status: Obese (BMI > 29.0): G1: Smokers, < IOM G2: Smokers, within IOM G3: Smokers, > IOM G4: Nonsmokers, < IOM G5: Nonsmokers, within IOM G6: Nonsmokers, > IOM Normal weight (BMI 19.8–26.0): G7: Smokers, < IOM G8: Smokers, within IOM G9: Smokers, > IOM G10: Nonsmokers, < IOM G11: Nonsmokers, within IOM G12: Nonsmokers, > IOM Obese:	Frequencies of SGA,%: G1: 13.3 G2: 10.0 G3: 7.7 G4: 5.5 G5: 4.7 G6: 3.6 G7: 28.6 G8: 10.9 G9: 3.6 G10: 8.9 G11: 6.5 G12: 6.4 G13: 10.7 G14: 6.6 G15: 6.0 G16: 4.0 G17: 5.3 *P* = 0.115 for G13–G17 G18: 15.9 G19: 7.5 G20: 5.7 *P* = 0.001 for G18–G20 For obese women,	Maternal age, pregravid BMI, infant sex, race, parity, prenatal alcohol use, prenatal illicit drug use, adequacy of prenatal care, gestational hypertension, GDM, gestational age

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
		G13: Lost/no gain G14: 0.5–6.5 kg G15: 7–11.5 kg G16: 12–16 kg G17: > 16 kg Normal weight: G18: < 11.5 kg G19: 11.5–16 kg G20: > 16 kg	compared to nonsmokers who gained 7–11.5 kg, smokers who gained < 7 kg were at significantly higher risk of SGA OR: 3.2 (95% CI, 1.1–10.1) For normal weight women, compared to nonsmokers who gained 11.5–16 kg, smokers who gained < 11.5 kg were at significantly higher risk of SGA OR: 4.3 (95% CI, 1.8–10.3)	
Nielsen et al., 2006[153] USA, hospitals (African American adolescents) 815 All weight/BMI Good	Pregravid weight: Self-report Total weight gain: Measured	G1: BMI < 19.8 G2: BMI 19.8–26.0 G3: BMI > 26.0 G4: < IOM G5: Lower half of IOM G6: Upper half of IOM G7: > IOM	SGA,%: G1: 22.3 G2: 15.6 G3: 11.5 *P* < 0.01 for G1–G3 AOR (95% CI) for SGA: G4: 2.31 (1.22–4.37) G5: 1.00 (reference) G6: 0.88 (0.41–1.89) G7: 0.68 (0.34–1.35) *P* < 0.01 for G4–G7	Parity, pregravid BMI, time between last weight measure and delivery, height
Devader et al., 2007[25] USA, birth certificate data 94,696 Normal weight/BMI 19.8–26 Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories (lbs): G1: < 25 G2: 25–35 G3: > 35	AOR (95% CI) for SGA: G1: 2.14 (2.01–2.27) G2: 1.0 (reference) G3: 0.48 (0.45–0.50)	Age, race, education, income, alcohol use, height, prior pregnancy, inadequate prenatal care use, smoking, child's gender, birth year
Edwards et al., 1996[55] USA, hospital	Pregravid weight: Self-report	Obese BMI > 29 (kg): G1: Lost weight/no change G2: 0.5–6.5 G3: 7–11.5	% SGA for obese: G1: 10.7% G2: 6.6% G3: 6.0%	Age, parity, pregravid BMI, GDM, PIH, prenatal adequacy, alcohol use, drug use, smoking, gestational

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
1,443 Normal/Obese weight/BMI Fair	Total weight gain: Measured	G4: 12–16 G5: > 16 Normal weight 19.8–26 G6: < 11.5 G7: 11.5–16 G8: > 16	G4: 4.0% G5: 5.3% *P* = 0.11 For normal weight: G6: 15.9% G7: 7.5% G8: 5.7% *P* = 0.001 AOR (95% CI) Obese: G1 vs. G3 2.9 (1.1–8.4) Normal weight: G6 vs. G7 1.7 (0.9–3.4)	age
Kiel et al., 2007[4] USA, birth registry 120,170 Obese BMI > 30 Fair	Pregravid weight: Self-report Total weight gain: Medical record	Maternal weight gain categories stratified by prepregnancy obesity status, Obese Class I (BMI 30–34.9), Obese Class II (BMI 35–39.9), Obese Class III (BMI ≥ 40): G1: ≤ -10 lbs G2: -2 to -9 lbs G3: No change G4: 2–9 lbs G5: 10–14 lbs G6: 15–25 lbs G7: 26–35 lbs G8: > 35 lbs	For Obese Class I: AOR (95% CI) for SGA were significantly greater (> 1.00, G6 was reference) for G1– G5 and significantly lower for G7–G8. For Obese Class II: AOR (95% CI) for SGA were significantly greater (> 1.00, G6 was reference) for G1– G5 and significantly lower for G7–G8 For Obese Class III: AOR (95% CI) for SGA were significantly greater (> 1.00, G6 was reference) for G1 and G3 and significantly lower for G7–G8	Age, race, parity, education, poverty (enrollment in Medicaid, WIC, food stamp programs), tobacco use, chronic hypertension
Parker and Abrams, 1992[118] USA, hospital (California) 6,690 All weight/BMI	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories: G1: < IOM G2: Within IOM G3: > IOM BMI IOM	AOR (95% CI) for SGA: G1: 1.78 (1.39–2.27) G2: 1.00 (reference) Incidence of SGA in nonobese women,%: G1: 3.25 G2: 6.14 G3: 13.11	Age, race, parity, pregravid BMI, height, maternal high and low weight gain, smoking, gestational age, birthweight

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Fair			Incidence of SGA in obese women,%: G1: 11.76 G2: 3.09	
Stevens-Simon and McAnarney, 1992[154] USA, adolescent maternity program 141 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories (kg/wk): G1: < 0.23 G2: 0.23–0.40 G3: > 0.40	Distribution of SGA, %: G1: 7.1 G2: 9.1 G3: 2.1 *P* = NS	None
Stotland et al., 2006[129] USA, university hospital 20,465 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Prenatal record	Maternal weight gain categories: G1: < IOM G2: Within IOM G3: > IOM G4: < 7 kg G5: > 18 kg	Unadjusted rates of SGA: G1: 11.74 *P*< 0.001 vs. G2 G2: 7.05 G3: 3.70 *P*< 0.001 vs. G2 G4: 13.99 *P*< 0.05 vs. G2 G5: 3.87 *P*< 0.05 vs. G2 AOR (95% CI) for SGA: G1: 1.66 (1.44–1.92) G2: 1.00 (reference) G3: 0.51 (0.44–0.59) G4: 2.26 (1.76–2.90) G5: 0.50 (0.42–0.60)	Age, race, parity, pregravid BMI, PIH, date of delivery, mode of delivery, length of first and second stages of labor, smoking, gestational age, birthweight
Bianco et al., 1998[54] USA, medical center 11,926 Nonobese (BMI 19–27) and morbidly obese (BMI > 35)	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories among morbidly obese (BMI > 35): G1: Weight loss/no change G2: 1–15 lbs G3: 16–25 lbs G4: 26–35 lbs G5: > 35 lbs	Distribution of SGA,%: G1: 4.0 G2: 3.9 G3: 5.6 G4: 3.1 G5: 3.8 *P* = No testing due to small numbers in each cell	Race, parity, clinic service, substance abuse, preexisting medical condition

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Poor				

AOR, adjusted odds ratio; BMI, body mass index; CI, confidence interval; G, group; GDM, gestational diabetes mellitus; kg, kilogram; IOM, Institute of Medicine; NS, not sufficient; OR, odds ratio; PIH, pregnancy-induced hypertension; SGA, small for gestational age; USA, United States of America; WIC, The Special Supplemental Nutrition Program for Women, Infants, and Children.

Overview of results for SGA infant weight Evidence from 10 studies (3 good,[116,153,159] 6 fair,[4,25,55,118,129,154] one poor[54]) supports an association between weight gains below the recommended IOM guidelines and the risk of having an SGA infant.

Detailed results of SGA infant weight With respect to gaining less than the IOM guidelines, two studies found statistically significant higher odds for women giving birth to an SGA infant across all pregravid BMI categories.[118,129] One of these studies also examined the odds for excessive weight gain, which was statistically significantly protective.[129]

Among normal-weight women, two studies found that excessive weight gain decreased the SGA risk by half, whereas inadequate weight gain doubled the SGA risk.[25,55] Among obese women, those who gained below the IOM guidelines were at nearly three times the risk of having an SGA infant[55,118,159] compared with those who gained within the recommended range. In the one study that was able to examine classes of obesity (Class I, BMI 30.0–34.9; Class II, BMI 35.0–39; and Class III, BMI ≥ 40), the risk of SGA increased for all classes in a linear fashion as weight gain fell below the IOM recommendation of at least 15 to 25 pounds.[4]

The good study conducted among black adolescents that examined total weight gain[153] found an increased odds for SGA associated with gaining less than the IOM guidelines compared with gaining at the lower half of the guidelines (AOR, 2.31; 95% CI, 1.22–4.37) and no significantly protective effect with weight gains in the upper half or greater than the IOM.

Two studies examined the rate of weight gain.[116,154] One, among black adolescents,[154] found no difference in the prevalence of SGA among rate of weight gain categories (slow, < 0.23 kg/week; average, 0.23 to 0.40 kg/week; and rapid, > 0.4 kg/week). In the other study, after adjustment for multiple confounders, increasing rates of weight gain were associated with a reduced risk of SGA.[116] This study calculated the expected change in the incidence of SGA by preventing inadequate weight gain to be -1.17 percent and -0.44 percent for black and white women, respectively.

Apgar Scores

Study characteristics Three studies, all rated fair quality, dealt with Apgar scores (Evidence Table 46; Table 36).[125,129,154]

Overview of results Three fair studies provide insufficient evidence to support an association between weight gain and low Apgar scores.

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TABLE 36. Weight change relative to IOM thresholds and Apgar scores

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Nixon et al., 1998[125] USA, county nurse-midwifery services 2,228 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Data records	Gestational weight gain categorized by IOM recommendations BMI IOM	Maternal weight gain by IOM guidelines was not not a signigicant predictor of Apgar scores (details-none reported)	None
Stevens-Simon and McAnarney, 1992[154] USA, adolescent maternity program 141 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories (kg/wk): G1: < 0.23 G2: 0.23–0.40 G3: > 0.40	Distribution of 1-minute Apgar score ≤ 4,%: G1: 25.0 G2: 4.5 G3: 14.9 *P* = 0.02 for G1 vs. G2 or G3 Distribution of 5-minute Apgar score ≤ 4,%: G1: 3.5 G2: 0 G3: 0 *P* = NS	None
Stotland et al., 2006[129] USA, university hospital 20,465 All weight/BMI (using IOM definitions) Fair	Pregravid weight: Self-report Total weight gain: Prenatal records	Maternal weight gain categories: G1: < IOM G2: Within IOM G3: > IOM G4: < 7 kg G5: > 18 kg	Unadjusted rates of 5-minute Apgar score < 7: G1: 1.94 G2: 1.58 G3: 2.14 (*P*< 0.05, G3 vs. G2) G4: 2.39 G5: 2.16 (*P*< 0.05, G5 vs. G2) AOR (95% CI) for 5-minute Apgar score < 7: G1: 1.18 (0.84–1.66) G2: 1.00 (reference) G3: 1.33 (1.01–1.76) G4: 1.29 (0.70–2.39)	Age, race, parity, pregravid BMI, pregnancy-induced hypertension, date of delivery, mode of delivery, length of first and second stage of labor, smoking, gestational age, birthweight

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
			G5: 1.30 (0.95–1.77)	

BMI, body mass index; G, group; IOM, Institute of Medicine; kg/wk, kilogram per week; NS, not significant; USA, United States of America.

Detailed Results on Apgar Scores Three studies included investigation of Apgar scores and adherence to the IOM recommendations.[125,129,154] In one study,[129] total weight gain above the IOM guidelines increased the risk of having a 5-minute Apgar score of < 7 by 33 percent (AOR, 1.33; 95% CI, 1.01–1.76), whereas a weight gain below the IOM guidelines was not associated with a low 5-minute Apgar score (AOR, 1.18; 95% CI, 0.84–1.66). Another study[125] found no effect of maternal weight gain with the outcome defined as a 1-minute Apgar score of < 7. The third study[154] was conducted among black adolescents and found a slow rate of weight gain (< 0.23 kg/week) to be associated with a 1-minute Apgar score of ≤ 4 compared to higher rates of weight gain (> 0.23 kg/week).

Infant Outcomes

Perinatal mortality

Study characteristics One U.S. study of a hospital database examined perinatal mortality (Evidence Table 47).[127] The study included overall perinatal mortality and adverse perinatal outcome, which was defined as an infant death between delivery and discharge, delivery before 37 completed weeks of gestation, LBW, or stillbirth.[127]

Overview of results One fair study did not conduct multivariable modeling using the IOM cutpoints, and therefore provides weak evidence on the association between weight gain and perinatal mortality.[127]

Detailed results The authors reported, using only bivariate analysis, that infants of mothers who gained below the IOM recommendations had a significantly higher proportion of adverse perinatal outcomes (14 percent) and perinatal mortality (1.1 percent) than the infants whose mothers gained within or above the recommendations (8.5 percent and 0.4 percent respectively; P < 0.001 for all comparisons).

Infant hypoglycemia

Study characteristics Two studies from hospital databases examined hypoglycemia in the infant (Evidence Table 48).[110,129] One study had many other outcomes such as birth trauma, admission to the special care nursery, neonatal infection, seizure, polycythemia, meconium aspiration syndrome, respiratory distress syndrome, and a hospital stay of 5 and 10 days.[129]

Overview of results Two studies, of good[110] and fair quality,[129] respectively, found moderate evidence that high maternal weight gain is associated with an increased risk of neonatal hypoglycemia for weight gain above IOM recommendations and were consistent in demonstrating a lack of association between weight gain below IOM recommendations and neonatal hypoglycemia.

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Detailed results Two studies included infant hypoglycemia as an outcome of interest.[110,129] The good study used a case-control design for women who delivered singletons at Kaiser Permanente Medical Center from 1996 to 1998.[110] Cases (N = 328) were defined as infants with plasma glucose < 40 mg/dl; controls were infants born to women with no GDM. Cases had a significantly higher odds of having mothers who gained more than the IOM guidelines (AOR, 1.38; 95% CI, 1.01–1.89); weight gains below the IOM were not associated with infant hypoglycemia.

Stratification by race (in the good study) showed that among infants born to non-Hispanic white women, a pregnancy weight gain below the IOM guidelines was significantly associated with a decreased odds of hypoglycemia (OR, 0.39; 95% CI, 0.18–0.84); among infants born to women of minority groups (undefined), weight gain below the IOM guidelines was significantly associated with an increased risk of hypoglycemia (OR, 1.69; 95% CI, 1.08–2.64).[110] This study also stratified by pregravid BMI and did not find any significant effect that suggested the effect of weight gain varied by pregravid BMI.

The second study reported that women who gained above the IOM guidelines were significantly more likely to have an infant with hypoglycemia (AOR, 1.52; 95% CI, 1.06–2.16)[129] but that women with weight gain below the guidelines had no such association. This study found significant associations only for weight gains above the IOM and the following outcomes: infant seizure (AOR, 6.5; 95% CI, 1.43–29.65), polycythemia (AOR, 1.44; 95% CI, 1.06–1.94), and meconium aspiration (AOR; 1.79, 95% CI, 1.12–2.86). Data were adjusted for maternal race, prepregnancy BMI, parity, age, gestational hypertension, smoking, gestational age at delivery, model of delivery, length of each stage of labor, and birthweight.

NICU admissions

Study characteristics Two studies, rated fair, dealt with admission to the neonatal intensive care unit (NICU); (Evidence Table 49).[129,154] One study was a cohort of black adolescents.154

Overview of results Two fair studies using different measures of weight gain provided weak inconsistent evidence on neonatal hospitalization.

Detailed results on admission to NICU One study found that decreased risk of NICU admission was significantly associated with weight gain below IOM guidelines (AOR, 0.66; 95% CI, 0.46–0.96) but not with weight gains above the IOM guidelines (AOR, 1.03; 95% CI, 0.79–1.35).[129] In the other study, among black adolescents a slow rate of weight gain (< 0.23 kg/week) was significantly associated with NICU admission (P = 0.01).[154]

Child Outcomes

Childhood weight status

Study characteristics Only one study of fair quality was found[24] that examined weight gain according to the IOM and childhood weight status (Evidence Table 50). This study involved 1,585 women from a single HMO in Boston who were part of pregnancy study and then enrolled in a follow-up study. A total of 1,110 children completed a visit at age 3, at which time study staff measured their weight and height; maternal weight and pregravid weight status were obtained via questionnaire. This study did not specify singleton-only births, but it did note that preterm births and infants weighing < 2,500 kg were excluded because of their different growth trajectories in the first year of life. Maternal weight gain was calculated as the difference between weight measured near delivery obtained from the prenatal record and self-reported pregravid weight. The study reported on the effect of total weight gain, net weight gain

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(excluding infant birthweight) and weight gain classified by IOM guidelines. Child BMI percentiles were grouped as follows: below 50th (referent category), 50th to 84th, 85th to 94th, and 95th or higher.

Results Using children born to women who gained inadequately as the referent, children born to women who gained adequately or excessively had higher odds of being in higher percentile categories. The AORs for children born to women who gained adequately were as follows: 50th to 84th percentile, 1.85 (1.17–2.92); 85th to 94th percentile, 2.09 (1.12–3.92), and 95th percentile and above, 3.77 (1.38–10.27). AORs for children born to mothers who gained excessively were similar: respectively, 1.84 (1.17–2.88), 2.03 (1.11–3.72) and 4.35 (1.69–11.24) Both models adjusted for maternal pregravid BMI, prenatal smoking, race/ethnicity, household income, martial status, glucose tolerance, paternal BMI, gestational length, and child's sex.

Short- and Long-term Maternal Outcomes

Lactation performance

Study characteristics Three studies (four articles) reported on the effects of weight gain on lactation performance (Evidence Table 51, Table 37).[166–169] One study was done using the Danish National Birth Cohort;[166] another study (2 articles) used a U.S. hospital database for years 1988 to 1997;[168,169] and the third used data from the U.S. Pediatric Nutrition Surveillance System and the Pregnancy Nutrition Surveillance System.[167] Lactation performance was defined as initiated breastfeeding,[167,168] duration of any breastfeeding,[166–168] and exclusive breastfeeding.[166,168,169] Weight gain was defined as the difference between weight at delivery and self-reported pregravid weight[168,169] or was based simply on self-reported total weight gain.[166,167] The two U.S. studies used total weight gain as categorical variables corresponding to the IOM guidelines;[167–169] the Danish study used categories corresponding to the following cutpoints: < 8 kg, 8 to 15.9 kg (the reference group), and ≥ 16 kg.[166]

Overview of results These studies (all fair quality) support an association between weight gains below the IOM guidelines and lower likelihood of breastfeeding initiation; they also suggest a shorter duration of exclusive breastfeeding among obese women. They provide only inconsistent evidence of an association between weight gain in relation to the IOM guidelines and initiation of breastfeeding.

TABLE 37. Weight change relative to IOM thresholds and breastfeeding

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Baker et al., 2007[166]	Pregravid weight: Prepregnant weight Self reported	G1: < 8 kg G2: 8–15.9 kg G3: ≥ 16 kg	Overall higher risk of terminating full or any breastfeeding with higher pregravid BMI.	BMI
Denmark-National Birth			Unadjusted RR full BF G1: 1.13 (95% 1.08–1.18) G3: 1.05 (1.03–1.08).	
37,459	Total weight gain:		Any BF	
All wt/BMI	Self-reported		G1: RR 1.16 (1.11–1.22)	

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Under wt BMI < 18.5 Normal BMI 18.5–24.9 Overweight BMI 25–29 Obese BMI ≥ 30 Fair			G3: 1.05 (1.03–1.08). GWG not a predictor of full or any when BMI was in the model.	
Li et al., 2003[167] USA WIC clinics 51,329 All wt/BMI (using IOM definitions) Fair	Pregravid weight: Prepregnant weight Self reported Total weight gain: Self-reported	G1: < IOM G2: within IOM G3: > IOM	Adjusted OR for failure to initiate BF by BMI: Under, normal and overweight G1: groups had a significant increased odds of failure to initiate BF compared to G2: within BMI strata. Obese women regardless of weight gain had increased odds of failure to initiate compared to normal wt G2. Adjusted mean duration of BF ($P < 0.01$)* G1: 12.9 wk* G2: 13.6 wk (ref) G3: 12.8 wk*	Age, race, parity, pregravid BMI, maternal education, marital status, prenatal care, poverty-income ratio, gestational weight gain, smoking, gestational age, birthweight
Rasmussen et al., 2002[168] Hilson et al., 2006[169] USA, hospital 2,494 All wt/BMI (using IOM definitions) Fair	Pregravid weight: Prepregnant weight Self reported Total weight gain: Prenatal records	G1: < IOM G2: within IOM G3: > IOM	OR unsuccessful initiation of BF (normal wt G2: ref) Underweight no significant diff Normal wt G3: 1.66 (1.05–2.63) Overwt no significant diff Obese G3: 2.89 (1.78–4.69) Hazard OR discontinuing exclusive BF (normal wt G2: ref) Underwt G3: 1.39 (1.01–1.92) Normal wt-no signif differences Overwt G3: 1.27 (1.03–1.56) Obese G1: 1.37 (1.01–1.84) G2: 1.50 (1.11–2.03) G3: 1.78 (1.48–2.14) Hazard OR discontinuing any BF (normal wt G2: ref) Underwt-no sign difference	Age, parity, participation in WIC/PCAP, type of delivery, mother attended college, smoking

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
			Normal wt-no sign difference Overwt- no sign difference Obese G2: 1.57 (1.14–2.18), G3: 1.99 (1.64–2.43)	

BF, breastfeeding; BMI, body mass index; G, group; IOM, Institute of Medicine; OR, odds ratio; overwt, over weight; PCAP, Prenatal Care Assistance Program; RR, relative risk; wt, weight; WIC, The Special Supplemental Nutrition Program for Women, Infants, and Children.

Detailed results on breastfeeding initiation Obese women, regardless of weight gain, had higher odds of never initiating breastfeeding than women of normal weight in one US study.[167] For women who were underweight or of normal weight, greater weight gain was associated with a lower odds of never initiating breastfeeding; for overweight and obese women, there was no such association.[167] Finally, for all three categories of women classified by BMI, weight gain below the IOM guidelines (as compared with weight gain within the guidelines) was associated with higher odds of never initiating breastfeeding.

The second study (2 articles) examined initiation of breastfeeding at 4 days postpartum among women who intended to breastfeed.[168,169] Compared with normal-weight women who gained within the IOM guidelines, normal-weight women who gained more than the IOM guidelines and obese women regardless of weight gain had significantly higher odds of not breastfeeding.[168] Among obese women, unsuccessful initiation of breastfeeding was limited to those who gained more than IOM guidelines[169] compared with normal-weight women who gained within the guidelines. This study also reported a nonsignificant tendency of failing to initiate breastfeeding successfully with weight gain less than the IOM guidelines.

Detailed results on duration of exclusive breastfeeding The two studies (three articles) examining the length of exclusive breastfeeding all showed statistically significant shorter durations among obese women.[166,168,169] The association between weight gain and duration of full breastfeeding did not differ by BMI status in two studies.[166,169]

U.S. women who gained above the IOM guidelines had a statistically significant shorter median duration of exclusive breastfeeding than women who gained within the guidelines according to multivariate models.[169] For those who gained above the guidelines, the median duration of exclusive breastfeeding was 1 week shorter for underweight and overweight women and 3 weeks shorter for obese women.

In the Danish study,[166] weight gain was a statistically significant predictor of full breastfeeding at 1, 16, and 20 weeks postpartum. In unadjusted models, both low weight gain (< 8 kg) and high weight gain (≥ 16 kg) were associated with early termination of full breastfeeding Once the authors adjusted for pregravid BMI, however, this association was no longer significant.

Detailed results on duration of any breastfeeding Shorter duration of any breastfeeding was associated with maternal obesity.[166,167,169]

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In the two U.S. studies, gaining weight above the IOM guidelines was associated with shorter duration of any breastfeeding (in the range of 1 to 2.5 weeks less) in bivariate and multivariate analysis.[167,169] In one study, gaining weight below the IOM guidelines was also associated with shorter length of any breastfeeding (~1 week).[167]

In the Danish study, weight gain was a statistically significant predictor of terminating any breastfeeding at 16 and 20 weeks postpartum but not at 1 week.[166] In unadjusted models, both low and high weight gains were associated with early termination of any breastfeeding. Once models were adjusted for pregravid BMI, this finding was no longer significant.

Fat retention

Study characteristics Two studies in the United States examined differences in the amount of fat retained in the postpartum period by IOM categories of weight gain (Evidence Table 52).[16,97] One study reported on 63 pregnant women (17 underweight, 34 normal weight, 12 overweight/obese) from a convenience sample of 124 nonsmoking women ages 18 to 40.[97] The study conducted body composition measurements using dual-energy x-ray absorptiometry both before and after pregnancy and weighed the women before, during, and after pregnancy. The second study was conducted among a convenience sample of 196 nonsmoking women between 19 and 36 years recruited from three prenatal clinics. These investigators used self-reported pregravid weight and conducted body composition measurements starting at 12 to 16 weeks of gestation, at 37 weeks, and/or at 2 to 4 weeks postpartum with hydrodensitometry (underwater weighing) and deuterium dilution volume.[16] Total body bone mineral was measured at 2 to 4 weeks postpartum using dual-energy absorptiometery. They applied a four-compartment model (incorporating measurements of total body water, body density, body weight, and bone mineral content) to estimate total body fat.

Overview of results Evidence from two fair studies suggests that fat retention was higher among women whose weight gains exceeded IOM guidelines.

Detailed results In one study, fat retention was significantly higher among women who gained above the IOM guidelines (5.3 kg) than among women who gained within (2.3 kg) or below (-0.5kg).[97] In the second study, changes in body fat from 14 to 37 weeks of gestation stratified by pregravid BMI showed that women who gained below the IOM guidelines had the lowest amount of fat gain; those within an intermediate level and those above had the highest fat gain.[16] The investigators did not report significance tests. Among obese women who gained within the IOM guidelines, the percentage of body fat change (-0.6 kg) was significantly lower than among other BMI groups who also gained within the recommendations (6.0 kg for underweight, 3.8 kg for normal weight, and 2.8 kg for overweight women).

Short-term weight retention

Study characteristics Four studies examined weight gain and weight retention in the short term[104,154,158,170] (Evidence Table 53, Table 38). Three studies reported on results at 6 weeks postpartum.[154,158,170] All used a cohort design involving mostly low-income women; two included Hispanic, black, and white women,[158,170] and the third included only black adolescents.[154] One study used total weight gain as the exposure;[170] two examined the rate of weight gain.[154,158] A fourth study examined the possible association 2 days after term delivery (37 to 43 weeks' gestation).[104]

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Overview of results Evidence from four fair studies supports an association between weight gain in excess of the IOM recommendations and higher weight retention in the immediate postpartum period.[104,154,158,170]

TABLE 38. Weight change relative to IOM thresholds and short-term weight retention

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Luke et al., 1996[104] USA, clinic 487 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain < IOM recommendations: G1: BMI < 19.8 G2: BMI 19.8–26.0 G3: BMI > 26.0 Maternal weight gain within IOM recommendations: G4: BMI < 19.8 G5: BMI 19.8–26.0 G6: BMI > 26.0 Maternal weight gain > IOM recommendations: G7: BMI < 19.8 G8: BMI 19.8–26.0 G9: BMI > 26.0	Mean (SEM) retained weight (defined as 2-day postpartum weight minus pregravid weight, kg): G1: 3.2 (0.5) $P < 0.05$ compared to G4 G2: 0.8 (0.4) $P < 0.05$ compared to G5 G3: -5.0 (0.7) $P < 0.05$ compared to G6 G4: 8.2 (0.7) G5: 7.0 (0.4) G6: 1.4 (0.8) G7: 15.5 (0.9) $P < 0.05$ compared to G4 G8: 12.9 (0.4) $P < 0.05$ compared to G5 G9: 9.5 (0.5) $P < 0.05$ compared to G6	Age, parity, race, smoking, gestation duration, fetal sex
Scholl et al., 1995[158] USA Camden Study 274 Normal weight/BMI 19.8–26 Fair	Pregravid weight: Self report Total weight gain: Measured	Maternal weight gain categories (kg/wk): G1: ≤ 0.34 G2: > 0.34–0.68 G3: > 0.68	Mean (SEM) change in weight (kg) from pregravid to 6 weeks postpartum: G1: 3.1 (0.80) G2: 3.9 (0.51) G3: 9.4 (0.70) $P < 0.001$, G3 vs. G1,G2	Age, parity, race, height, lactation status, smoking
Stevens-Simon and McAnarney, 1992[154] USA, adolescent	Pregravid weight: Self-report Total weight gain:	Maternal weight gain categories (kg/wk): G1:< 0.23 G2: 0.23–0.40 G3: > 0.40	Short term weight retention (2–11 weeks postpartum), total kg: G1: -1.7 (SD 2.9) G2: 2.9 (SD 2.9) G3: 9.6 (SD 5.6) $P < 0.0001$	Age, pregravid BMI, level of physical activity, timing of first prenatal and postpartum visits substance use, body habitus

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
maternity program 141 All weight/BMI Fair	Measured		AOR (95% CI) for subsequent maternal obesity (> 120% ideal weight for height): G3: 190.94 (7.55–4,779.02)	
Walker et al., 2004[170] USA, Austin New Mothers Study 419 All weight/BMI (using IOM definitions) Fair	Pregravid weight: Self-report Total weight gain: Self-report	Maternal weight gain categories: G1: < IOM G2: Within IOM G3: > IOM	Mean (SD) weight (kg) retained at 6 weeks postpartum: G1: -0.34 (3.44) G2: 3.86 (3.45) G3: 10.55 (6.14) *P* = 0.000 % Women who attained pregravid weight at 6 weeks postpartum: G1: 48.8 G2: 14.3 G3: 2.3 Correlation of gestational weight gain, excluding infant weight, (continuous variable) and weight retained at 6 weeks postpartum: r = 0.90 *P* = 0.000 Multiple regression analysis predicted a mean increase in retained weight of 0.88 kg for each 1 kg increase in maternal weight gain (B = 0.88, SE = 0.02, *P* = 0.000)	Race, parity, pregravid BMI, gestational weight gain, gestational age

B,beta; BMI, body mass index; CI, confidence interval; G, group; IOM, Institute of Medicine; kg, kilogram; kg/wk, kilogram per week; SD, standard deviation; SE, standard error; SEM, standard error of mean.

Detailed results In one study, women who gained more than recommended levels retained, at 6 weeks, statistically significantly more weight than women who gained within or below IOM guidelines.[170] The 2-day post-delivery analyses, stratified by pregravid BMI, showed that for each BMI grouping, women who gained above the IOM guidelines retained statistically significantly more weight than women who gained within the guidelines; women who gained below the IOM guidelines retained significantly less than those who gained within them.[104]

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Two studies examined the rate of weight gain. One defined < 0.23 kg per week as slow weight gain and > 0.4 kg per week as rapid,[154] and the other defined low as < 0.34 kg per week and excessive as > 0.68 kg week.[158] In both studies, the amount of weight retained was highest among women who had an excessive rate of weight gain compared with women who had lower rates.[154,158]

Weight retention during the first year postpartum

Study characteristics Six studies examined the effect of weight gain according to IOM classifications on weight retained during the first year postpartum (Evidence Table 54, Table 39).[158,171–175] Five studies were from the United States, and one was from Sweden.[171] Five used a cohort design;[158,171,172,174,175] one U.S. study was done in a representative sample of births.[173]

Overview of results The evidence from five fair studies[158,171,172,174,175] and one poor study[173] supports an association between excessive weight gain and weight retention within the first year postpartum.

TABLE 39. Weight change relative to IOM thresholds and weight retention during the first year ostpartum

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Amorim et al., 2007[171] Sweden, hospital 483 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Obstetric records	Maternal weight gain categories: < IOM Within IOM > IOM	A mixed ANOVA with one repeated measures factor (weight before pregnancy, 6 months, 1, and 15 years) and one between-subjects factor (< IOM, within IOM, > IOM) showed a main effect of time [F (9.024) = 113.7, *P* = 0.000] and a significant time group interaction [F(6,12) = 77.23, *P* = 0.000] The weight of women who gained excessive during pregnancy was significantly greater at each time-point [main effect of group: F (10.55) = 870.0, *P* = 0.000]	Pregravid BMI
Rooney et al., 2002[174] USA, hospital 540 All weight/BMI Fair	Pregravid weight: Measured at first visit Total weight gain: Measured	Maternal weight gain categories: G1: < IOM G2: Within IOM G3: > IOM	Average weight change between prepregnancy and 6 months postpartum (kg): G1: -0.61 G2: 1.8 G3: 4.2 *P* = 0.01 Regression coefficient (95% CI)	Duration of breastfeeding, postpartum aerobic exercise, weight loss by 6 months

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
		BMI IOM	for weight at 6 months postpartum: G1: -1.53 (-3.36–0.30) G2: Reference G3: 1.24 (-0.63–3.11)	
Scholl et al., 1995[158] USA, Camden Study 274 Normal weight/BMI 19.8–26 Fair	Pregravid weight: Self report Total weight gain: Measured	Maternal weight gain categories (kg/wk): G1: ≤ 0.34 G2: > 0.34–0.68 G3: > 0.68	Mean (SEM) change in weight (kg) from pregravid to 6 months postpartum: G1: 3.2 (0.95) G2: 3.8 (0.61) G3: 7.9 (0.83) *P* < 0.001, G3 vs. G1, G2 Mean (SEM) change in weight (kg) from 6 weeks to 6 months postpartum: G1: 0.13 (0.64) G2: -0.05 (0.41) G3: -1.48 (0.56) *P* < 0.05, G3 vs. G1, G2 AOR (95% CI) for becoming overweight (BMI > 26.0) at 6 months postpartum: G1, G2: 1.0 (reference) G3: 2.89 (1.36–6.00)	Age, race, parity, pregravid BMI, lactation, height, smoking
Walker, 1996[172] USA, mail survey 88 Underweight/Normal/Overweight (using IOM definitions) Fair	Pregravid weight: Self report Total weight gain: Self report	Maternal weight gain categories: G1: < IOM G2: Within IOM G3: > IOM	Mean weight retention at 6 months postpartum, lbs: G1: 0.4 G2: 3.7 G3: 13.5 *P* < 0.001 Maternal weight gain was significantly related to weight at 6 months postpartum: r = 0.60, *P* < 0.001 Mean weight retention at 18 months postpartum, lbs: G1, G2: 0.7 G3: 11.0 *P* < 0.01 Maternal weight gain was significantly related to weight at	Mode of delivery, infant sex, breastfeeding, infant birthweight, pregravid BMI

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
			18 months postpartum: r = 0.49, *P* < 0.001	
Olson 2002[175] USA, hospital and primary care clinic system 622 All weight/BMI Fair	Pregravid weight: Measured during first trimester Total weight gain: Measured	Maternal weight gain categories: G1: < IOM G2: Within IOM G3: > IOM G4: Interaction for > IOM and income ≤ 185% federal poverty line	Regression coefficient (SE) for weight change from early pregnancy to 1 year postpartum (kg): G1: -1.50 (0.62) *P* = 0.016 G2: reference G3: 0.32 (0.65) *P* = 0.621 G4: 3.41 (0.91) *P* < 0.001 AOR (95% CI) for major weight gain (≥ 10 lbs) at 1 year postpartum: G1: 0.33 (0.13–0.83) G2: 1.00 (reference) G3: 1.47 (0.73–2.94) G4: 3.23 (1.25–9.08) Compared to normal-weight women (BMI 19.8–26.0) in G2, normal weight, overweight (BMI 26.1–29.0) and obese (BMI > 29.0) women in G3 retained significantly more weight at 1 year postpartum (all *P* < 0.01)	Exercise, food intake, breastfeeding, pregravid BMI, age, marital status, income, postpartum month that weight was measured
Keppel 1993[173] USA, 1988 National Maternal and Infant Health Survey 2,944 Non obese/BMI < 29 Poor	Pregravid weight: Self-report Total weight gain: Self-report	Categories of amount of weight retained (lbs) at 10–18 months postpartum: G1: Lost weight G2: 0–3 G3: 4–8 G4: 9–13 G5: ≥ 14	The percent distribution of women in G1–G5 stratified by maternal weight gain categories showed that both black and white women who gained < IOM or within the IOM guidelines retained less weight (10–18 months postpartum) than women who gained > IOM recommendations. Irrespective of maternal weight gain, black women retained more weight than white women	None

ANOVA, analysis of variance; BMI, body mass index; CI, confidence interval; G, group; kg, kilogram; kg/wk, kilogram per week; SE, standard error.

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Detailed results Regardless of when postpartum weight was measured—at 6[158,171,172,174] or at 10 to 18 months[171,173,175]—women who gained above the IOM recommendations retained more weight than those who gained within them. Women who gained below recommendations did not always retain less weight than those who gained within them, according to one fair[158] and one poor study.[173] In the poor study, which stratified results by race, this pattern of weight retention by weight gain held true for white and black women.[173] One study calculated women had statistically significant odds of becoming overweight at 6 months given rates of weight gain above IOM guidelines.[158]

Another study used a mixed ANOVA with a one-repeated-measure factor (time of the weight measurement: before pregnancy, 6 months, 1 year, and 15 years after) and one between-subject factor (below, within, above the IOM guidelines).[171] The weight of women who gained excessively during pregnancy was statistically significantly higher at each time point adjusted for pregravid BMI.

Long-term weight retention

Study characteristics Four articles from three databases examined weight retention after several years[171,174,176] or until the second pregnancy[177] (Evidence Table 55, Table 40). One study (2 articles) was in a U.S. medical center in Wisconsin,[174,176] one was done in another U.S. hospital;[177] and one was conducted in Sweden.[171] Three studies were rated fair quality; one was rated good.[177]

TABLE 40. Weight change relative to IOM thresholds and long-term weight retention

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Gunderson, 2000[177] USA, hospital 1,300 All weight/BMI (using IOM definitions) Good	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories: G1: < IOM/ within IOM G2: > IOM	AOR (95% CI) for becoming overweight between baseline (pregravid weight at start of index pregnancy) and start of second study pregnancy (median interval time = 1.5 years): G1: Reference G2: 2.95 (1.67–5.24)	Smoking, PIH, education, parity, marital status, age at menarche, interval to first birth
Rooney, 2005[176] USA, hospital 484 All weight/BMI	Pregravid weight: Measured at first prenatal visit Total weight gain: Measured	Categories of maternal weight gain: G1: < IOM G2: within IOM G3: > IOM	Multivariable regression coefficient (95% CI) for BMI at 15 years postpartum: G1: -0.57 (-0.57–1.21) G2: reference G3: 1.69 (0.79–2.58) Multivariable regression coefficient (95% CI) for change in weight between	Marital status at delivery, change in marital status, current parity, insurance status at delivery, current insurance status, baseline BMI, weight gain at index pregnancy, retained weight at 6 months postpartum, participation in postpartum aerobic exercise, duration of breastfeeding

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Fair			baseline and 15 years postpartum: G1: 0.43 (-1.87–2.73) G2: reference G3: 4.19 (1.88–6.51)	
Amorim et al., 2007[171] Sweden, hospital 483 All weight/BMI (using IOM definitions) Fair	Pregravid weight: Self-report Total weight gain: Obstetric records	Maternal weight gain categories: G1: < IOM G2: Within IOM G3: > IOM	Mean (SD) change in weight at 15 years postpartum, kg: G1: 6.2 (6.8) G2: 6.7 (6.8) G3: 10.3 (8.5) *P* = 0.000 Mean (SD) BMI at 15 years postpartum: G1: 23.5 (3.7) G2: 23.6 (3.0) G3: 25.9 (3.9) *P* = 0.000 Multiple regression coefficient, B (95% CI) for 15 year BMI status: G1: 0.01 (-0.56–0.59) G2: Reference G3: 0.72 (0.15–1.30) *P* = 0.033 Multiple regression coefficient (95% CI) for change in BMI status between pregravid and 15 years postpartum: G1: 0.02 (-0.56–0.59) G2: Reference G3: 0.68 (0.11–1.24) *P* = 0.042	Education, lactation, weight retention at 6 months postpartum, weight gain between 6 months and 1 year postpartum, pregravid BMI
Rooney et al., 2002[174] USA, hospital 540 All weight/BMI (using IOM definitions) Fair	Pregravid weight: Measured at first visit Total weight gain: Measured	Maternal weight gain categories: G1: < IOM G2: Within IOM G3: > IOM	Average weight change between prepregnancy and ~8.5 years postpartum (kg): G1: 4.1 G2: 6.5 G3: 8.4 *P* = 0.01 Regression coefficients (95% CI) for BMI at ~8.5 years postpartum: G1: -3.86 (-5.56 – -2.16) G2: Reference G3: -0.70 (-2.13–0.74)	Duration of breastfeeding, postpartum aerobic exercise, weight loss by 6 months

AOR, adjusted odds ratio; B, beta; BMI, body mass index; CI, confidence interval; G, group; IOM, Institute of

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Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Results	Confounders and Effect Modifiers Included in Analysis
Medicine; PHI, pregnancy-induced hypertension; SD, standard deviation; USA, United States of America.				

Overview of results Evidence from one good article[177] and three fair articles[171,174,176] supports an association between excessive weight gain and higher weights later in life.

Detailed results The results for the Sweden study were reported above.[171] In the Wisconsin study, the average amount of weight retained at a mean of 8.5 years later was statistically significantly higher among women who gained more than recommended guidelines than among women who gained within or below guidelines.[174,176] In the regression model predicting long-term weight (at 8.5 years and 14.7 years), weight gain during pregnancy was a significant predictor of weight retention.

In the other U.S. study, the incidence of overweight at the second pregnancy was statistically significantly higher among women who had gained above the IOM in the prior pregnancy than among those who gained within or below IOM recommendations.[177] The adjusted odds of becoming overweight between baseline and the start of the second pregnancy was nearly threefold for women gaining above recommendations.

KQ 5: Anthropometrics of Weight Measurement

Nearly all of the 150 studies included in this review estimated adiposity using body weight or BMI. Ten studies collected data from other anthropometric measurements and incorporated them into varying body composition equations or models to estimate body fat (Evidence Table 56).[16,97,102,115,143,178–182] These measurements included bioelectrical impedance analysis (BIA),178 dual energy X-ray absorptiometry (DEXA, formerly referred to as DXA),[16,97,180] skinfold thicknesses,[102,143,180] circumferences (arm, thigh, radius, upper chest, chest, elbow, waist, upper iliac, wrist, knee, calf, and ankle),[102,115,143,178,180] total body water,[16,97,178,180] total body nitrogen,[97] total body potassium,[97] magnetic resonance imaging (MRI),[179,181] and underwater weighing.[16,97,180] Studies that used DEXA or MRI methods[16,97,179,180] recorded measurements only during the postpartum period.

Collectively, these studies do not provide sufficient evidence to judge whether alternate methods of weight measurement are more informative or predictive of infant and maternal outcomes than standard body weight and height measurements.

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Appendix F

Data Tables

APPROACH TO GATHERING EVIDENCE

In order to review the most relevant scientific literature available, the committee and staff conducted thorough searches of several online bibliographic databases, including Medline, Science Direct, and WorldCat/First Search. General searches on pregnancy, gestational weight gain, and outcomes of pregnancy were first conducted to identify primary literature. Using the results of the primary search, key search terms were developed and secondary searches were then conducted. Search terms were chosen based on relevance to the report outline and topics included in the previous IOM report, *Nutrition During Pregnancy* (IOM, 1990). Although initial searches were general, subsequent searches focused on retrieving studies that were not covered by the evidence-based review conducted by Viswanathan et al. (2008). To identify studies that fell outside of the scope of that report, searches were limited to publication dates prior to 1990 and after October 2007. Similar to the methodology used by Viswanathan et al. (2008), searches were limited to English. As the study progressed, focused searches were conducted as needed and general searches were carried out to identify newly published articles. See Box F-1 for an example of how searches were conducted.

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BOX F-1 Examples of Searches Using Key Words to Identify Relevant Literature (PubMed)

General search (limited to English)
#1 Search Pregnancy
#2 Search Weight Gain
#3 Search #1 and #2
#4 Search gestational weight
#5 Search #3 OR #4
#6 Search #5 AND obesity
#7 Search #5 AND BMI OR body mass index

Focused search: Consequences of GWG for the Mother (limited to publication date prior to 1990 or after October 2007 and English)
#8 Search #5 AND antepartum outcomes
#9 Search #5 AND consequences
#10 Search #1 AND maternal age
#11 Search #1 AND BMI OR body mass index
#12 Search #5 AND depression OR mental health
#13 Search #5 AND delivery OR cesarean section OR labor OR induced labor OR complications at delivery OR hemorrhage OR anesthesia OR coagulation OR forceps OR infection OR protracted labor
#14 Search #13 AND obesity OR BMI
#15 Search #7 AND weight retention OR postpartum weight
#16 Search #6 AND lactation
#17 Search #5 AND maternal health OR chronic disease OR morbidity
#18 Search #5 AND maternal mortality

Focused search: Consequences of GWG for the Child (limited to publication date prior to 1990 or after October 2007 and English)
#19 Search #5 AND child outcomes OR infant outcomes
#20 Search #5 AND small-for-gestational age OR SGA OR IUGR
#21 Search #5 AND large-for-gestational age OR LGA
#22 Search #20 AND cognitive development OR neurodevelopment OR academic performance OR school OR cognition OR neurology
#23 Search #21 AND obesity OR child obesity OR metabolic syndrome
#24 Search #5 AND preterm birth
#25 Search #5 AND fetal growth OR fetal development OR body composition
#26 Search #7 AND infant outcomes
#27 Search #5 AND child mental health
#28 Search #5 AND birth defects OR congenital anomalies
#29 Search #5 AND fetal death OR infant death OR neonatal death OR miscarriage OR stillbirth OR infant mortality
#30 Search #5 AND child health OR infant health OR asthma OR cancer OR chronic disease OR morbidity

The focus of this appendix is literature that addresses the consequences of gestational weight gain. Table F-1 includes studies on the consequences of gestational weight gain for the mother

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and for the child, as discussed in Chapter 5, *Consequences of Gestational Weight Gain for the Mother*, and Chapter 6, *Consequences of Gestational Weight Gain for the Child*. This table is not inclusive of all the literature covered in this report nor does the report address each of the studies listed in the table. The table only includes studies that examined the consequences of gestational weight gain that were considered by the committee throughout the duration of the project.

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STUDIES ON CONSEQUENCES OF GESTATIONAL WEIGHT GAIN FOR THE MOTHER AND CHILD

TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
Author, year: Abrams et al., 1989 **Country/Setting:** USA (Perinatal Nutrition Project, San Diego, CA) **Enrollment period:** Jan. 1978 to Dec. 1986 **Study Objective:** To examine the relationship between maternal weight gain and preterm delivery.	**Design:** • Cohort • Retrospective **Total Study N:** 2,163 **Group Description:** G1: Preterm births G2: Term births **Group N:** G1: 118 G2: 2,045 **Inclusion criteria:** • Low income • Prepregnancy underweight or prepregnancy obesity • Low pregnancy weight gain • Anemia	Preterm birth: delivery between 26-37 weeks' gestation Gestational age: maternal estimate of the last menstrual period, antenatal sonography before 28 weeks' gestation, Dubowitz score, or a combination of these. Prepregnancy weight: maternal recall at first visit (prepregnancy weight for height based on the 1959 Metropolitan Insurance standards of desirable weight). Total pregnancy weight gain: estimated by subtracting the prepregnancy weight from the last measured weight before delivery.	**Outcomes description:** • Maternal weight gain • Preterm delivery **Results:** Women with low rate of weight gain (< 0.27 kg/wk) had a 60% higher risk for spontaneous preterm birth compared with those with average weight gain (0.27-0.52 kg/wk). They were also more than twice as likely to deliver preterm as women with a high rate of gain (>0.52 kg/wk) (OR = 2.54; 95% CI 1.49, 4.88). **Maternal confounders/effect modifiers:** • Dietary intake • Use of cigarettes, alcohol, and illicit drugs • Pre-pregnancy weight/height • Age • Income • Marital status • Race • Use of vitamin supplements • Whether or not pregnancy was planned

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
	• History of obstetric complications • Concurrent medical complication **Exclusion criteria:** • Antepartum death • Twin gestation • Major congenital anomalies • Induced deliveries (not preceded by spontaneous labor or rupture of membranes)		**Infant and child confounders/effect modifiers:** NR
Author, year: Abrams and Laros, 1986 **Country/Setting:** USA (San Francisco, CA) **Enrollment period:** Sept. 1980 to Dec. 1983 **Study Objective:** To study the effect of maternal weight gain on birth weight.	**Design:** • Cohort **Total Study N:** 2,946 **Group Description:** G1: Prepregnancy, underweight G2: Prepregnancy, ideal weight G3: Prepregnancy, moderately overweight G4: Prepregnancy, very overweight	Pregnancy weight gain = measured weight at last prenatal visit – reported prepregnancy weight Low gain: total gain of <7 kg Excessive gain: total gain of >20 kg	**Outcomes description:** • Total maternal weight gain • Infant birth weight **Results:** Mean weight gain (kg) G1: 14.3 G2: 15.2 G3: 15.2 G4: 14.1 Birth weight (gm) G1: 3,290 G2: 3,414 G3: 3,521 G4: 3,593

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
	Group N: G1: 268 G2: 1,535 G3: 901 G4: 224 **Inclusion criteria:** • Singleton pregnancies • ≥ 37 weeks' gestation • Live infant was delivered at study hospital **Exclusion criteria:** • Maternal transfers • Transports • Intrauterine transfusions • Fetal surgeries		**Maternal confounders/effect modifiers:** • Race • Parity • Maternal age • Number of cigarettes smoked/day • Prepregnancy weight/height • SES
Author, year: Berkowitz, 1981 **Country/Setting:** USA **Enrollment period:** 1977 **Study Objective:** To study the epidemiology of preterm delivery.	**Design:** • Case-control **Total Study N:** 488 **Group Description:** G1: preterm deliveries, < 37 weeks G2: term deliveries, 37 weeks or later	Demographics: G1: Married: 64% Race: White, 65.7% Black, 29.1% Hispanic, 4.0% Mean age: 24.9 G2: Married: 77.3% Race:	**Outcomes description:** • Risk factors associated with preterm delivery **Results:** The following risk factors carried the highest relative risk for a preterm delivery: Previous preterm (OR=29.8) Antepartum hemorrhage and placental abnormalities (OR=25.9) Third trimester urinary tract infection (OR=6.2) Low SES (OR=5.5) Previous pregnancy terminating in an induced abortion

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
	Group N: G1: 175 G2: 313 **Inclusion criteria:** • Singleton infants • Delivered before 37 wks gestation • Spoke English • Were interviewed during postpartum stay • Had not placed infant up for adoption **Exclusion criteria:** • Deliveries that were induced or surgically assisted without prior spontaneous labor or spontaneous rupture of membranes • Women who were referred to outlying hospitals or physicians	White, 72.8% Black, 22.7% Hispanic, 3.2% Mean age: 26.2	(OR=4.6) Inadequate GWG (OR=4.3) Other significant risk factors included: low pregravid weight, history of infertility, vaginal bleeding during pregnancy, lack of leisure time activity during pregnancy, alcohol consumption prior to third trimester, and negative attitude toward pregnancy. **Maternal confounders/effect modifiers:** • Race/ethnicity • Age • Marital status • SES • Age of menarche onset • Gravidity • Pregnancy order • Birth order • Range of menstrual cycles • Infertility history • History of induced abortion **Infant and child confounders/effect modifiers:** NR
Author, year: Billewicz and Thomson, 1957 **Country/Setting:** UK	**Design:** • Cohort **Total Study N:** 4,214	Weight was recorded at first antenatal visit and again 4-6 wks later. Most were weighed monthly from 28-30 wks, and more often during final weeks. Data from first trimester is sparse.	**Outcomes description:** • Preeclampsia with or without albuminuria • Other hypertensive complications **Results:** Women with preeclampsia tended to have an increased

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
Enrollment period: 1949-1954 **Study Objective:** To examine the clinical significance of weight trends during pregnancy.	**Group Description:** G1: Preeclampsia with albuminuria G2: Other hypertensive complications G3: Normotensive **Group %:** G1: 6.5 G2: 25.4 G3: 68.1 **Inclusion criteria:** • Women pregnant with first child • Residents of Aberdeen, delivered at Aberdeen Maternity Hospital **Exclusion criteria:** • Multiple gestations		rate of gain as the pregnancy progressed, and was greater in all stages than in the normotensive group. The incidence of preeclampsia also increases as the rate of gain increases. Favorable outcomes in women with preeclampsia are seen when the rate of gain is moderate, at least in the second half of pregnancy. **Maternal confounders/effect modifiers:** • Social class • Height • Age **Infant and child confounders/effect modifiers:** NR
Author, year: Butte et al., 1984 **Country/Setting:** USA **Enrollment period:** NR **Study Objective:**	**Design:** • 4 month Longitudinal **Total Study N:** 45 **Group Description:** Middle-upper SES, presumably well-nourished women	Maternal weight was measured between 10:00 am and 4:00 pm using a balance beam (clothed in bathing suits); maternal height was measured using an upright extension meter. Skinfold measurements were also taken at the triceps, biceps, suprailiac and subscapular sites. Body fat was calculated using water displacement.	**Outcomes description:** • Milk production • Maternal dietary intake • Anthropometry • Body composition • Maternal energy balance **Results:** Adequate GWG 14.4 (3.3 kg) was a good indicator of good nutritional status during pregnancy.

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
To examine the influence of maternal diet and body composition on lactational performance.	**Inclusion criteria:** • Healthy mothers and infants • Nonsmoking • 18-36 yrs of age • No chronic medications • Parity one or two • Intent of breastfeeding exclusively for at least 4 mos • Term deliveries • AGA infant **Exclusion criteria:** NR		Maternal weights were 16 (6%) [range 0-29%] above prepregnancy weights at the onset of lactation; maternal weights were then 5 (7%) [range -10-21%] above prepregnancy weights after 4 mos of lactation. Mean weight loss of 3.8 (2.3 kg) occurred during the 1st month postpartum, followed by a mean weight loss of 0.67 (0.11 kg)/month. Over all, successful lactation is compatible with gradual weight loss (with energy intakes less than current NRC RDAs. **Maternal confounders/effect modifiers:** • Education • Income **Infant and child confounders/effect modifiers:** NR
Author, year: Chen et al., 2008 **Country/Setting:** USA **Enrollment period:** 1995-2000 **Study Objective:** To examine the association between teenage pregnancy and neonatal and postneonatal mortality.	**Design:** • Cohort • Retrospective **Total Study N:** 4,037,009 **Group Description:** G1: Nulliparous women, under aged 10-15 G2: Nulliparous women, under aged 16-17 G3: Nulliparous women, under aged 18-19	Low weight gain = < 0.16 kg/wk	**Outcomes description:** • Neonatal and postneonatal mortality and morbidity **Results:** Teenage pregnancy (G1, G2, G3) was associated with increased neonatal mortality (OR: 1.20, 95% CI = 1.16-1.24) and postneonatal mortality (OR: 1.47, 95% CI = 1.41-1.54). There was still an association of increased risk of neonatal and postneonatal mortality after adjusting for GWG (OR 1.23, 95% CI = 1.19-1.28 and OR: 1.48, 95% CI = 1.42-1.55 respectively). No association was seen with gestational age at birth and neonatal mortality and teenage pregnancy (OR: 0.98, 95% CI = 0.95-1.02), but there was a significant

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
	G4: Nulliparous women, under aged 20-24 **Group N:** G1: 183,977 live births G2: 674,026 live births G3: 1,098,111 live births G4: 2,080,895 live births **Inclusion criteria:** • Singleton live births • 10-24 years of age • Nulliparous **Exclusion criteria:** • Subjects with missing data on prenatal care and/or gestational age		association between gestational age at birth, teenage pregnancy, and postneonatal mortality (OR: 1.40, 95% CI = 1.34-1.46). **Maternal confounders/effect modifiers:** • Education level (defined as appropriate or inappropriate for age) • Prenatal care (intensive, adequate, or inadequate) • Race • Tobacco and alcohol use during pregnancy • Mode of delivery **Infant and child confounders/effect modifiers:** • Birth defect • Gestational age (< 32 wks, 32-36 weeks, ≥ 37 wks)
Author, year: Frentzen et al., 1988 **Country/Setting:** USA (Florida) **Enrollment period:** Jan. 1982 to Dec. 1984 **Study Objective:** To compare the influence of pregnancy weight gain on infant birth weight and	**Design:** • Cohort • Retrospective **Total Study N:** 135 **Group Description:** G1: Control (wt/ht 90-120% of standard) G2: Overweight (wt/ht ≥ 135% of standard)	Race: G1: 53% black, 45% white, 1 Hispanic woman G2: 61% black, 39% white Mean age at conception: G1: 22.4 G2: 25.2	**Outcomes description:** • Apgar score ≤ 6 at 5 min • Admission to NICU • Birth weight (≤ 2,500 g and ≥ 4,000 g) • SGA, AGA, LGA • Preterm, Postterm **Results:** Mean GWG G1: 14.2 kg (± SD 6.9) G2: 11.2 kg (± SD 7.7) Mean birth weight (g)

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
outcome among indigent women who were highly overweight before pregnancy and those who were of average weight.	**Group N:** G1:57 G2: 78 **Inclusion criteria:** • Delivered liveborn • Singleton infants **Exclusion criteria:** NR		G1: 3,236 (± SD 689) G2: 3,434 (± SD 565) Apgar score ≤ 6 at 5 min G1: 9% G2: 3% Admission to NICU G1: 12% G2: 6% Birth weight ≤ 2,500 g G1: 12% G2: 3% Birth weight ≥ 4,000 g G1: 11% G2: 14% SGA G1: 0 G2: 0 AGA G1: 84% G2: 73% LGA G1: 16% G2: 27% Preterm ≤ 37 wks G1: 16% G2: 11% Postterm ≥ 42 wks G1: 10% G2: 11% **Maternal confounders/effect modifiers:** • Maternal age

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
			• Parity • Smoking status • SES **Infant and child confounders/effect modifiers:** NR
Author, year: Geelhoed et al., 2008 **Country/Setting:** Rotterdam, The Netherlands **Enrollment period:** Apr 2002 to Jan 2006 **Study Objective:** To examine the associations of maternal anthropometrics during pregnancy and left ventricular mass in infancy.	**Design:** • Cohort • Prospective **Total Study N:** 791 **Inclusion criteria:** • Singleton infants • Aged 6 wks and 6 mos **Exclusion criteria:** • Multiple gestations • Pregnancies resulting in intrauterine or perinatal death	Measurements were taken at early visits (< 18 wks gestation), mid pregnancy (18-25 wks), and late pregnancy (> 25 wks). Pregravid weight was self-reported. Weight gain= late pregnancy weight – prepregnancy weight	**Outcomes description:** • Maternal anthropometrics • Infant cardiac structure **Results:** No associations were seen between maternal weight gain during pregnancy and LVM at 6 wks of age, however weight gain during pregnancy was positively correlated with postnatal LVM at 6 mos of age. For each kg increase in weight during pregnancy, LVM at age 6 mos increased by 0.08 g (95% CI: 0.02, 0.15). Weight gain in late pregnancy is associated with larger LVM at 6 mos. **Maternal confounders/effect modifiers:** • Age • Height • Prepregnancy weight • Prepregnancy BMI • Weight in late pregnancy **Infant and child confounders/effect modifiers:**

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
			• Gender • Birth weight/length
Author, year: Haiek and Lederman, 1988 **Country/Setting:** USA (New York, NY) **Enrollment period:** January 1981 to May 1985 **Study Objective:** To examine the relationship between maternal weight for height and term birth weight.	**Design:** • Cohort **Total Study N:** 180 **Group Description:** G1: Adult women, 19-30 y G2: Teens, < 16 y **Group N:** G1: 90 G2: 90 **Inclusion criteria:** • Gave birth at St. Luke's Hospital • Live infants • Received prenatal care **Exclusion criteria:** • Delivery occurred before 37 weeks' gestation • Factors known to affect fetal growth were present	Data obtained from a standard prenatal and intrapartum form included in the medical record.	**Outcomes Description:** Term birth weight **Results:** Mean birth weight was lower in the teen group than compared with the adult group. Birth weight also increased with increasing maternal prepregnancy weight, weight gain, and percent of standard weight for height at term for both groups. Overall, the teen group gave birth to smaller babies than the adult group. **Maternal confounders/effect modifiers:** • Marital status • Education • Race • Date of registration for prenatal care • Number of prenatal visits • Height • Prepregnancy weight • Weight at delivery • Smoking and drinking habits • Obstetric history and complications • Type of delivery • Duration of pregnancy **Infant and child confounders/effect modifiers:** • None stated

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
Author, year: Harrison et al., 1980 **Country/Setting:** USA (Arizona) **Enrollment period:** Dec 1976 to June 1978 **Study Objective:** To examine the relationship between maternal obesity, weight gain, and infant birth weight.	**Design:** • Cohort • Prospective **Total Study N:** 327 **Inclusion criteria:** • Delivered normal, singleton, full term infants at study hospital **Exclusion criteria:** • Lived outside immediate geographic area • Addicted to alcohol or drugs • Refused follow-up • Diabetic	Maternal age, parity, race/ethnicity	**Outcomes description:** • Maternal weight • Birth weight **Results:** Obese mothers had a higher incidence of inadequate weight gain, as compared to nonobese mothers. Massively obese mothers had a markedly higher risk of delivering an infant over 4 kg. For women with gains over 12 lbs, maternal obesity was directly related to mean birth weight. Mean birth weight was higher for infants of massively obese mothers when weight gain was adequate. **Maternal confounders/effect modifiers:** NR **Infant and child confounders/effect modifiers:** NR
Author, year: Hedderson et al., 2008 **Country/Setting:** USA (Northern California) **Enrollment period:** Jan 1, 1996 to June 30, 1998	**Design:** • Nested case-control **Total Study N:** 455 **Group Description:** G1: GDM Cases G2: Controls	Age < 25 yrs = G1: 6.4%, G2: 15.2% 25-29 yrs = G1: 11.6 %, G2: 16.7% 30-34 yrs = G1: 36.3%, G2: 34.8% ≥ 35 yrs = G1: 45.8 %, G2: 33.3% Race-ethnicity Non-Hispanic White = G1:41.8%, G2: 64.2%	**Outcomes description:** • GDM • Rate of weight gain **Results:** Gains of 1.1-2.2 kg/yr were associated with a small increased risk of GDM (OR 1.63, 85% CI 0.95-2.81). Gains of 2.3-10.0 kg/yr were associated with a 2.5-fold increased risk of GDM (OR 2.61, 95% CI 1.5-4.57) as

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
Study Objective: To evaluate obesity and rate of weight change during 5 years prior to pregnancy and risk of GDM	**Group N:** G1: 251 G2: 204 **Inclusion criteria:** • Pregnancies resulting in a singleton, live birth • Women did not have recognized diabetes prior to pregnancy • Screening test performed at 24-28 wks' gestation **Exclusion criteria:** • Multiple births • History of GDM or diabetes	Hispanic = G1: 22.3%, G2: 10.8% Asian = G1: 13.9%, G2: 5.9% African-American = G1: 7.2%, G2: 11.8% Marital Status Never married = G1: 13.7%, G2: 13.3% Married = G1: 80.2%, G2: 73.9% Widowed, Divorced, Separated = G1: 4.4%, G2: 3.0% Education ≤ 12 yrs = G1: 37.5%, G2: 32.4% 13-15 yrs = G1: 29.9%, G2: 37.3% 16 yrs = G1: 20.7%, G2: 15.2% ≥ 17 yrs = G1: 10.8%, G2: 14.2% Parity 0 = G1: 40.2%, G2: 36.8% 1 = G1: 31.1%, G2: 39.2% ≥ 2 = G1: 28.7%, G2: 24.0% Body weight was recorded at baseline and before pregnancy. Baselines = earliest measured weight during a nonpregnancy state recorded in the medical record during the 5 yrs prior to study pregnancy but after age 18.	compared with stable weight). **Maternal confounders/effect modifiers:** • Age • Baseline BMI • Prepregnancy BMI • Parity • Education • Note of infertility (y or n) • Amenorrhea (y or n) • PCOS (y or no) • Hypothyroid (y or n) • Family history of diabetes (y or n) • Smoking prior to pregnancy (y or n) **Infant and child confounders/effect modifiers:** NR

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
		Prepregnancy weight = self-reported Rate of weight change = change in body weight and prepregnancy weight divided by the time in years between two weights	
Author, year: Hediger et al., 1989 **Country/Setting:** USA (Camden, NJ) **Enrollment period:** NR **Study Objective:** To examine the relationship between maternal weight gain in adolescents and birth weight and length of gestation.	**Design:** • Cohort **Total Study N:** 1,790 **Group Description:** G1: Adequate weight gain G2: Early inadequate weight gain (< 4.3 kg/wk by 24 weeks' gestation) G3: Late inadequate weight gain G4: Both inadequate weight gain **Group N:** G1: 955 G2: 304 G3: 387 G4: 144 **Inclusion criteria:** • Teens • Registered in Camden	**Total study characteristics** Race: Puerto Rican, 23.1% Black, 40.6% White, 36.3% Age: ≤ 15, 16.7% 16-17, 44.4% 18-19, 38.9% BMI: ≤ 19.5, 27.6% 19.6-24.5, 55.8% > 24.5, 16.6% Prenatal Care: Inadequate, 5.5% Intermediate, 64.4% Adequate, 30.1% Payment Status: Medicaid, 75.9% Private Insurance, 24.1%	**Outcomes description:** • Low birth weight (< 2,500 g) • Preterm delivery (before 37 weeks' gestation) • SGA status (10th percentile) **Results:** Mean total weight gain = 14.8 ± 6.1 kg (range -3.8 to 43.9 kg) G2 was associated with a significantly increased risk of having an SGA infant (OR = 1.88, 95% CI 1.08-3.27). G3 was associated with preterm delivery, whether or not the total gain was adequate for gestation (OR = 1.69, 95% CI 1.12-2.55). **Maternal confounders/effect modifiers:** • Age • Ethnicity • Prepregnant weight • Measured height • Cigarette smoking • Weight gain during pregnancy

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
	County Adolescent Family Life program • Initial OB/GYN exam before 24 weeks' gestation • Delivered live born singleton infant • One of five affiliated hospitals • Delivery after 24 weeks' gestation **Exclusion criteria:** • Entered prenatal care after 24 weeks' gestation	Smoked: 32.5% Prepregnant weight: recalled at entry to prenatal care (used to calculate prepregnancy BMI). Weight during pregnancy was measured at each prenatal visit. Total pregnancy weight gain: interview or abstracting from clinical prenatal records	**Infant and child confounders/effect modifiers:** NR
Author, year: Langford et al., 2008 **Country/Setting:** USA; Missouri birth certificate data **Enrollment period:** 1990-2004 **Study Objective:** To examine the relationship between GWG and adverse maternal and infant outcomes for overweight women.	**Design:** • Population-based Cohort **Total study N:** 34,143 **Group Description:** G1: GWG below IOM recs (< 15 lbs) G2: GWG within IOM recs (15-25 lbs) G3: GWG above IOM recs (>25 lbs) **Group N:** G1: 1,787	Study population was further divided into eight categories to represent 10-lb gain increments. Prepregnancy BMI: height and weight reported on birth certificate (taken from medical records or self-reported). **Age:** G1: 18-24 yrs: 57.1% 25-30 yrs: 30.8% 31-35 yrs: 12.1% G2: 18-24 yrs: 52.5%	**Outcomes description:** Adjusted relative risks • Preeclampsia • Cesarean section • Macrosomia • Low birth weight (LBW) • Perinatal death **Groups** G1: GWG below IOM recs G2: GWG within IOM recs G3: GWG above IOM recs ---- G4: GWG <5lbs G5: GWG 6-14 lbs

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
	G2: 7,205 G3: 25,151 **Inclusion criteria:** • Singleton, full term deliveries • Nulliparous • Missouri residents • Aged 18-35 • Prepregnancy BMIs 26-29 kg/m^2 **Exclusion criteria:** • NR	25-30 yrs: 34.7% 31-35 yrs: 12.8% G3: 18-24 yrs: 53.8% 25-30 yrs: 34.5% 31-35 yrs: 11.7% **Race/ethnicity** G1: White, non-Hispanic: 78.8% Black, non-Hispanic: 15.8% G2: White, non-Hispanic: 81.2% Black, non-Hispanic: 14.3% G3: White, non-Hispanic: 84.0% Black, non-Hispanic: 12.7%	G6: GWG 15-24 lbs G7: GWG 25-34 lbs G8: GWG 35-44 lbs G9: GWG 45-54 lbs G10: GWG 55-64 lbs G11: GWG ≥ 65 lbs **Results:** Preeclampsia, RR (95% CI): G1: 0.78 (0.61-0.99) G2: Reference G3: 1.71 (1.54-1.89) G4: 0.72 (0.69-1.33) G5: 0.83 (0.64-1.07) G6: Reference G7: 1.31 (1.15-1.50) G8: 1.68 (1.47-1.91) G9: 2.04 (1.78-2.34) G10: 2.70 (2.31-3.15) G11: 3.35 (2.82-3.98) C-Section, RR (95% CI): G1: 0.92 (0.83-1.01) G2: Reference G3: 1.30 (1.24-1.36) G4: 1.10 (NA) G5: 0.90 (0.88-1.37) G6: 1.0 (0.80-1.00) G7: Reference G8: 1.29 (1.07-1.21) G9: 1.43 (1.22-1.37) G10: 1.62 (1.34-1.52)

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	**Protocol** Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
			G11: 1.79 (1.50-1.75) Macrosomia, RR (95% CI: G1: 0.60 (0.47-0.77) G2: 1.0 G3: 2.13 (1.94-2.33) G4: 0.79 (1.46-1.36) G5: 0.59 (0.45-0.78) G6: Reference G7: 1.54 (1.37-1.73) G8: 2.05 (1.83-2.30) G9: 2.72 (2.42-3.06) G10: 3.11 (2.72-3.56) G11: 3.73 (3.21-4.33) LBW: G1: 1.71 (1.35-2.17) G2: 1.0 G3: 0.60 (0.51-0.70) G4: 1.83 (1.10-3.06) G5: 1.65 (1.27-2.15) G6: Reference G7: 0.74 (0.61-0.91) G8: 0.51 (0.41-0.63) G9: 0.51 (0.39-0.67) G10: 0.59 (0.42-0.85) G11: 0.64 (0.41-1.00) Perinatal death: G1: 1.88 (0.77-4.62) G2: 1.0 G3: 1.09 (0.62-1.92)

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
			Maternal confounders/effect modifiers: • Age • Race/ethnicity • Education • Medicaid status • WIC status • Tobacco and alcohol use • Chronic hypertension • Cardiac disease • Insulin-dependent diabetes • Inadequate prenatal care **Infant and child confounders/effect modifiers:** • Child's sex
Author, year: Lof et al., 2008 **Country/Setting:** Sweden **Enrollment period:** Apr 2000 to Nov 2003 **Study Objective:** To examine the effects of pre-pregnancy physical activity and maternal BMI on GWG and birth weight.	**Design:** • Cohort • Prospective **Total Study N:** 223 **Inclusion criteria:** • Healthy women **Exclusion criteria:** • History of hypertension, diabetes, or thyroid problems	Body weight was measured at gestational weeks 12, 25, and 33. GWG during the second (weeks 12-25) and third trimesters (weeks 25-33) was determined (kg/week). BMI was calculated as body weight in gestational week 12 and was divided by self-reported squared height. Prepregnancy physical activity level, prepregnancy BMI, maternal age, parity, education, smoking status	**Outcomes description:** • Gestational weight gain • Infant birth weight **Results:** BMI and GWG, but not pre-pregnancy physical activity level, were linked to birth weight. GWG during gestational weeks 12 and 33 was correlated with elevated birth weight. **Maternal confounders/effect modifiers:** • Parity • Smoking status • Education level • Pregnancy physical activity level

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
			Infant and child confounders/effect modifiers: NR
Author, year: Manios et al., 2008 **Country/Setting:** Greece **Enrollment period:** Apr 2003 to July 2004 **Study Objective:** To examine the effect of maternal obesity on initiation and duration of breastfeeding.	**Design:** • Cross-sectional **Total Study N:** 2,374 **Inclusion criteria:** • Greek preschool aged children, 12 to 60 mos • Participants in GENESIS (Growth, Exercise, ad Nutrition Epidemiological Study In preschoolers) **Exclusion criteria:** NR	Prepregnancy weight was self-reported. GWG was self-reported and categorized based on IOM recs. BMI categories: underweight (<19.8); normal (19.8-26); overweight (>26-29); obese (>29)	**Outcomes description:** • Breastfeeding initiation and duration **Results:** A higher percentage of mothers with increased prepregnancy BMI or high GWG failed to initiate breastfeeding, as compared to normal weight mothers. With women who initiated breastfeeding, no significance differences were seen in breastfeeding duration in women with different gestational weight gains. **Maternal confounders/effect modifiers:** • Parental age • Education level of population • Parental anthropometric data • Parity • Smoking and alcohol consumption during pregnancy • Weight status before, during and after pregnancy **Infant and child confounders/effect modifiers:** • Feeding patterns • Gestational age
Author, year: Mitchell and Lerner, 1989	**Design:** • Cohort	Initial weight/BMI: recorded at first prenatal visit	**Outcomes description:** • Brith weight • Gestational age

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
Country/Setting: USA **Enrollment period:** NR **Study Objective:** To compare pregnancy outcome in overweight and normal weight women.	**Total Study N:** 152 **Inclusion criteria:** • Singleton pregnancies • Patients at one private practice • Entered prenatal care prior to 12th week • Seen regularly throughout gestation **Exclusion criteria:** NR	Gestational weight gain: difference between weight at first prenatal visit (initial weight) and weight recorded at final antepartum visit (≤ 5 days before delivery).	• Apgar scores at 1 and 5 min • Incidence of infant or maternal complications • Gestational weight gain **Results:** A significant linear relationship was seen between maternal weight gain and birth weight in normal and overweight pregnancies. Infants of overweight mothers had higher birth weights at each weight gain level. Overweight mothers also gained significantly less weight than normal weight mothers. **Maternal confounders/effect modifiers:** • Age • Height • Parity • Race • Smoking habits **Infant and child confounders/effect modifiers:** NR
Author, year: Naeye, 1981 **Country/Setting:** USA **Enrollment period:** 1959-1966	**Design:** • Cohort **Total Study N:** 13,830 **Inclusion criteria:** • Black • Singleton infants	Mothers of infants were placed into age categories: 10-14, 15-16, 17-19, and 20-32 years and grouped according to pregravid weight for height (recalled at first antenatal visit). Net pregnancy gain was calculated by subtracting the weight of the	**Outcomes description:** • Pregravid weight for height • Net pregnancy weight gain, • Birth weight and length • Head circumference **Results:** Mothers aged 10-16 years (normal or underweight before pregnancy) had growth retarded infants by

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
Study Objective: To determine whether the growth needs of young mothers compete with the growth needs of their fetuses for available nutrients.	• Maternal age of 10-32 years **Exclusion criteria:** • Maternal diabetes mellitus • Placenta previa • Hydramnios • Oligohydramnios • Congenital malformations of the infant	neonate and the placenta from the maternal weight at the end of the pregnancy.	comparison to older mothers. This was not seen in overweight young mothers. A greater percentage of young mothers (10-14 years) had acetonuria (2+ greater acetone of the urine). **Maternal confounders/effect modifiers:** • Parity • Gynecologic age • Cigarette smoking **Infant and child confounders/effect modifiers:** NR
Author, year: Naeye, 1979 **Country/Setting:** USA **Enrollment period:** 1959-1966 **Study Objective:** To determine if a 24 to 27 lb weight gain is optimal for all singleton pregnancies or requires modification for specific subgroups of mothers based on pregnancy outcome.	**Design:** • Cohort **Total Study N:** 44,565 **Inclusion criteria:** • Singleton infants **Exclusion criteria:** • Hydramnios • Oligohydramnios • One or more maternal hematocrit values less than 20% • Diabetes • Heart disease • Tuberculosis	Prepregnancy weight was recorded during interviews at first or second antenatal visits for medical care. Total pregnancy gain was divided by the optimal weight gain value for the length of pregnancy (NAS guidelines). This was multiplied by 100 to give the mother's weight gain in percent of the optimal value.	**Outcomes description:** • Preterm delivery • Pregnancy/delivery complications • Fetal/placental complications • Neonatal mortality **Results:** Mothers who began pregnancy overweight had the fewest fetal and neonatal deaths with a 16 lb gain at term. The optimal weight gain for normally proportioned mothers was 20 lbs and 30 lbs for underweight mothers. For all three groups, perinatal mortality rates increased weight gain less or more than optimal values. Extreme gains (low or high) had modest influence on the occurrence of common placental and fetal disorders, but once a disorder was present, mortality rates increased significantly when mothers had extreme gains (low or

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
	• Alcoholism • Drug addiction • Uterine leiomyomas • Prepregnancy weigh and height not recorded		high). **Maternal confounders/effect modifiers:** • Parity • Gynecologic age • Cigarette smoking **Infant and child confounders/effect modifiers:** NR
Author, year: Nohr et al., 2008 **Country/Setting:** Denmark (Danish National Birth Cohort) **Enrollment period:** 1996-2002 **Study Objective:** To examine the associations between prepregnancy BMI and gestational weight gain with pregnancy outcomes.	**Design:** • Cohort • Retrospective **Total Study N:** 60,892 **Group Description:** G1: Low GWG (< 10 kg) G2: Medium GWG (10-15 kg) G3: High GWG (16-19 kg) G4: Very high GWG (≥ 20 kg) **Groups % of N:** G1: 12.6% G2: 44.7% G3: 20.9% G4: 21.9% **Inclusion criteria:**	Prepregnancy BMI: self-reported at first pregnancy interview. BMI Categories: underweight (<18.5); normal (18.5-25); overweight (25-30); obese (≥30). Gestational weight gain: self-reported (postpartum interview 6 mos after birth)	**Outcomes description:** *Maternal outcomes:* • Preeclampsia • GDM • Hypertensive disorders • Cesarean section before labor • Instrumental deliveries • Postpartum weight retention • Postpartum weight loss *Neonatal outcomes:* • SGA • LGA • Apgar score **Maternal Outcomes Results: OR (95% CI)** Preeclampsia G1: 0.7 (0.6-0.9) G2: Reference G3: 1.6 (1.3-1.8) G4: 2.8 (2.4-3.2)

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
	• Liveborn, singleton infants • Delivered ≥37 wks gestation • Mothers participated in first pregnancy interview and first postpartum interview **Exclusion criteria:** • Women with type 1 diabetes • Women younger than 18 years of age • Missing information on important study variables • Missing data on postpartum weight retention 6 mos after birth		Gestational diabetes G1: 2.3 (1.9-2.8) G2: Reference G3: 0.8 (0.7-1.1) G4: 1.1 (0.9-1.4) Hypertensive disorders G1: 1.0 (0.8-1.2) G2: Reference G3: 1.1 (0.9-1.4) G4: 1.4 (1.1-1.7) Cesarean section before labor G1: 0.9 (0.8-1.0) G2: Reference G3: 1.0 (0.9-1.1) G4: 1.2 (1.1-1.3) Cesarean section during labor G1: 0.8 (0.8-0.9) G2: Reference G3: 1.2 (1.1-1.3) G4: 1.4 (1.3-1.5) Instrumental deliveries G1: 0.9 (0.8-1.0) G2: Reference G3: 1.1 (1.0-1.2) G4: 1.2 (1.1-1.3) Postpartum weight retention (5 kg +) G1: 0.4 (0.3-0.4) G2: Reference G3: 2.3 (2.2-2.4) G4: 6.2 (5.8-6.5) Postpartum weight loss (2 kg +) G1: 2.8 (2.7-3.0)

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
			G2: Reference G3: 0.5 (0.5-0.5) G4: 0.3 (0.3-0.3) **Neonatal Outcomes Results: OR (95% CI)** SGA G1: 1.8 (1.6-2.0) G2: Reference G3: 0.7 (0.7-0.8) G4: 0.5 (0.5-0.5) LGA G1: 0.7 (0.6-0.8) G2: Reference G3: 1.6 (1.5-1.7) G4: 2.6 (2.4-2.8) Low Apgar Score G1: 0.8 (0.6-1.1) G2: Reference G3: 1.2 (1.0-1.5) G4: 1.3 (1.0-1.6) **Maternal confounders/effect modifiers:** • Age at conception • Parity • Lifestyle habits in the first part of pregnancy (smoking, alcohol intake, physical exercise) • Social status (education and occupation) • Duration of breastfeeding **Infant and child confounders/effect modifiers:** NR

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
Author, year: Papiernik and Kaminski, 1974 **Country/Setting:** France (Paris) **Enrollment period:** 1969 **Study Objective:** To examine multiple factors in relationship to the risk of premature delivery.	**Design:** • Case-control **Total Study N:** 365 **Group Description:** G1: infant weighing < 2,500 g G2: infant weighing > 2,500 g **Group N:** G1: 149 G2: 216 **Inclusion criteria:** • Delivered infant at the same obstetrical department **Exclusion criteria:** NR	Data was collected using medical records completed at prenatal consultation. Groups were further divided by length of gestation: G1: >37 weeks (weight >2,599 g) G2: ≤ 37 weeks (weight > 2,500 g) G3: ≤ 37 weeks (weight ≤ 2,500 g) and > 35 weeks (weight > 2,000 g) G4: ≤ 35 weeks (weight ≤ 2,000 g) G5: > 37 weeks (weight ≤ 2,500 g) **Subgroup N:** G1: 198 G2: 18 G3: 56 G4: 46 G5: 47 General Factors: G1: Unwed: 10% Weight < 45 kg: 6% Height < 150 cm: 1% > 2 children w/o domestic help: 3% Age < 20 or > 40: 14% Low SES: 12% G2: Unwed: 11% Weight < 45 kg: 6% Height < 150 cm: 0%	**Outcomes description:** • Risk of prematurity (32nd wk of gestation) • Length of gestation; obstetric risks **Results:** The principle factors of prematurity and/or intrauterine growth retardation were determined to be: Pathology of the cervix or isthmus, unfavorable obstetrical and gynecological antecedent; signs of imminent delivery; low eight gain; fatigue; toxemia; short, thin women **Maternal confounders/effect modifiers:** • Marital status • Weight/height • Having more than two children without domestic help • Age • Social class • Factors of fatigue **Infant and child confounders/effect modifiers:** NR

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
		> 2 children w/o domestic help:6% Age < 20 or > 40:22% Low SES: 28 G3: Unwed: 14% Weight < 45 kg: 5% Height < 150 cm: 2% > 2 children w/o domestic help: 2% Age < 20 or > 40: 18% Low SES: 18% G4: Unwed: 9% Weight < 45 kg: 2% Height < 150 cm: 0% > 2 children w/o domestic help: 13% Age < 20 or > 40: 7% Low SES: 31% G5: Unwed: 13% Weight < 45 kg: 15% Height < 150 cm: 2% > 2 children w/o domestic help: 4% Age < 20 or > 40: 13% Low SES: 21%	
Author, year: Rodriquez et al., 2008 **Country/Setting:** Sweden and Denmark **Enrollment period:**	**Design:** • Follow-up of prospective cohort **Total Study N:** 12,556	Prepregnancy BMI: taken from medical chart at time of booking (rounded to the nearest whole number) by the midwife Maternal weight: recorded at delivery or in late gestation for all women and	**Outcomes description:** • Average weekly weight gain (stratified by prepregnancy BMI) • ADHD symptoms **Results:** GWG outside of the IOM guidelines was not related to

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
Follow-up: 2001-2002 in Sweden, 1993-1994 in Denmark **Study Objective:** To examine the relationship between pregnancy weight and core symptoms of ADHD in offspring	**Group Description:** School-aged children **Inclusion criteria:** • Live born, singleton infants **Exclusion criteria:** NR	subtracted from prepregnancy weight to obtain GWG Average weekly gain: divided weight gain by the number of completed gestational weight	ADHD symptoms (below recs: OR: 0.96, 95% CI 0.81,1.14; above recs: OR: 0.98, 95% CI: 0.82,1.16). Analyses found significant associations between prepregnancy overweight/obese and high ADHD symptoms scoring in offspring (OR range: 1.37 [95% CI 1.07, 1.75] to 1.89 [95% CI 1.13,3.15). Offspring of overweight women who had high GWG had a 2-fold risk of ADHD symptoms (OR: 2.10, 95% CI 1.19,3.72) when compared to normal weight women. **Maternal confounders/effect modifiers:** • Smoking status during pregnancy • Weight gain • Education • Family structure • Age • Cohort country of origin **Infant and child confounders/effect modifiers:** • Gestational age • Birth weight • Sex
Author, year: Rudra et al., 2008 **Country/Setting:** USA (Washington State) **Enrollment period:**	**Design:** • Cohort • Prospective **Total Study N:** 2,468	Pregravid BMI: using self-reported height and weight during the three months before pregnancy. Weight gain during pregnancy= rate of gain between prepregnancy and 18-22 wks gestation	**Outcomes description:** • Preterm delivery • Weight gain rate • Prepregnancy BMIs **Results:** There was no association between spontaneous preterm

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
1996-2005 **Study Objective:** To examine the relationship between prepregnancy weight and gestational weight gain and preterm delivery.	**Inclusion criteria:** • Attended prenatal care clinics affiliated with two hospitals • Started prenatal care before 20 wks gestation • 18 years of age or older • Speak or read English • Planned to carry pregnancy to term and to deliver at one of two affiliated hospitals **Exclusion criteria:** • Had early pregnancy loss • Multiple gestations • Missing prepregnancy weight or height data in interviews • Missing weight data mid-pregnancy • Extreme weight loss during pregnancy	Weight gain rate in early pregnancy = [(weight at 18-22 wks – prepregnancy weight)/weeks' gestation at weight measurement] Preterm delivery = delivery before 37 completed weeks of gestation Prepregnancy BMI: Normal, 71.0% Underweight, 4.0% Overweight, 15.9% Obese, 9.1% Age: 18-20 yrs, 1.1% 21-35 yrs, 75.8% 36-48 yrs, 23.1% Nulliparous, 63.1% Race/ethnicity: Non-Hispanic White, 85.1% Non-Hispanic Black, 1.7% Hispanic, 3.2% Asian/Pacific Islander, 7.0% Married, 91.1% High School education, 95.8% Household income:	delivery and BMI (before and after adjustment for age, race/ethnicity, and parity) (OR per 5 kg/m^2 increase: 1.01, 95% CI, 0.80-1.29). There was an association between PPROM and BMI (after adjustment for age, race/ethnicity, and parity) (OR per 5 kg/m^2 increase: 1.12, 95% CI 0.87-1.43). There was also an association between indicated delivery and BMI (after adjustment for maternal characteristics and weight gain). Each 5 kg/m^2 was associated with a 71% increase (95% CI, 1.4-2.06). However, the association was weakened when adjustments for diabetes or hypertensive conditions were made. There was an inverse association between early pregnancy weight gain and spontaneous preterm delivery (before and after adjustment for age, race/ethnicity, prepregnancy BMI, and parity) (OR per 0.1 kg/wk increase: 0.88, 95% CI 0.78-1.00). Early prepregnancy weight gain was not strongly associated with PPROM. Weight gain per week was associated with indicated preterm delivery(after adjustment for age, race/ethnicity, BMI, and parity) (OR per 0.1 kg/wk increase: 1.13, 95% CI 1.01-1.26). **Maternal confounders/effect modifiers:** • Maternal age • Parity • Race/ethnicity

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
		< 30,000, 3.7% 30,000-69,999, 22.3% ≥ 70,000, 71.3%	• Marital status • Education level • Income • Smoking status • Prenatal vitamin use • Prior preterm delivery • Prepregnancy hypertension • Prepregnancy diabetes • Preeclampsia • Pregnancy induced hypertension • Gestational diabetes **Infant and child confounders/effect modifiers:** NR
Author, year: Scott et al., 1981 **Country/Setting:** UK **Enrollment period:** 1964 to 1977 **Study Objective:** To assess the relative contributions of different maternal factors in SGA pregnancies.	**Design:** • Cohort • Retrospective **Total Study N:** 855 **Group Description:** G1: SGA G2: AGA **Group N:** G1: 488 G2: 367 **Inclusion criteria:**	Net pregnancy gain = Final weight – (prepregnancy weight, fetal weight, placental weight)	**Outcomes description:** • SGA **Results:** G1: Mean maternal height: 159.4 cm (OR: 2.03 [RR: 1.3-3.1]) Mean prepregnancy weight: 55.5 kg (OR: 1.84 [RR: 1.2-2.8]) Mean net GWG: 7.3 kg (OR: 1.78 (1.1-2.8]) Previous liveborn infant > 1 SD below mean: 64.0% (OR: 7.98 [RR: 4.7-13.5]) Manual social classes: 67.5% (OR: 1.08 [RR 0.8-1.5]) Smoking during pregnancy: 59.1%

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
	• Gave birth to an SGA infant in one of two study hospitals • Personally interviewed within 3 days of delivery • Babies examined by medical members of research team • Birth weight more than 2 SDs below the mean for gestational age and sex (British Perinatal Mortality Survey) • Had to be sure of dates, with a regular menstrual cycle not exceeding 32 days • Last period was not a withdrawal bleeding from the pill • Liveborn, singletons **Exclusion criteria:** • Infants with major congenital anomalies		(OR: 3.04 [RR 2.2-4.2]) Hypertension without preeclampsia: 28.5% (OR: 2.84 [RR 1.9-4.2]) Preeclampsia: 10.5% (OR: 15.78 [RR: 6.2-40.4]) G2: Mean maternal height: 162.0 cm Mean prepregnancy weight: 58.5 kg Mean net GWG: 9.0 kg Previous liveborn infant > 1 SD below mean: 15.2% Manual social classes: 54.1% Smoking during pregnancy: 32.4% Hypertension without preeclampsia: 18.3% Preeclampsia: 1.6% **Maternal confounders/effect modifiers:** • Maternal height/weight (prepregnancy) • Previous live born infants more than 1 SD below mean • Manual social class • Smoking during pregnancy • Hypertension without preeclampsia • Preeclampsia **Infant and child confounders/effect modifiers:** NR
Author, year: Segal et al., 2008 **Country/Setting:**	**Design:** • Cross-sectional	Prepregnancy BMI categories: normal (≤24); overweight (25-29.9); obese (≥30).	**Outcomes description:** • Obstetrical outcomes (infant birth weight, LGA)

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
Canada **Enrollment period:** NR **Study Objective:** To examine the maternal factors that determine infant birth weight.	**Total Study N:** 86 **Inclusion criteria:** • Healthy Caucasian women • Singleton pregnancies • Attended outpatient obstetrics study clinics • Had been referred for a 100-g oral glucose tolerance test (OGTT) following an abnormal result in a screening test. • Were recruited in late second/early third trimester • Women with normal glucose tolerance or impaired glucose tolerance **Exclusion criteria:** • Women diagnosed with GDM on the OGTT	Mean Age: 33.1 yrs Mean prepregnancy BMI: 29.6 kg/m^2 BMI Class: Normal, 58.1% Overweight, 27.9% Obese, 14.0% Nulliparous, 53.5%	**Results:** Mean birth weight, 3,519 g Macrosomic infants: 16.3% LGA infants, 16.3% There was a positive association between prepregnancy BMI and birth weight (after adjustment for length of gestation) ($r = 0.31$, $p = 0.0063$). There was negative association between birth weight and maternal serum levels of adipocentin ($r= -0.3$, $p = 0.0084$). Prepregnancy BMI was found to be a positive predictor of LGA (OR: 1.25, 95% CI, 1.05-1.49). A positive association was also seen between GWG preceding the OGTT and LGA (OR: 1.14, 95% CI 0.98-1.34). **Maternal confounders/effect modifiers:** • Family history of diabetes (type 2 DM), • Age • Prepregnancy BMI • GWG preceding OGTT • Current smoking • Area under the glucose curve • Index of insulin sensitivity • Parity • Adipocentin **Infant and child confounders/effect modifiers:** NR
Author, year:	**Design:**	Data was pulled from medical	**Outcomes description:**

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Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
Shepard et al., 1986 **Country/Setting:** USA **Enrollment period:** May 1980 to Aug 1982 **Study Objective:** To study maternal weight gain as a proportion of prepregnant weight and to examine its relationship to complications of pregnancy, labor, and delivery	• Cohort • Retrospective **Total study N:** 1,396 **Group Description:** by proportional weight gain (weight gain/prepregnant weight) G1: gains ≤ 15 % G2: gains 16-25% G3: gains 26-35% G4: gains >35% **Group N:** G1: 118 G2: 548 G3: 565 G4: 165 **Inclusion criteria:** • Delivered at study hospital • Obtained prenatal care at one of several study sites • Singleton infant delivered between 37-42 wks gestation • No history of diabetes, hypertension, CVD,	records. Proportional weight gain = weight gain/prepregnant weight Prepregnant Weight: G1: ≤ 110 lbs: 4.2% 111-120 lbs: 18.6% 121-130 lbs: 18.6% 131-140 lbs: 28.8% > 140 lbs: 29.7% G2: ≤ 110 lbs: 8.2% 111-120 lbs: 19.7% 121-130 lbs: 30.7% 131-140 lbs: 19.3% > 140 lbs: 22.1% G3: ≤ 110 lbs: 14.5% 111-120 lbs: 28.9% 121-130 lbs: 23.4% 131-140 lbs: 19.3% > 140 lbs: 14.0% G4: ≤ 110 lbs: 27.3% 111-120 lbs: 30.3% 121-130 lbs: 21.2% 131-140 lbs: 12.7% > 140 lbs: 8.5%	• Maternal weight gain • Complications of pregnancy, labor, and delivery **Results:** Gestational hypertension: G1: 4.2% G2: 2.6% G3: 4.1% G4: 9.1% Preeclampsia: G1: 2.5% G2: 1.8% G3: 4.3% G4: 7.0% Vaginal bleeding: G1:7.6% G2: 5.1% G3: 3.5% G4: 3.0% Cesarean delivery: G1: 17.0% G2:12.0% G3: 16.8% G4: 20.6% Infant birth weight: G1: ≤ 2,500 g: 1.7% 2,501-4,000 g: 94.9% > 4,000 g: 3.4% G2: ≤ 2,500 g: 1.1% 2,501-4,000 g: 90.0%

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
	seizure, thyroid problems **Exclusion criteria:** • Infants delivered with severe congenital malformations • Women who lost weight during pregnancy • Women with missing height/weight information on medical records		> 4,000 g: 8.9% G3: ≤ 2,500 g: 0.4% 2,501-4,000 g: 86.2% > 4,000 g: 13.5% G4: ≤ 2,500 g: 1.2% 2,501-4,000 g: 77.6% > 4,000 g: 21.2% **Maternal confounders/effect modifiers:** • Marital status • Age • Race/ethnicity • Education level • Smoking status • Alcohol consumption • Gravidity • Prepregnant weight/height **Infant and child confounders/effect modifiers:** • Gestational age • Infant sex
Author, year: Tavris and Read, 1982 **Country/Setting:** USA (San Francisco, CA) **Enrollment period:**	**Design:** • Cohort • Retrospective **Total Study N:** 2,590	Groups were compared by maternal weight gain categories: -24 to -16 lbs, 1-15 to -11 lbs, -10 to -6 lbs, -5 to -1 lbs, 0 to 4 lbs, 5 to 9 lbs, 10 to 14 lbs, 15 to 19 lbs, 20 to 24 lbs, 25 to 29 lbs, 30 to 34 lbs, ≥ 35 lbs.	**Outcomes description:** • Fetal and neonatal deaths • Other deaths before age 5 • Cognitive development (Raven Colored Progressive Matrices test scores) **Results:**

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
April 1, 1964 to April 1966 **Study Objective:** To examine the effect of maternal weight gain on fetal, infant, and childhood death and on cognitive development.	**Group Description:** G1: Fetal deaths G2: Neonatal deaths G3: Infant or childhood deaths **Group N:** G1: 145 G2: 45 G3: 32 **Inclusion criteria:** • Pregnant members of the Kaiser Permanente Foundation Health Plan **Exclusion criteria:** • Not weighed within 120 days following last menstrual period and again within 10 days of delivery		Low weight gain categories had a higher percentage of fetal and neonatal death than the high weight gain categories ($p < 0.001$). When confining delivery to 35 weeks' or more, the relationship between gain and fetal and neonatal deaths was no longer significant. Raven Coloured Progressive Matrices scores at age 5 were better for children with mothers who gained between 5 and 29 lbs as compared with mothers who gained below 5 lbs or above 29 lbs. No significant differences in test scores were seen in the weight gain group of 5 to 29 lbs. **Maternal confounders/effect modifiers:** • Maternal age • Race • Parity • Education • Prepregnancy weight/height ratio • Time interval since last pregnancy • Paternal education • Annual income of parents **Infant and child confounders/effect modifiers:** • Gestational age at time of delivery
Author, year: Tenovuo et al., 1988 **Country/Setting:**	**Design:** • Cohort • Prospective	Gestational age = G1: 38.8 wks; G2: 38.8 wks Birth weight = G1: 2,452 g; G2: 3,378 g	**Outcomes description:** • Gestational age • Birth weight/length • Head circumference

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
Finland **Enrollment period:** 1985 **Study Objective:** To determine the risk factors associated with severely SGA neonates.	**Total Study N:** 236 **Group Description:** G1: Severely SGA neonates G2: Control **Group N:** G1: 118 G2: 118 **Inclusion criteria:** • Born within study period within study area **Exclusion criteria:** NR	Birth length = G1: 46.6 cm; G2: 50.2 cm Head circumference = 32.7 cm; G2: 34.5 cm Maternal age = G1: 27 yrs; G2: 27 yrs Paternal age = G1: 30 yrs; G2: 29 yrs Maternal height = G1: 163 cm; G2: 165 cm Paternal height = G1: 177 cm; G2: 179 cm	**Results:** **Effects of Risk Factors, Odds Ratio (p value)** Previous SGA infant: Mild = 2.69 Severe = 5.39 (0.008) Low social class: 2.67 (0.054) Low prepregnancy weight (kg): 1.04 (0.012) Toxemia: 4.58 (0.004) Smoking: 1-9 cigarettes/day = 1.58 >10 cigarettes/day = 3.4 (0.042) Poor GWG (kg): 1.10 (0.015) The most important prepregnancy risk factors for SGA were low maternal prepregnancy weight and a maternal history of previous SGA infant. The most important pregnancy-related risk factors for SGA were poor GWG, toxemia, and smoking. **Maternal confounders/effect modifiers:** • Smoking • Previous SGA infant • Toxemia • Parity • Previous pregnancy-related complications • Social class • Maternal/paternal age and height **Infant and child confounders/effect modifiers:**

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
			NR
Author, year: Tilton et al., 1989 **Country/Setting:** Santiago, Chile **Enrollment period:** NR **Study Objective:** To examine the influence of obesity on obstetric performance, pregnancy outcome, and lactational performance.	**Design:** • Cohort • Retrospective **Total Study N:** 326 **Group Description:** G1: Obese G2: Normal weight **Group N:** G1: 163 G2: 163 **Inclusion criteria:** • Obese gravidas • Singleton pregnancies • 20 + years of age • Had first prenatal visit no later than 18 weeks gestation **Exclusion criteria:** • Lack of adequate match to obese group	Obesity was defined as weight/height equivalent to 120% and over of standard weight at 20 wks of gestations. Mean age (yrs) G1: 29.9; G2: 29.9 Mean weight at first visit (kg) G1: 72.46; G2: 54.76 Mean height (cm) G1: 156.1; G2: 156.1 Mean parity G1: 1.12; G2: 1.15 Mean gestational age at first visit (wks) G1: 11.7; G2: 11.4 Percent primiparous G1: 30.7; G2: 32.5 Prepregnancy risk factors: Smoker G1: 28.4%; G2: 15.63% Previous cesarean G1: 20.9%; G2: 16.0% Chronic hypertension G1: 3.7%; G2: 0.6% Nephropathy G1: 2.5%; G2: 2.5% Cardiopathy G1: 1.2%; G2: 0.6%	**Outcomes description:** • Obstetric performance • Pregnancy outcome • Lactational performance **Results:** Obese gravidas had significantly increased incidences of gestational hypertension, inadequate pregnancy weight gain, cesarean section, postpartum infections, and LGA infants. No significant increase was seen on obese gravidas for incidence of diabetes, toxemia, breech presentation, postpartum hemorrhage, infant morbidity, or lactational failure. **Maternal confounders/effect modifiers:** • Age • Height • Parity • Gestational age at first visit **Infant and child confounders/effect modifiers:** • Birth weight • Age-weight classification of infant • Postpartum infections • Mode of delivery

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
		Chronic diabetes G1: 0.6%	
Author, year: Udal et al., 1978 **Country/Setting:** USA (Arizona) **Enrollment period:** NR **Study Objective:** To examine the relationship between maternal and neonatal obesity.	**Design:** • Cohort **Total Study N:** 109 **Group Description:** G1: Obese mothers G2: Nonobese mothers **Group N:** G1: 33 G2: 76 **Inclusion criteria:** • Nondiabetic mothers • Infants born at 37-43 weeks' gestation • Examined within 72 hours of birth **Exclusion criteria:** • Infants of diabetic mothers • Twins • Neonates with known congenital or metabolic abnormalities	Prepregnant weight: obtained from maternal history or medical chart review. Obese prepregnant weight >120% of median weight for height. Weight at term: admitting obstetric nurse Neonatal fatness was calculated by the sum of eight skin fold measurements (SSFT). Race: White, n = 98 Black, n = 5 American Indian, n = 6	**Outcomes description:** • Neonatal obesity **Results:** Parameters of Infants Born to Mothers Birth weight (gm) = G1: 3,471 ± 739; G2: 3,279 ± 494 (p value NS) SSFTs (mm) = G1: 30.2 ± 9.1; G2: 26.0 ± 5.2 (p value < 0.05) Head circumference (cm) = G1: 34.7 ± 1.9; G2; 34.3 ± 1.3 (p value NS) Length (cm) = G1: 50.5 ± 3.3; G2: 50.2 ± 2.2 (p value NS) LGA infants tended to have higher skin fold thickness measurements (sum of eight skin fold measurements) and obese mothers had infants with significantly increased skin fold thickness measurements. GWG was associated with increased neonatal fatness and length, while prepregnancy weight for height was associated with neonatal fatness independent of length. GWG (kg) = 26 ± 18 in fatter LGA infants as compared to 14 ± 7 in other LGA infants (p value < 0.01). **Maternal confounders/effect modifiers:** • GWG • Parity

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
			• Prepregnancy weight/height • Cigarette smoking • Family history of diabetes • Gestational age **Infant and child confounders/effect modifiers:** • Gestational age • Birth weight • Bilateral mid-arm circumference • Eight skin fold thickness measurements
Author, year: Varma, 1984 **Country/Setting:** UK (London) **Enrollment period:** 1978-1980 **Study Objective:** To assess the relationship between maternal weight at booking in the first trimester and the total weight gain during pregnancy and birth weight, complications, and mode of delivery.	**Design:** • Cohort • Retrospective **Total Study N:** 3,002 **Group Description:** G1: GWG ≤ 2.5 kg G2: GWG 2.5-5.9 kg G3: GWG 6.0-10.9 kg G4: GWG 11.0-15.9 kg G5: 16.0-20.9 kg G6: 21+ kg **Group N:** G1: 182 G2: 272 G3: 1,114 G4: 1,028	Maternal booking weight: recorded under standardized conditions in clinic, every 4 wks up to 28 wks then every 2 wks from 28-36 wks and weekly from 36 wks-delivery.	**Outcomes description:** • Total weight gain • Maternal weight • Pregnancy complications • Mode of delivery • Birth weight • Fetal status in labor (cardiotocographic tracing and fetal scalp pH) **Results:** Preeclampsia G1: 8.8% G2: 5.9% G3: 9.7% G4: 9.7% G5: 10.3% G6: 11.1% Preexisting hypertension G1: 4.4% G2: 4.4%

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
	G5: 252 G6: 154 **Inclusion criteria:** • Seen in antenatal clinic during first trimester **Exclusion criteria:** • Diabetes • Multiple pregnancy • Polyhydramnios • Gastrointestinal disorders		G3: 6.1% G4: 6.8% G5: 11.9% G6: 13.9% IUGR G1: 29.7% G2: 14.7% G3: 5.8% G4: 7.0% G5: 6.3% G6: 6.9% Premature Labor G1: 8.8% G2: 4.4% G3: 2.5% G4: 2.5% G5: 2.4% G6: 4.1% Antepartum hemorrhage G1: 5.5% G2: 3.3% G3: 2.5% G4: 3.1% G5: 3.2% G6: 3.5% Fetal distress in labor G1: 12.1% G2: 5.1% G3: 4.1% G4: 4.9% G5: 6.3% G6: 6.3%

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
			Normal delivery G1: 73.6% G2: 73.5% G3: 72.4% G4: 68.6% G5: 69.8% G6: 55.6% Forceps delivery G1: 13.2% G2: 14.7% G3: 13.1% G4: 16.9% G5: 17.4% G6: 27.8% C-Section G1: 13.2% G2: 11.8% G3: 14.5% G4: 14.4% G5: 15.1% G6: 16.6% **Maternal confounders/effect modifiers:** NR **Infant and child confounders/effect modifiers:** NR
Author, year: Voldner et al., 2008 **Country/Setting:**	**Design:** • Cohort • Prospective	BMI and maternal anthropometric measures were collected at each visit. Gestational age, gender of child,	**Outcomes description:** • Macrosomic infant (≥ 4,200 g) **Results:**

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
Norway **Enrollment period:** 2002-2005 **Study Objective:** To examine the modifiable determinants of fetal macrosomia, specifically lifestyle-related factors.	**Total Study N:** 553 **Inclusion criteria:** • Healthy women • Singleton pregnancies • Scandinavian heritage • Delivered at study hospital	parity, maternal age, maternal height, smoking habits, marital status, education level, work outside the home	Weight gain < 10.2 kg Univariate analyses: OR = 1.0 Multivariate analyses: OR = 1.0 Weight gain ≥ 10.2 kg Univariate analyses: OR = 1.7 (95% CI 1.0-2.8; p value 0.04) Multivariate analyses: OR = 1.7 (95% CI 0.9-3.2; p value 0.09) BMI, weight gain, plasma glucose and gestational age were independent determinants of macrosomia (if physical activity was left out of the analysis). Once physical activity was included (low level, prepregnancy), this became a significant determinant for macrosomia (OR 2.9, 95% CI: 1.9, 7.3). **Maternal confounders/effect modifiers:** • Maternal BMI • Maternal subcutaneous fat at visit one • Weight gain in pregnancy • Plasma glucose values (visit one and three) • Intake of energy and energy providing nutrients • Smoking and level of physical activity before and during pregnancy **Infant and child confounders/effect modifiers:** • Gestational age
Author, year: Wolff et al., 2008	**Design:** • Randomized control trial	Weight, height, blood pressure and heart rate were measured at inclusion and at 27 and 36 weeks' gestation.	**Outcomes description:** • Dietary intake and weight development • Glucose metabolism

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
Country/Setting: Denmark **Enrollment period:** NR **Study Objective:** To examine the effects of dietary counseling on GWG and glucose metabolism in obese pregnant women.	**Total Study N:** 50 **Group Description:** G1: Intervention (nondiabetic obese pregnant women) G2: Control **Group N:** G1: 23 G2: 27 **Inclusion criteria:** • Obese pregnant women (BMI ≥ 30 kg/m^2) • Nondiabetic • 15 ± 3 weeks' gestation at enrollment **Exclusion criteria:** • Smoked • Age < 18 years or > 45 years • Multiple pregnancy • Medical complications known to affect fetal growth or weight gain	Prepregnancy weight, weight gain from 36 weeks' gestation until delivery, and postpartum weight (1st, 2nd, and 3rd weeks), were self reported. Weight at 4 weeks postpartum was measured at the department. Total GWG was calculated as the difference between self-reported prepregnancy weight and weight just before delivery. G1: received 10 one-hour consultations with a trained dietician during the pregnancy; were instructed to eat a healthy diet; and limit energy intake based on individual requirements (estimated by energetic cost of fetal growth).	• Birth outcome **Results:** G1: Average GWG = 6.6 ± 5.5 kg G2: Average GWG = 13.3 ±7.5 kg (mean difference of 6.7 kg; 95 % CI: 2.6-10.8, P = 0.002) G1: Average gain/wk = 0.26 ± 0.15 kg/wk G2: Average gain/wk = 0.44 ± 0.21 kg/wk (mean difference of 0.18 kg/wk (95% CI: 0.07-0.30, P = 0.02) G1 also had a 20% reduction in both s-insulin and s-leptin as compared to G2 at week 27. Birth weight (g) G1: 3,757 ± 617 G2: 3,895 ±485 Infant length (cm) G1: 52 ± 3 G2: 53 ± 2 Gestational age (days) G1: 281 ± 13 G2: 280 ± 11 Placental weight (g) G1: 701 ± 131 G2: 771 ± 161 Head circumference (cm) G1: 35 ± 1 G2: 36 ± 2 Abdominal circumference (cm) G1: 35 ± 1

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
			G2: 34 ± 2 GDM G1: 0 (0%) G2: 3 (10%) Pregnancy-induced hypertension G1: 1 (4%) G2: 4 (1%) Preeclampsia G1: 0 (0%) G2: 1 (4%) Prolonged pregnancy G1: 3 (13%) G2: 4 (15%) Cesarean delivery G1: 2 (9%) G2: 3 (11%) **Maternal confounders/effect modifiers:** NR **Infant and child confounders/effect modifiers:** NR
Author, year: Wrotniak et al., 2008 **Country/Setting:** USA (National Collaborative Perinatal Project) **Enrollment period:**	**Design:** • Cohort • Retrospective **Total Study N:** 10,226 **Inclusion criteria:** • Singleton, term	Prepregnancy weight was self-reported at enrollment. GWG and height were measured at time of delivery and were used to calculate GWG. Median Prepregnancy BMI: 21.9 kg/m^2	**Outcomes description:** • Offspring overweight status • GWG **Results:** Median GWG: 9.5 kg Median birth weight: 3,230 g Median gestational age: 40 wks Median BMI at 7 yr assessment: 15.7 kg/m^2

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TABLE F-1 Consequences of Gestational Weight Gain

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics	Outcomes/Results/Confounders
1595 to 1972 **Study Objective:** To examine the association of GWG with offspring overweight at 7 years of age.	pregnancies **Exclusion criteria:** • Mothers with GDM • Gestational age < 37 weeks or > 42 weeks	Median Age: 23 yrs Race: Black, 47.4% White, 50.5% Hispanic, 1.3% Other, 0.8%	Overweight status at 7 yr assessment (BMI at or above 95th percentile): 5.7% *Adjusted Association between GWG and overweight at 7 yrs* GWG by 1 kg of weight gain: OR 1.03 (95% CI: 1.02, 1.05) Excessive GWG vs Recommended GWG (IOM): OR 1.48 (95% CI: 1.06, 2.06) Insufficient weight gain vs Recommended weight gain (IOM): OR 0.88 (95% CI: 0.68, 1.14) The association between GWG and overweight in offspring was strongest for women underweight before pregnancy. **Maternal confounders/effect modifiers:** • Race • Age • Prepregnancy BMI • Number of cigarettes smoked/day **Infant and child confounders/effect modifiers:** • Sex • First-born status • Study site • Gestational age

NR = Not reported.

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Appendix G

Consultant Reports

As part of its approach, the committee commissioned analyses from consultants to aid in decision making by providing information not readily available in current literature. Dr. Ellen Nohr from Aarhus University, Denmark, provided analyses from the Danish National Birth Cohort on low and very high categories of gestational weight gain (GWG), as well as data for obese class I, II and III women. Additionally, she provided information on subgroups pregnant women, such as primiparous, short and young women, and smokers (see Part I). Dr. Amy Herring, University of North Carolina, provided data from the 1988 National Maternal and Infant Health Survey (NMIHS) on the association between GWG and pregnancy outcomes by race. She provided additional analyses on the association between GWG and postpartum weight retention by linking the 1988 NMIHS to its 1991 follow-up (see Part II). Dr. Cheryl Stein, Mount Sanai School of Medicine, provided data from on adverse outcomes associated with GWG stratified by racial/ethnic group using births data from 1995-2003 in New York City (see Part III). Dr. James Hammitt, Harvard University, conducted a quantitative analysis of risk trade-offs between maternal and child health outcomes associated with GWG (see Part IV).

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PART I: ANALYSES FROM DR. NOHR

COMBINED ASSOCIATIONS OF PREPREGNANCY BODY MASS INDEX AND GESTATIONAL WEIGHT GAIN WITH THE OUTCOME OF PREGNANCY. ANALYSES BASED ON THE DANISH NATIONAL BIRTH COHORT

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The combined associations of prepregnancy body mass index (BMI) and gestational weight gain on pregnancy outcomes have until recent years mostly focused on birth weight. Large data collections with detailed information about maternal characteristics and pregnancy outcomes are now available which makes it possible to investigate these associations in a broader range of maternal and neonatal outcomes while adjusting for important maternal life style factors. Such a study based on the Danish National Birth Cohort (DNBC) was presented for the Committee to Reexamine IOM Pregnancy Weight Guidelines in June 2008 (Nohr et al., 2008) along with a number of analyses that focused on the BMI-specific association between GWG and all outcomes included in the study. These supplementary analyses are presented in the following in the 'First DNBC Report'. At the meeting in June, the IOM committee requested new analyses for some outcomes where very low and very high categories of GWG as well as obese class I and obese class II + III were included. This work is presented in the 'Second DNBC Report'. In August 2008, additional analyses were presented for the IOM committee that provided information in subgroups of women defined by parity, height, smoking and young age. These results are presented in the 'Third DNBC Report'.

First DNBC Report

Study population

The Danish National Birth Cohort (DNBC) is a nationwide study of 100,419 pregnancies among 92,274 women recruited 1996-2002. More detailed descriptions of the study methods and the recruitment were previously published (Olsen et al., 2001; Nohr et al., 2006; Danish National Birth Cohort homepage, Available online: http://www.ssi.dk/sw9314.asp [accessed February 2009]). Briefly, data were collected during 2 telephone interviews during pregnancy at approximately 16 and 30 wk of gestation, and 2 telephone interviews after birth when the child was approximately 6 and 18 months old. The women included in the cohort were mostly Caucasians as only 4 percent were born outside Scandinavia.

This study used information about 60,892 liveborn, full-term singleton (≥ 37 wk of gestation) infants whose mothers had participated in the first pregnancy and the first postpartum interview and provided information about prepregnancy BMI, GWG and postpartum weight retention 6 months after birth. In the following, the data and methods of the study will be shortly presented. A more detailed description has been published (Nohr et al., 2008).

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Independent variables

The main exposures were prepregnancy BMI and GWG. In the first pregnancy interview, the women reported their prepregnancy weight and height, which was used to calculate their prepregnancy BMI and categorize them according to the World's Health Organization's definitions as underweight (BMI < 18.5 kg/m^2), normal weight (18.5 ≤ BMI < 25 kg/m^2), overweight (25 ≤ BM I < 30 kg/m^2), and obese (BMI ≥ 30 kg/m^2) (WHO, 2000). Gestational weight gain was based on information from the telephone interview 6 months after birth. At this time, the woman was asked "How much (in kg) was your total gain in pregnancy?" Her response was divided into 4 categories: low (<10 kg) medium (10-15 kg), high (16-19 kg, and very high (≥ 20 kg). The medium category, which has been associated with minimum infant mortality in other populations (IOM, 1990) was used as reference.

From the first pregnancy interview, we also used information about the mother's age at conception, parity, smoking, alcohol intake and physical exercise during pregnancy, and social status defined by education and occupation. Information about duration of breastfeeding was reported by the women in the first postpartum interview. The categorization of these variables is described in greater detail elsewhere (Nohr et al., 2008).

Maternal outcomes

Pregnancy outcomes during late pregnancy included preeclampsia/eclampsia, chronic/gestational hypertension and gestational diabetes and were identified through linkage to the National Hospital Discharge Register. Because we suspected some underreporting of gestational diabetes, we added self-reported information about this disease from the pregnancy interviews.

Birth complications were also identified in the National Hospital Discharge Register and included instrumental deliveries, which in nearly all cases covered vacuum extraction, and planned and emergency cesarean deliveries. The latter type covered cesarean section carried out when the woman was in labor.

Postpartum weight retention was calculated as the difference between the woman's prepregnancy weight and her weight 6 months postpartum as reported in the first postpartum interview. Postpartum weight retention was summarized by 2 variables defined as postpartum weight loss (loss ≥ 2 kg) and postpartum weight retention (gain of ≥ 5 kg) relative to a woman's prepregnancy weight. In the same way, postpartum weight retention at 18 months was calculated for those women in the study population who participated in the second postpartum interview, who had not given birth again and who were not pregnant again (39,776 women).

Neonatal outcomes

Neonatal outcomes were identified in the National Birth Register and included birth weight, length, gestational age as recorded at birth, and Apgar score after 5 minutes. Birth weight was standardized by gestational age according to the reference curve of Marsal et al. (1996). Standardized birth weight was dicotomized into either a small-for-gestational age (SGA) infant (z-score < 10th percentile) or a large-for-gestational age (LGA) infant (z-score > 90th percentile). Additionally, results for SGA defined as a z-score < 2.5th percentile and for birth weight > 4000 gram were presented.

To estimate the relative fat tissue of the infant, we calculated ponderal index of the newborn (birth weight in grams divided by the birth length in cm cubed). We defined low ponderal index

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as values < 10th percentile and high ponderal index as values > 90th percentile. Low Apgar score was defined as a value < 8 after 5 min.

Statistical methods

A BMI- and GWG-specific variable was generated by cross-classifying BMI group (4 categories) and GWG group (4 categories). In multiple logistic regression models, the associations between this variable and pregnancy outcomes were estimated. This corresponds to the full model with an interaction term between the original BMI and GWG variables. Normal weight women with medium GWG (10-15 kg) were used as reference. These models were adjusted for a number of maternal characteristics and lifestyle factors and for gestational age at birth. In the analyses of birth complications, neonatal complications, and postpartum weight retention, women with preeclampsia and gestational diabetes were excluded (n = 1787). In the analyses of emergency cesarean deliveries, women with a planned cesarean were excluded, and in the analyses of instrumental deliveries, all women with cesarean deliveries were excluded. In all adjusted models, Wald's test with 9 degrees of freedom and a significance level of 0.05 (two sided p-value) was used to assess the hypothesis that there was no effect modification by BMI group of the association between GWG and pregnancy outcomes.

Because we observed that background risks of most pregnancy outcomes increased with increasing BMI groups in a way that was not well reflected in a multiplicative model, we also used an additive approach to the data. Thus, we used the calculated odds ratios from the above models to compute 16 absolute adjusted risks for each pregnancy outcome according to each category within the BMI-and GWG-specific variable for a woman with a given set of confounder categories: She was primiparous, 25-29 years old, 1.60-1.69 m tall, reported no smoking, no alcohol intake and no exercise during pregnancy, was of high social status and gave birth after 280 days of conception. For postpartum weight retention, she breastfed < 14 weeks.

Results

Figures 1-18 in this report are supplementary to the study by Nohr et al. (2008). The first 17 figures display odds ratios and adjusted absolute risks for different outcomes. In Figure 18, the absolute risks for 4 important outcomes are stratified on BMI group and combined to evaluate the 'trade-off' between mother and infant according to GWG:

Odds ratios, displayed in the upper part of all figures, showed that:

- Except for birth weight and postpartum weight retention, prepregnancy BMI was by far the strongest predictor of the outcomes under study.
- There was little evidence of interaction between BMI and GWG in a multiplicative model. It was only present for birth weight and postpartum weight retention ($p < 0.01$), and although statistical important, it was judged to be of minor clinical importance.

In the lower part of all figures, BMI- and GWG-specific adjusted absolute risks for all included pregnancy outcomes showed that:

- Across BMI groups, background risks varied highly, which led to highly varying risk differences when moving from low to high GWG. Especially the risk of SGA and LGA

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were related to both increasing BMI and increasing GWG (Figures 4-7). In contrast, the absolute risk of postpartum weight retention was highly responsive to GWG, but not to BMI.

- These observations support the idea of BMI-specific recommendations. According to Figure 18, especially underweight women may benefit from very high GWG to prevent having a small baby while heavier women may benefit from avoiding high and very high GWG which only brings a slight increase of growth restriction for the infant.

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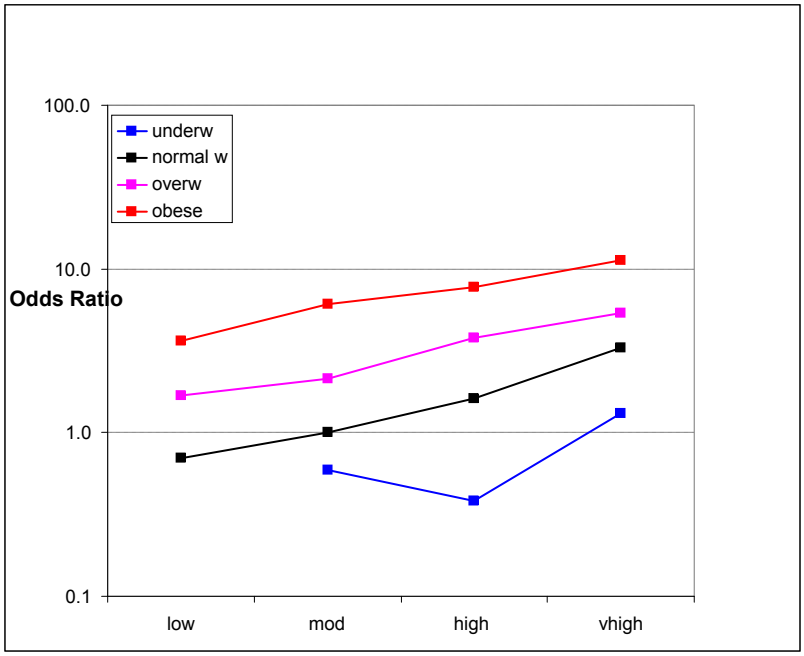

FIGURE 1A Preeclampsia
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 1A Preeclampsia, adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	0.0	0.6	0.4	1.3
Normal weight	0.7	1.0	1.6	3.3
Overweight	1.7	2.1	3.8	5.4
Obese	3.6	6.1	7.7	11.2

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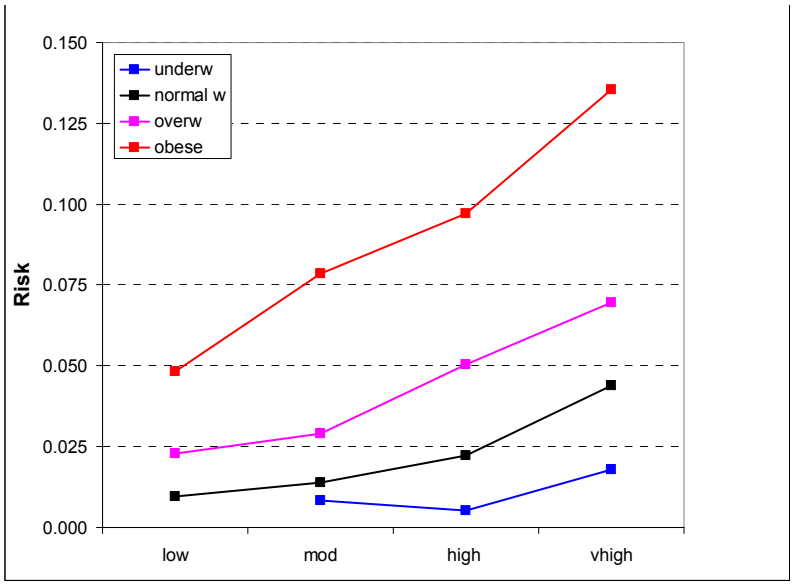

FIGURE 1B Preeclampsia
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 1B Preeclampsia, adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight		0.8%	0.5%	0.8%
Normal weight	1.0%	1.4%	2.2%	4.4%
Overweight	2.3%	2.9%	5.0%	7.0%
Obese	4.8%	7.9%	9.7%	13.6%

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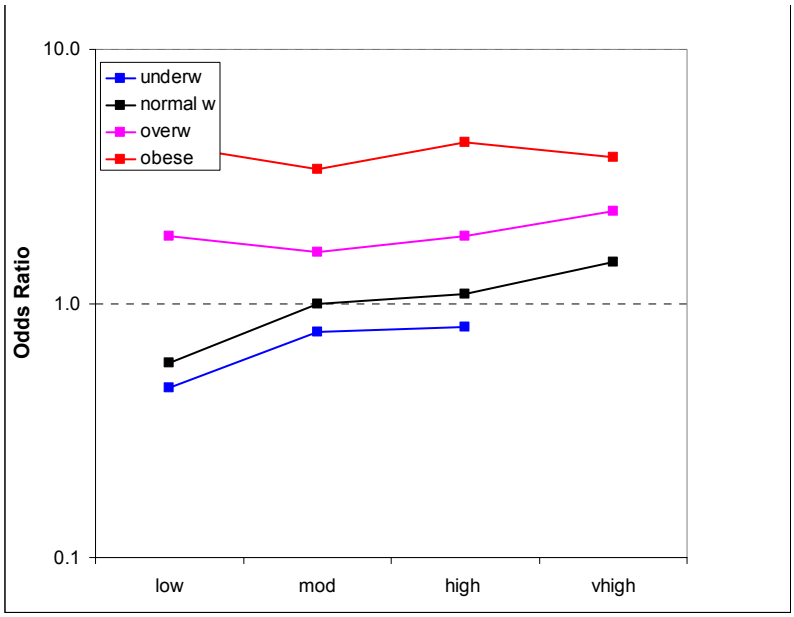

FIGURE 2A Hypertensive disorders
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 2A Hypertensive disorders, adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	0.5	0.8	0.8	0.0
Normal weight	0.6	1.0	1.1	1.5
Overweight	1.8	1.6	1.8	2.3
Obese	4.2	3.4	4.3	3.8

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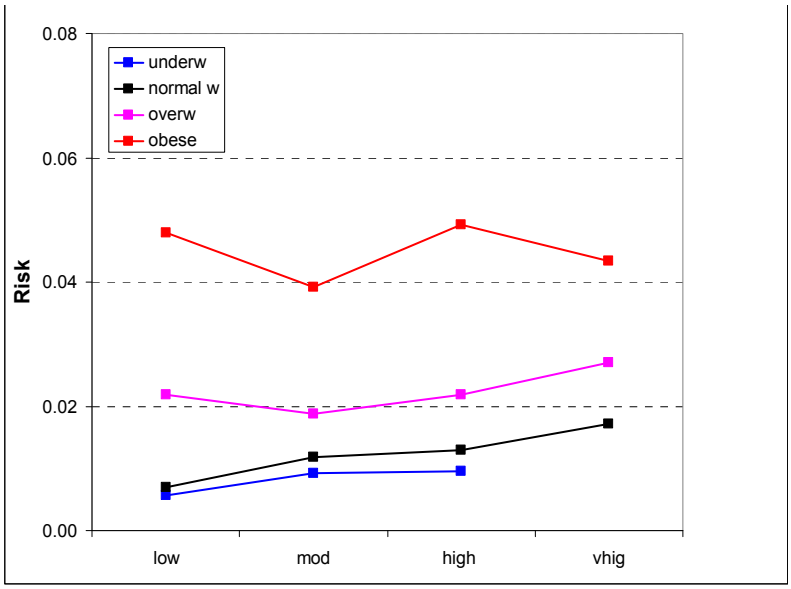

FIGURE 2B Hypertensive disorders
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 2B Hypertensive disorders, adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	0.6%	0.9%	1.0%	
Normal weight	0.7%	1.2%	1.3%	1.7%
Overweight	2.2%	1.9%	2.2%	2.7%
Obese	4.8%	3.9%	4.9%	4.3%

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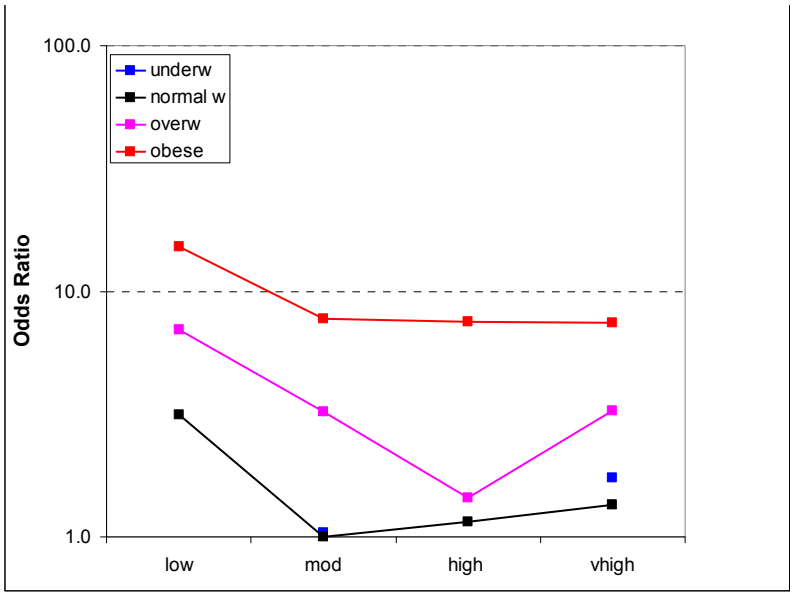

FIGURE 3A Gestational diabetes
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 3A Gestational diabetes, adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	0.0	1.0	0.0	1.7
Normal weight	3.2	1.0	1.2	1.4
Overweight	7.0	3.2	1.4	3.2
Obese	15.1	7.7	7.5	7.4

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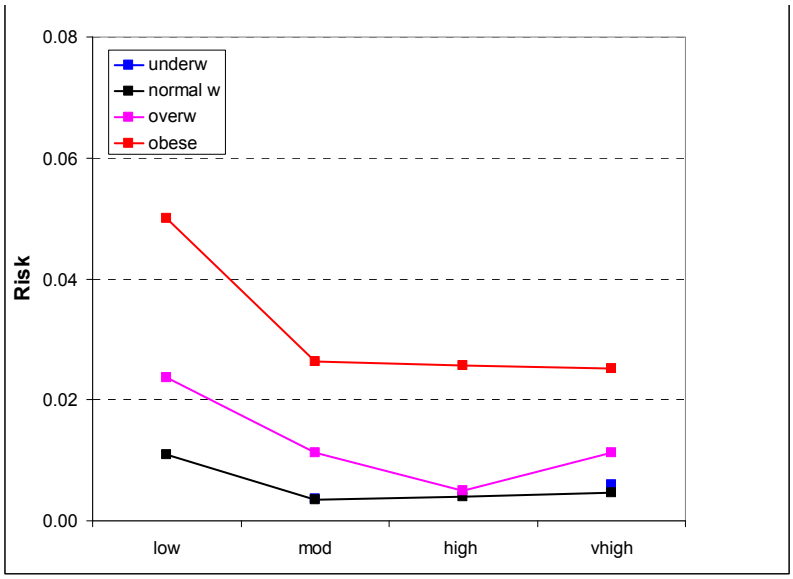

FIGURE 3B Gestational diabetes
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 3B Gestational diabetes, adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight		0.4%		0.6%
Normal weight	1.1%	0.4%	0.4%	0.5%
Overweight	2.4%	1.1%	0.5%	1.1%
Obese	5.0%	2.6%	2.6%	2.5%

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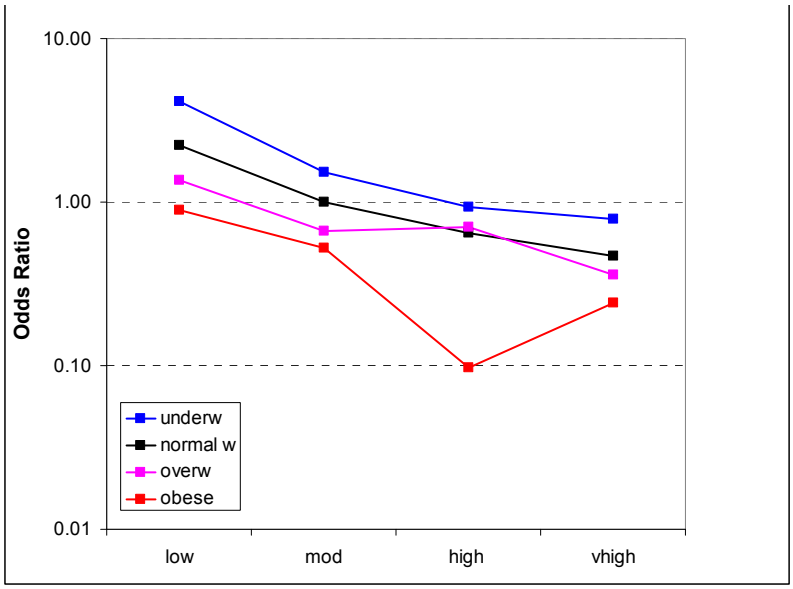

FIGURE 4A Small-for-gestational-age infant (< 2.5 percent)
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 4A Small-for-gestational-age infant (< 2.5 percent), adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	4.1	1.5	0.9	0.8
Normal weight	2.2	1.0	0.6	0.5
Overweight	1.4	0.7	0.7	0.4
Obese	0.9	0.5	0.1	0.2

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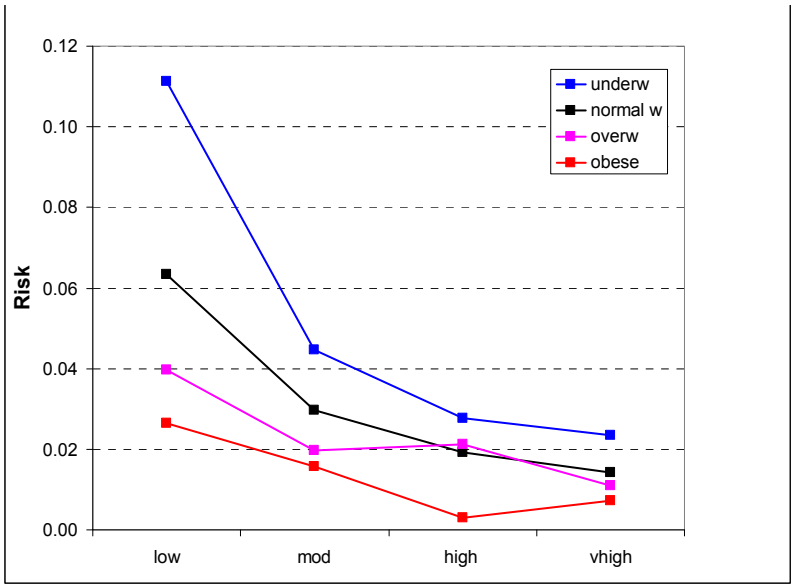

FIGURE 4B Small-for-gestational-age infant (< 2.5 percent)
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 4B Small-for-gestational-age infant (< 2.5 percent), adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	11.1%	4.5%	2.8%	2.4%
Normal weight	6.3%	3.0%	1.9%	1.4%
Overweight	4.0%	2.0%	2.1%	1.1%
Obese	2.7%	1.6%	0.3%	0.7%

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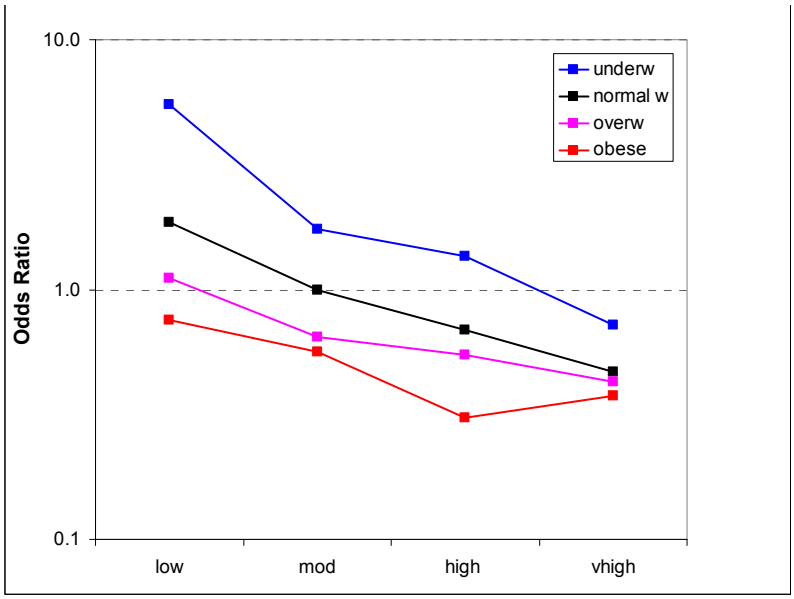

FIGURE 5A Small-for-gestational-age infant (< 10 percent)
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 2A Small-for-gestational-age infant (< 10 percent), adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	5.5	1.7	1.4	0.7
Normal weight	1.9	1.0	0.7	0.5
Overweight	1.1	0.6	0.5	0.4
Obese	0.8	0.6	0.3	0.4

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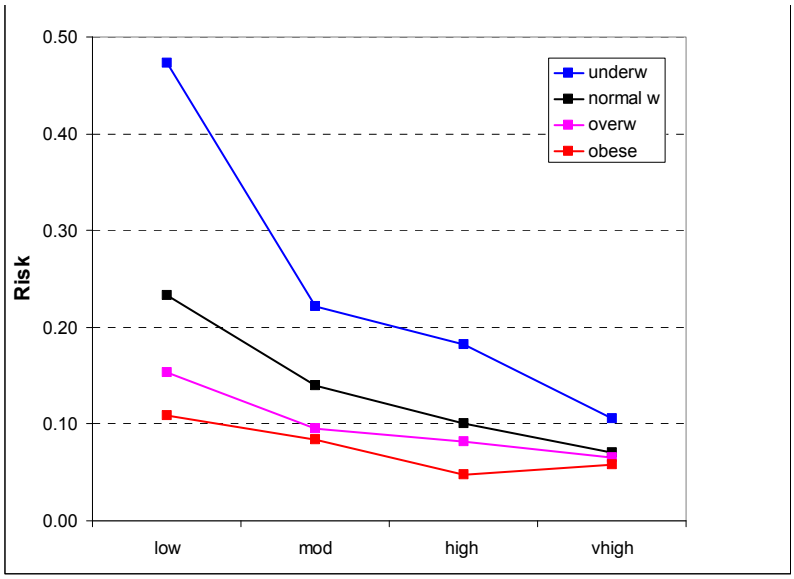

FIGURE 5B Small-for-gestational-age infant (< 10 percent)
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 5B Small-for-gestational-age infant (< 10 percent), adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	47.3%	22.1%	18.2%	10.5%
Normal weight	23.3%	14.0%	10.1%	7.1%
Overweight	15.4%	9.5%	8.2%	6.5%
Obese	10.9%	8.4%	4.8%	5.8%

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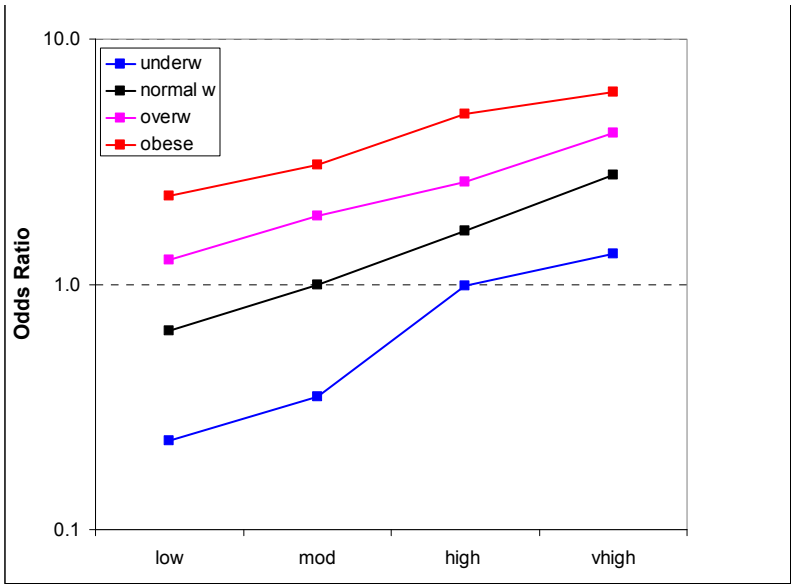

FIGURE 6A Large-for-gestational-age infant (> 90 percent)
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 6A Large-for-gestational-age infant (> 90 percent), adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	0.2	0.3	1.0	1.3
Normal weight	0.6	1.0	1.6	2.8
Overweight	1.3	1.9	2.6	4.1
Obese	2.3	3.1	5.0	6.1

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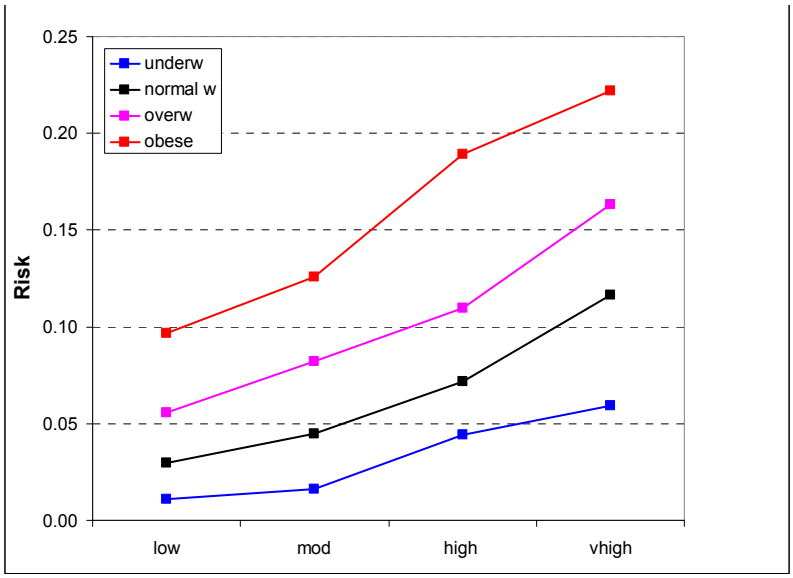

FIGURE 6B Large-for-gestational-age infant (> 90 percent)
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 6B Large-for-gestational-age infant (> 90 percent), adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	1.1%	1.6%	4.4%	5.9%
Normal weight	2.9%	4.5%	7.2%	11.6%
Overweight	5.6%	8.2%	11.0%	16.3%
Obese	9.7%	12.6%	18.9%	22.2%

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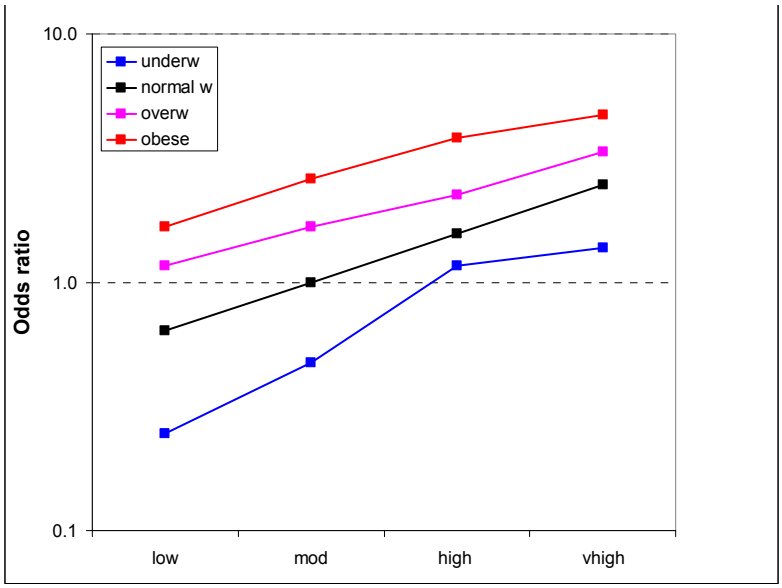

FIGURE 7A Birth weight > 4,000 g
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 7A Birth weight > 4,000 g, adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	0.2	0.5	1.2	1.4
Normal weight	0.6	1.0	1.6	2.5
Overweight	1.2	1.7	2.6	3.4
Obese	1.7	2.6	3.8	4.7

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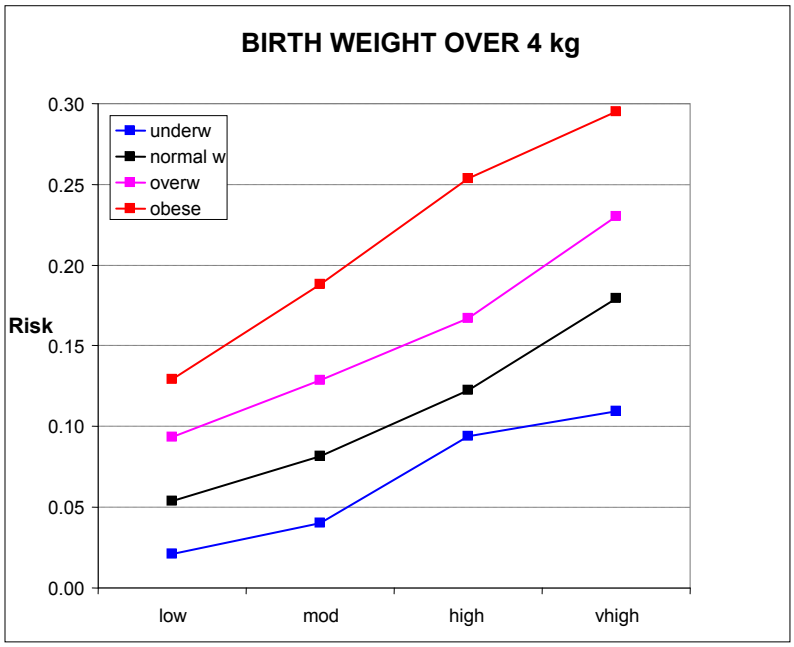

FIGURE 7B Birth weight > 4,000 g
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 7B Adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	2.1%	4.0%	9.4%	10.9%
Normal weight	5.4%	8.2%	12.3%	17.9%
Overweight	9.4%	12.9%	16.7%	23.0%
Obese	12.9%	18.8%	25.4%	29.5%

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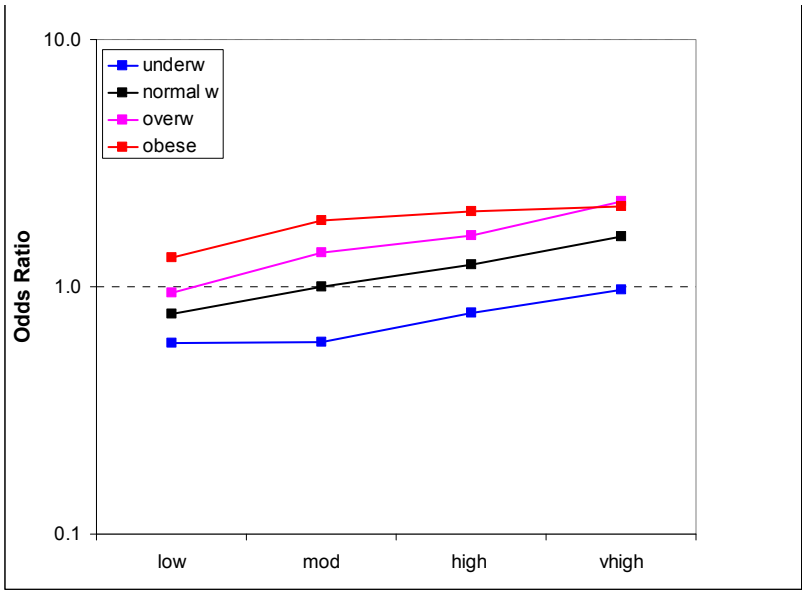

FIGURE 8A High Ponderal Index (> 90 percent)
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 8A High Ponderal Index (> 90 percent), adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	0.6	0.6	0.8	1.0
Normal weight	0.8	1.0	1.2	1.6
Overweight	0.9	1.4	1.6	2.2
Obese	1.3	1.9	2.0	2.1

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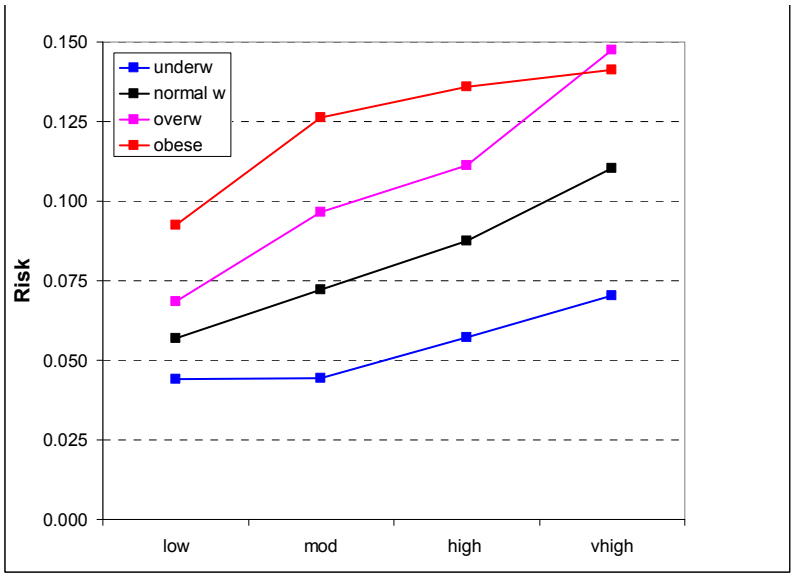

FIGURE 8B High Ponderal Index (> 90 percent)
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 8B High Ponderal Index (> 90 percent), adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	4.4%	4.4%	5.7%	7.0%
Normal weight	5.7%	7.2%	8.8%	11.0%
Overweight	6.8%	9.7%	11.1%	14.7%
Obese	9.2%	12.6%	13.6%	14.1%

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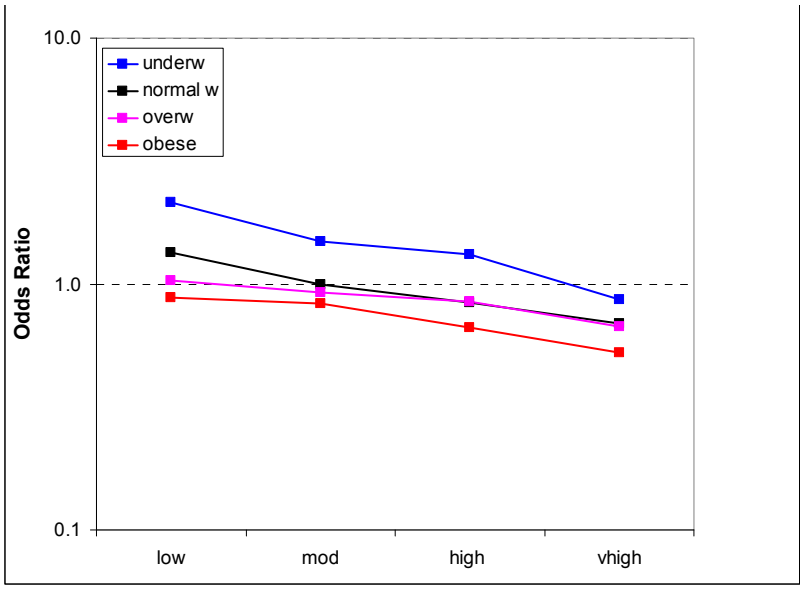

FIGURE 9A Low Ponderal Index (< 10 percent)
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 9A Low Ponderal Index (< 10 percent), adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	2.1	1.5	1.3	0.9
Normal weight	1.3	1.0	0.8	0.7
Overweight	1.0	0.9	0.9	0.7
Obese	0.9	0.8	0.7	0.5

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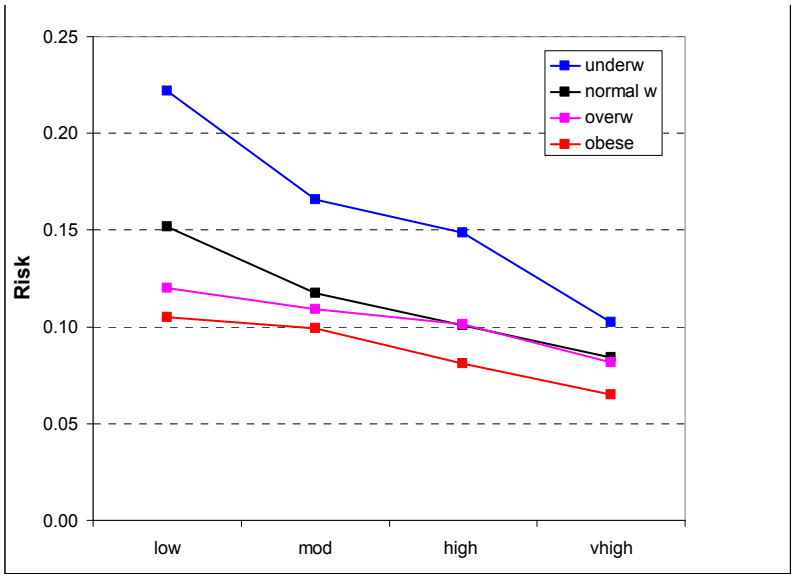

FIGURE 9B Low Ponderal Index (< 10 percent)
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 9B Adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	22.2%	16.6%	14.9%	10.3%
Normal weight	15.2%	11.7%	10.1%	8.4%
Overweight	12.0%	10.9%	10.2%	8.2%
Obese	10.5%	9.9%	8.1%	6.5%

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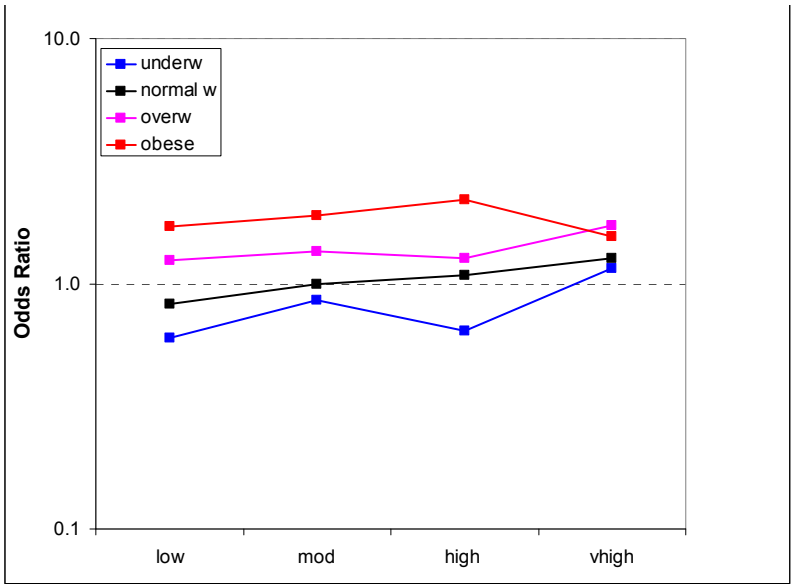

FIGURE 10A Cesarean delivery before labor
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 10A Cesarean delivery before labor, adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	0.6	0.9	0.6	1.2
Normal weight	0.8	1.0	1.1	1.3
Overweight	1.2	1.4	1.3	1.7
Obese	1.7	1.9	2.2	1.6

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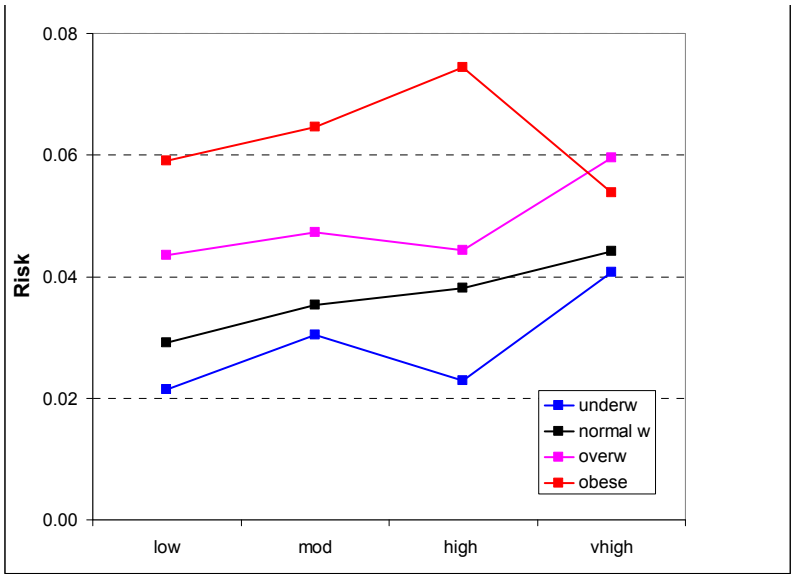

FIGURE 10B Cesarean delivery before labor
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 10B Cesarean delivery before labor, adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	2.2%	3.1%	2.3%	4.1%
Normal weight	2.9%	3.5%	3.8%	4.4%
Overweight	4.4%	4.7%	4.4%	6.0%
Obese	5.9%	6.5%	7.4%	5.4%

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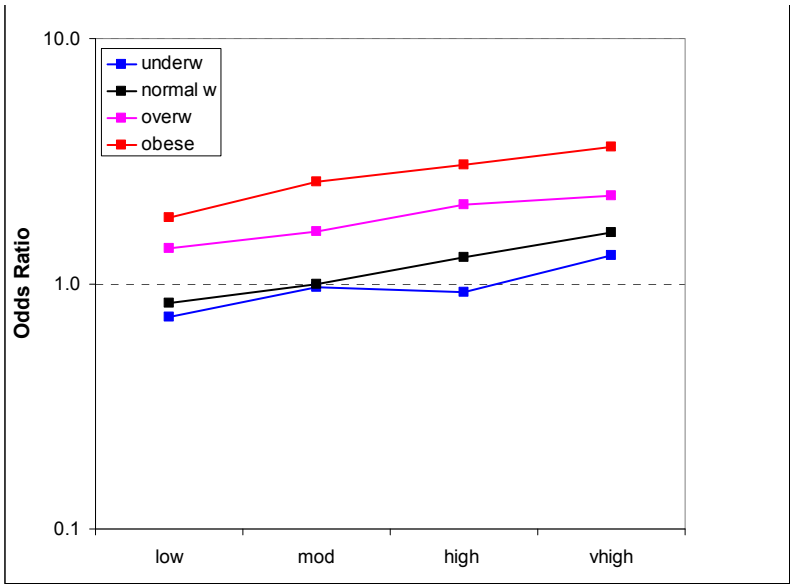

FIGURE 11A Cesarean delivery during labor
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 11A Cesarean delivery during labor, adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	0.7	1.0	0.9	1.3
Normal weight	0.8	1.0	1.3	1.6
Overweight	1.4	1.6	2.1	2.3
Obese	1.9	2.6	3.0	3.6

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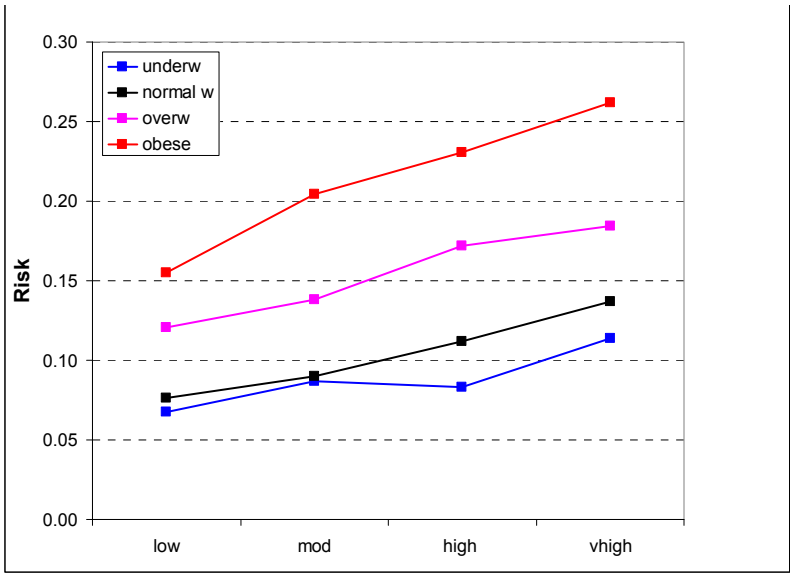

FIGURE 11B Cesarean delivery during labor
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 11B Cesarean delivery during labor, adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	6.7%	8.7%	8.3%	11.4%
Normal weight	7.6%	9.0%	11.2%	13.7%
Overweight	12.1%	13.8%	17.2%	18.4%
Obese	15.5%	20.4%	23.1%	26.2%

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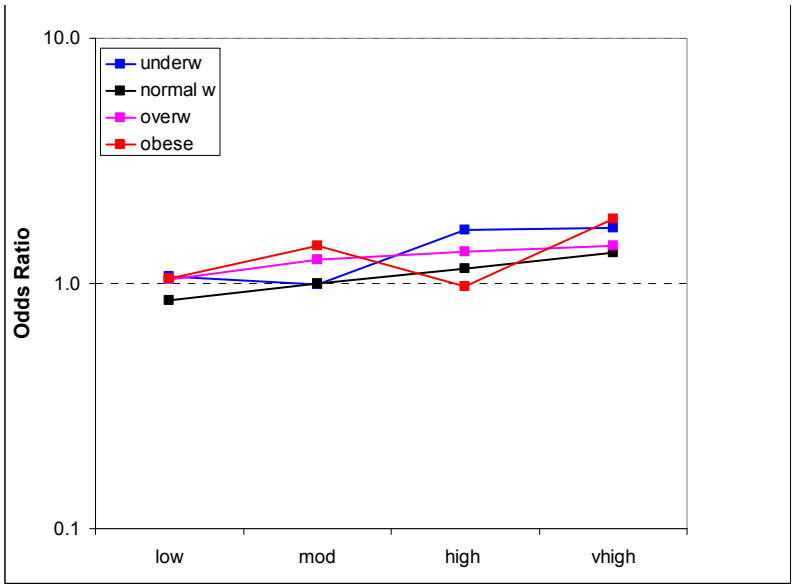

FIGURE 12A Instrumental deliveries
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 12A Instrumental deliveries, adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	1.1	1.0	1.7	1.7
Normal weight	0.9	1.0	1.1	1.3
Overweight	1.0	1.3	1.4	1.4
Obese	1.0	1.4	1.0	1.8

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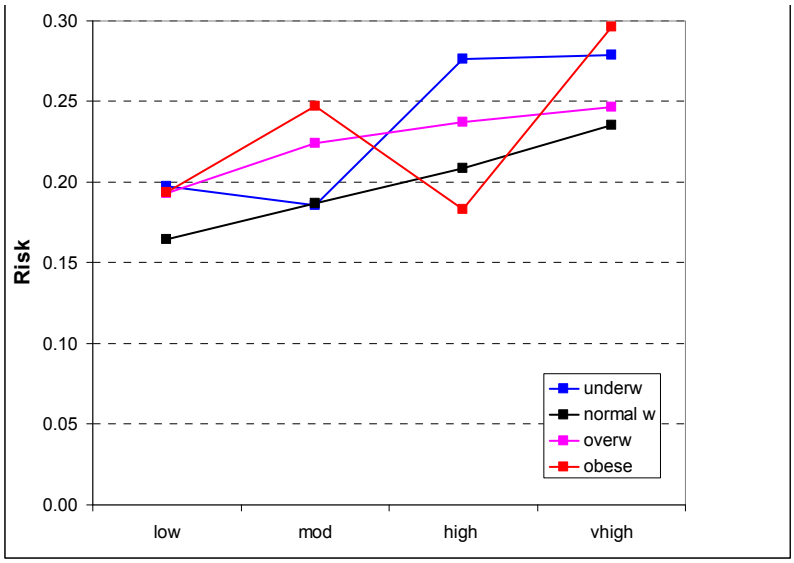

FIGURE 12B Instrumental deliveries
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 12B Instrumental deliveries, adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	19.7%	18.6%	27.6%	27.9%
Normal weight	16.4%	18.7%	20.9%	23.6%
Overweight	19.3%	22.4%	23.8%	24.7%
Obese	19.4%	24.7%	18.3%	29.6%

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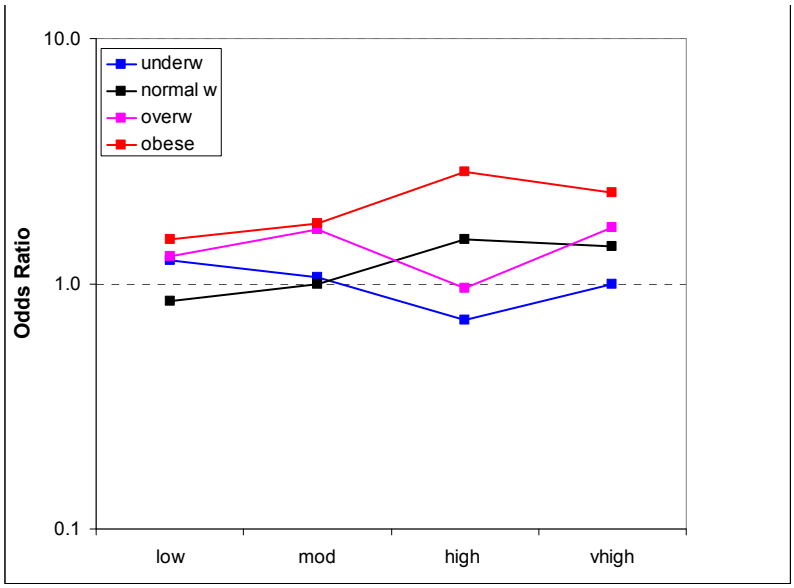

FIGURE 13A Low Apgar score (< 8 after 5 minutes)
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 13A Low Apgar score (< 8 after 5 minutes), adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	1.2	1.1	0.7	1.0
Normal weight	0.8	1.0	1.5	1.4
Overweight	1.3	1.7	1.0	1.7
Obese	1.5	1.8	2.8	2.4

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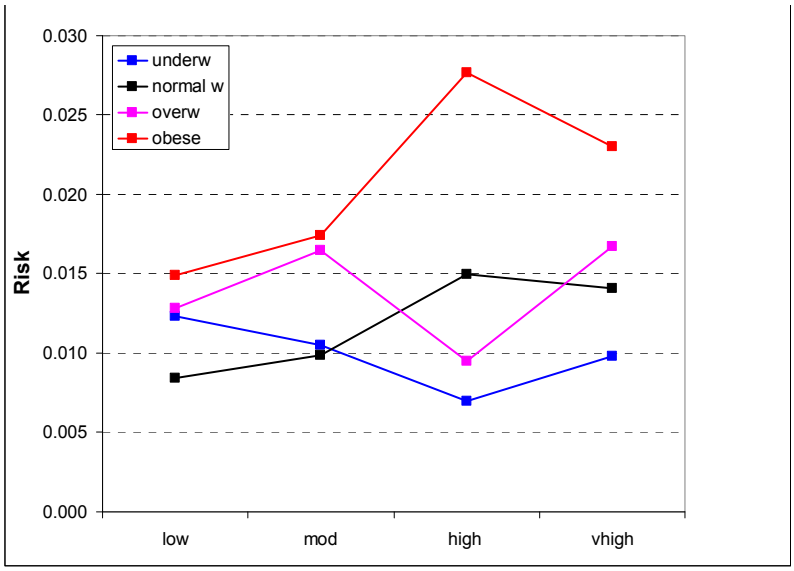

FIGURE 13B Low Apgar score (< 8 after 5 minutes)
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 13B Low Apgar score (< 8 after 5 minutes), adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	1.2%	1.1%	0.7%	1.0%
Normal weight	0.8%	1.0%	1.5%	1.4%
Overweight	1.3%	1.7%	1.0%	1.7%
Obese	1.5%	1.7%	2.8%	2.3%

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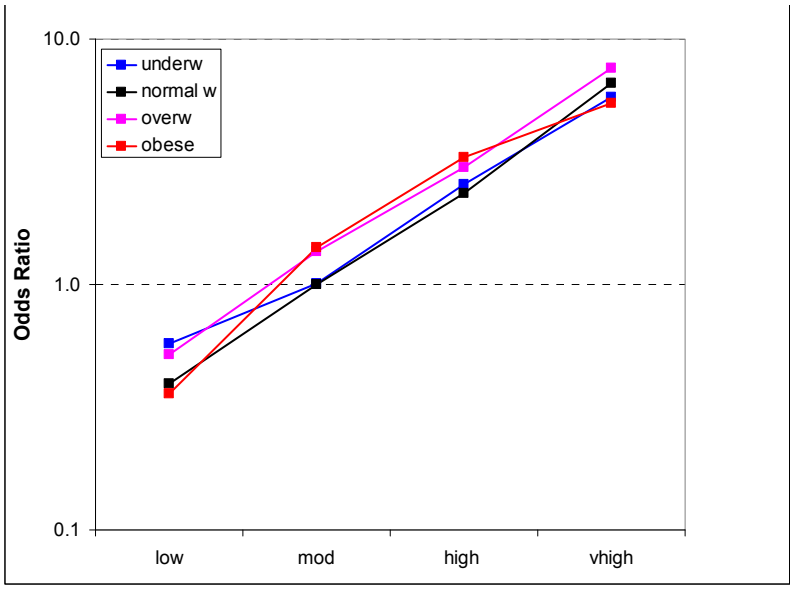

FIGURE 14A Post partum weight retention ≥ 5 kg at 6 months
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 14A Post partum weight retention ≥ 5 kg at 6 months, adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	0.6	1.0	2.6	5.8
Normal weight	0.4	1.0	2.4	6.6
Overweight	0.5	1.4	3.0	7.6
Obese	0.4	1.4	3.3	5.5

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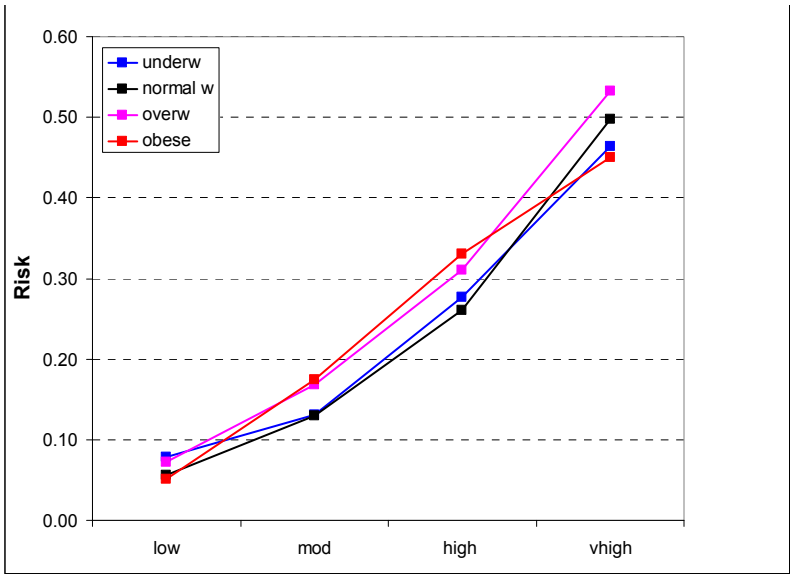

FIGURE 14B Post partum weight retention ≥ 5 kg at 6 months
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exerrcise, 280 days of gestation.

TABLE 14B Post partum weight retention ≥ 5 kg at 6 months, adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	7.9%	13.1%	27.6%	46.5%
Normal weight	5.6%	13.0%	26.1%	49.7%
Overweight	7.2%	16.9%	31.1%	53.2%
Obese	5.1%	17.5%	33.0%	45.0%

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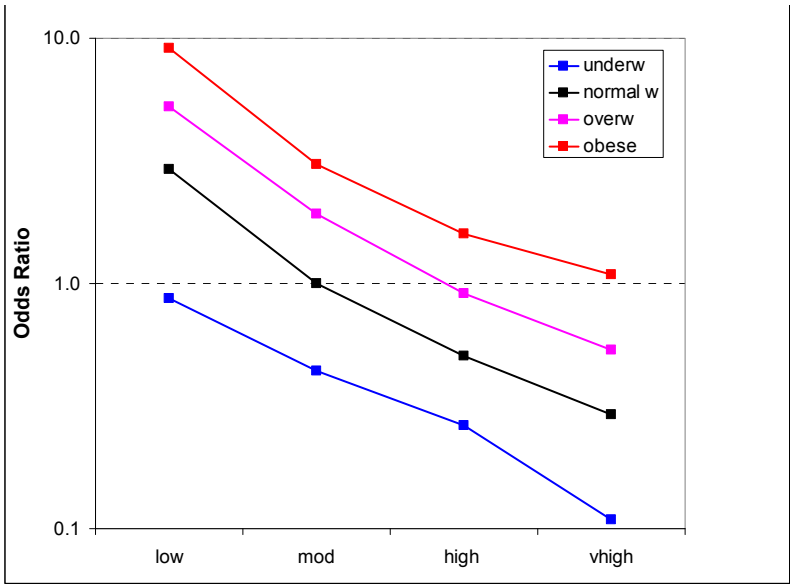

FIGURE 15A Post partum weight loss ≥ 2 kg at 6 months
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 15A Post partum weight loss ≥ 2 kg at 6 months, adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	0.9	0.4	0.3	0.1
Normal weight	2.9	1.0	0.5	0.3
Overweight	5.3	1.9	0.9	0.5
Obese	9.1	3.1	1.6	1.1

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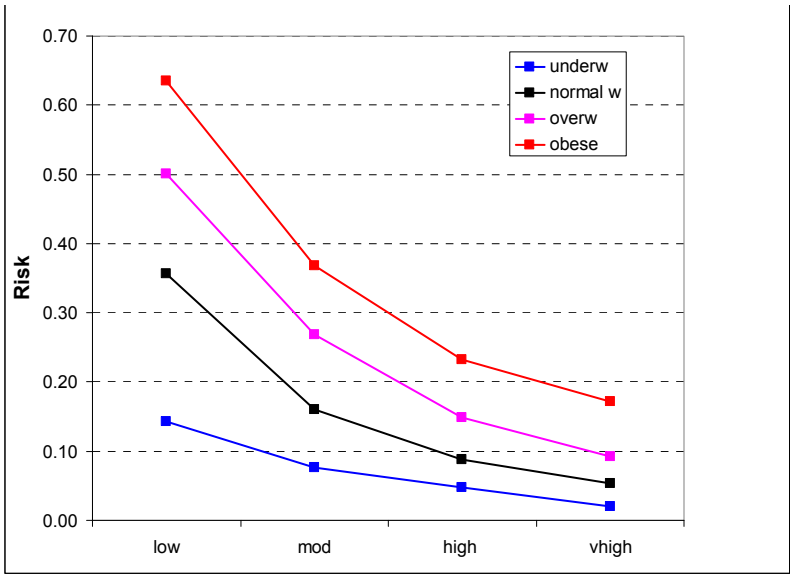

FIGURE 15B Post partum weight loss ≥ 2 kg at 6 months
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 1B Post partum weight loss ≥ 2 kg at 6 months, adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	14.3%	7.7%	4.8%	2.0%
Normal weight	35.7%	16.0%	8.8%	5.3%
Overweight	50.1%	26.9%	14.8%	9.3%
Obese	63.4%	36.8%	23.3%	17.2%

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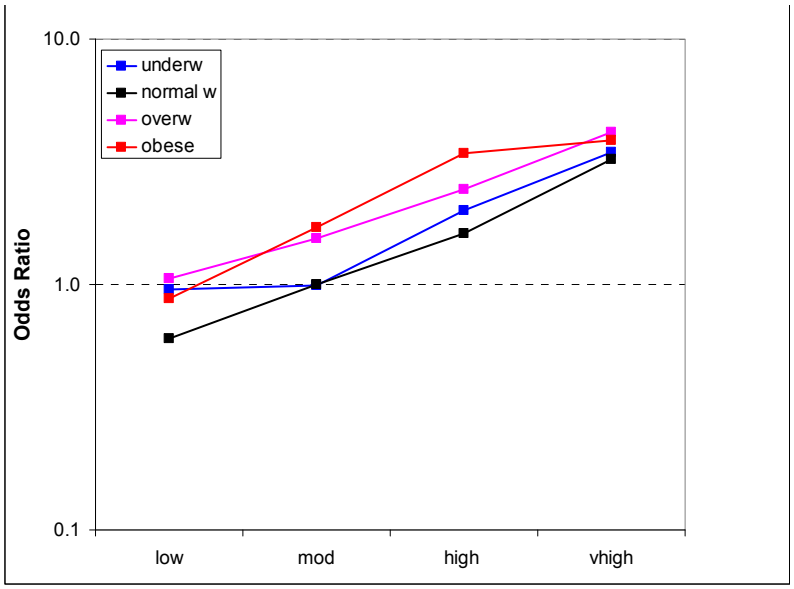

FIGURE 16A Post partum weight retention ≥ 5 kg at 18 months
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 16A Post partum weight retention ≥ 5 kg at 18 months, adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	1.0	1.0	2.0	3.5
Normal weight	0.6	1.0	1.6	3.2
Overweight	1.1	1.5	2.4	4.2
Obese	0.9	1.7	3.4	3.9

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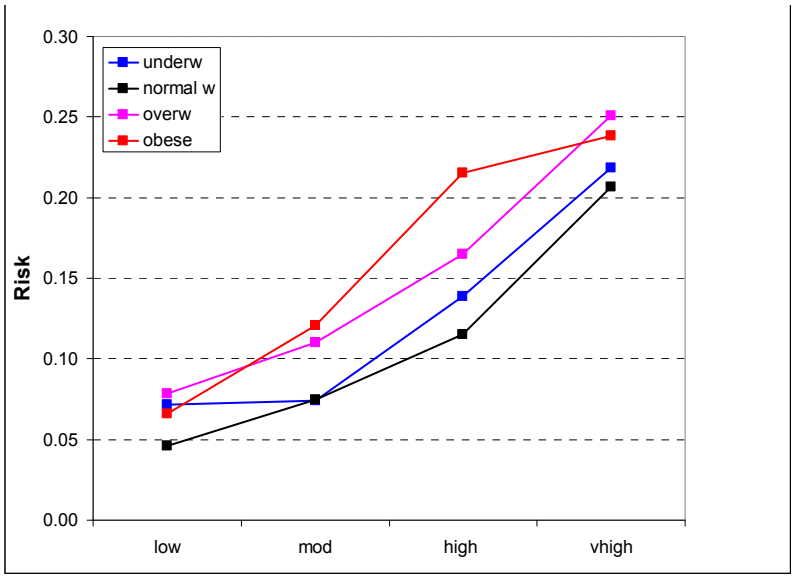

FIGURE 16B Post partum weight retention ≥ 5 kg at 18 months
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 16B Post partum weight retention ≥ 5 kg at 18 months, adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	7.1%	7.4%	13.9%	21.8%
Normal weight	4.6%	7.5%	11.5%	20.7%
Overweight	7.8%	11.0%	16.5%	25.1%
Obese	6.6%	12.1%	21.6%	23.8%

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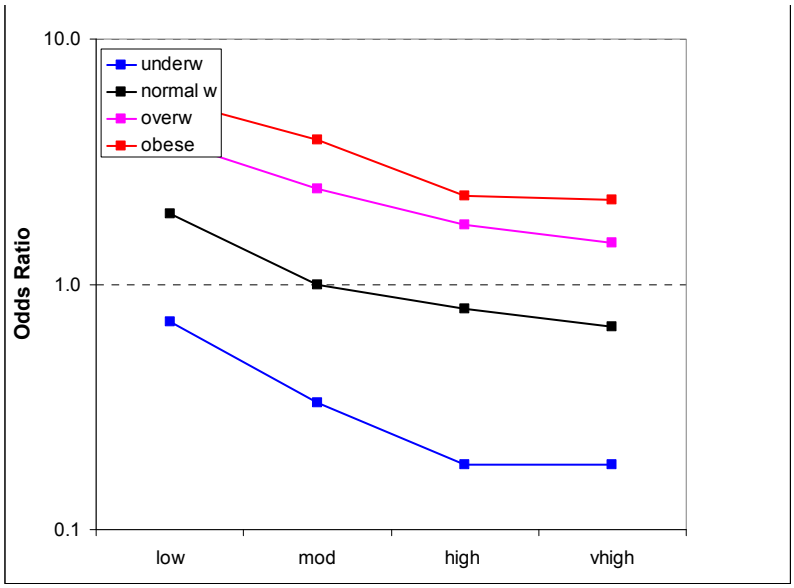

FIGURE 17A Post partum weight loss ≥ 2 kg at 18 months
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE 17A Post partum weight loss ≥ 2 kg at 18 months, adjusted odds ratios (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	0.7	0.3	0.2	0.2
Normal weight	1.9	1.0	0.8	0.7
Overweight	3.8	2.4	1.7	1.5
Obese	5.6	3.9	2.3	2.2

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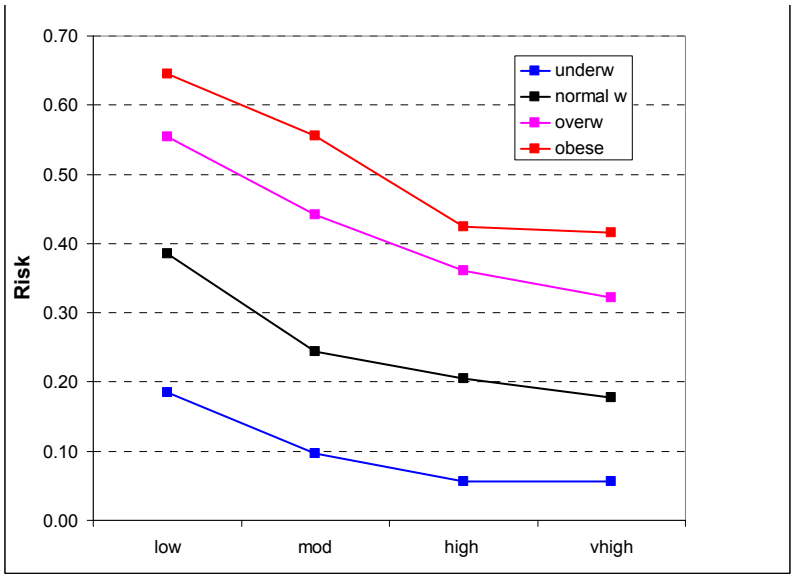

FIGURE 17B Post partum weight loss ≥ 2 kg at 18 months
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exerrcise, 280 days of gestation.

TABLE 17B Post partum weight loss ≥ 2 kg at 18 months, adjusted risks (gestational weight gain by BMI)

	Low	Moderate	High	Very High
Underweight	18.5%	9.6%	5.6%	5.6%
Normal weight	38.6%	24.4%	20.5%	17.8%
Overweight	55.4%	44.1%	36.1%	32.3%
Obese	64.5%	55.5%	42.5%	41.6%

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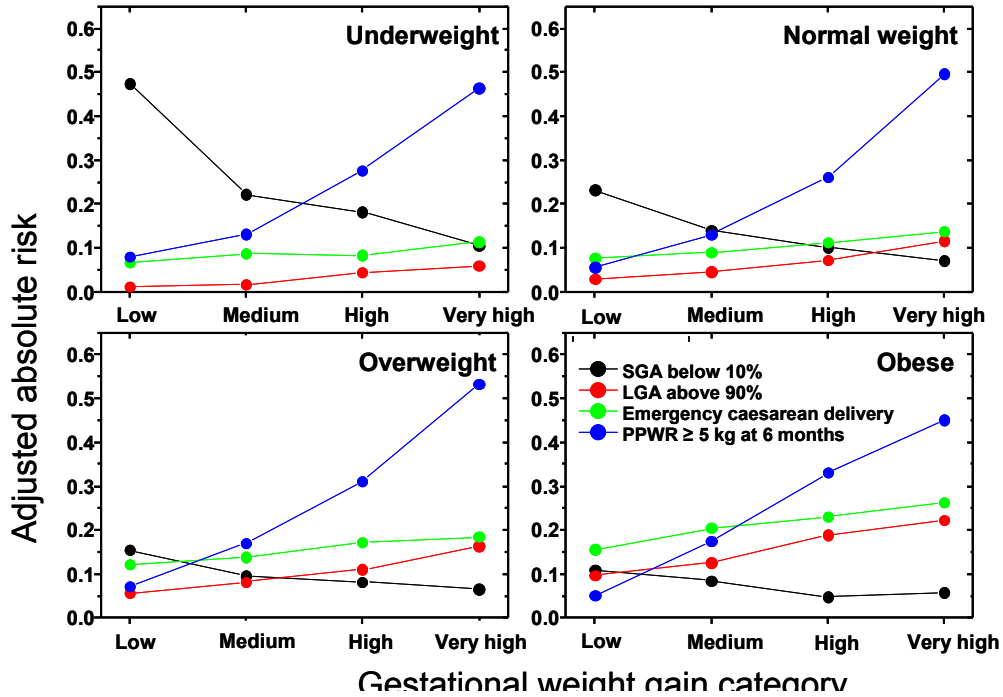

FIGURE 18 GWG-specific absolute risks for SGA, LGA, emergency cesarean delivery and postpartum weight retention within each group

NOTE: Points present risks of a primiparous woman, aged 25-29, height 160-69, nonsmoker, no alcohol consumption, high social status, no exercise and 280 days of gestation. For PPWR, she breastfed < 14 weeks.

TABLE 18A GWG-specific absolute risks for SGA, LGA, emergency cesarean delivery and postpartum weight retention for underweight women

	< 10 kg	10-15 kg	16-19 kg	≥ 20 kg
SGA	47.3%	22.1%	18.2%	10.5%
LGA	1.1%	1.6%	4.4%	5.9%
Emergency CD	6.7%	8.7%	8.3%	11.4%
PPWR	7.9%	13.1%	27.6%	46.5%

NOTE: SGA = small-for-gestational age; LGA = large-for-gestational age; CD = cesarean delivery; PPWR = postpartum weight retention

TABLE 18B GWG-specific absolute risks for SGA, LGA, emergency cesarean delivery and postpartum weight retention for normal weight women

	< 10 kg	10-15 kg	16-19 kg	≥ 20 kg
SGA	23.3%	14.0%	10.1%	7.1%
LGA	2.9%	4.5%	7.2%	11.6%
Emergency CD	7.6%	9.0%	11.2%	13.7%
PPWR	5.6%	13.0%	26.1%	49.7%

NOTE: SGA = small-for-gestational age; LGA = large-for-gestational age; CD = cesarean delivery; PPWR = postpartum weight retention

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TABLE 18C GWG-specific absolute risks for SGA, LGA, emergency cesarean delivery and postpartum weight retention for overweight women

	< 10 kg	**10-15 kg**	**16-19 kg**	**≥ 20 kg**
SGA	15.4%	9.5%	8.2%	6.5%
LGA	5.6%	8.2%	11.0%	16.3%
Emergency CD	12.1%	13.8%	17.2%	18.4%
PPWR	7.2%	16.9%	31.1%	53.2%

NOTE: SGA = small-for-gestational age; LGA = large-for-gestational age; CD = cesarean delivery; PPWR = postpartum weight retention

TABLE 18D GWG-specific absolute risks for SGA, LGA, emergency cesarean delivery and postpartum weight retention for obese women

	< 10 kg	**10-15 kg**	**16-19 kg**	**≥ 20 kg**
SGA	10.9%	8.4%	4.8%	5.8%
LGA	9.7%	12.6%	18.9%	22.2%
Emergency CD	15.5%	20.4%	23.1%	26.2%
PPWR	5.1%	17.5%	33.0%	45.0%

NOTE: SGA = small-for-gestational age; LGA = large-for-gestational age; CD = cesarean delivery; PPWR = postpartum weight retention

Second DNBC Report

At the IOM workshop in Washington June 2008, the IOM committee found the additive approach with presentation of absolute adjusted risks across BMI groups useful and informative. They asked for additional analyses of some of the most important outcomes where one more obese class and 2 more GWG groups were included.

Methods

The study population and the methods for deriving the adjusted odds ratios and absolute risks were the same as for the First DNBC Report.

The BMI categories were expanded by dividing the obese group into obese class I (30 ≤ BMI > 35) and obese class 2+3 (BMI ≥ 35) (WHO, 2000). These categories are denoted obese and extremely obese in the figures. The GWG categories were expanded with 2 groups and included now the 6 following categories: 2 low categories (<5 kg, and 5-9 kg), 1 medium category (10-15 kg) and 3 high categories (16-19 kg, 20-24 kg and ≥ 25 kg). The analyses were carried out for the following 4 outcomes:

- Small-for-gestational-age infant (<10th percentile)
- Large-for-gestational-age infant (> 90th percentile)
- Emergency cesarean delivery
- Postpartum weight retention of ≥ 5 kg at 6 months

Finally, the results were stratified on BMI group and combined in one figure to evaluate the 'trade-off' between mother and infant:

- GWG-specific absolute risks for SGA, LGA, emergency cesarean delivery and postpartum weight retention within each BMI group.

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Results

- In all BMI groups, risk of SGA responded to increasing GWG throughout the entire spectrum of gain categories. The same was seen for LGA except for extremely obese women with GWG ≥ 25 kg, which did not increase risk of LGA further.
- Only for underweight, normal weight and overweight women was GWG <5 kg associated with substantial risk of SGA.
- Extremely obese women had risks similar to obese women except for emergency cesarean delivery. Here, data indicated high and increasing risk with increasing GWG.
- The data did not suggest deleterious consequences of GWG < 5 kg in obese and extremely obese women.

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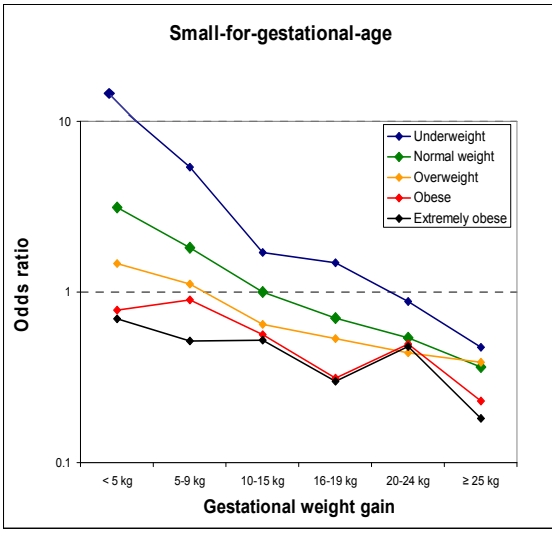

FIGURE 19A Small-for-gestational age infant (< 10 percentile)
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, exercise, social status, gestational age in days (p = 0.0001 [Wald's test]).

TABLE 19A Small-for-gestational age, adjusted odds ratios (by BMI and gestational weight gain)

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
< 18.5	12.5	5.4	1.7	1.5	0.9	0.5
	(3.9; 39.8)	(3.9; 7.5)	(1.4; 2.0)	(1.1; 1.9)	(0.6; 1.3)	(0.2; 0.9)
18.5-24.9	3.1	1.8	1.0	0.7	0.5	0.4
	(2.2; 4.5)	(1.6; 2.0)	(ref)	(0.6; 0.8)	(0.5; 0.6)	(0.3; 0.4)
25.0-29.9	1.5	1.1	0.6	0.5	0.4	0.4
	(1.0; 2.0)	(0.9; 1.3)	(0.6; 0.7)	(0.4; 0.7)	(0.3; 0.6)	(0.3; 0.5)
30-34.9	0.8	0.9	0.3	0.3	0.5	0.2
	(0.5; 1.1)	(0.7; 1.2)	(0.4; 0.7)	(0.2; 0.6)	(0.3; 0.9)	(0.1; 0.5)
35+	0.7	0.5	0.5	0.3	0.5	0.2
	(0.4; 1.1)	(0.3; 0.9)	(0.3; 0.8)	(0.1; 1.3)	(0.1; 1.7)	(0.0; 1.3)

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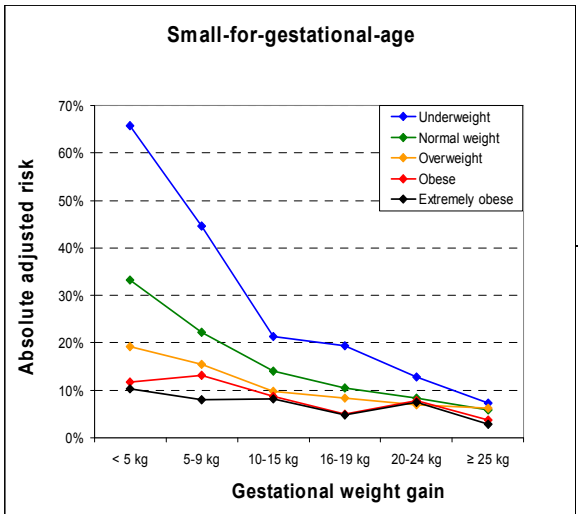

FIGURE 19B Small-for-gestational age infant (< 10 percentile)
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 19B Small-for-gestational age, adjusted risks (by BMI and gestational weight gain)

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
< 18.5	66%	45%	21%	19%	13%	7%
	(38-86)	(37-53)	(18-24)	(16-24)	(9-18)	(4-13)
18.5-24.9	33%	22%	14%	11%	8%	6%
	(26-42)	(20-25)	(13-15)	(9-12)	(7-10)	(5-7)
25.0-29.9	19%	15%	10%	8%	7%	6%
	(15-25)	(13-18)	(9-11)	(7-10)	(5-9)	(5-8)
30-34.9	12%	13%	9%	5%	8%	4%
	(8-16)	(10-17)	(7-11)	(3-9)	(5-13)	(2-8)
35+	10%	8%	8%	5%	7%	3%
	(7-15)	(5-13)	(5-13)	(2-18)	(2-22)	(0-18)

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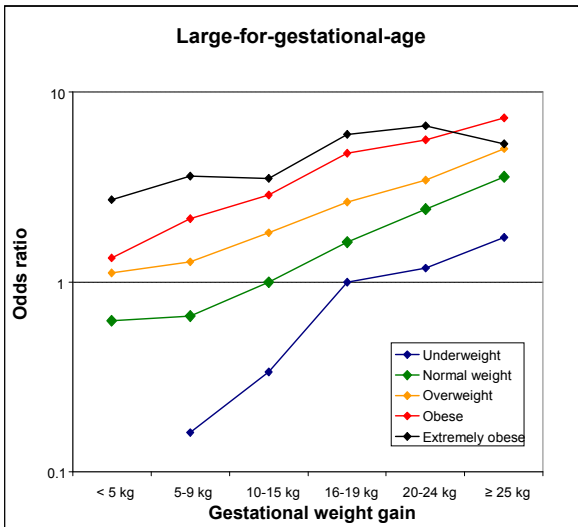

FIGURE 20A Large-for-gestational age infant (> 90 percentile)
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, exercise, social status, gestational age in days (p = 0.0001 [Wald's test]).

TABLE 20A Large-for-gestational age, adjusted odds ratios (by BMI and gestational weight gain)

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
< 18.5	---	0.2	0.3	1.0	1.2	1.7
		(0.1; 0.5)	(0.2; 0.5)	(0.7; 1.4)	(0.8; 1.8)	(1.0; 3.0)
18.5-24.9	0.6	0.7	1.0	1.6	2.4	3.6
	(0.3; 1.2)	(0.6; 0.8)	(ref)	(1.5; 1.8)	(2.2; 2.7)	(3.2; 4.0)
25.0-29.9	1.1	1.3	1.8	2.6	3.4	5.0
	(0.8; 1.6)	(1.1; 1.5)	(1.6; 2.0)	(2.3; 3.0)	(3.0; 4.0)	(4.2; 6.0)
30-34.9	1.3	2.2	2.9	4.8	5.6	7.3
	(0.9; 1.9)	(1.7; 2.7)	(2.4; 3.4)	(3.7; 6.2)	(4.1; 7.6)	(5.0; 10.5)
35+	2.7	3.6	3.5	6.0	6.6	5.3
	(1.9; 3.8)	(2.7; 4.9)	(2.6; 4.7)	(3.3; 10.9)	(3.2; 13.8)	(2.5; 11.5)

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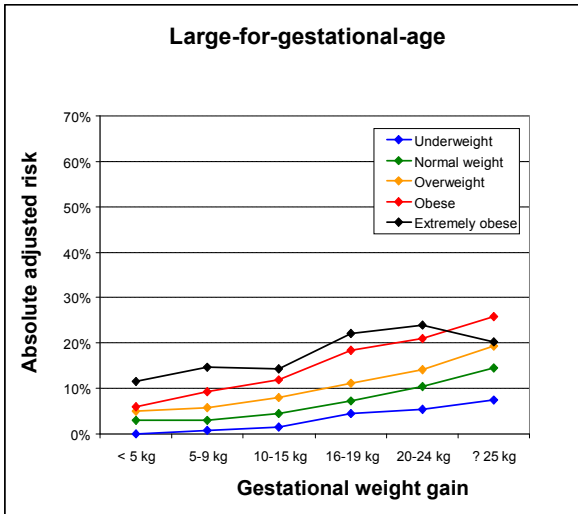

FIGURE 20B Large-for-gestational age infant (> 90 percentile)
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 20B Large-for-gestational age, adjusted risks (by BMI and gestational weight gain)

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
< 18.5	---	1%	2%	5%	5%	8%
		(0-2)	(1-2)	(3-6)	(4-8)	(4-12)
18.5-24.9	3%	3%	5%	7%	10%	15%
	(1-6)	(3-4)	(4-5)	(7-8)	(9-11)	(13-16)
25.0-29.9	5%	6%	8%	11%	14%	19%
	(4-7)	(5-7)	(7-9)	(10-13)	(12-16)	(17-22)
30-34.9	6%	9%	12%	18%	21%	26%
	(4-8)	(8-11)	(10-14)	(15-23)	(16-27)	(19-33)
35+	11%	15%	14%	22%	24%	20%
	(8-15)	(11-19)	(11-18)	(14-34)	(13-40)	(10-35)

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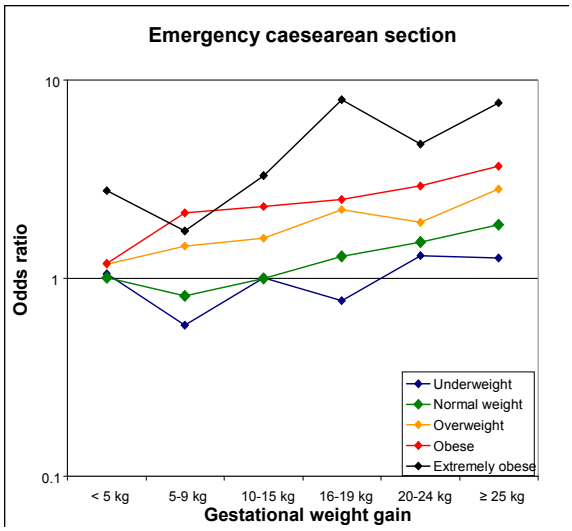

FIGURE 21A Emergency cesarean deliveries
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, exercise, social status, gestational age in days (p = 0.23 [Wald's test]).

TABLE 21A Emergency cesarean deliveries, adjusted odds ratios (by BMI and gestational weight gain)

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
< 18.5	1.1	0.6	1.0	0.8	1.3	1.3
	(0.2; 6.7)	(0.2; 1.4)	(0.8; 1.3)	(0.5; 1.2)	(0.8; 2.1)	(0.6; 2.5)
18.5-24.9	1.0	0.8	1.0	1.3	1.5	1.9
	(0.5; 2.1)	(0.7; 1.0)	(ref)	(1.2; 1.4)	(1.3; 1.7)	(1.6; 2.2)
25.0-29.9	1.2	1.5	1.6	2.2	1.9	2.8
	(0.7; 1.9)	(1.2; 1.8)	(1.4; 1.8)	(1.9; 2.6)	(1.6; 2.3)	(2.2; 3.5)
30-34.9	1.2	2.1	2.3	2.5	2.9	3.7
	(0.8; 1.8)	(1.6; 2.8)	(1.9; 2.8)	(1.7; 3.6)	(2.0; 4.3)	(2.4; 5.7)
35+	2.8	1.7	3.3	7.9	4.7	7.7
	(1.9; 4.1)	(1.1; 2.7)	(2.3; 4.6)	(4.1; 15.4)	(2.0; 11.2)	(3.4; 17.5)

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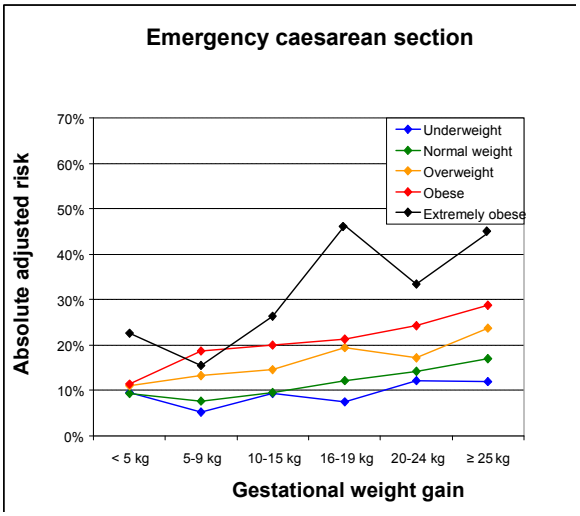

FIGURE 21B Emergency cesarean deliveries
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 21B Emergency cesarean deliveries, adjusted risks (by BMI and G gestational weight gain)

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
< 18.5	9%	5%	9%	7%	12%	12%
	(2-40)	(2-12)	(7-12)	(5-11)	(8-18)	(6-21)
18.5-24.9	9%	8%	9%	12%	14%	17%
	(5-17)	(6-9)	(9-10)	(11-13)	(13-16)	(15-19)
25.0-29.9	11%	13%	15%	19%	17%	24%
	(7-17)	(11-16)	(13-16)	(17-22)	(15-21)	(20-28)
30-34.9	11%	19%	20%	21%	24%	29%
	(8-17)	(15-23)	(17-24)	(16-28)	(18-32)	(21-38)
35+	23%	16%	26%	46%	33%	45%
	(16-30)	(10-23)	(20-34)	(31-63)	(17-54	(26-65)

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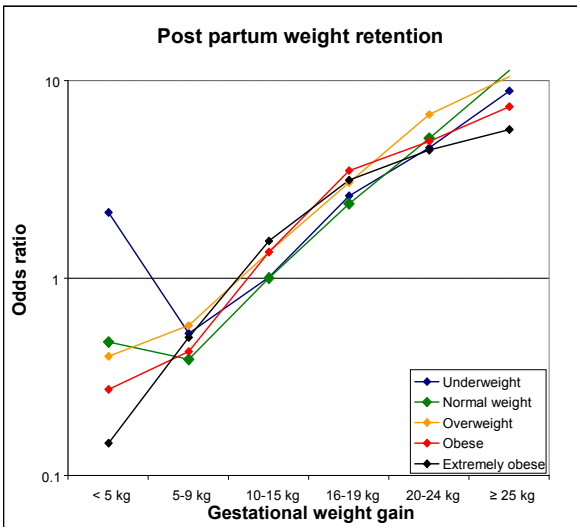

FIGURE 22A Postpartum weight retention ≥ 5 kg at 6 months
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, exercise, social status, gestational age in days (p = 0.001 [Wald's test]).

TABLE 22A Postpartum weight retention ≥ 5 kg at 6 months, adjusted odds ratios (by BMI and gestational weight gain)

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
< 18.5	2.1	0.5	1.0	2.6	4.6	8.9
	(0.7; 6.6)	(0.3; 1.0)	(0.8; 1.2)	(2.1; 3.2)	(3.6; 5.8)	(6.4; 12.4)
18.5-24.9	0.5	0.4	1.0	2.4	5.1	11.3
	(0.3; 0.9)	(0.3; 0.5)	(ref)	(2.2; 2.6)	(4.7; 5.5)	(10.3; 12.4)
25.0-29.9	0.4	0.6	1.4	3.0	6.7	10.4
	(0.3; 0.6)	(0.5; 0.7)	(1.2; 1.5)	(2.7; 3.4)	(6.0; 7.6)	(9.0; 12.1)
30-34.9	0.3	0.4	1.4	3.5	4.9	7.4
	(0.2; 0.5)	(0.3; 0.6)	(1.1; 1.6)	(2.7; 4.5)	(3.8; 6.5)	(5.4; 10.1)
35+	0.1	0.5	1.5	3.1	4.5	5.6
	(0.1; 0.3)	(0.3; 0.8)	(1.1; 2.1)	(1.8; 5.5)	(2.4; 8.2)	(2.9; 10.9)

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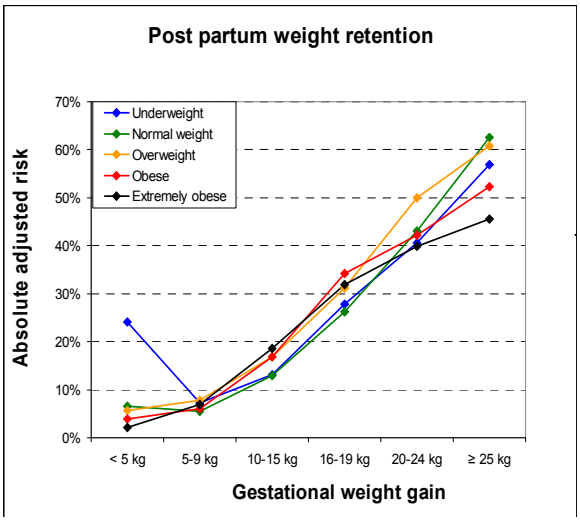

FIGURE 22B Postpartum weight retention ≥ 5 kg at 6 months
NOTE: Absolute risks dervied from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE 22B Postpartum weight retention ≥ 5 kg at 6 months, adjusted risks (by BMI and gestational weight gain)

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
< 18.5	24%	7%	13%	28%	41%	57%
	(9-50)	(4-12)	(11-15)	(24-32)	(35-47)	(48-65)
18.5-24.9	7%	5%	13%	26%	43%	63%
	(4-12)	(5-7)	(12-14)	(25-28)	(41-45)	(60-65)
25.0-29.9	6%	8%	17%	31%	50%	61%
	(4-9)	(6-10)	(15-18)	(28-34)	(47-53)	(57-65)
30-34.9	4%	6%	17%	34%	42%	52%
	(2-6)	(4-8)	(15-19)	(29-40)	(36-49)	(44-60)
35+	2%	7%	19%	32%	40%	46%
	(0-5)	(4-11)	(15-24)	(21-45)	(26-55)	(30-62)

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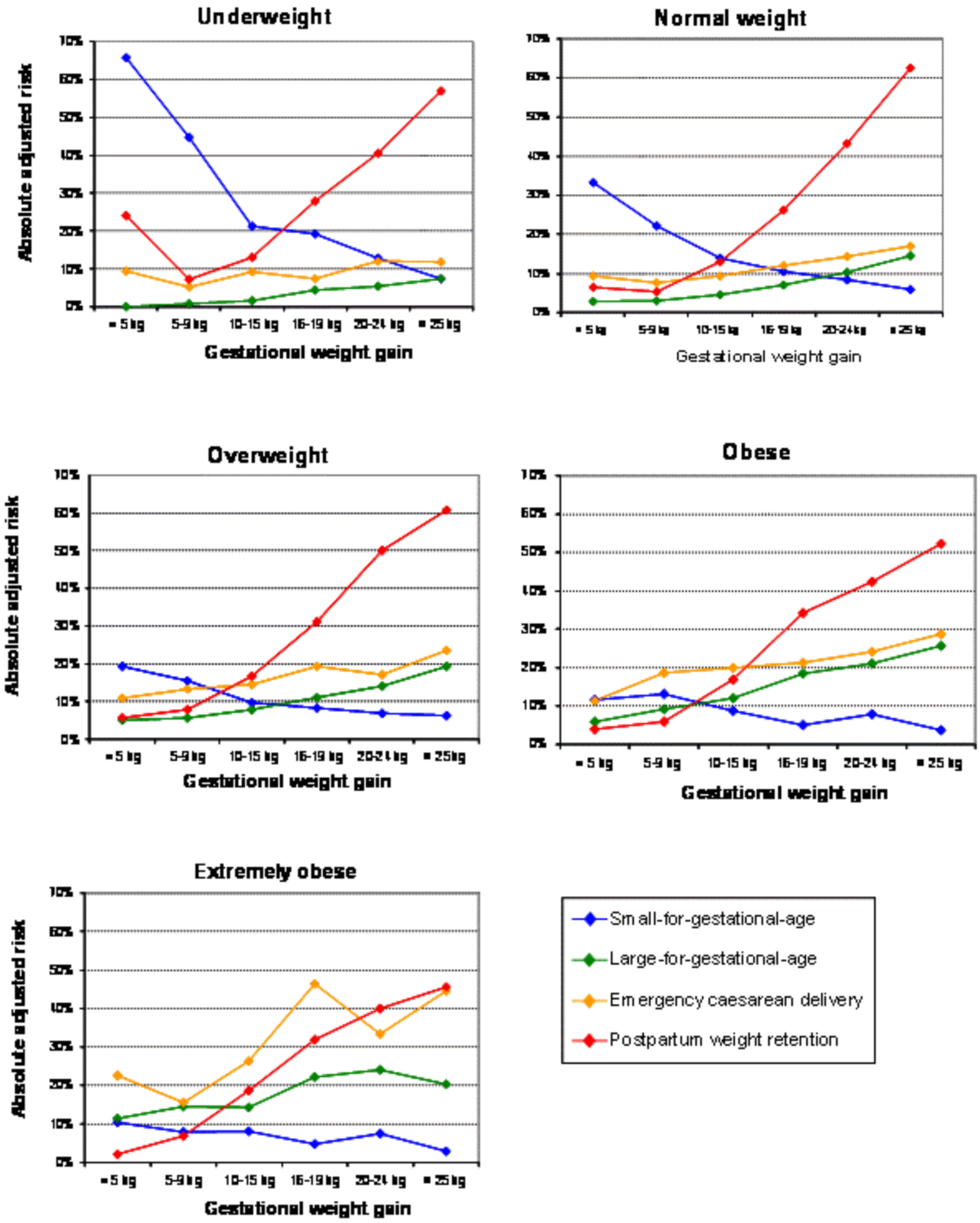

FIGURE 23 GWG-specific absolute risks for SGA, LGA, emergency cesarean delivery and postpartum weight retention within each BMI group.
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, non smoker, no alcohol consumption, high social status, no exercise, 280 days of gestation. For PPWR, she is breastfeeding less than 14 weeks.

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Third DNBC Report

Because the 1990 IOM Guidelines did not provide sufficient data about GWG in subpopulations of interest, the committee requested additional information about subgroups of pregnant women, defined by parity, height, age, and smoking. Also, the committee asked for analyses of the association between GWG and emergency cesarean delivery and postpartum weight retention with and without adjustment for birth weight. These results are presented in the Third DNBC Report. The methods and analyses are presented below. More details are available in 'Nohr et al', in preparation.

Study population

The initial study population was similar to the one used in the First and Second DNBC Report. However, in this study, women < 18 y old (n = 71) were included and women with diagnosed preeclampsia (n = 1,118) and gestational diabetes (n = 690) were excluded. As a result, there were 59,147 women in the final study population.

Independent variables

The main exposures, self-reported prepregnancy BMI and GWG, were defined in the same way as in the previous reports. For this report, BMI was categorized into 4 categories: underweight (BMI < 18.5 kg/m^2), normal weight (18.5 ≤ BMI < 25 kg/m^2), overweight (25 ≤ BMI < 30 kg/m^2), and obese (BMI ≥ 30 kg/m^2) (21). Gestational weight gain was divided into 6 categories: 2 low categories (<5 kg, and 5-9 kg), 1 medium category (10-15 kg) and 3 high categories (16-19 kg, 20-24 kg and ≥ 25 kg). Other covariates in the models were similar to those used in previous analyses.

Pregnancy outcomes

Pregnancy outcomes used in these analyses were:

- Small-for-gestational-age infant (<10th percentile)
- Large-for-gestational-age infant (> 90th percentile)
- Emergency cesarean delivery
- Postpartum weight retention of ≥ 5 kg at 6 months

Information about the definition of these outcomes can be found in the First DNBC report.

Statistical methods

A BMI- and GWG-specific variable was generated by cross-classifying BMI group (4 categories) and GWG group (6 categories). Few underweight women reported a low GWG, so for this group the 2 lowest categories were combined into 1 category, which was defined as a gain < 10 kg. Thus, this BMI- and GWG-specific variable consisted of 23 categories.

We divided the study population into primiparous (n = 27,030) and multiparous (n = 32,117) women to investigate the associations among BMI, GWG and selected pregnancy outcomes within each of these strata. In multiple logistic regression models, the BMI- and GWG-specific

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variable and the covariates of age, height and smoking (yes/no) were mutually adjusted to estimate their independent associations with the pregnancy outcomes of interest within each of these subpopulations. The reference category was defined as normal weight women with a medium GWG (10-15 kg), 25-29 years old at conception, height of 1.60-1.69 m who did not smoke during pregnancy. In these models, we also adjusted for alcohol consumption and exercise in pregnancy, social status, and gestational age at delivery in days. In the analysis of postpartum weight retention, duration of breastfeeding was added to the model. In the analysis of emergency cesarean delivery, we excluded women who had a cesarean section before labor (1,485 primiparous and 2,429 multiparous women).

Within the groups of primiparous and multiparous women, we used the calculated odds ratios from these models to compute absolute adjusted risks for pregnancy outcomes according to each category within the BMI-and GWG-specific variable (which produced 23 different absolute risks for each pregnancy outcome). This was done for 4 different sets of characteristics among primiparous women and 3 different sets among multiparous women, which created a total of 7 different types of women. In each of these models, "a reference woman" was 25-29 years old, 1.60-1.69 m tall and did not smoke or consume alcohol during pregnancy. This woman, which will be denoted "an unexposed woman" in the following, performed a moderate amount of exercise during pregnancy, was of high social status and had a gestational length of 280 days. For postpartum weight retention, she breastfed <14 weeks. The same characteristics applied for 'a short woman', only she was < 1.60 m tall. 'A smoking woman' was also defined as a reference woman, only she was a smoker. Among primiparous women, we also defined 'a young woman', who was similar to the reference woman, only was she < 20 years old.

Results

First, the absolute risks are presented in 7 figures, one for each subtype of woman, to evaluate if the 'trade-off' between mother and infant differed across different types of women. Every figure is accompanied with a table with estimates and 95% confidence intervals corresponding to all points in the figure:

- Unexposed primiparae: GWG-specific risks of pregnancy outcomes
- Short primiparae: GWG-specific risks of pregnancy outcomes
- Smoking primiparae: GWG-specific risks of pregnancy outcomes
- Young primiparae: GWG-specific risks of pregnancy outcomes
- Unexposed multiparae: GWG-specific risks of pregnancy outcomes
- Short multiparae: GWG-specific risks of pregnancy outcomes
- Smoking multiparae: GWG-specific risks of pregnancy outcomes

To evaluate the differences between subtypes of women within each BMI group, these results were also combined in 4 new figures, one for each BMI group.

- GWG-specific risk of pregnancy outcomes in subtypes of underweight women
- GWG-specific risk of pregnancy outcomes in subtypes of normal weight women
- GWG-specific risk of pregnancy outcomes in subtypes of overweight women
- GWG-specific risk of pregnancy outcomes in subtypes of obese women

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For emergency cesarean delivery and postpartum weight retention, the above analyses were repeated with adjustment for birth weight. When adjusted for birth weight, the presented absolute risk was that of a woman giving birth to a 3,500-3,999 g baby. These results are presented in:

- Underweight women: Risks before and after adjustment for birth weight
- Normal weight women: Risks before and after adjustment for birth weight
- Overweight women: Risks before and after adjustment for birth weight
- Obese women: Risks before and after adjustment for birth weight.

In summary, the findings showed that, in addition to prepregnancy BMI, other characteristics were associated with a woman's risk of important pregnancy outcomes.

Parity

- The mean GWG in primiparae was higher than in multiparae (15.7 kg vs. 14.6 kg), which may be needed to eliminate excess risk of giving birth to a SGA baby. Thus, risk of SGA was 46 percent and 22 percent in underweight and normal weight primiparae with GWG < 10 kg.
- In contrast, the average risk of SGA was much lower among multiparous women. Among underweight and normal weight multiparae, an absolute risk at or below 10 percent was reached at 2-3 GWG categories lower than among primiparae.
- Risk of postpartum weight retention increased steeply with increasing gain irrespective of parity.
- Although LGA was responsive to increasing GWG, a considerable excess risk of LGA was only present in obese primiparae and multiparous women.
- These findings suggest that a multiparous woman may reach an overall favorable pregnancy outcome at a lower GWG than needed for a primiparous woman, and that recommendations for GWG could be lower in multiparous than in primiparous women.

Height

- These data could not confirm the idea included in the IOM (1990) guidelines that short (< 157 cm) women should gain at the lower end of the recommended range. Only risk of emergency cesarean deliveries was uniquely high in short primiparae, which was probably related to pelvic size and prepregnancy BMI and not to gain, since the risk did not vary with GWG.

Young age

- Young primiparae in these data had better outcomes than primiparae aged 25-29 years. It was suggested in the IOM (1990) guidelines that adolescents should gain more weight to avoid SGA, but these findings suggest that this is not necessary, at least not among those at ages (mean=18.4 years) studied here. However, this may not be true among younger teens, which was poorly presented in the DNBC.

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Smokers

- Smokers had a substantial excess risk of SGA, which was only eliminated in multiparous women with high prepregnant BMI values and high gains. However, smokers retained weight just like non-smokers. Thus, smoking cessation still seems the best way to improve birth outcomes and reduce the risk of excessive postpartum weight retention among smokers.

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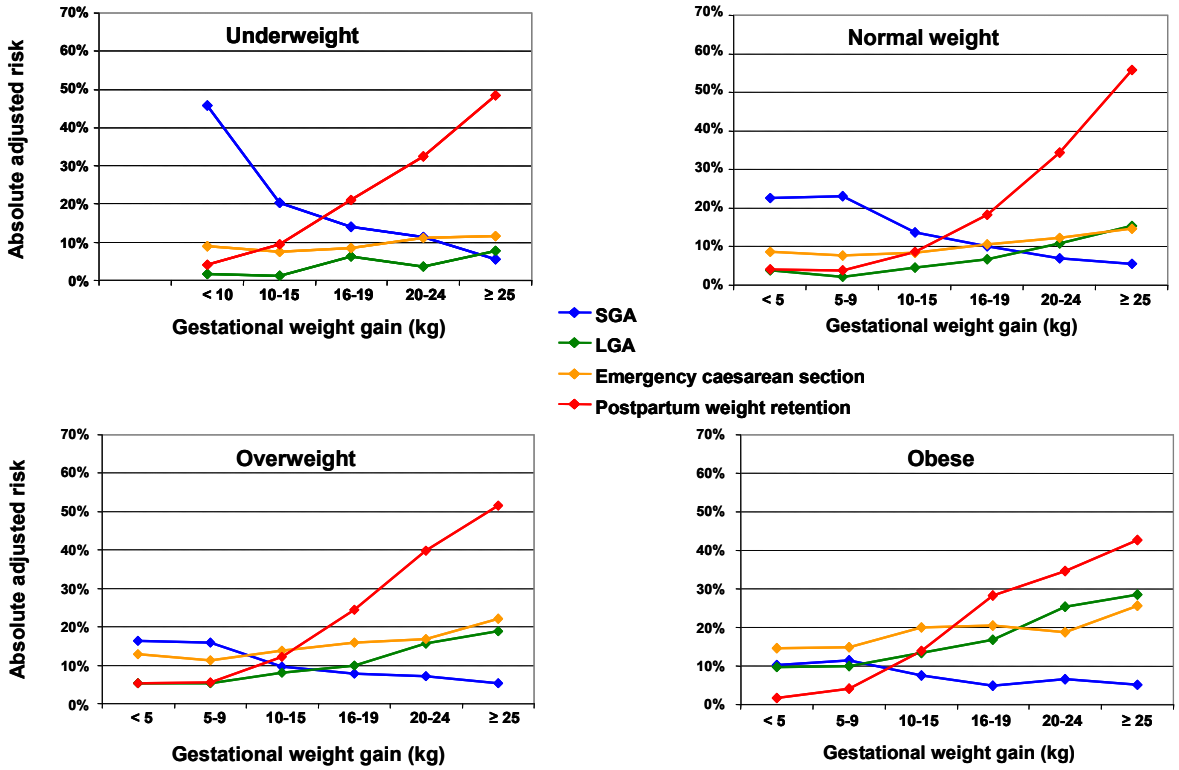

FIGURE 24 Unexposed primiparae, GWG –specific risks of pregnancy outcomes.
NOTE: Age 25-29, height 160-169 cm, high social status. In pregnancy: no smoking, no alcohol, moderate exercise. For PPWR, she breastfed > 14 weeks.

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TABLE 24 Unexposed primiparae, GWG –specific risks of pregnancy outcomes

	< 5 kg	< 10 kg	10-15 kg	16-19 kg	20-24 kg	≥ 25 kg
Underweight						
SGA		0.46	0.20	0.14	0.11	0.06
		(0.36; 0.56)	(0.17; 0.24)	(0.10; 0.19)	(0.08; 0.17)	(0.03; 0.11)
LGA		0.02	0.01	0.06	0.04	0.08
		(0.00; 0.07)	(0.01; 0.03)	(0.04; 0.10)	(0.02; 0.08)	(0.04; 0.16)
Emergency		0.09	0.08	0.09	0.11	0.12
CS		(0.04; 0.18)	(0.05; 0.10)	(0.06; 0.13)	(0.07; 0.17)	(0.06; 0.21)
PPWR		0.04	0.09	0.21	0.32	0.48
		(0.02; 0.10)	(0.07; 0.12)	(0.17; 0.26)	(0.26; 0.40)	(0.38; 0.59)
Normal weight						
SGA	0.23	0.23	0.14	0.10	0.07	0.06
	(0.15; 0.34)	(0.20; 0.26)	(0.13; 0.15)	(0.09; 0.11)	(0.06; 0.08)	(0.05; 0.07)
LGA	0.04	0.02	0.04	0.07	0.11	0.16
	(0.01; 0.11)	(0.02; 0.03)	(0.04; 0.05)	(0.06; 0.08)	(0.09; 0.12)	(0.13; 0.18)
Emergency	0.09	0.08	0.08	0.11	0.12	0.15
CS	(0.04; 0.19)	(0.06; 0.10)	(0.08; 0.09)	(0.09; 0.12)	(0.11; 0.14)	(0.12; 0.17)
PPWR	0.04	0.04	0.09	0.18	0.34	0.56
	(0.02; 0.11)	(0.03; 0.05)	(0.08; 0.09)	(0.17; 0.20)	(0.32; 0.37)	(0.53; 0.59)
Overweight						
SGA	0.16	0.16	0.10	0.08	0.07	0.05
	(0.11; 0.24)	(0.13; 0.20)	(0.08; 0.11)	(0.06; 0.10)	(0.06; 0.09)	(0.04; 0.08)
LGA	0.05	0.05	0.08	0.10	0.16	0.19
	(0.02; 0.11)	(0.04; 0.08)	(0.07; 0.10)	(0.08; 0.12)	(0.13; 0.19)	(0.15; 0.23)
Emergency	0.13	0.11	0.14	0.16	0.17	0.22
CS	(0.08; 0.21)	(0.09; 0.15)	(0.12; 0.16)	(0.13; 0.19)	(0.14; 0.20)	(0.18; 0.26)
PPWR	0.05	0.05	0.12	0.24	0.40	0.51
	(0.03; 0.10)	(0.04; 0.08)	(0.11; 0.14)	(0.21; 0.27)	(0.36; 0.44)	(0.47; 0.56)
Obese						
SGA	0.10	0.12	0.07	0.04	0.07	0.05
	(0.07; 0.14)	(0.09; 0.15)	(0.06; 0.10)	(0.02; 0.08)	(0.04; 0.11)	(0.02; 0.09)
LGA	0.10	0.10	0.13	0.17	0.26	0.29
	(0.06; 0.14)	(0.07; 0.14)	(0.11; 0.17)	(0.12; 0.23)	(0.19; 0.34)	(0.21; 0.40)
Emergency	0.15	0.15	0.20	0.21	0.19	0.26
CS	(0.11; 0.20)	(0.12; 0.19)	(0.17; 0.23)	(0.15; 0.28)	(0.13; 0.26)	(0.19; 0.36)
PPWR	0.02	0.04	0.14	0.29	0.35	0.42
	(0.01; 0.04)	(0.03; 0.06)	(0.11; 0.17)	(0.23;0.35)	(0.28; 0.43)	(0.34; 0.51)

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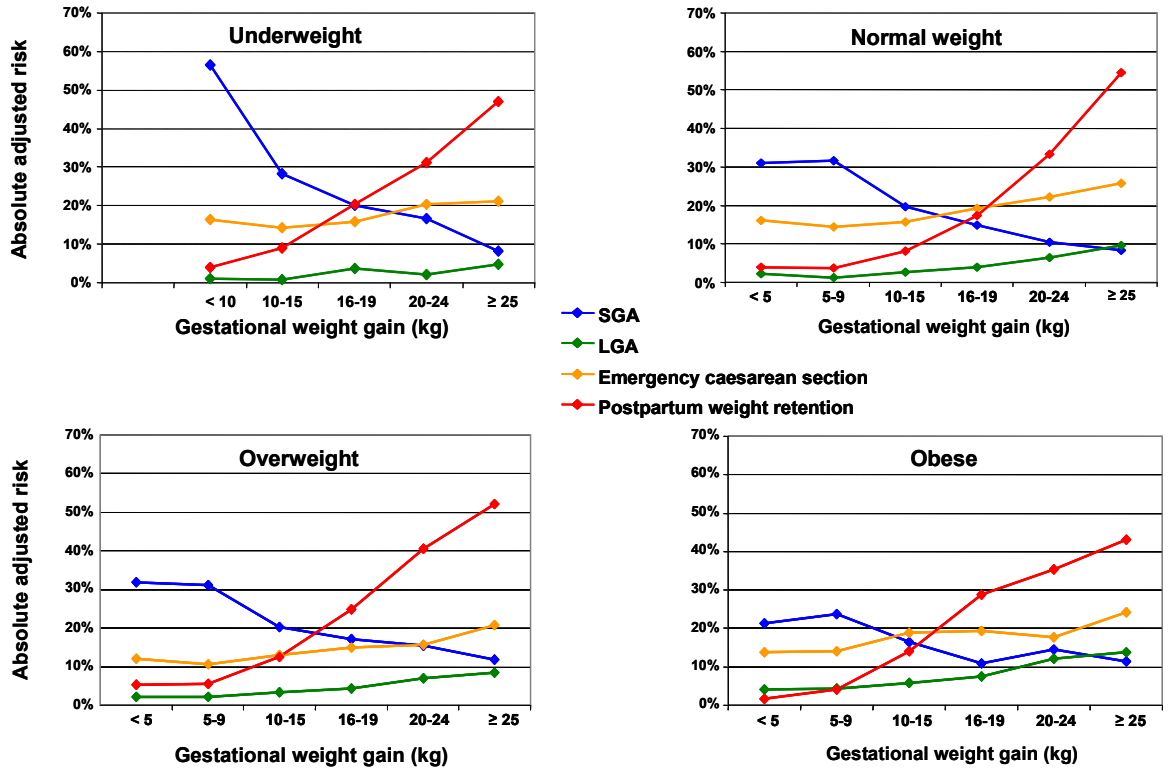

FIGURE 25 Short primiparae, GWG –specific risks of pregnancy outcomes.
NOTE: Age 25-29, height < 160 cm, high social status. In pregnancy: no smoking, no alcohol, moderate exercise. For PPWR, she breastfed > 14 weeks.

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TABLE 25 Short primiparae, GWG –specific risks of pregnancy outcomes

	< 5 kg	< 10 kg	10-15 kg	16-19 kg	20-24 kg	≥ 25 kg
Underweight						
SGA		0.57	0.28	0.20	0.17	0.08
		(0.46; 0.67)	(0.23; 0.34)	(0.15; 0.27)	(0.11; 0.24)	(0.04; 0.17)
LGA		0.01	0.01	0.04	0.02	0.05
		(0.00; 0.04)	(0.00; 0.02)	(0.02; 0.07)	(0.01; 0.05)	(0.02; 0.10)
Emergency		0.17	0.14	0.16	0.20	0.21
CS		(0.08; 0.31)	(0.10; 0.19)	(0.10; 0.24)	(0.13; 0.30)	(0.12; 0.36)
PPWR		0.04	0.09	0.20	0.31	0.47
		(0.02; 0.09)	(0.07; 0.12)	(0.15; 0.26)	(0.24; 0.39)	(0.36; 0.58)
Normal weight						
SGA	0.31	0.32	0.20	0.15	0.11	0.09
	(0.21; 0.45)	(0.28; 0.36)	(0.17; 0.22)	(0.13; 0.17)	(0.09; 0.13)	(0.07; 0.11)
LGA	0.02	0.01	0.03	0.04	0.07	0.10
	(0.01; 0.07)	(0.01; 0.02)	(0.02; 0.04)	(0.03; 0.06)	(0.05; 0.09)	(0.07; 0.13)
Emergency	0.16	0.15	0.16	0.19	0.22	0.26
CS	(0.07; 0.33)	(0.12; 0.18)	(0.14; 0.18)	(0.17; 0.22)	(0.19; 0.26)	(0.22; 0.30)
PPWR	0.04	0.04	0.08	0.17	0.33	0.55
	(0.01; 0.10)	(0.03; 0.05)	(0.07; 0.09)	(0.15; 0.20)	(0.29; 0.37)	(0.50; 0.59)
Overweight						
SGA	0.23	0.23	0.14	0.12	0.11	0.08
	(0.16; 0.33)	(0.18; 0.28)	(0.12; 0.17)	(0.09; 0.15)	(0.08; 0.14)	(0.06; 0.11)
LGA	0.03	0.03	0.05	0.06	0.10	0.12
	(0.01; 0.07)	(0.02; 0.05)	(0.04; 0.07)	(0.04; 0.08)	(0.07; 0.13)	(0.08; 0.16)
Emergency	0.23	0.21	0.24	0.28	0.29	0.36
CS	(0.14; 0.35)	(0.16; 0.26)	(0.21; 0.28)	(0.23; 0.33)	(0.24; 0.35)	(0.30; 0.43)
PPWR	0.05	0.05	0.12	0.23	0.38	0.50
	(0.03; 0.09)	(0.04; 0.07)	(0.10; 0.14)	(0.20; 0.27)	(0.33; 0.44)	(0.44; 0.56)
Obese						
SGA	0.15	0.17	0.11	0.07	0.10	0.07
	(0.11; 0.21)	(0.13; 0.22)	(0.08; 0.14)	(0.04; 0.12)	(0.06; 0.16)	(0.04; 0.13)
LGA	0.06	0.06	0.08	0.11	0.17	0.19
	(0.04; 0.09)	(0.04; 0.09)	(0.06; 0.12)	(0.07; 0.16)	(0.11; 0.24)	(0.12; 0.29)
Emergency	0.26	0.27	0.33	0.35	0.32	0.42
CS	(0.19; 0.34)	(0.21; 0.33)	(0.28; 0.39)	(0.26; 0.44)	(0.23; 0.42)	(0.32; 0.53)
PPWR	0.02	0.04	0.13	0.27	0.34	0.41
	(0.01; 0.03)	(0.02; 0.06)	(0.11; 0.16)	(0.21;0.35)	(0.26; 0.42)	(0.32; 0.51)

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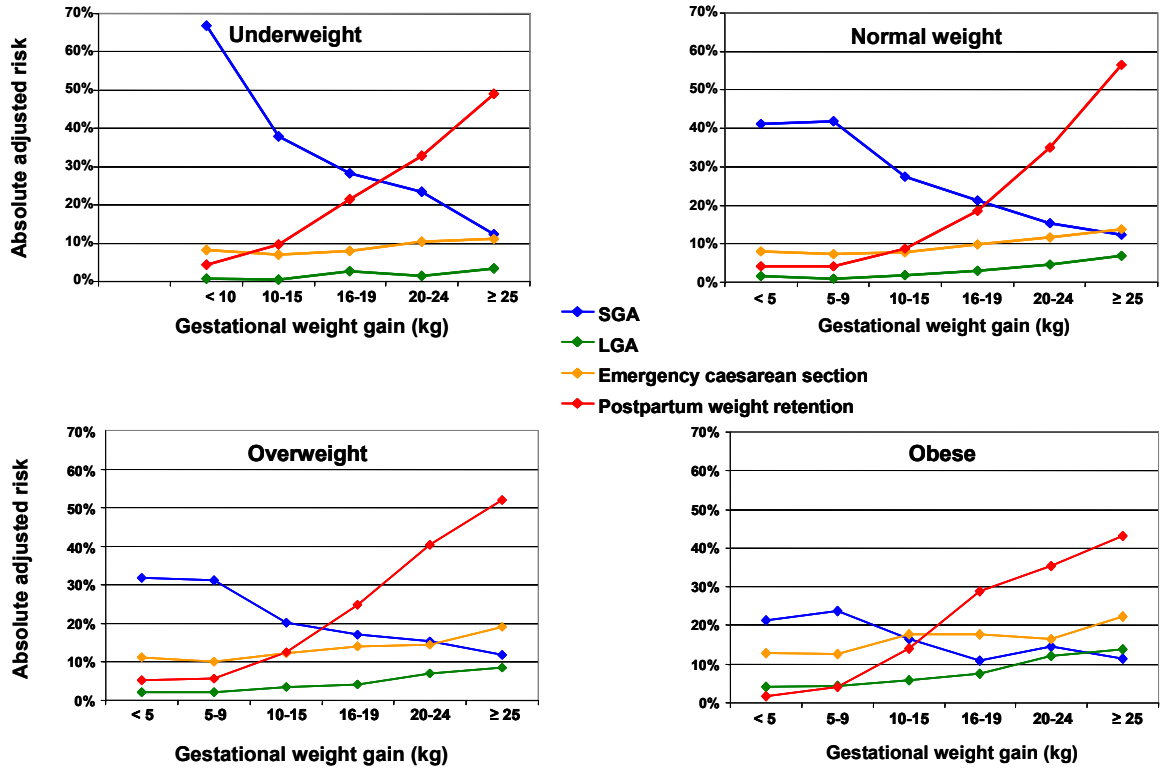

FIGURE 26 Smoking primiparae, GWG –specific risks of pregnancy outcomes.
NOTE: Age 25-29, height 160-169 cm, high social status. Smoked in pregnancy, no alcohol, moderate exercise. For PPWR, she breastfed > 14 weeks.

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TABLE 26 Smoking primiparae, GWG –specific risks of pregnancy outcomes

	< 5 kg	< 10 kg	10-15 kg	16-19 kg	20-24 kg	≥ 25 kg
Underweight						
SGA		0.67	0.38	0.28	0.24	0.12
		(0.57; 0.76)	(0.32; 0.44)	(0.22; 036)	(0.17; 0.32)	(0.06; 0.23)
LGA		0.01	0.01	0.03	0.02	0.03
		(0.00; 0.03)	(0.00; 0.01)	(0.02; 0.04)	(0.01; 0.03)	(0.01; 0.07)
Emergency		0.08	0.07	0.08	0.11	0.11
CS		(0.04; 0.17)	(0.05; 0.10)	(0.05; 0.12)	(0.07; 0.17)	(0.06; 0.20)
PPWR		0.04	0.10	0.21	0.33	0.49
		(0.02; 0.10)	(0.07; 0.12)	(0.17; 0.27)	(0.26; 0.40)	(0.38; 0.59)
Normal weight						
SGA	0.41	0.42	0.28	0.21	0.15	0.13
	(0.29; 0.55)	(0.38; 0.46)	(0.25; 0.30)	(0.19; 0.24)	(0.13; 0.18)	(0.10; 0.15)
LGA	0.02	0.01	0.02	0.03	0.05	0.07
	(0.00; 0.05)	(0.01; 0.01)	(0.01; 0.02)	(0.02; 0.04)	(0.04; 0.06)	(0.06; 0.09)
Emergency	0.08	0.07	0.08	0.10	0.11	0.14
CS	(0.03; 0.18)	(0.06; 0.09)	(0.07; 0.09)	(0.09; 0.12)	(0.10; 0.13)	(0.11; 0.16)
PPWR	0.04	0.04	0.09	0.18	0.35	0.56
	(0.02; 0.11)	(0.03; 0.05)	(0.08; 0.10)	(0.17; 0.21)	(0.32; 0.87)	(0.53; 0.60)
Overweight						
SGA	0.32	0.23	0.20	0.17	0.15	0.12
	(0.31; 0.33)	(0.26; 0.37)	(0.17; 0.23)	(0.14; 0.21)	(0.12; 0.19)	(0.09; 0.16)
LGA	0.02	0.02	0.03	0.04	0.07	0.08
	(0.01; 0.05)	(0.01; 0.03)	(0.03; 0.04)	(0.03; 0.06)	(0.05; 0.09)	(0.06; 0.11)
Emergency	0.12	0.11	0.13	0.15	0.16	0.20
CS	(0.07; 0.20)	(0.08; 0.14)	(0.11; 0.15)	(0.12; 0.18)	(0.13; 0.19)	(0.16; 0.25)
PPWR	0.05	0.06	0.12	0.25	0.40	0.52
	(0.03; 0.10)	(0.04; 0.08)	(0.11; 0.14)	(0.21; 0.28)	(0.36; 0.45)	(0.47; 0.57)
Obese						
SGA	0.22	0.24	0.16	0.10	0.15	0.10
	(0.16; 0.29)	(0.19; 0.30)	(0.13; 0.20)	(0.06; 0.17)	(0.09; 0.23)	(0.05; 0.19)
LGA	0.04	0.04	0.06	0.08	0.12	0.14
	(0.03; 0.06)	(0.03; 0.06)	(0.04; 0.08)	(0.05; 0.11)	(0.08; 0.17)	(0.09; 0.21)
Emergency	0.14	0.14	0.19	0.20	0.18	0.25
CS	(0.10; 0.19)	(0.11; 0.19)	(0.15; 0.23)	(0.14; 0.27)	(0.12; 0.25)	(0.18; 0.34)
PPWR	0.02	0.04	0.14	0.29	0.35	0.43
	(0.01; 0.04)	(0.02; 0.06)	(0.11; 0.17)	(0.23;0.36)	(0.28; 0.43)	(0.34; 0.52)

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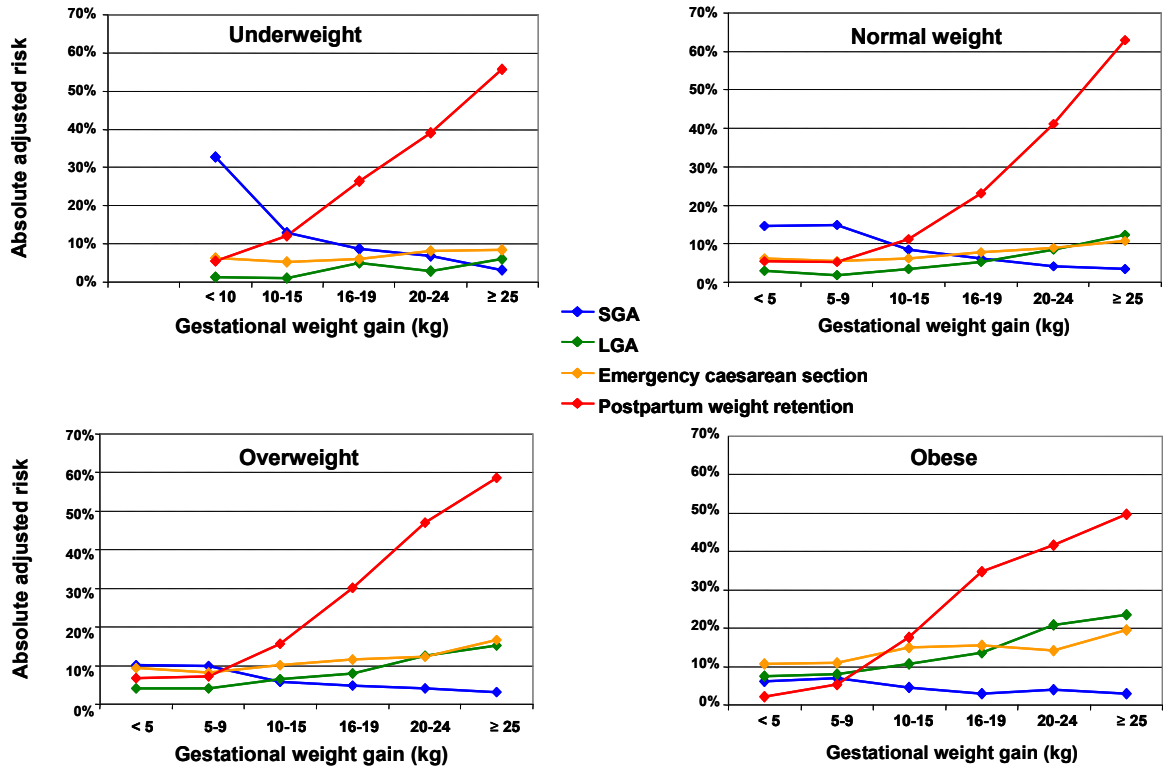

FIGURE 27 Young primiparae, GWG –specific risks of pregnancy outcomes.
NOTE: Age < 20, height 160-169 cm, high social status. In pregnancy: no smoking, no alcohol, moderate exercise. For PPWR, she breastfed > 14 weeks.

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TABLE 27 Young primiparae, GWG –specific risks of pregnancy outcomes

	< 5 kg	< 10 kg	10-15 kg	16-19 kg	20-24 kg	≥ 25 kg
Underweight						
SGA		0.33	0.13	0.09	0.07	0.03
		(0.22; 0.46)	(0.09; 0.18)	(0.06; 0.14)	(0.04; 0.12)	(0.01; 0.07)
LGA		0.01	0.01	0.05	0.03	0.06
		(0.00; 0.06)	(0.00; 0.02)	(0.03; 0.09)	(0.01; 0.07)	(0.03; 0.14)
Emergency		0.06	0.05	0.06	0.08	0.09
CS		(0.03; 0.14)	(0.03; 0.09)	(0.03; 0.11)	(0.05; 0.14)	(0.04; 0.17)
PPWR		0.06	0.12	0.27	0.39	0.56
		(0.02; 0.13)	(0.09; 0.17)	(0.20; 0.35)	(0.30; 0.49)	(0.44; 0.67)
Normal weight						
SGA	0.15	0.15	0.09	0.06	0.04	0.03
	(0.08; 0.24)	(0.11; 0.21)	(0.06; 0.12)	(0.04; 0.09)	(0.03; 0.06)	(0.02; 0.05)
LGA	0.03	0.02	0.03	0.05	0.08	0.12
	(0.01; 0.10)	(0.01; 0.03)	(0.02; 0.05)	(0.03; 0.08)	(0.05; 0.13)	(0.08; 0.19)
Emergency	0.06	0.06	0.06	0.08	0.09	0.11
CS	(0.02; 0.15)	(0.04; 0.89)	(0.04; 0.09)	(0.05; 0.11)	(0.06; 0.13)	(0.07; 0.15)
PPWR	0.05	0.05	0.11	0.23	0.41	0.63
	(0.02; 0.14)	(0.04; 0.07)	(0.09; 0.14)	(0.19; 0.28)	(0.35; 0.48)	(0.56; 0.69)
Overweight						
SGA	0.10	0.10	0.06	0.05	0.04	0.03
	(0.06; 0.17)	(0.07; 0.15)	(0.04; 0.08)	(0.03; 0.07)	(0.03; 0.07)	(0.02; 0.05)
LGA	0.04	0.04	0.06	0.08	0.13	0.15
	(0.02; 0.09)	(0.02; 0.07)	(0.04; 0.10)	(0.05; 0.12)	(0.08; 0.19)	(0.10; 0.23)
Emergency	0.09	0.08	0.10	0.12	0.13	0.16
CS	(0.05; 0.17)	(0.05; 0.13)	(0.07; 0.14)	(0.08; 0.17)	(0.09; 0.18)	(0.11; 0.23)
PPWR	0.07	0.07	0.16	0.30	0.47	0.59
	(0.03; 0.13)	(0.05; 0.11)	(0.12; 0.20)	(0.24; 0.37)	(0.40; 0.54)	(0.51; 0.66)
Obese						
SGA	0.06	0.07	0.05	0.03	0.04	0.03
	(0.04; 0.10)	(0.05; 0.11)	(0.03; 0.07)	(0.01; 0.05)	(0.02; 0.07)	(0.01; 0.06)
LGA	0.08	0.08	0.11	0.14	0.21	0.24
	(0.04; 0.13)	(0.05; 0.13)	(0.07; 0.17)	(0.08; 0.22)	(0.13; 0.33)	(0.15; 0.38)
Emergency	0.11	0.11	0.15	0.16	0.14	0.20
CS	(0.07; 0.17)	(0.07; 0.17)	(0.10; 0.21)	(0.10; 0.24)	(0.09; 0.22)	(0.13; 0.30)
PPWR	0.02	0.05	0.18	0.35	0.42	0.50
	(0.01; 0.05)	(0.03; 0.09)	(0.13; 0.23)	(0.27; 0.45)	(0.32; 0.52)	(0.39; 0.60)

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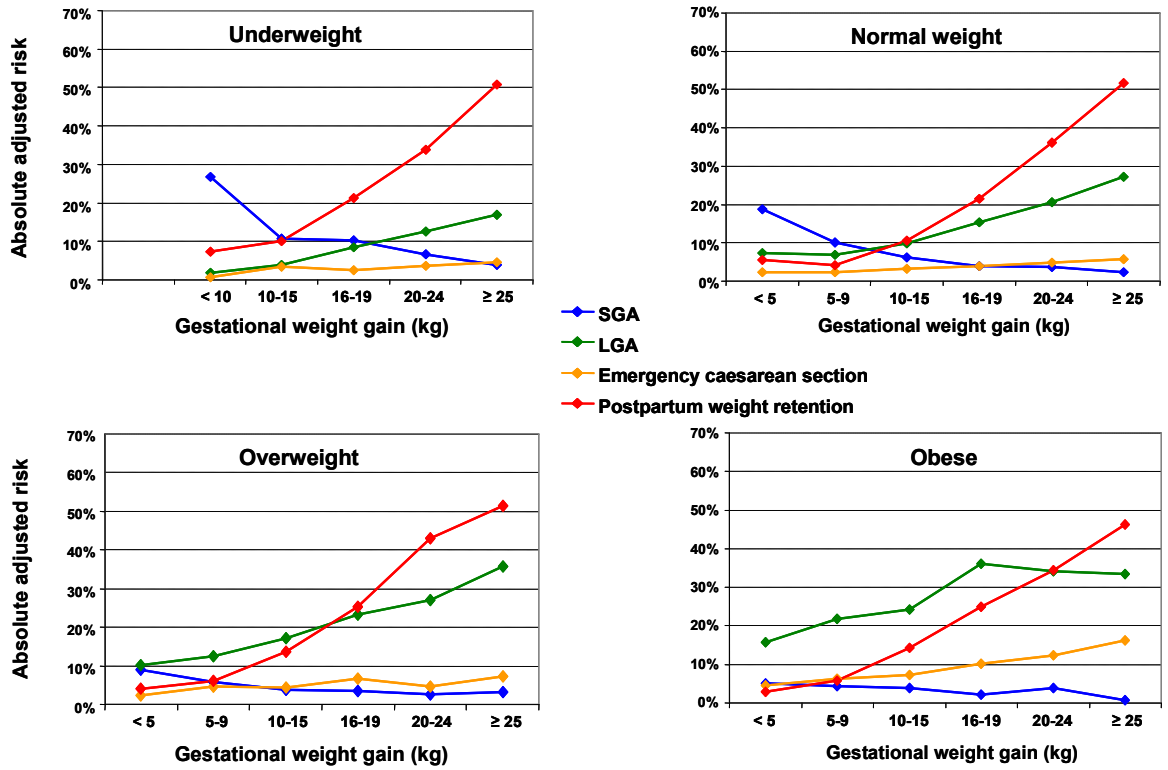

FIGURE 28 Unexposed multiparae, GWG –specific risks of pregnancy outcomes.
NOTE: Age 25-29, height 160-169 cm, high social status. In pregnancy: no smoking, no alcohol, moderate exercise. For PPWR, she breastfed > 14 weeks.

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TABLE 28 Unexposed multiparae, GWG –specific risks of pregnancy outcomes

	< 5 kg	< 10 kg	10-15 kg	16-19 kg	20-24 kg	≥ 25 kg
Underweight						
SGA		0.27	0.11	0.10	0.07	0.04
		(0.19; 0.36)	(0.09; 0.14)	(0.08; 0.14)	(0.04; 0.11)	(0.01; 0.10)
LGA		0.02	0.04	0.08	0.13	0.17
		(0.01; 0.06)	(0.03; 0.06)	(0.06; 0.12)	(0.08; 0.19)	(0.09; 0.30)
Emergency		0.01	0.03	0.03	0.04	0.04
CS		(0.00; 0.05)	(0.02; 0.05)	(0.01; 0.05)	(0.02; 0.08)	(0.01; 0.13)
PPWR		0.07	0.10	0.21	0.34	0.51
		(0.04; 0.13)	(0.08; 0.13)	(0.17; 0.26)	(0.27; 0.41)	(0.39; 0.62)
Normal weight						
SGA	0.19	0.10	0.06	0.04	0.04	0.02
	(0.13; 0.27)	(0.08; 0.12)	(0.05; 0.07)	(0.03; 0.05)	(0.03; 0.05)	(0.02; 0.03)
LGA	0.07	0.07	0.10	0.15	0.20	0.27
	(0.04; 0.14)	(0.06; 0.08)	(0.09; 0.11)	(0.14; 0.17)	(0.18; 0.23)	(0.24; 0.31)
Emergency	0.02	0.02	0.03	0.04	0.05	0.06
CS	(0.01; 0.07)	(0.02; 0.03)	(0.03; 0.04)	(0.03; 0.05)	(0.04; 0.06)	(0.04; 0.07)
PPWR	0.05	0.04	0.11	0.22	0.36	0.52
	(0.03; 0.11)	(0.03; 0.05)	(0.10; 0.12)	(0.20; 0.23)	(0.33; 0.39)	(0.48; 0.55)
Overweight						
SGA	0.09	0.06	0.04	0.03	0.02	0.03
	(0.06; 0.13)	(0.04; 0.07)	(0.03; 0.05)	(0.02; 0.05)	(0.01; 0.04)	(0.02; 0.05)
LGA	0.10	0.13	0.17	0.23	0.27	0.36
	(0.07; 0.15)	(0.10; 0.15)	(0.15; 0.19)	(0.20; 0.26)	(0.24; 0.31)	(0.31; 0.41)
Emergency	0.02	0.05	0.05	0.07	0.04	0.07
CS	(0.01; 0.05)	(0.03; 0.06)	(0.04; 0.06)	(0.05; 0.09)	(0.03; 0.07)	(0.05; 0.11)
PPWR	0.04	0.06	0.14	0.25	0.43	0.51
	(0.02; 0.07)	(0.05; 0.08)	(0.12; 0.15)	(0.22; 0.28)	(0.39; 0.47)	(0.46; 0.56)
Obese						
SGA	0.05	0.04	0.05	0.02	0.04	0.01
	(0.03; 0.07)	(0.03; 0.06)	(0.03; 0.06)	(0.01; 0.05)	(0.02; 0.08)	(0.00; 0.05)
LGA	0.16	0.22	0.24	0.36	0.34	0.35
	(0.13; 0.20)	(0.19; 0.26)	(0.21; 0.28)	(0.30; 0.44)	(0.26; 0.42)	(0.26; 0.45)
Emergency	0.05	0.06	0.07	0.10	0.12	0.16
CS	(0.03; 0.07)	(0.04; 0.09)	(0.05; 0.10)	(0.06; 0.16)	(0.08; 0.20)	(0.09; 0.26)
PPWR	0.03	0.06	0.14	0.25	0.34	0.46
	(0.02; 0.05)	(0.04; 0.08)	(0.12; 0.17)	(0.20; 0.31)	(0.27; 0.42)	(0.36; 0.56)

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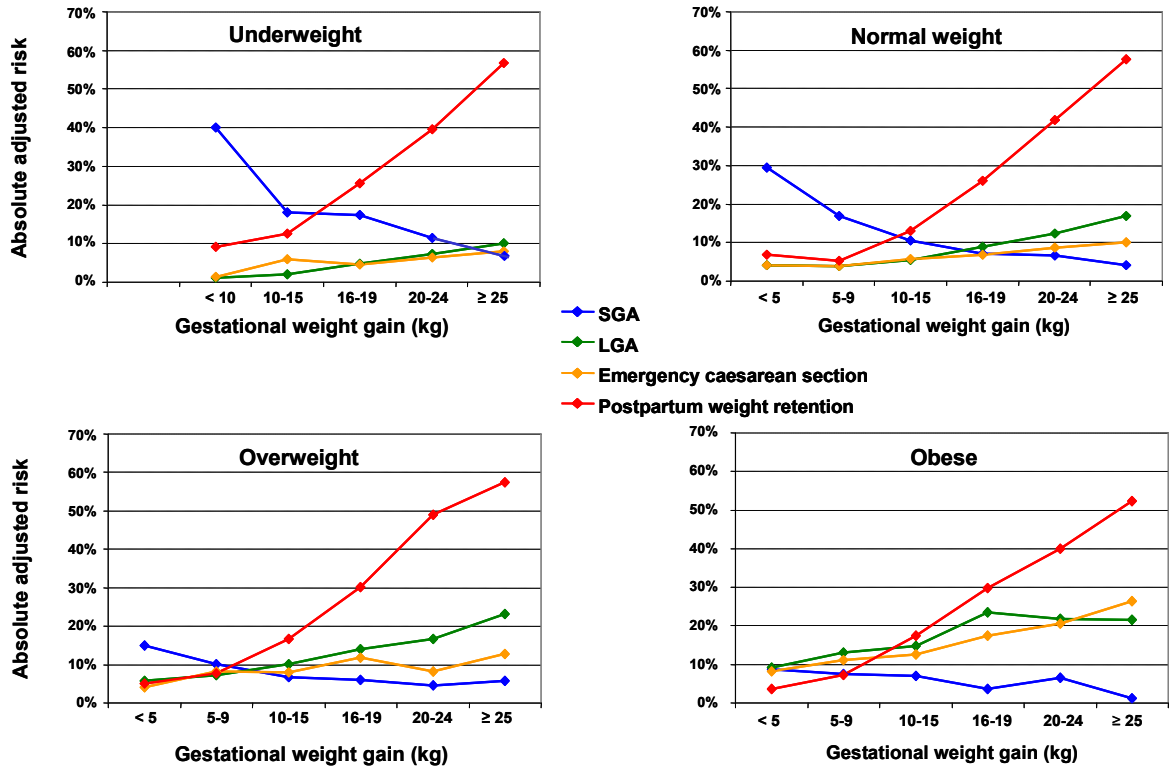

FIGURE 29 Short multiparae, GWG –specific risks of pregnancy outcomes.
NOTE: Age 25-29, height < 160 cm, high social status. In pregnancy: no smoking, no alcohol, moderate exercise. For PPWR, she breastfed > 14 weeks.

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TABLE 29 Short multiparae, GWG –specific risks of pregnancy outcomes

	< 5 kg	< 10 kg	10-15 kg	16-19 kg	20-24 kg	≥ 25 kg
Underweight						
SGA		0.40	0.18	0.18	0.12	0.07
		(0.30; 0.52)	(0.14; 0.23)	(0.13; 0.24)	(0.07; 0.19)	(0.03; 0.17)
LGA		0.01	0.02	0.05	0.07	0.10
		(0.00; 0.03)	(0.01; 0.03)	(0.03; 0.07)	(0.05; 0.11)	(0.05; 0.19)
Emergency		0.01	0.06	0.05	0.06	0.08
CS		(0.00; 0.09)	(0.04; 0.09)	(0.02; 0.09)	(0.03; 0.14)	(0.03; 0.22)
PPWR		0.09	0.13	0.26	0.39	0.57
		(0.05; 0.16)	(0.10; 0.16)	(0.20; 0.32)	(0.32; 0.48)	(0.45; 0.68)
Normal weight						
SGA	0.30	0.17	0.11	0.07	0.07	0.04
	(0.21; 0.41)	(0.14; 0.21)	(0.09; 0.13)	(0.06; 0.09)	(0.05; 0.09)	(0.03; 0.06)
LGA	0.04	0.04	0.06	0.09	0.12	0.17
	(0.02; 0.09)	(0.03; 0.05)	(0.05; 0.07)	(0.07; 0.11)	(0.10; 0.15)	(0.14; 0.21)
Emergency	0.04	0.04	0.06	0.07	0.09	0.10
CS	(0.01; 0.12)	(0.03; 0.06)	(0.04; 0.07)	(0.05; 0.09)	(0.07; 0.11)	(0.07; 0.14)
PPWR	0.07	0.05	0.13	0.26	0.42	0.58
	(0.03; 0.13)	(0.04; 0.07)	(0.11; 0.15)	(0.23; 0.29)	(0.38; 0.46)	(0.53; 0.62)
Overweight						
SGA	0.15	0.10	0.07	0.06	0.04	0.05
	(0.10; 0.21)	(0.08; 0.13)	(0.05; 0.09)	(0.04; 0.09)	(0.03; 0.07)	(0.03; 0.09)
LGA	0.06	0.07	0.10	0.14	0.17	0.23
	(0.04; 0.09)	(0.06; 0.09)	(0.08; 0.12)	(0.11; 0.17)	(0.14; 0.21)	(0.19; 0.29)
Emergency	0.04	0.08	0.08	0.12	0.08	0.13
CS	(0.02; 0.09)	(0.06; 0.12)	(0.06; 0.10)	(0.09; 0.16)	(0.05; 0.12)	(0.09; 0.19)
PPWR	0.05	0.08	0.17	0.30	0.49	0.57
	(0.03; 0.09)	(0.06; 0.10)	(0.14; 0.19)	(0.26; 0.34)	(0.44; 0.54)	(0.51; 0.63)
Obese						
SGA	0.09	0.07	0.07	0.04	0.07	0.01
	(0.06; 0.13)	(0.05; 0.10)	(0.05; 0.10)	(0.02; 0.09)	(0.03; 0.14)	(0.00; 0.09)
LGA	0.09	0.13	0.15	0.24	0.21	0.22
	(0.07; 0.12)	(0.10; 0.17)	(0.12; 0.18)	(0.18; 0.31)	(0.15; 0.29)	(0.15; 0.31)
Emergency	0.08	0.11	0.13	0.18	0.21	0.26
CS	(0.05; 0.13)	(0.08; 0.15)	(0.09; 0.17)	(0.11; 0.27)	(0.13; 0.32)	(0.15; 0.40)
PPWR	0.04	0.07	0.18	0.30	0.40	0.52
	(0.02; 0.06)	(0.05; 0.10)	(0.14; 0.21)	(0.23; 0.37)	(0.31; 0.48)	(0.42; 0.62)

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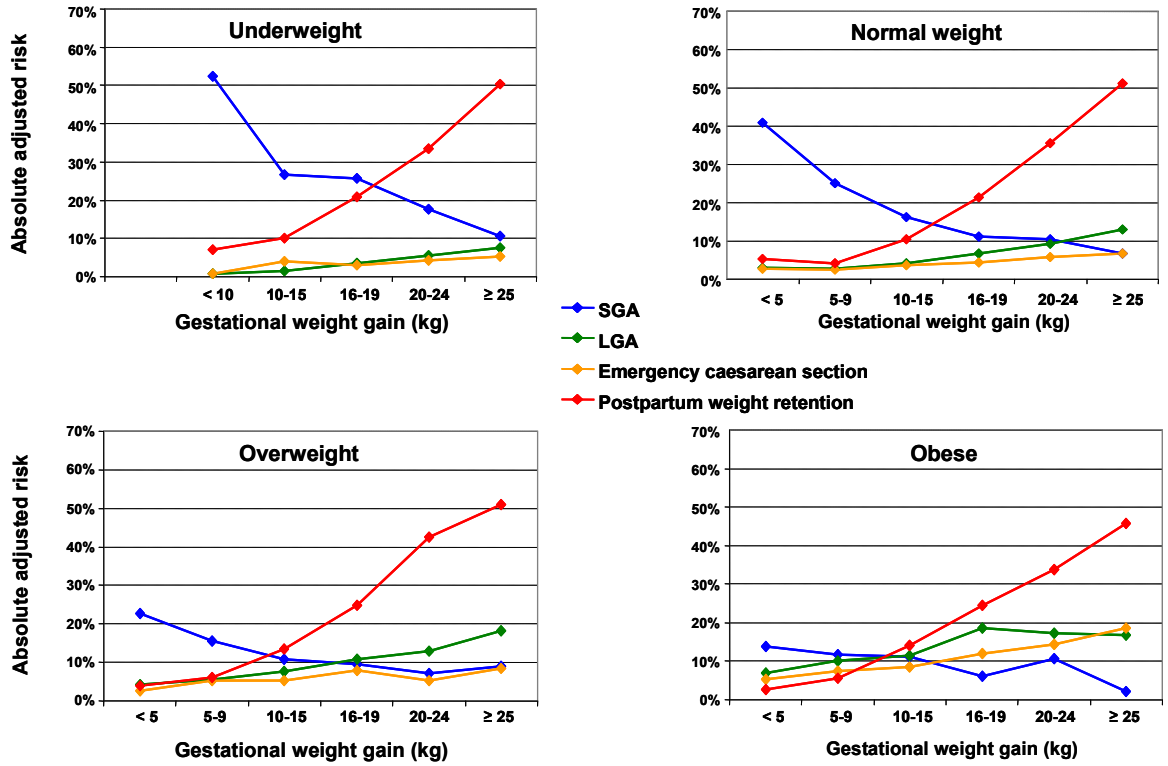

FIGURE 30 Smoking multiparae, GWG –specific risks of pregnancy outcomes.
NOTE: Age 25-29, height 160-169 cm, high social status. Smoked during pregnancy, no alcohol, moderate exercise. For PPWR, she breastfed > 14 weeks.

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TABLE 30 Smoking multiparae, GWG –specific risks of pregnancy outcomes

	< 5 kg	< 10 kg	10-15 kg	16-19 kg	20-24 kg	≥ 25 kg
Underweight						
SGA		0.53	0.27	0.26	0.18	0.11
		(0.42; 0.64)	(0.22; 0.33)	(0.20; 0.33)	(0.11; 0.27)	(0.04; 0.25)
LGA		0.01	0.02	0.04	0.05	0.108
		(0.00; 0.02)	(0.01; 0.03)	(0.02; 0.05)	(0.03; 0.09)	(0.04; 0.14)
Emergency		0.01	0.04	0.03	0.04	0.05
CS		(0.00; 0.06)	(0.03; 0.06)	(0.01; 0.06)	(0.02; 0.09)	(0.02; 0.15)
PPWR		0.07	0.10	0.21	0.33	0.50
		(0.04; 0.13)	(0.08; 0.12)	(0.17; 0.26)	(0.27; 0.41)	(0.38; 0.62)
Normal weight						
SGA	0.41	0.25	0.16	0.11	0.11	0.07
	(0.30; 0.53)	(0.21; 0.29)	(0.14; 0.19)	(0.09; 0.13)	(0.09; 0.13)	(0.05; 0.09)
LGA	0.03	0.03	0.04	0.07	0.09	0.13
	(0.02; 0.06)	(0.02; 0.04)	(0.04; 0.05)	(0.06; 0.08)	(0.08; 0.11)	(0.11; 0.15)
Emergency	0.03	0.03	0.04	0.04	0.06	0.07
CS	(0.01; 0.08)	(0.02; 0.04)	(0.03; 0.05)	(0.04; 0.06)	(0.04; 0.07)	(0.05; 0.09)
PPWR	0.05	0.04	0.10	0.21	0.36	0.51
	(0.03; 0.10)	(0.03; 0.05)	(0.09; 0.12)	(0.19; 0.24)	(0.33; 0.39)	(0.47; 0.55)
Overweight						
SGA	0.22	0.16	0.11	0.10	0.07	0.09
	(0.16; 0.30)	(0.12; 0.20)	(0.09; 0.13)	(0.07; 0.13)	(0.04; 0.10)	(0.05; 0.14)
LGA	0.04	0.05	0.08	0.11	0.13	0.18
	(0.03; 0.07)	(0.04; 0.07)	(0.07; 0.09)	(0.09; 0.13)	(0.11; 0.16)	(0.15; 0.22)
Emergency	0.03	0.05	0.05	0.08	0.05	0.09
CS	(0.01; 0.06)	(0.04; 0.08)	(0.04; 0.7)	(0.06; 0.11)	(0.03; 0.08)	(0.06; 0.13)
PPWR	0.04	0.06	0.13	0.25	0.43	0.51
	(0.02; 0.07)	(0.05; 0.08)	(0.12; 0.15)	(0.22; 0.28)	(0.38; 0.47)	(0.45; 0.56)
Obese						
SGA	0.13	0.12	0.11	0.06	0.11	0.02
	(0.09; 0.19)	(0.08; 0.16)	(0.08; 0.15)	(0.03; 0.14)	(0.05; 0.21)	(0.00; 0.25)
LGA	0.07	0.10	0.11	0.19	0.17	0.17
	(0.05; 0.09)	(0.08; 0.13)	(0.09; 0.14)	(0.14; 0.24)	(0.12; 0.23)	(0.12; 0.31)
Emergency	0.05	0.07	0.08	0.12	0.14	0.18
CS	(0.03; 0.09)	(0.05; 0.10)	(0.06; 0.11)	(0.07; 0.19)	(0.09; 0.23)	(0.10; 0.29)
PPWR	0.03	0.06	0.14	0.25	0.33	0.45
	(0.02; 0.05)	(0.04; 0.08)	(0.12; 0.17)	(0.19; 0.31)	(0.26; 0.42)	(0.36; 0.56)

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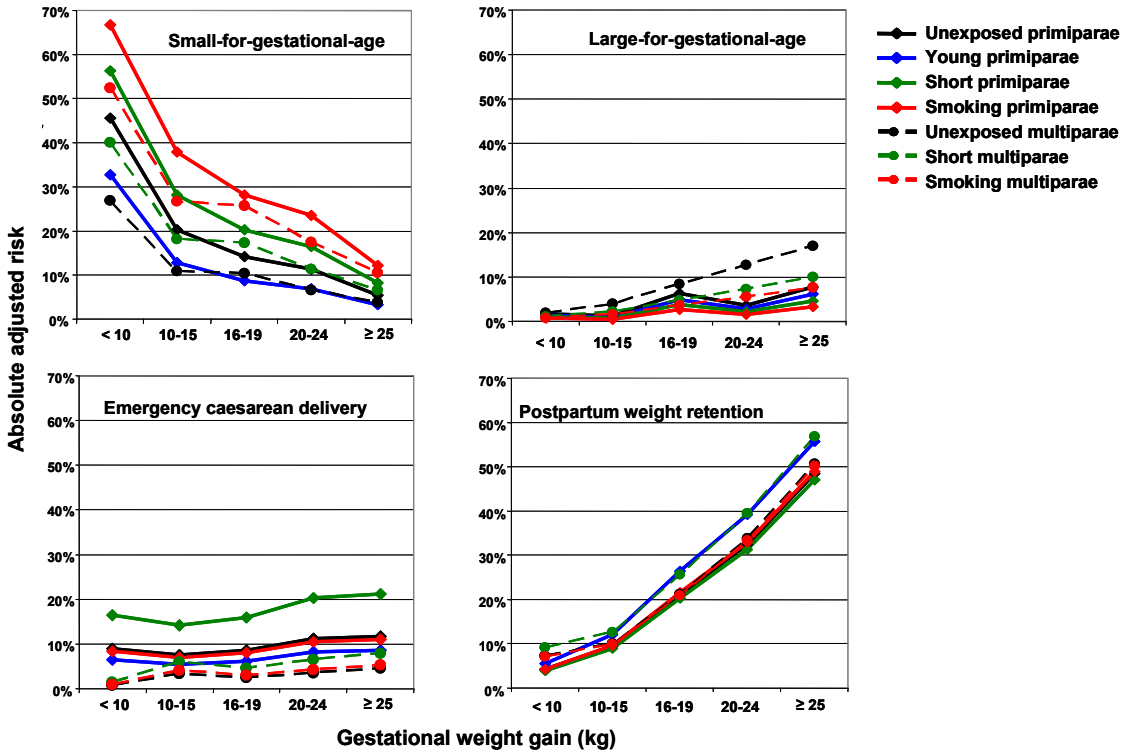

FIGURE 31 GWG-specific risk of pregnancy outcomes in subtypes of underweight women.

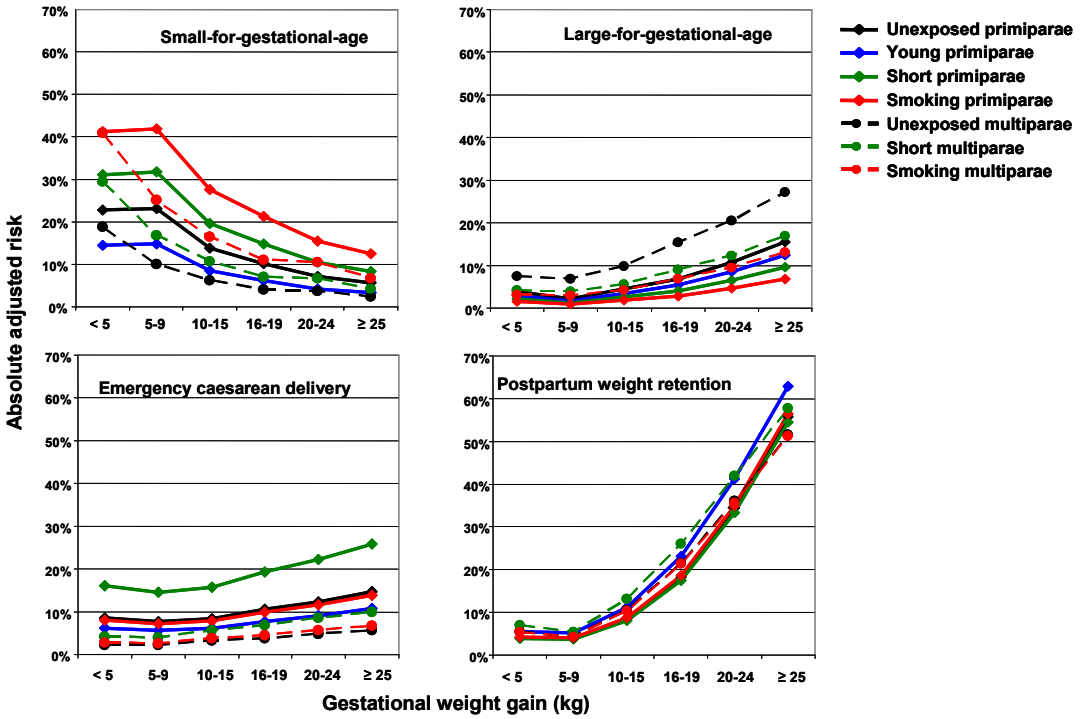

FIGURE 32 GWG-specific risk of pregnancy outcomes in subtypes of normal weight women.

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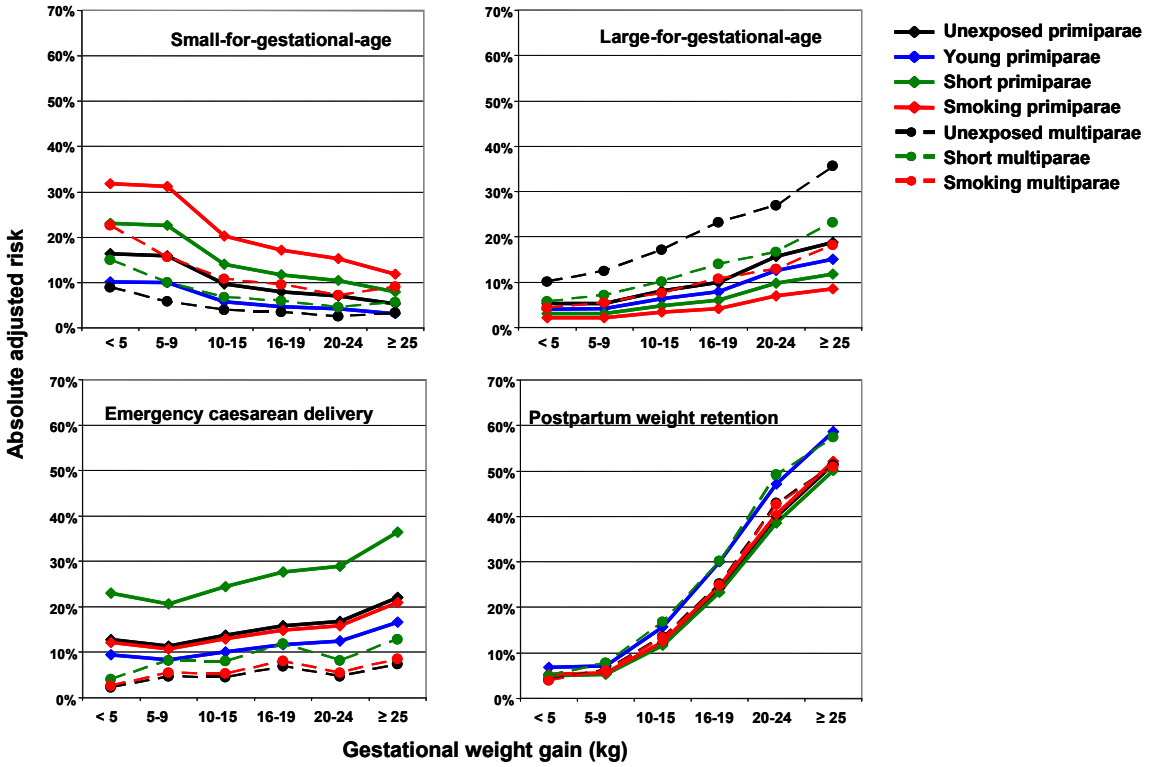

FIGURE 33 GWG-specific risk of pregnancy outcomes in subtypes of overweight women.

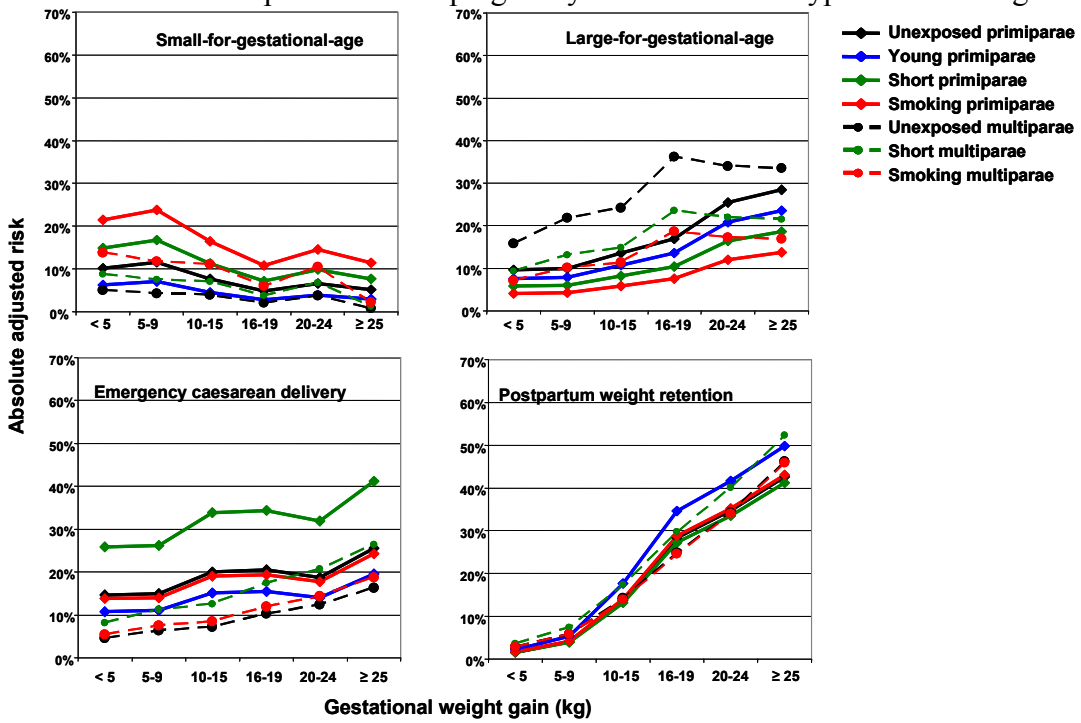

FIGURE 34 GWG-specific risk of pregnancy outcomes in subtypes of obese women.

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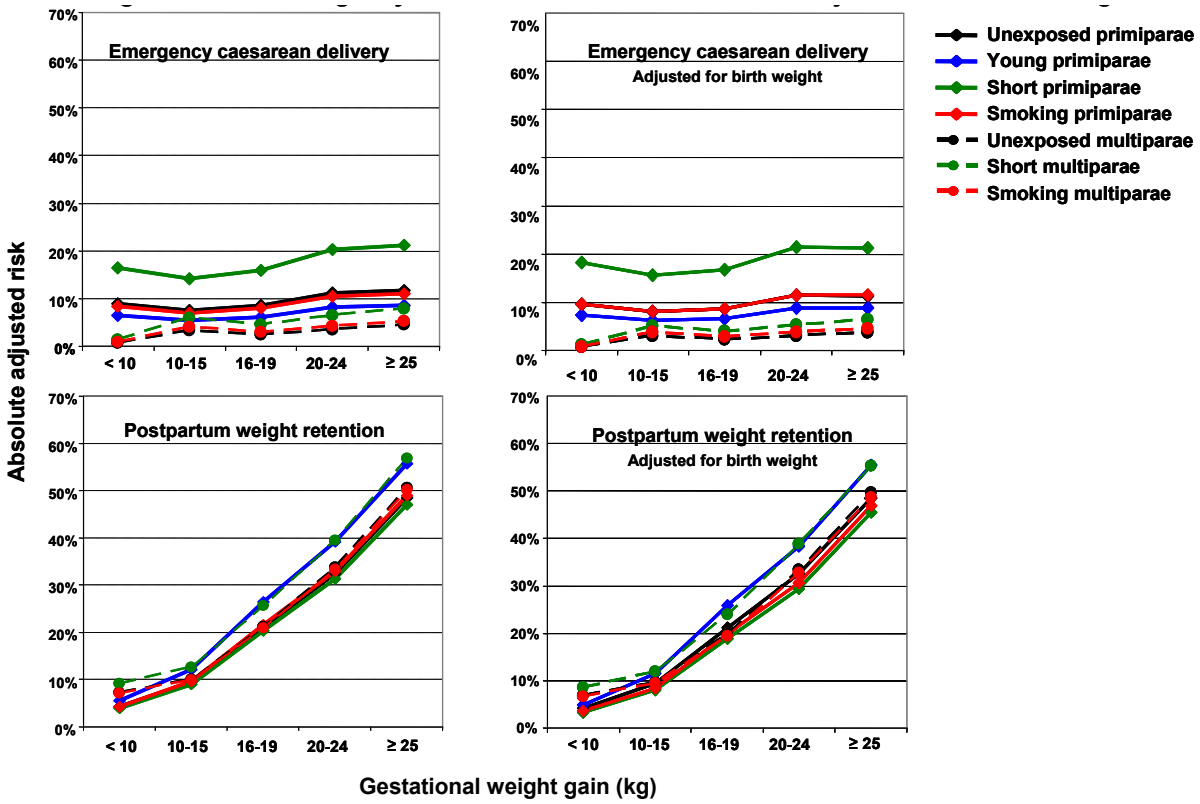

FIGURE 35 Underweight women, emergency cesarean delivery (CS) and postpartum weight retention (PPWR) with and without adjustment for birth weight.
NOTE: Absolute risks in different types of underweight women. When adjusted for birth weight, the presented risk is that of a women giving birth to a 3,500-3,999 g baby.

TABLE 35A Emergency cesarean delivery (CS) in different types of underweight women by GWG

	< 10 kg	10-15 kg	16-19 kg	20-24 kg	≥ 25 kg
Unexposed primipara	0.09	0.08	0.09	0.11	0.12
Young primipara	0.06	0.05	0.06	0.08	0.09
Short primipara	0.16	0.14	0.16	0.20	0.21
Smoking primipara	0.08	0.07	0.08	0.11	0.11
Unexposed multipara	0.01	0.03	0.03	0.04	0.04
Short multipara	0.01	0.06	0.05	0.06	0.08
Smoking multipara	0.01	0.04	0.03	0.04	0.05

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TABLE 35B Emergency cesarean delivery (CS) with adjustment for birth weight in different types of underweight women by GWG

	< 10 kg	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
Unexposed primipara	0.10	0.08	0.09	0.12	0.11
Young primipara	0.07	0.06	0.07	0.09	0.09
Short primipara	0.18	0.16	0.17	0.22	0.21
Smoking primipara	0.10	0.08	0.09	0.12	0.11
Unexposed multipara	0.01	0.03	0.02	0.03	0.04
Short multipara	0.01	0.05	0.04	0.05	0.06
Smoking multipara	0.01	0.04	0.03	0.04	0.05

TABLE 35C Postpartum weight retention (PPWR) in different types of underweight women by GWG

	< 10 kg	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
Unexposed primipara	0.04	0.09	0.21	0.32	0.48
Young primipara	0.06	0.12	0.26	0.39	0.56
Short primipara	0.04	0.09	0.20	0.31	0.47
Smoking primipara	0.04	0.10	0.22	0.33	0.49
Unexposed multipara	0.07	0.10	0.21	0.34	0.51
Short multipara	0.09	0.13	0.26	0.39	0.57
Smoking multipara	0.07	0.10	0.21	0.33	0.50

TABLE 35D Postpartum weight retention (PPWR) with adjustment for birth weight in different types of underweight women by GWG

	< 10 kg	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
Unexposed primipara	0.04	0.09	0.21	0.32	0.48
Young primipara	0.05	0.12	0.26	0.38	0.55
Short primipara	0.03	0.08	0.19	0.29	0.45
Smoking primipara	0.04	0.08	0.20	0.31	0.47
Unexposed multipara	0.07	0.10	0.20	0.33	0.50
Short multipara	0.09	0.12	0.24	0.39	0.55
Smoking multipara	0.07	0.09	0.19	0.33	0.49

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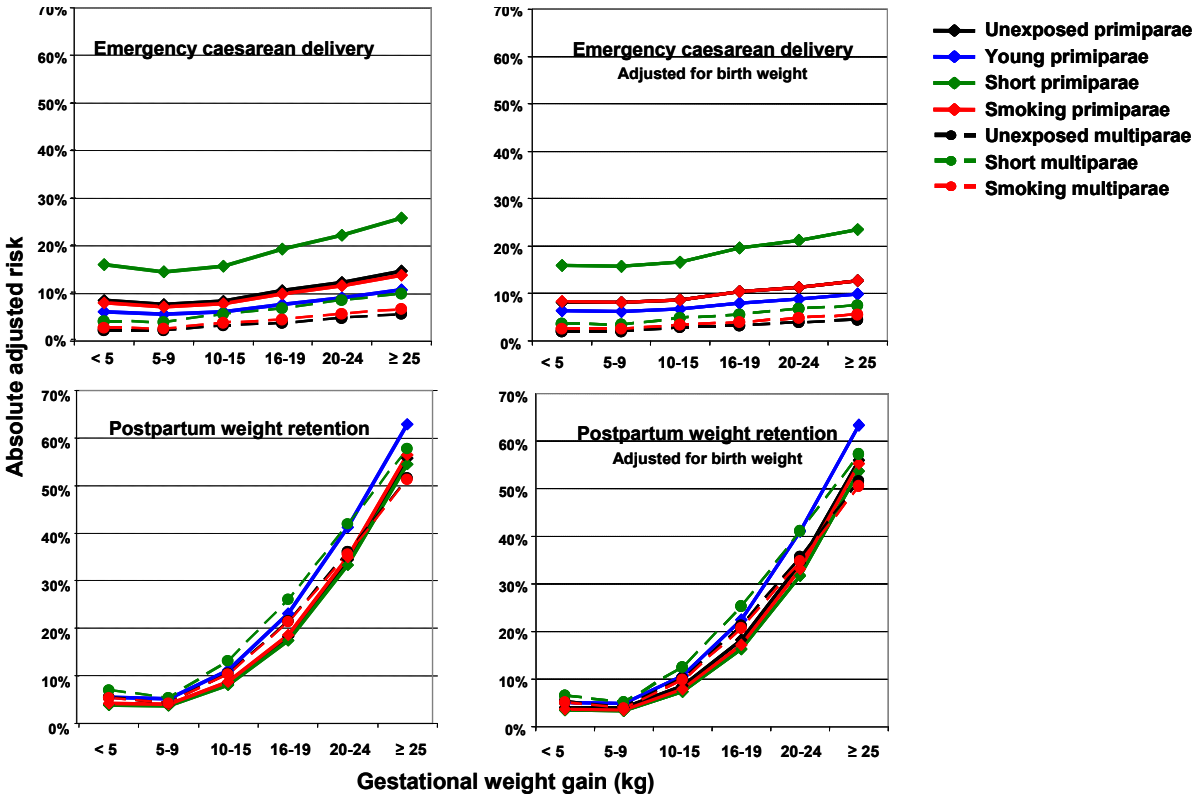

FIGURE 36 Normal weight women, emergency cesarean delivery (CS) and postpartum weight retention (PPWR) with and without adjustment for birth weight.
NOTE: Absolute risks in different types of normal weight women. When adjusted for birth weight, the presented risk is that of a women giving birth to a 3,500-3,999 g baby.

TABLE 36A Emergency cesarean delivery (CS) in different types of normal weight women by GWG

	< 5 kg	5-9 kg	10-15 kg	16-19 kg	20-24 kg	25 + kg
Unexposed primipara	0.09	0.08	0.08	0.11	0.12	0.15
Young primipara	0.06	0.06	0.06	0.08	0.09	0.11
Short primipara	0.16	0.15	0.16	0.19	0.22	0.26
Smoking primipara	0.08	0.07	0.08	0.10	0.12	0.14
Unexposed multipara	0.02	0.02	0.03	0.04	0.05	0.06
Short multipara	0.04	0.04	0.06	0.07	0.09	0.10
Smoking multipara	0.03	0.03	0.04	0.04	0.06	0.07

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TABLE 36B Emergency cesarean delivery (CS) with adjustment for birth weight in different types of normal weight women by GWG

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**25 + kg**
Unexposed primipara	0.08	0.08	0.09	0.10	0.11	0.13
Young primipara	0.06	0.06	0.07	0.08	0.09	0.10
Short primipara	0.16	0.16	0.17	0.20	0.21	0.23
Smoking primipara	0.08	0.08	0.09	0.10	0.11	0.13
Unexposed multipara	0.02	0.02	0.03	0.03	0.04	0.04
Short multipara	0.03	0.03	0.05	0.06	0.07	0.08
Smoking multipara	0.02	0.02	0.03	0.04	0.05	0.05

TABLE 36C Postpartum weight retention (PPWR) in different types of normal weight women by GWG

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**25 + kg**
Unexposed primipara	0.04	0.04	0.09	0.18	0.34	0.56
Young primipara	0.05	0.05	0.11	0.23	0.41	0.63
Short primipara	0.04	0.04	0.08	0.18	0.33	0.54
Smoking primipara	0.04	0.04	0.09	0.19	0.35	0.56
Unexposed multipara	0.05	0.04	0.11	0.22	0.36	0.52
Short multipara	0.07	0.05	0.13	0.26	0.42	0.58
Smoking multipara	0.05	0.04	0.10	0.21	0.36	0.51

TABLE 36D Postpartum weight retention (PPWR) with adjustment for birth weight in different types of normal weight women by GWG

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**25 + kg**
Unexposed primipara	0.04	0.04	0.09	0.18	0.34	0.56
Young primipara	0.05	0.05	0.11	0.23	0.41	0.63
Short primipara	0.03	0.03	0.07	0.16	0.32	0.54
Smoking primipara	0.04	0.04	0.08	0.17	0.33	0.55
Unexposed multipara	0.05	0.04	0.10	0.21	0.36	0.52
Short multipara	0.07	0.05	0.12	0.25	0.41	0.57
Smoking multipara	0.05	0.04	0.10	0.21	0.35	0.51

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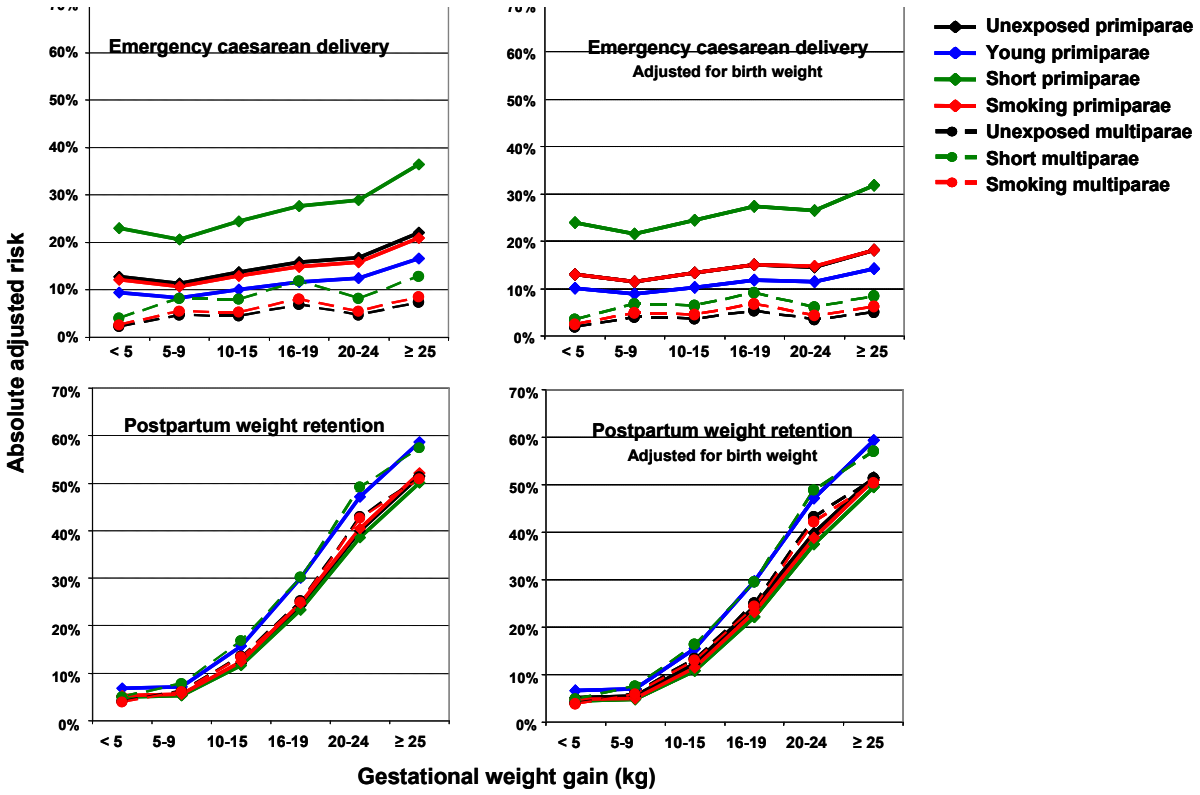

FIGURE 37 Overweight women, emergency cesarean delivery (CS) and postpartum weight retention (PPWR) with and without adjustment for birth weight.
NOTE: Absolute risks in different types of overweight women. When adjusted for birth weight, the presented risk is that of a women giving birth to a 3,500-3,999 g baby.

TABLE 37A Emergency cesarean delivery (CS) in different types of overweight women by GWG

	< 5 kg	5-9 kg	10-15 kg	16-19 kg	20-24 kg	≥ 25 kg
Unexposed primipara	0.13	0.11	0.14	0.16	0.17	0.22
Young primipara	0.09	0.08	0.10	0.12	0.12	0.17
Short primipara	0.23	0.21	0.25	0.28	0.29	0.36
Smoking primipara	0.12	0.11	0.13	0.15	0.16	0.21
Unexposed multipara	0.02	0.05	0.05	0.07	0.05	0.07
Short multipara	0.04	0.08	0.08	0.12	0.08	0.13
Smoking multipara	0.03	0.05	0.05	0.08	0.05	0.09

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TABLE 37B Emergency cesarean delivery (CS) with adjustment for birth weight in different types of overweight women by GWG

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
Unexposed primipara	0.13	0.11	0.13	0.15	0.15	0.18
Young primipara	0.10	0.09	0.10	0.12	0.11	0.14
Short primipara	0.24	0.21	0.24	0.27	0.27	0.32
Smoking primipara	0.13	0.12	0.13	0.15	0.15	0.18
Unexposed multipara	0.02	0.04	0.04	0.05	0.03	0.05
Short multipara	0.03	0.07	0.06	0.09	0.06	0.08
Smoking multipara	0.02	0.05	0.05	0.07	0.04	0.06

TABLE 37C Postpartum weight retention (PPWR) in different types of overweight women by GWG

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
Unexposed primipara	0.05	0.05	0.012	0.24	0.40	0.52
Young primipara	0.07	0.07	0.16	0.30	0.47	0.59
Short primipara	0.05	0.05	0.12	0.23	0.39	0.50
Smoking primipara	0.05	0.06	0.12	0.25	0.40	0.52
Unexposed multipara	0.04	0.06	0.14	0.25	0.43	0.51
Short multipara	0.05	0.08	0.17	0.30	0.49	0.57
Smoking multipara	0.04	0.06	0.13	0.25	0.43	0.51

TABLE 37D Postpartum weight retention (PPWR) with adjustment for birth weight in different types of overweight women by GWG

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
Unexposed primipara	0.05	0.05	0.12	0.24	0.40	0.51
Young primipara	0.07	0.07	0.15	0.30	0.47	0.59
Short primipara	0.05	0.05	0.11	0.22	0.37	0.49
Smoking primipara	0.05	0.05	0.12	0.23	0.39	0.51
Unexposed multipara	0.04	0.06	0.13	0.25	0.43	0.51
Short multipara	0.05	0.08	0.16	0.29	0.49	0.57
Smoking multipara	0.04	0.06	0.13	0.24	0.42	0.50

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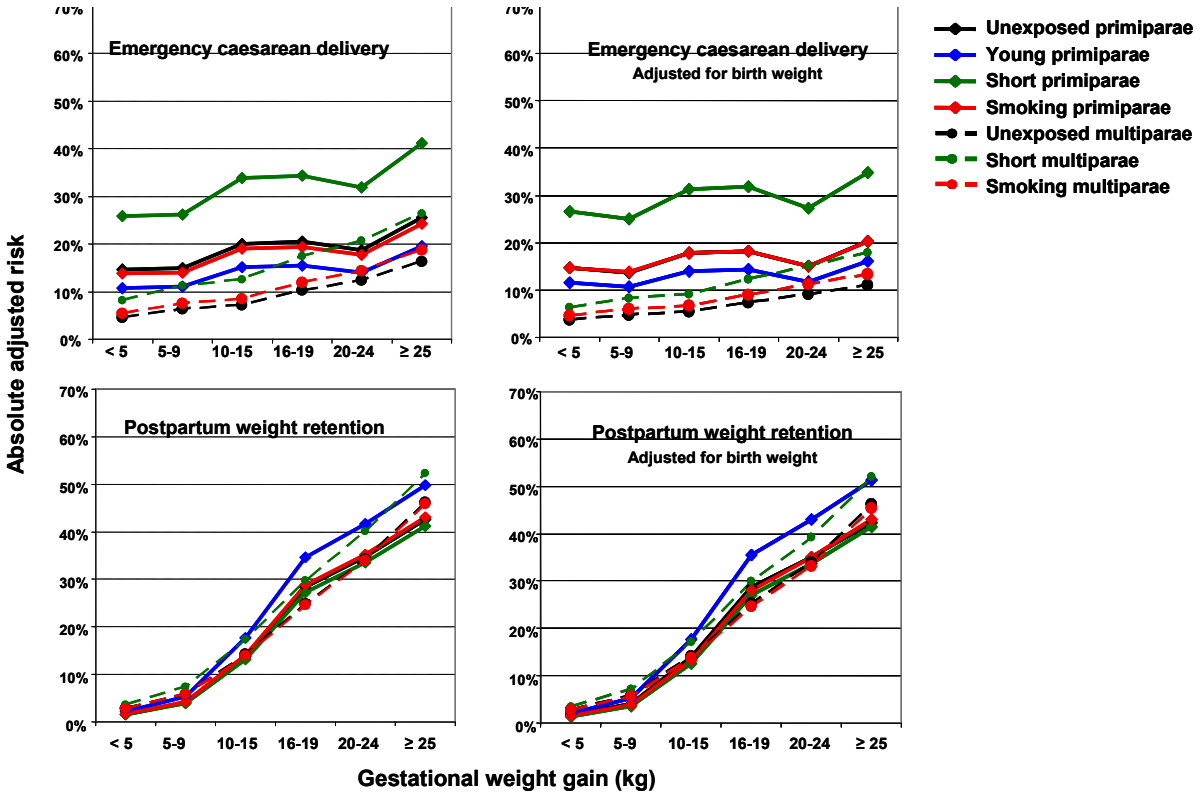

FIGURE 38 Obese women, emergency cesarean delivery (CS) and postpartum weight retention (PPWR) with and without adjustment for birth weight.
NOTE: Absolute risks in different types of obese women. When adjusted for birth weight, the presented risk is that of a women giving birth to a 3500-3999 g baby.

TABLE 38A Emergency cesarean delivery (CS) in different types of obese women by GWG

	< 5 kg	5-9 kg	10-15 kg	16-19 kg	20-24 kg	≥ 25 kg
Unexposed primipara	0.15	0.15	0.20	0.21	0.19	0.26
Young primipara	0.11	0.11	0.15	0.15	0.14	0.20
Short primipara	0.26	0.26	0.34	0.34	0.32	0.41
Smoking primipara	0.14	0.14	0.19	0.19	0.18	0.24
Unexposed multipara	0.05	0.06	0.07	0.10	0.12	0.16
Short multipara	0.08	0.11	0.13	0.17	0.21	0.26
Smoking multipara	0.05	0.07	0.08	0.12	0.14	0.19

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TABLE 38B Emergency cesarean delivery (CS) with adjustment for birth weight in different types of obese women by GWG

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
Unexposed primipara	0.15	0.14	0.018	0.18	0.15	0.20
Young primipara	0.11	0.11	0.14	0.14	0.12	0.16
Short primipara	0.27	0.25	0.31	0.32	0.27	0.35
Smoking primipara	0.15	0.14	0.18	0.18	0.15	0.20
Unexposed multipara	0.04	0.05	0.05	0.07	0.09	0.11
Short multipara	0.06	0.08	0.09	0.12	0.15	0.18
Smoking multipara	0.05	0.06	0.07	0.09	0.11	0.13

TABLE 38C Postpartum weight retention (PPWR) in different types of obese women by GWG

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
Unexposed primipara	0.02	0.04	0.14	0.28	0.35	0.43
Young primipara	0.02	0.05	0.18	0.35	0.42	0.50
Short primipara	0.02	0.04	0.13	0.27	0.34	0.41
Smoking primipara	0.02	0.04	0.14	0.29	0.35	043
Unexposed multipara	0.03	0.06	0.14	0.25	0.34	0.46
Short multipara	0.04	0.07	0.17	0.30	0.40	0.52
Smoking multipara	0.03	0.06	0.14	0.25	0.34	0.46

TABLE 38D Postpartum weight retention (PPWR) with adjustment for birth weight in different types of obese women by GWG

	< 5 kg	**5-9 kg**	**10-15 kg**	**16-19 kg**	**20-24 kg**	**≥ 25 kg**
Unexposed primipara	0.02	0.04	0.14	0.29	0.35	0.42
Young primipara	0.02	0.05	0.18	0.35	0.43	0.51
Short primipara	0.01	0.04	0.13	0.27	0.34	0.42
Smoking primipara	0.02	0.04	0.13	0.28	0.35	0.43
Unexposed multipara	0.03	0.06	0.14	0.25	0.34	0.46
Short multipara	0.03	0.07	0.17	0.30	0.39	0.52
Smoking multipara	0.03	0.06	0.14	0.25	0.33	0.45

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PART II: ANALYSES FROM DR. HERRING

ASSOCIATION BETWEEN GESTATIONAL WEIGHT CHANGES AND ADVERSE PREGNANCY OUTCOMES IN THE 1988 NATIONAL MATERNAL AND INFANT HEALTH SURVEY AND 1991 FOLLOW-UP SURVEY

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Improvement of maternal, fetal, and child health are key public health goals. In an effort to achieve these objectives, the Institute of Medicine (IOM) report *Nutrition During Pregnancy* offered recommendations in 1990 for weight gain during pregnancy based on pre-pregnancy maternal body mass index (BMI). Since publication of the IOM reports, the population of U.S. women of childbearing age has become more diverse. New health concerns have arisen, including the greater prevalence of women who are overweight or obese entering pregnancy, which puts them at high risk for pregnancy complications. More women are becoming pregnant at an older age and enter pregnancy with chronic conditions such as type 2 diabetes, which also puts them at risk for pregnancy complications and may lead to increased morbidity during their post-pregnancy years. In addition to adverse outcomes for the mother, there are risks for the child associated with gestational weight gain outside recommended levels.

The Committee to Reexamine IOM Pregnancy Weight Guidelines requested an analysis based on the 1988 National Maternal and Infant Health Study and its 1991 longitudinal follow-up. Data from the 1988 National Maternal and Infant Health Survey (NMIHS) and its 1991 longitudinal follow-up study were used to generate

- Descriptions of gestational weight gain distributions in the general population as well as in specific subgroups of interest
- Descriptions of distributions of pregnancy, birth, and maternal and child health outcomes, including cesarean delivery, preterm birth, birth weight among term births, small for gestational age, large for gestational age, breastfeeding initiation, duration of breastfeeding, postpartum weight retention, and childhood weight status
- Results from statistical modeling of relationships between gestational weight gain, pregravid body mass index, and outcomes of interest
- Predictions from these outcomes based on weight gain scenarios, including current (observed in data) gain, gain according to the current IOM recommendations, and gain according to proposed recommendations
- Outcome risk estimates, averaged over other exposures, by pregravid BMI and adequacy of weight gain

Women included in the analysis had singleton pregnancies ending in live births as defined by NMIHS (NMIHS distinguishes live births from fetal and infant deaths). Due to the presence of numerous extreme outliers, data were cleaned by excluding (1) subjects with birth weights

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further than three standard deviations from the mean birth weight for each gestational age at delivery, (2) subjects with gestational weight gain greater than 40 kg or with gestational weight loss greater than -10 kg, and (3) deliveries before 26 weeks gestation nor after 42 weeks gestation. Due to poor quality of data on GDM, PIH, and preeclampsia, these outcomes were not analyzed in further detail.

Gestational Weight Gain

Gestational weight gain in NMIHS is available from either maternal self-report at the time of questionnaire (mean 17 months postpartum with range 6 to 31 months) or from medical care provider report. For these analyses, medical care provider was used when available, and maternal self-report was used when provider report was unavailable. Pregravid weight, used to calculate gestational weight gain, was largely based on self-reported data unless the provider reported a measured pregravid weight (this is possible but not indicated in the data set). In addition, gestational age at delivery is based on vital records data and is not of uniform quality; there are numerous cases of extreme outliers in birth weight that may be due to incorrect pregnancy dating. Birth weight was thus cleaned by eliminating observations more than three standard deviations from the mean birth weight at each gestational age week.

The original gestational weight gain variable has mean 30.5 pounds and ranged from 217 pounds lost to 235 pounds gained. For purposes of this analysis, data were cleaned by excluding the top 1 percent and bottom 1 percent of this variable. The resulting variable had range limited to 22 pounds lost to 79 pounds gained. The (unweighted) empirical density of weight gain is presented in Figure 1; 29 percent of women had inadequate gain; 26 percent of women had adequate gain, and 45 percent of women had excessive gain based on the current IOM recommendations for weight gain and WHO cutoffs for BMI.

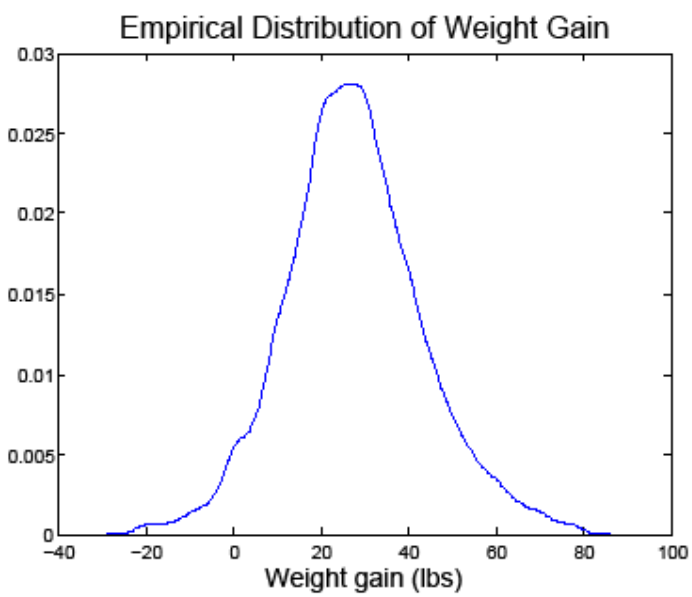

FIGURE 1 Empirical distribution of weight gain in NMIHS

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Weight gain adequacy was related to pregravid BMI category, as described below in Table 1. In particular, underweight women tended to have inadequate or adequate gain, while the majority of normal weight, overweight, and obese women had excessive gain. Interestingly, fewer overweight women had inadequate gain than women in any other group.

In all analysis models, predicted outcomes are obtained for the following three scenarios.

1. Observed weight gain
2. Weight gain according to the IOM (1990) recommendations
3. Weight gain as indicated by the Oken et al. (2008) analysis

In order to determine whether weight gain was according to the current IOM recommendations, women were classified into one of four pregravid BMI groups. Within each BMI group, the current IOM recommended weight gain range at 40 weeks was linearly extrapolated (after accounting for recommended first trimester gain) to a range at each week of gestation, so that each woman could be classified as having adequate weight gain (within the IOM recommended range), inadequate gain, or excessive gain, specific to her pregravid BMI and the gestational age of her child at delivery. Scenario 2 was created by first determining whether a woman's weight gain was adequate or not. For women with adequate gain (that is, gain within the recommended range), weight gain values were unaltered. For women with inadequate or excessive gain, a new gestational weight gain was randomly sampled from a uniform distribution on the IOM recommended weight gain range specific to her pregravid BMI and gestational week at delivery. Scenario 3 was created by taking the Oken et al.(2008) values based on the IOM (1990) recommended first trimester gain, extrapolating them to gestational ages other than 40 weeks. Then all women were assigned to the exact weight gain recommended specific to that gestational week and pregravid BMI.

TABLE 1 Adequacy of Weight Gain (Current IOM Guidelines) by Pregravid BMI (WHO Cutoffs)

	Weight Gain Adequacy (%)		
Pregravid BMI	**Inadequate**	**Adequate**	**Excessive**
Underweight	33.7	41.2	25.1
Normal	29.8	28.7	41.5
Overweight	19.4	18.8	61.8
Obese	32.9	7.7	59.5

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Overall risk estimates of outcomes of interest are presented in Figure 2.

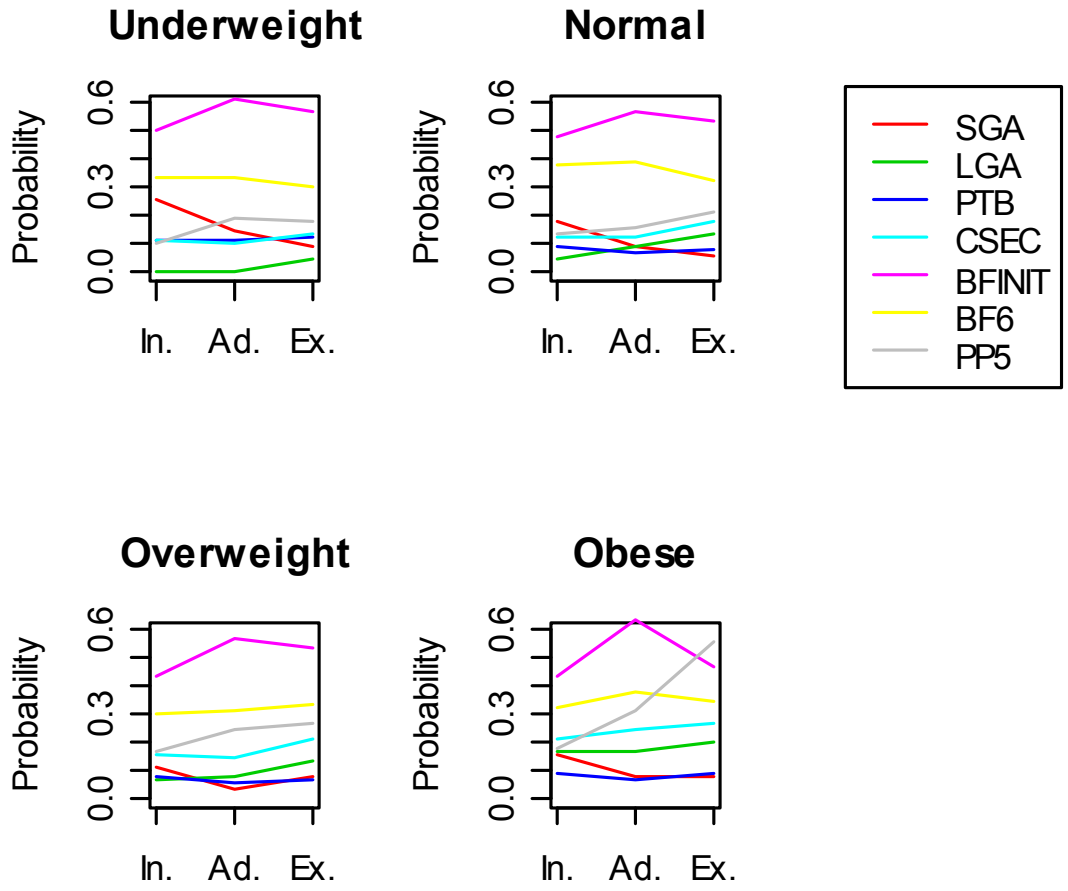

FIGURE 2 Risks, by NHLBI BMI and IOM weight gain (inadequate, adequate, excessive) categories, of SGA, LGA, PTB, cesarean delivery, breastfeeding initiation (BFI), breastfeeding 6 months among initiators (BF6), and postpartum weight retention > 5kg (PP5).

Cesarean Delivery

Analysis was limited to women who had not had a prior cesarean delivery. Predictors were selected in the logistic regression model based on backward selection, with the following predictors retained in the final model: maternal pregravid BMI (WHO categories), maternal weight gain, maternal race (black versus non-black), maternal height (< 63 in, 63-66 in, ≥ 67 in), maternal age (< 20 years, 20-24 years, 25-29 years, 30-34 years, ≥ 35 years), maternal smoking during 12 months prior to delivery (none, 1-10 cigarettes per day, > 10 cigarettes per day), maternal employment during pregnancy, parity (multiparous versus nulliparous), gestational age at delivery (linear) and birth weight.

While weight gain was significantly related to cesarean delivery probability, this relationship was not very precise, as illustrated in Figure 3.

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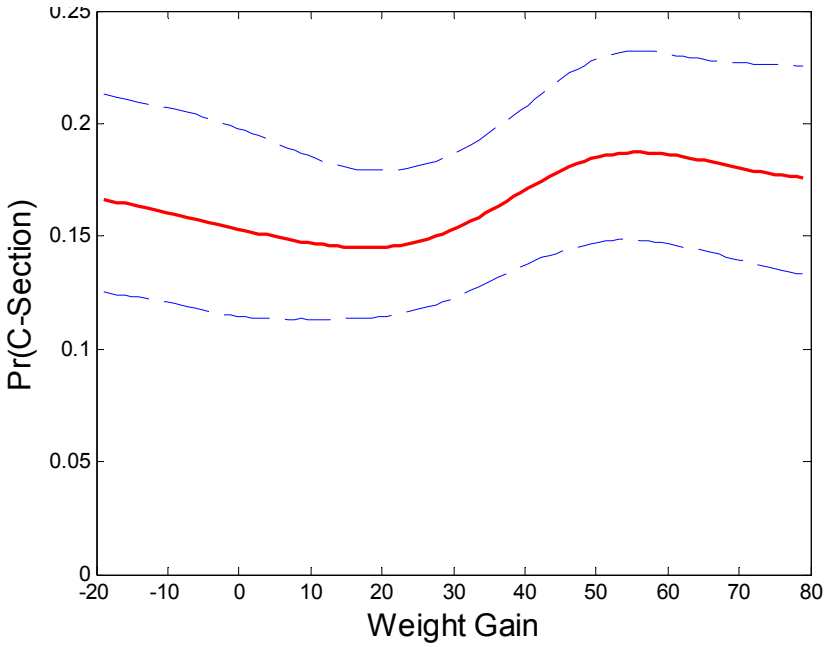

FIGURE 3 Weight gain (lbs) and probability of cesarean delivery.

The probability of cesarean delivery did vary across recommendations, with a probability of 0.23 (0.22, 0.24) in the observed data, of 0.25 (0.21, 0.30) under the IOM recommendations, and 0.25 (0.21, 0.29) under the alternate recommendations. Predicted probabilities by pregravid BMI are presented in Table 2.

TABLE 2 Predicted cesarean delivery Probabilities by Pregravid BMI

	Cesarean Delivery Probabilities (95% CI)		
Pregravid BMI	**Observed Data**	**IOM Gain**	**Oken Gain**
Underweight	0.17 (0.14, 0.21)	0.19 (0.14, 0.25)	0.19 (0.14, 0.25)
Normal weight	0.21 (0.19, 0.22)	0.22 (0.19, 0.28)	0.22 (0.19, 0.28)
Overweight	0.30 (0.27, 0.33)	0.32 (0.26, 0.38)	0.29 (0.25, 0.34)
Obese	0.35 (0.30, 0.40)	0.38 (0.32, 0.46)	0.38 (0.32, 0.46)

Preterm Birth

Preterm birth was defined as birth before 37 completed weeks of gestation, with duration of gestation obtained from the vital record. Predictors were selected in a logistic regression model based on backward selection, and the following were included in the final model: maternal pregravid BMI (WHO categories), maternal weight gain rate, maternal race (black versus non-black), education (< 12 years, 12 years, 13-15 years, 16 or more years), maternal height (< 63 in, 63-66 in, ≥ 67 in), maternal age (< 20 years, 20-24 years, 25-29 years, 30-34 years, ≥ 35 years), and maternal smoking during 12 months prior to delivery (none, 1-10 cigarettes per day, > 10 cigarettes per day).

Clearly, weight gain will be greater for longer pregnancies, so a relationship between lower gains and higher preterm birth probability should be apparent. In this model, the rate of weight gain per week was used as the predictor of interest in an attempt to control for the known relationship between weight gain and duration of gestation. This relationship is depicted in Figure 4, which shows the preterm birth probability as a function of gestational weight gain for

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women of normal pregravid BMI who were non-black, college educated, 5'3"-5'6", 20-24 years old, and nonsmokers. The relationship between rate of weight gain and preterm birth was not statistically significant.

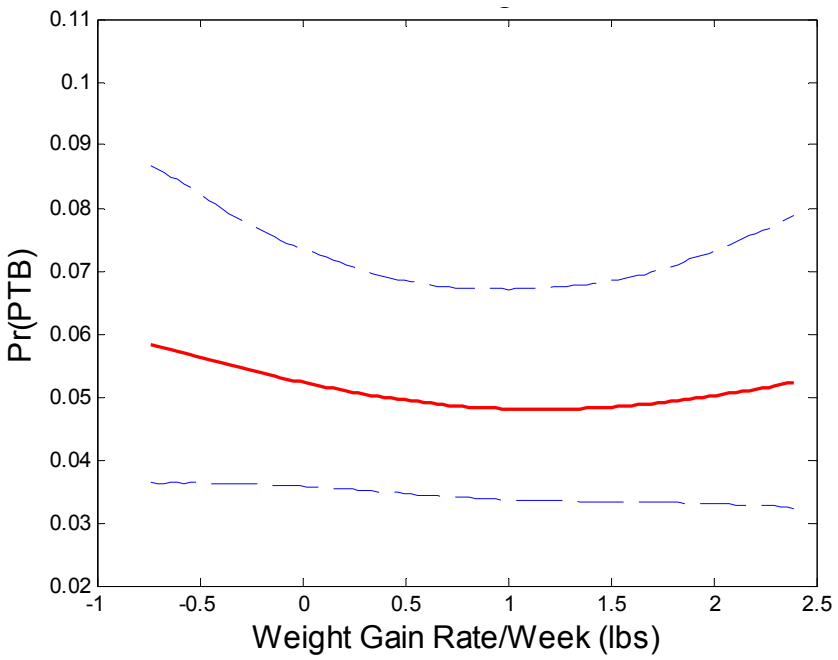

FIGURE 4 Relationship of weight gain to preterm birth probability

Preterm birth was not strongly associated with suggested changes in weight gain. In the observed data, the predicted probability of preterm birth is 0.08 (0.08, 0.10), while under the current IOM recommendations and Oken recommendations, the predicted probability is 0.08 (0.08, 0.99). Preterm birth probabilities by pregravid BMI are below.

TABLE 3 Predicted Probabilities of Preterm Birth by Pregravid BMI

	Preterm Probabilities (95% CI)		
Pregravid BMI	**Observed Data**	**IOM Gain**	**Oken Gain**
Underweight	0.11 (0.09, 0.14)	0.11 (0.09, 0.14)	0.11 (0.09, 0.14)
Normal weight	0.08 (0.07, 0.09)	0.08 (0.07, 0.09)	0.08 (0.07, 0.09)
Overweight	0.07 (0.05, 0.08)	0.07 (0.05, 0.08)	0.07 (0.05, 0.09)
Obese	0.08 (0.06, 0.11)	0.08 (0.06, 0.11)	0.09 (0.06, 0.13)

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Birth Weight Among Term Births

Birth weight was analyzed among births ranging from 37 to 42 weeks gestation. Due to numerous outliers even after cleaning, the regression model used was not the traditional normal (Gaussian) regression model but a regression based on t distributed outcomes with degrees of freedom estimated in the modeling procedure. T-regression is much less sensitive to outliers and was used to avoid trimming the outcome data based eliminating observations in the tails of the birth weight distribution. Predictors were selected in the birth weight regression model based on backward selection; predictors retained in the final model include gestational age at delivery, maternal pregravid BMI (WHO categories), maternal weight gain, maternal race (black versus white), education (< 12 years, 12 years, 13-15 years, 16 or more years), maternal height (< 63 in, 63-66 in, ≥ 67 in), maternal smoking during 12 months prior to delivery (none, 1-10 cigarettes per day, > 10 cigarettes per day), parity (multiparous versus nulliparous), infant gender, and the interaction between pregravid BMI and weight gain.

The association between weight gain and birth weight among terms is illustrated in Figure 5. Among underweight and normal weight women, in the range of (5, 55) pounds gained among normal weight women, birth weight steadily increases, and then birth weight declines slightly after around 55 pounds gained. This trend flattens among overweight and obese women so that there is less association between gestational weight gain and birth weight.

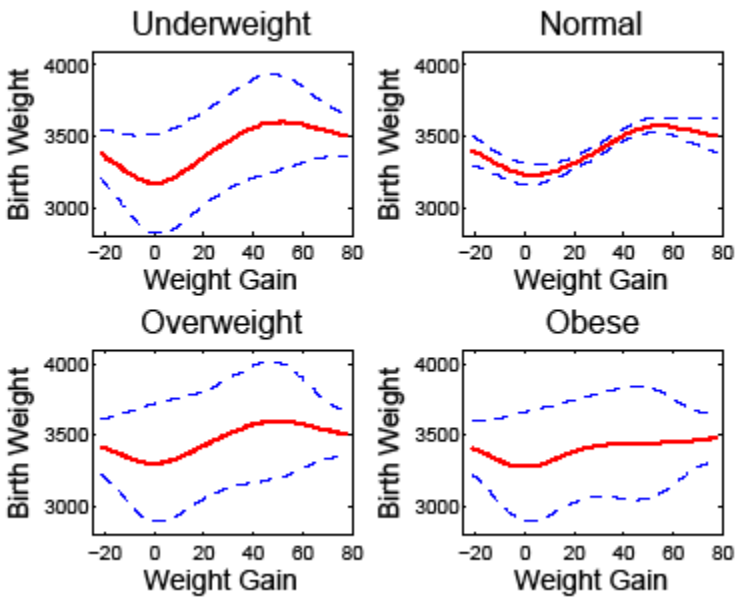

FIGURE 5 Birth weight by weight gain (lbs)

Figure 6 presents the estimated birth weight density among term births observed in the NMIHS data (blue curve and confidence bands); among term births assuming compliance to current IOM recommendations (red); and among term births assuming compliance to the Oken et al. (2008) values. When analysis was restricted to smokers, we saw the same general trends with respect to weight gain, though mean birth weights were lower in this group, as expected.

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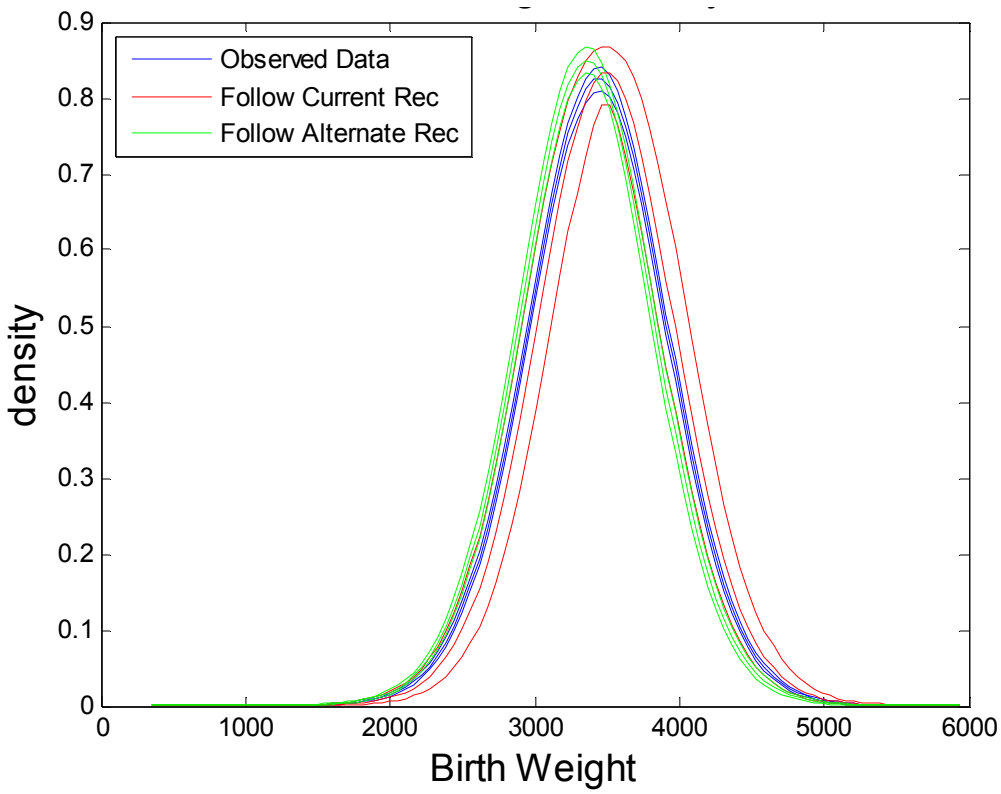

FIGURE 6 Birth weight density, predicted birth weight distribution by hypothetical weight gain

Small-for-Gestational Age

Analysis of small-for-gestational age births involved white and black infants born in the range of 24-42 completed weeks of gestation. The exclusion of other infants is due to the lack of known standards for determining SGA status. The Zhang and Bowes (1995) criteria were used for determining SGA status. Predictors were selected in the SGA logistic regression model based on backward selection; predictors retained in the final model include maternal pregravid BMI (WHO categories), maternal weight gain, maternal race (black versus non-black), maternal education (< 12 years, 12 years, 13-15 years, 16+ years) maternal height (< 63 in, 63-66 in, ≥ 67 in), maternal age (< 20 years, 20-24 years, 25-29 years, 30-34 years, ≥ 35 years), maternal smoking in 12 months prior to delivery (none, 1-10 cigarettes per day, > 10 cigarettes per day), maternal exercise during pregnancy, gestational age, maternal employment during pregnancy, and the following interactions: pregravid BMI by weight gain, race by weight gain, race by maternal height, race by maternal age, and race by exercise. As illustrated in Figures 7 and 8, weight gain was significantly associated with SGA risk. Non-black women who were underweight, normal weight, or overweight were somewhat more likely to have a SGA birth if their weight gain was inadequate. The association between weight gain and SGA risk was considerably muted as pregravid BMI increased.

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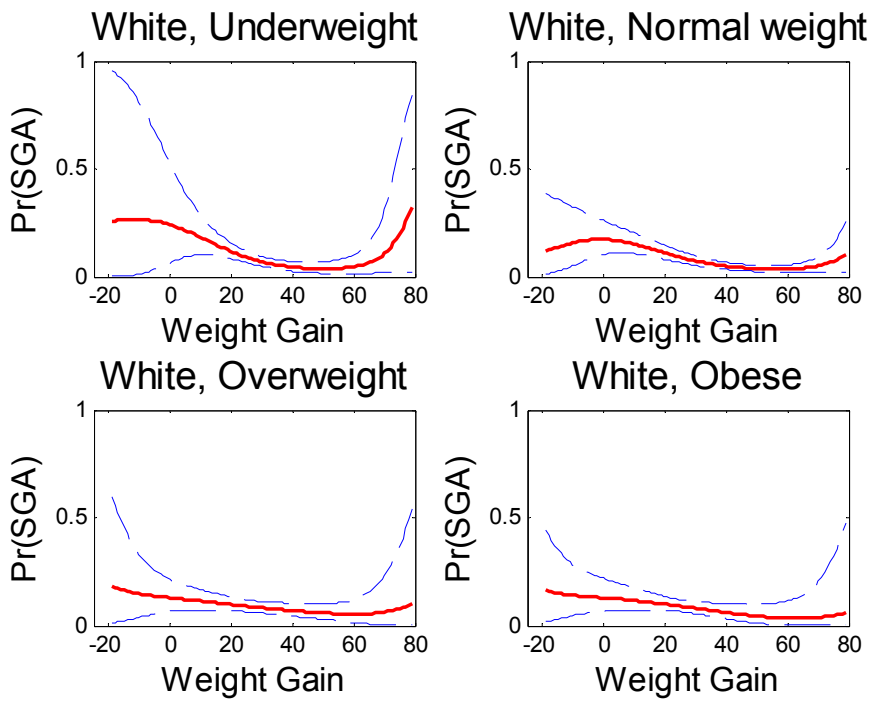

FIGURE 7 SGA risk among white women by weight gain (lbs) and pregravid BMI

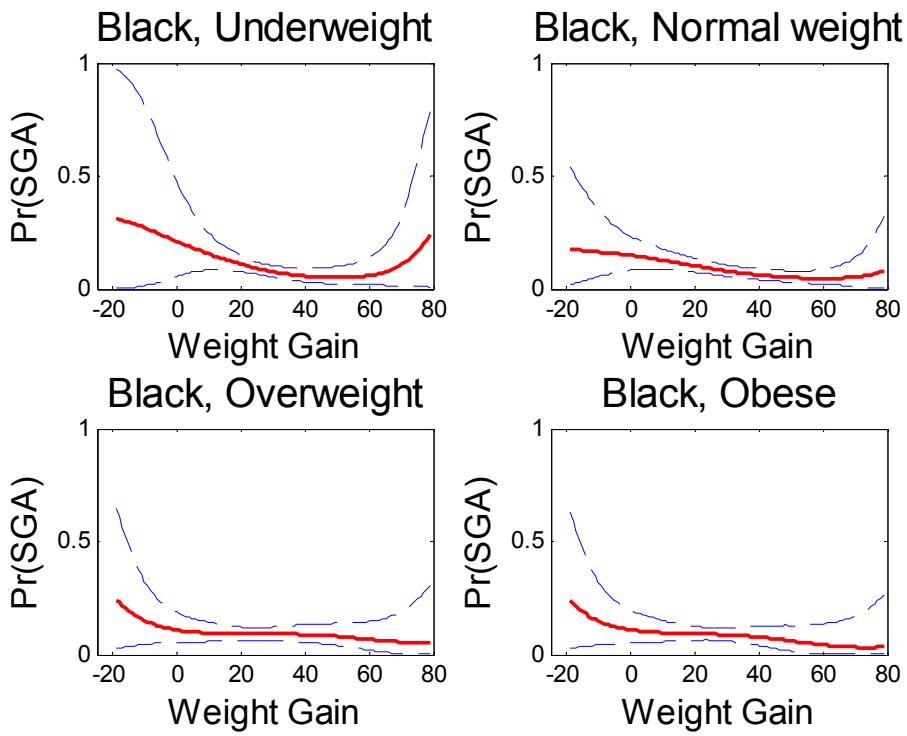

FIGURE 8 Risk of SGA birth in black women by weight gain (lbs) and pregravid BMI

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The SGA density does vary slightly across weight gain recommendations. Using the observed data, 11 percent (10 percent, 12 percent) of births are SGA. Under the IOM recommendations, 11 percent (10 percent, 12 percent) of births are SGA. Under the alternate values, 13 percent (12 percent, 16 percent) of births are SGA. Probabilities of SGA birth by pregravid BMI categories are below in Table 4.

TABLE 4 Predicted Probabilities of SGA Birth by Pregravid BMI

	SGA Probabilities (95% CI)		
Pregravid BMI	**Observed Data**	**IOM Gain**	**Oken Gain**
Underweight	0.17 (0.14, 0.19)	0.14 (0.11, 0.18)	0.19 (0.15, 0.21)
Normal weight	0.11 (0.10, 0.12)	0.11 (0.10, 0.12)	0.12 (0.11, 0.13)
Overweight	0.09 (0.07, 0.11)	0.10 (0.08, 0.12)	0.12 (0.07, 0.20)
Obese	0.10 (0.08, 0.13)	0.11 (0.08, 0.15)	0.17 (0.04, 0.35)

Large-for-Gestational Age

Zhang and Bowes (1995) cutoffs were used to determine LGA status. Predictors were selected in the LGA logistic regression model based on backward selection. Predictors retained in the final model include maternal pregravid BMI (WHO categories), maternal weight gain, maternal race (black versus non-black), maternal height (< 63 in, 63-66 in, ≥ 67 in), maternal age (<20 years, 20-24 years, 25-29 years, 30-34 years, ≥ 35 years), maternal smoking during 12 months prior to delivery (none, 1-10 cigarettes per day, > 10 cigarettes per day), gestational age at delivery, and the following two-way interactions: pregravid BMI by weight gain, race by pregravid BMI, race by weight gain, race by height, and race by smoking.

The probability of LGA birth is associated with weight gain, though interval estimates are wide. Figures 9 and 10 show this probability as a function of race and pregravid BMI.

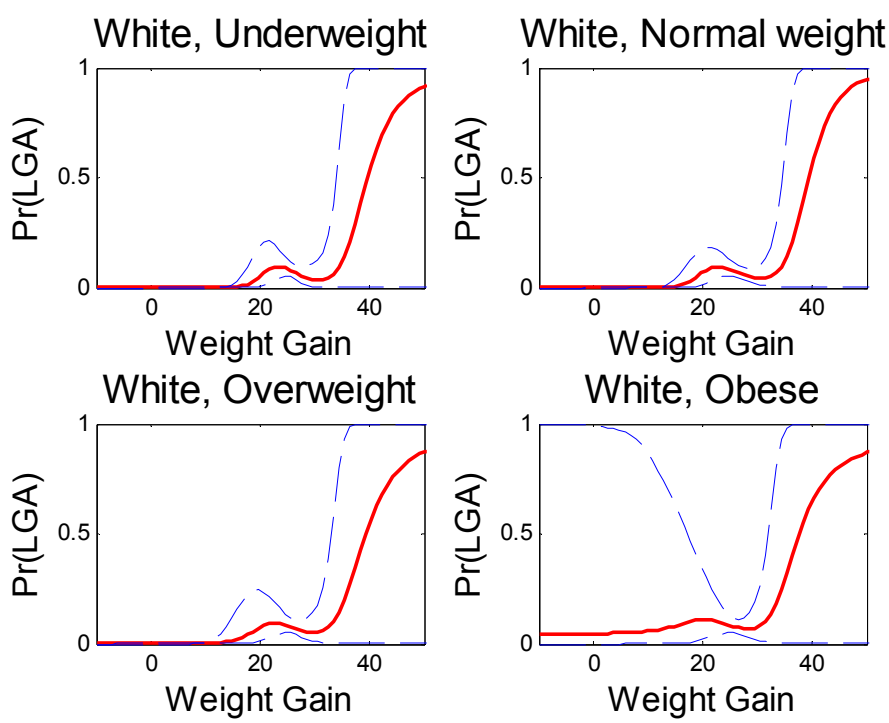

FIGURE 9 Probability of LGA birth by pregravid BMI and weight gain (lbs) in whites

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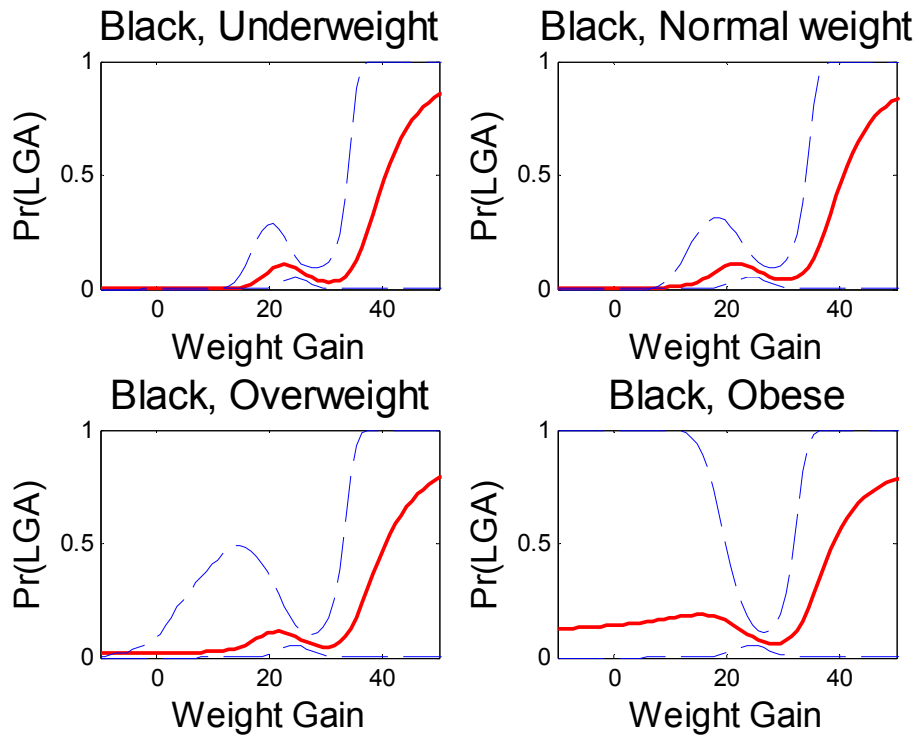

FIGURE 10 Probability of LGA birth by BMI and weight gain (lbs) in blacks

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The LGA density does vary slightly across weight gain recommendations. Using the observed data, 11 percent (10 percent, 11 percent) of births are LGA. Under the IOM (1990) recommendations, 8 percent (8 percent, 9 percent) of births are LGA. Under the alternate recommendations, 8 percent (7 percent, 9 percent) of births are LGA. Predicted probabilities of LGA by pregravid BMI category are in Table 5.

TABLE 5 Predicted Probabilities of LGA Birth by Pregravid BMI

	LGA Probabilities (95% CI)		
Pregravid BMI	**Observed Data**	**IOM Gain**	**Oken Gain**
Underweight	0.05 (0.03, 0.07)	0.04 (0.02, 0.06)	0.03 (0.01, 0.04)
Normal weight	0.10 (0.09, 0.12)	0.08 (0.07, 0.09)	0.07 (0.06, 0.08)
Overweight	0.12 (0.10, 0.14)	0.08 (0.06, 0.10)	0.10 (0.06, 0.14)
Obese	0.19 (0.15, 0.22)	0.16 (0.12, 0.19)	0.17 (0.12, 0.23)

Breastfeeding

Breastfeeding initiation and duration were not associated with pregnancy weight gain after confounder adjustment. While point estimates of the probabilities of initiation and of breastfeeding 6 months among initiators are provided in Figure 2, the interval estimates about these probabilities are quite wide. Analysis of these outcomes is not included due to space consideration (available upon request).

Postpartum Weight Retention

The quality of postpartum weight retention depends on the quality of the pregravid weight. Women self-reported postpartum weight on the questionnaire, which was administered at 6-31 months postpartum. The first analysis was of postpartum weight retention among only those subjects queried at 6-12 months postpartum. Among this subset, mean retention was 6.7 pounds.

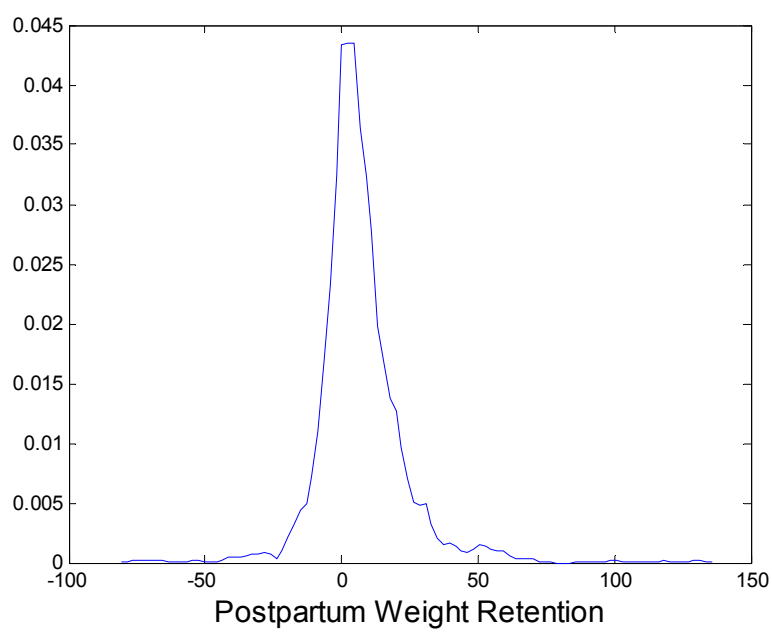

FIGURE 11 Postpartum weight retention, 6-12 months

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Due to numerous outliers, a regression based on t-distributed outcomes with degrees of freedom estimated in the modeling procedure was used. Predictors were selected in the postpartum weight retention regression model based on backward selection, with the following predictors in the final model: postpartum month of survey, maternal pregravid BMI (WHO categories), maternal weight gain, maternal race (black versus white), education (< 12 years, 12 years, 13-15 years, 16 or more years), maternal age (< 20 years, 20-24 years, 25-29 years, 30-34 years, ≥ 35 years), maternal smoking during postpartum (none, 1-10 cigarettes per day, >10 cigarettes per day), parity (multiparous versus nulliparous), duration of gestation, breastfeeding duration, and interactions between pregravid BMI and weight gain, pregravid BMI and race, pregravid BMI and parity, and pregravid BMI and month postpartum of survey. As illustrated in Figure 12, obese women tended to report more postpartum weight retention. Across all pregravid BMI groups, weight retention only seemed to increase substantially with weight gains greater than 20 pounds.

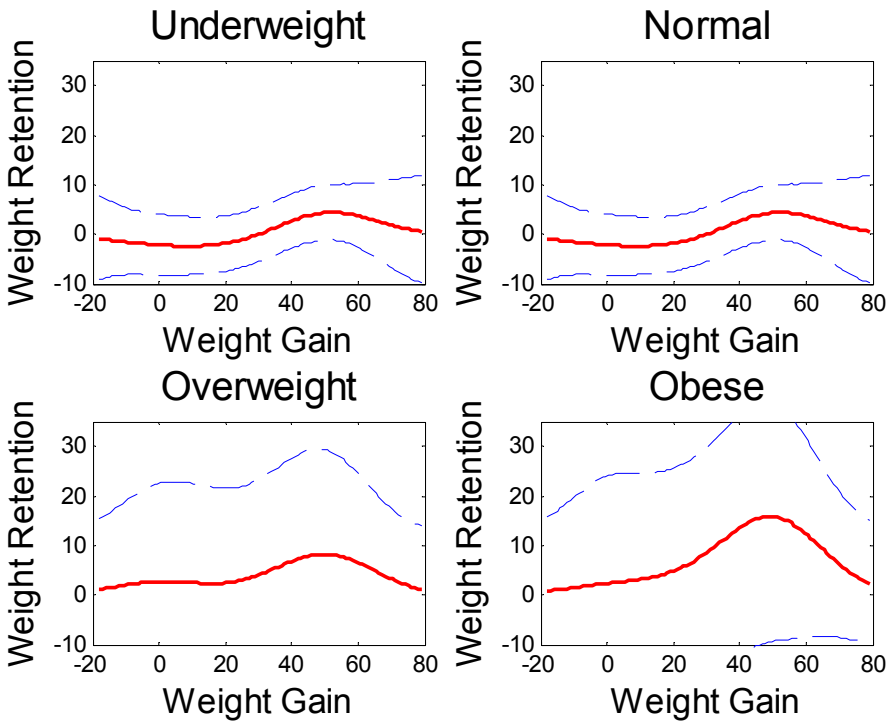

FIGURE 12 Postpartum weight retention (lbs), 6-12 months, by pregravid BMI and weight gain (lbs)

Predicted weight retention is subject to considerable uncertainty due to the relatively small sample size (n = 1,157) with reported postpartum weight in the 6-12 month interval.

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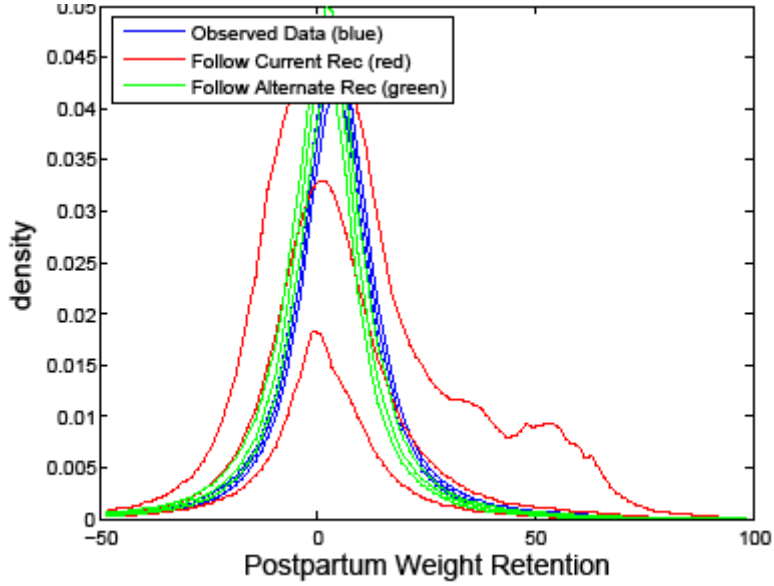

FIGURE 13 Predicted density of postpartum weight retention, 6-12 months

Child Weight Status at Age 3

Data access rights from NCHS obtained; pending linkage of 1988 and 1991 data (January). We have 1,663 children with height and weight measurements taken between 2 and 5 years of age, and we have 1099 children with height and weight measurements taken between 2.5 and 3.5 years of age. Please provide input on the age range of children to include in analysis. In addition, among children aged 2.5-3.5, BMI ranges from 1.05 to 147.7, with a median of 15.8. If we clean the data by eliminating the top and bottom 1 percent, the range becomes 11.43-27.01. Please provide input on cleaning of this variable as well.

Infant Mortality

TABLE 6 NMIHS-based Mortality per 100 Births (Through One Year) for Term Births without Congenital Malformations, Representative of 1988 U.S. Population

	Pregravid BMI			
Weight Gain (kg)	**Underweight**	**Normal weight**	**Overweight**	**Obese**
< 0	4.07	2.60	2.03	1.79
[0,5)	4.87	2.18	1.29	1.45
[5,10)	1.79	1.25	1.04	1.30
[10,15)	0.72	0.71	0.88	0.85
[15,20)	0.69	0.61	0.88	1.01
[20,25)	0.42	0.61	0.69	1.59
≥ 25	0.95	0.69	0.73	0.78

NOTE: For all NMIHS births, infant mortality was 1.0 per 100 births.

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PART III: ANALYSES FROM DR. STEIN

THE EFFECT OF MATERNAL RACE/ETHNICITY AND BMI ON THE ASSOCIATION BETWEEN GESTATIONAL WEIGHT GAIN AND BIRTH OUTCOME

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To examine the independent and joint effects of maternal race/ethnicity and BMI on the association between gestational weight gain (GWG) and birth outcome, New York City vital statistics birth data for 1995 to 2003 was linked to hospital discharge data from the Statewide Planning and Research Cooperative System (SPARCS). Of 1,173,053 birth records, 1,084,882 (92.5 percent) were successfully matched to a hospital discharge record. Unmatched records resulted from missing personal information needed for the matching algorithm. Singleton births were more likely to be matched to a hospital discharge record than infants from a multiple gestation. Of 1,133,020 vital records for singleton births, 1,067,356 (94.2 percent) were successfully linked to a hospital discharge record.

Inclusion Criteria

Of the 1,067,356 singleton births with matched vital records and hospital discharge data, 913,461 (85.6 percent) were potentially eligible for analysis. Inclusion criteria, and the corresponding percent lost, are GWG between -10 to 40 kg (10.7 percent), no birth defects (2.2 percent), non-missing outcome and covariate (maternal age, race/ethnicity, parity, education, smoking) data (1.2 percent), gestational age between 26 and 42 completed weeks (1.0 percent), and plausible combination of birth weight and gestational age (0.7 percent) (Alexander et al., 1996). Maternal height, needed to calculate body mass index (BMI), was reported for births to New York City residents in hospitals located elsewhere in New York State, which were only 34,307 (3.8 percent) of these 913,461 potentially eligible births. As indicated in Table 3, women with height reported had higher pre-pregnancy and delivery weights, more frequent primary cesarean sections, fewer term small-for-gestational age (SGA) and more term large-for-gestational age (LGA) births. Additionally, these women were more often from Queens and the Bronx, which likely accounts for the increased proportion of white non-Hispanic women.

Dependent Variables

Five birth outcomes were studied: preterm birth < 37 completed weeks gestation, spontaneous preterm birth < 37 completed weeks gestation, primary cesarean delivery, term SGA, and term LGA. Preterm birth < 37 weeks was examined as a dichotomous variable. Spontaneous preterm births were differentiated from medically indicated preterm births using International Classification of Diseases, Ninth Revision (ICD-9) hospital discharge diagnosis and procedure codes. Women with artificial rupture of membranes, induction of labor by artificial rupture of membranes, or other surgical or medical induction of labor (ICD-9 codes 73.0, 73.01, 73.09, 73.1, 73.4) were categorized as medically indicated preterm births. From the remaining women, those with PROM (658.1x; 658.2x) were categorized as spontaneous. We then added

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pre-labor cesarean deliveries to medically indicated births. To identify pre-labor cesareans, we looked for women with delivery by cesarean section (74.x), but without codes indicating labor or spontaneous delivery (644.0x; 644.1x; 644.2x). The remaining preterm births were classified as spontaneous. Medically indicated preterm births (692) were excluded from analyses comparing spontaneous preterm births < 37 weeks to term births. Vaginal births after cesarean (1,008) and repeat cesareans (3,502) were excluded from analyses comparing primary cesarean delivery to vaginal delivery as noted on the birth certificate. Term SGA was used to indicate term infants below the 10th percentile of birth weight for week of gestation; by the combination of infant gender, maternal race (black/non-black), and parity (nulliparous/multiparous) (Gregory et al., 2008). Term LGA corresponded to term infants above the 90th percentile of birth weight for week of gestation by the combination of infant gender, maternal race, and parity (Gregory et al., 2008).

Independent Variables

GWG was calculated as delivery weight minus pre-pregnancy weight as reported on the birth certificate, and then converted from pounds to kilograms. For analyses restricted to term births only (SGA and LGA), GWG was used as a categorical measure (0–9 kg, 10–14 kg, 15–19 kg, 20–40 kg) with 10–14 kg as the referent. Women who lost weight during pregnancy were excluded from these analyses because of small numbers (140). For the analyses not restricted to term births (preterm birth, spontaneous preterm birth, cesarean delivery), the rate of GWG was calculated as GWG divided by completed weeks gestation. Rate of GWG was categorized into tertiles, with the middle tertile as the referent. The equivalent weight gain at 40 completed weeks for the middle tertile was 12.1–16.4 kg.

BMI was computed as prepregnancy weight divided by height squared. The National Heart, Lung, Blood Institute cutoff points were used to categorize BMI as underweight (< 18.5), normal weight (18.5– < 25), overweight (25– < 30), and obese (≥ 30).

Maternal race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, Asian, Other) was self-reported on the birth certificate. Additional maternal demographic characteristics examined as covariates include maternal age (continuous and squared terms), number of previous pregnancies (0, ≥1), education (<12 years, 12 years, >12 years), and tobacco use during pregnancy (smoker, non-smoker).

Statistical Analysis

Analyses were restricted to singleton births with complete information on all measures and were performed using SAS Version 9.1 (SAS Institute, Cary, NC) and Stata Version 10 (Stata Corp, College Station, TX). Unconditional logistic regression was used to estimate odds ratios (OR) and 95 percent confidence intervals (CI) for the relation between GWG and birth outcome. For each birth outcome, the unadjusted association was calculated. To assess whether the effect of GWG on birth outcome varied by pre-pregnancy BMI, was included as a product interaction term between GWG and BMI and adjusted for race/ethnicity. To assess whether the effect of GWG on birth outcome varied by race/ethnicity, a product interaction term was included between GWG and race/ethnicity and adjusted for BMI. Finally, to look at the potential for joint effects between BMI and race/ethnicity, a three-level product interaction term was included containing GWG, BMI, and race/ethnicity. Regardless of the p-value for the product terms, results were tabulated stratified by BMI, adjusted for ethnicity, for ethnicity adjusted for BMI, and jointly stratified by BMI and ethnicity.

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In additional analyses, maternal age, parity, education and smoking were adjusted for and no substantive confounding was found. Spline regression (Zhang and Bowes, 1995) did not alter decisions about category cutoff points for GWG. Analyses that included the 879,154 births without height information were also performed. For these analyses, stratification by race/ethnicity was used and adjusted for prepregnancy weight (continuous and square terms).

TABLE 1 Characteristics of Singleton Births, New York City, 1995 – 2003, *n* = 34,307

Characteristic	**N (percent)**
Gestational weight gain, kg	
Mean (standard deviation)	14.4 (5.8)
Gestational weight gain, kg	
<0	140 (0.4)
0 – 4	1,408 (4.1)
5 – 9	5,861 (17.1)
10 – 14	12,950 (37.7)
15 – 19	8,953 (26.1)
20 – 24	3,590 (10.5)
>25	1,405 (4.1)
Gestational weight gain, kg	
0 – 9	7,269 (21.3)
10 – 14	12,950 (37.9)
15 – 19	8,953 (26.2)
>20	4,995 (14.6)
Gestational weight gain rate*	
Lower tertile (-0.35 – 0.30 kg/week)	11,250 (32.3)
Middle tertile (0.31 – 0.41 kg/week)	11,416 (33.3)
Upper tertile (0.42 – 1.19 kg/week)	11,641 (33.9)
Body mass index, pre-pregnancy	
Mean (standard deviation)	24.8 (5.3)
Body mass index, pre-pregnancy	
<18.5 (underweight)	1,632 (4.8)
18.5 – < 25 (normal weight)	19,892 (58.0)
25 – <30 (overweight)	7,893 (23.0)
30 – <35 (obese I)	3,077 (9.0)
35 – <40 (obese II)	1,166 (3.4)
40+ (obese III)	647 (1.9)
Body mass index, pre-pregnancy	
<18.5 (underweight)	1,632 (4.8)
18.5 – < 25 (normal weight)	19,892 (58.0)
25 – <30 (overweight)	7,893 (23.0)
30+ (obese)	4,890 (14.3)

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Characteristic	N (percent)
Preterm < 37 weeks	
Yes	2,430 (7.1)
No	31,877 (92.9)
Preterm < 37 weeks, delivery indication	
PROM or spontaneous	1,738 (71.5)
Medically indicated	692 (28.5)
****Primary cesarean delivery**	
Primary cesarean	6,279 (21.1)
Vaginal delivery	23,518 (78.9)
Term SGA < 10 percentile	
Yes	2,749 (8.6)
No	29,128 (91.4)
Term LGA > 90 percentile	
Yes	3,242 (10.2)
No	28,635 (89.8)
Maternal race/ethnicity	
Non-Hispanic white	16,291 (47.5)
Non-Hispanic black	9,209 (26.8)
Hispanic	4,953 (14.4)
Asian	3,558 (10.4)
Other	296 (0.9)
Maternal age, years	
Mean (standard deviation)	30.7 (5.3)
Parity	
0	15,926 (46.4)
1+	18,381 (53.6)
Education, years	
<12	1,968 (5.7)
12	8,676 (25.3)
>12	23,663 (69.0)
Tobacco use	
Yes	879 (2.6)
No	33,428 (97.4)

*Rate of gestational weight gain equivalent for 40 weeks gestation: lower tertile = -13.6 – 12 kg gain; middle tertile = 12.1 – 16.4 kg gain; upper tertile = 16.5 – 47.6 kg gain

**Excludes 3,502 repeat cesarean and 1,008 vaginal birth after cesarean deliveries

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TABLE 2 Bivariate Association between BMI and Characteristics of Singleton Births, New York City, 1995 – 2003, *n* = 34,307

	Body Mass Index			
	Underweight	**Normal Weight**	**Overweight**	**Obese**
	N=1,632	**N=19,892**	**N=7,893**	**N=4,890**
	N (percent)	**N (percent)**	**N (percent)**	**N (percent)**
Gestational weight gain, kg				
<0	0	11 (0.1)	26 (0.3)	103 (2.1)
0 – 9	217 (13.3)	3,063 (15.4)	2,030 (25.8)	1,959 (40.9)
10 – 14	716 (43.9)	7,973 (40.1)	2,779 (35.3)	1,482 (31.0)
15 – 19	466 (28.5)	5,804 (29.2)	1,876 (23.9)	807 (16.9)
>20	233 (14.3)	3,041 (15.3)	1,182 (15.0)	539 (11.3)
Gestational weight gain, kg				
<0	0	11 (0.1)	26 (0.3)	103 (2.1)
0 – 4	15 (0.9)	331 (1.7)	402 (5.1)	660 (13.5)
5 – 9	202 (12.4)	2,732 (13.7)	1,628 (20.6)	1,299 (26.6)
10 – 14	716 (43.9)	7,973 (40.1)	2,779 (35.2)	1,482 (30.3)
15 – 19	466 (28.5)	5,804 (29.2)	1,876 (23.8)	807 (16.5)
20 – 24	177 (10.9)	2,265 (11.4)	805 (10.2)	343 (7.0)
>25	56 (3.4)	776 (3.9)	377 (4.8)	196 (4.0)
Gestational weight gain rate*				
Lower tertile	414 (25.4)	5,300 (26.6)	2,909 (36.9)	2,627 (53.7)
Middle tertile	628 (38.5)	7,268 (36.5)	2,397 (30.4)	1,123 (23.0)
Upper tertile	590 (36.1)	7,324 (36.8)	2,587 (32.8)	1,140 (23.3)
Maternal race/ethnicity				
Non-Hispanic white	679 (41.6)	10,157 (51.1)	3,399 (43.1)	2,056 (42.0)
Non-Hispanic black	313 (19.2)	4,251 (21.4)	2,695 (34.1)	1,950 (40.0)
Hispanic	169 (10.4)	2,801 (14.1)	1,251 (15.9)	732 (15.0)
Asian	445 (27.3)	2,493 (12.5)	487 (6.2)	133 (2.7)
Other	26 (1.6)	190 (1.0)	61 (0.8)	19 (0.4)
Preterm < 37 weeks	150 (9.2)	1,232 (6.2)	595 (7.5)	453 (9.3)
Preterm, spontaneous**	114 (7.1)	874 (4.5)	433 (5.6)	317 (6.7)
Primary cesarean delivery***	234 (15.5)	3,350 (18.8)	1,519 (22.9)	1,176 (30.7)
Term SGA < 10 percentile****	233 (15.7)	1,737 (9.3)	499 (6.9)	262 (6.0)
Term LGA > 90 percentile****	60 (4.1)	1,518 (8.1)	916 (12.6)	740 (17.0)

*Rate of gestational weight gain equivalent for 40 weeks gestation: lower tertile = -13.6 – 12 kg gain; middle tertile = 12.1 – 16.4 kg gain; upper tertile = 16.5 – 47.6 kg gain

**Excludes 692 medically indicated preterm births

***Excludes 3,502 repeat cesarean and 1,008 vaginal birth after cesarean deliveries

****Excludes 2,430 preterm births

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TABLE 2b Bivariate Association between Rate of Gestational Weight Gain and Race/Ethnicity among Singleton Births, New York City, 1995 – 2003, *n* = 34,307

	Rate of Gestational Weight Gain*		
	Lower Tertile N=11,250 N (percent)	**Middle Tertile N=11,416 N (percent)**	**Upper Tertile N=11,641 N (percent)**
Maternal race/ethnicity			
Non-Hispanic white	4,922 (30.2)	5,560 (34.1)	5,809 (35.7)
Non-Hispanic black	3,451 (37.5)	2,807 (30.5)	2,951 (32.0)
Hispanic	1,597 (32.2)	1,540 (31.1)	1,816 (36.7)
Asian	1,157 (32.5)	1,412 (39.7)	989 (27.8)
Other	123 (41.5)	97 (32.8)	76 (25.7)

*Rate of gestational weight gain equivalent for 40 weeks gestation: lower tertile = -13.6 – 12 kg gain; middle tertile = 12.1 – 16.4 kg gain; upper tertile = 16.5 – 47.6 kg gain

TABLE 2c Bivariate Association between Gestational Weight Gain and Race/Ethnicity among Singleton Births, New York City, 1995 – 2003, *n* = 34,167

	Gestational Weight Gain			
	0 – 9 kg N=7,269 N (percent)	**10 – 14 kg N=12,950 N (percent)**	**15 – 19 kg N=8,953 N (percent)**	**20+ kg N=4,995 N (percent)**
Maternal race/ethnicity				
Non-Hispanic white	3,043 (18.7)	6,178 (38.1)	4,487 (27.6)	2,527 (15.6)
Non-Hispanic black	2,356 (25.7)	3,318 (36.3)	2,158 (23.6)	1,316 (14.4)
Hispanic	1,083 (21.9)	1,769 (35.9)	1,259 (25.5)	824 (16.7)
Asian	706 (19.9)	1,571 (44.2)	976 (27.5)	301 (8.5)
Other	81 (27.5)	114 (38.6)	73 (24.7)	27 (9.1)

TABLE 2d Bivariate Association between Rate of Gestational Weight Gain and Race/Ethnicity among Singleton Births, New York City, 1995 – 2003, *n* = 913,461

	Rate of Gestational Weight Gain*		
	Lower Tertile N=339,592 N (percent)	**Middle Tertile N=291,098 N (percent)**	**Upper Tertile N=282,771 N (percent)**
Maternal race/ethnicity			
Non-Hispanic white	91,741 (32.8)	101,031 (36.1)	86,706 (31.0)
Non-Hispanic black	93,127 (39.2)	67,288 (28.3)	77,119 (32.5)
Hispanic	110,550 (37.8)	86,361 (29.5)	95,548 (32.7)
Asian	42,713 (42.5)	35,156 (35.0)	22,573 (22.5)
Other	1,461 (41.2)	1,262 (35.6)	825 (23.3)

*Rate of gestational weight gain equivalent for 40 weeks gestation: lower tertile = -13.6 – 12 kg gain; middle tertile = 12.1 – 16.4 kg gain; upper tertile = 16.5 – 47.6 kg gain

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TABLE 2e Bivariate Association between Gestational Weight Gain and Race/Ethnicity among Singleton Births, New York City, 1995 – 2003, *n* = 913,320

	Gestational Weight Gain			
	0 – 9 kg N=234,764 N (percent)	**10 – 14 kg N=333,968 N (percent)**	**15 – 19 kg N=223,366 N (percent)**	**20+ kg N=121,192 N (percent)**
Maternal race/ethnicity				
Non-Hispanic white	56,817 (20.3)	112,814 (40.4)	75,274 (26.9)	34,517 (12.3)
Non-Hispanic black	69,294 (29.2)	77,868 (32.8)	54,412 (22.9)	35,899 (15.1)
Hispanic	78,528 (26.9)	99,705 (34.1)	70,694 (24.2)	43,513 (14.9)
Asian	29,086 (29.0)	42,137 (41.9)	22,251 (22.1)	6,964 (6.9)
Other	1069 (30.1)	1,444 (40.7)	735 (20.7)	299 (8.4)

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TABLE 3 Percent of Singleton Births with and without Maternal Height, New York City, 1995 – 2003

Characteristic	**Height Recorded (n=34,307)**	**Height Missing (n=879,154)**
Pre-pregnancy weight, kg (mean)	66.1	64.5
Delivery weight, kg (mean)	80.5	78.5
Gestational weight gain (mean)	14.4	14.0
Rate of gestational weight gain (kg/week)	0.37	0.36
Preterm < 37 weeks	7.1	7.1
Preterm PROM or spontaneous	5.2	5.6
Primary cesarean delivery	21.1	16.5
Term SGA < 10 percentile	8.6	10.4
Term LGA > 90 percentile	10.2	8.5
Maternal race/ethnicity		
Non-Hispanic white	47.5	29.9
Non-Hispanic black	26.8	26.0
Hispanic	14.4	32.7
Asian	10.4	11.0
Other	0.9	0.4
Tobacco use	2.6	3.4
Maternal age, years (mean)	30.7	28.8
Parity (mean)	0.9	1.0
Education, years (mean)	14.2	12.8
County of residence		
Manhattan	2.4	16.5
Brooklyn	5.1	31.4
Bronx	24.3	17.9
Queens	62.5	21.2
Staten Island	0.2	4.7
Outside NYC	4.4	5.0
Outside NYS	0.1	1.5
Unknown	1.1	1.9

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TABLE 4 Odds Ratios and 95% Confidence Intervals for the Association of Rate of Gestational Weight Gain with Preterm Birth < 37 Weeks vs Term Birth ≥37 Weeks among Singleton Births, New York City, 1995 – 2003, *n* = 34,307

	Rate of Gestational Weight Gain*		
	Lower Tertile	**Middle Tertile**	**Upper Tertile**
Overall, unadjusted	1.3 (1.2, 1.5)	1.0	1.1 (1.0, 1.3)
By BMI, adjusted for race/ethnicity (GWG*BMI p=0.14)			
<18.5 (underweight)	1.1 (0.7, 1.6)	1.0	0.8 (0.5, 1.2)
18.5 – < 25 (normal weight)	1.4 (1.2, 1.6)	1.0	1.2 (1.0, 1.3)
25 – <30 (overweight)	1.1 (0.9, 1.4)	1.0	1.1 (0.9, 1.3)
30+ (obese)	1.1 (0.8, 1.4)	1.0	1.2 (0.9, 1.7)
By race/ethnicity, adjusted for BMI (GWG*ethnicity p=0.49)			
Non-Hispanic white	1.1 (1.0, 1.4)	1.0	1.1 (0.9, 1.3)
Non-Hispanic black	1.2 (1.0, 1.4)	1.0	1.2 (1.0, 1.4)
Hispanic	1.3 (1.0, 1.7)	1.0	1.0 (0.8, 1.3)
Asian	1.4 (1.0, 2.0)	1.0	1.1 (0.7, 1.5)
By race/ethnicity (n=913,461), adjusted for pre-pregnancy weight (GWG*ethnicity p<0.001)			
Non-Hispanic white	1.1 (1.1, 1.1)	1.0	1.1 (1.1, 1.1)
Non-Hispanic black	1.2 (1.1, 1.2)	1.0	1.0 (1.0, 1.0)
Hispanic	1.1 (1.1, 1.2)	1.0	1.0 (1.0, 1.1)
Asian	1.0 (1.0, 1.1)	1.0	1.1 (1.0, 1.1)
By BMI and race/ethnicity (GWG*BMI*ethnicity p=0.82)			
Non-Hispanic white			
<18.5 (underweight)	1.0 (0.5, 1.9)	1.0	0.7 (0.4, 1.4)
18.5 – < 25 (normal weight)	1.3 (1.0, 1.7)	1.0	1.2 (0.9, 1.4)
25 – <30 (overweight)	1.1 (0.8, 1.6)	1.0	1.0 (0.7, 1.5)
30+ (obese)	1.0 (0.6, 1.6)	1.0	1.3 (0.8, 2.2)
Non-Hispanic black			
<18.5 (underweight)	1.3 (0.6, 2.9)	1.0	0.8 (0.4, 1.8)
18.5 – < 25 (normal weight)	1.2 (0.9, 1.6)	1.0	1.1 (0.9, 1.5)
25 – <30 (overweight)	1.2 (0.9, 1.6)	1.0	1.2 (0.8, 1.6)
30+ (obese)	1.3 (0.9, 1.8)	1.0	1.4 (0.9, 2.2)
Hispanic			
<18.5 (underweight)	1.1 (0.3, 4.3)	1.0	0.7 (0.2, 2.5)
18.5 – < 25 (normal weight)	1.5 (1.0, 2.2)	1.0	1.1 (0.7, 1.6)
25 – <30 (overweight)	1.2 (0.7, 1.9)	1.0	0.9 (0.5, 1.6)
30+ (obese)	1.0 (0.6, 1.8)	1.0	0.9 (0.5, 1.8)
Asian			
<18.5 (underweight)	0.3 (0.1, 1.4)	1.0	0.9 (0.3, 2.1)
18.5 – < 25 (normal weight)	1.8 (1.2, 2.8)	1.0	1.3 (0.8, 2.1)
25 – <30 (overweight)	1.1 (0.5, 2.3)	1.0	0.8 (0.3, 2.3)
30+ (obese)	0.4 (0.1, 1.3)	1.0	0.4 (0.1, 2.1)

*Rate of gestational weight gain equivalent for 40 weeks gestation: lower tertile = -13.6 – 12 kg gain; middle tertile = 12.1 – 16.4 kg gain; upper tertile = 16.5 – 47.6 kg gain

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TABLE 5 Odds Ratios and 95% Confidence Intervals for the Association of Rate of Gestational Weight Gain with Spontaneous Preterm Birth < 37 Weeks vs. Term Birth ≥ 37 Weeks among Singleton Births, New York City, 1995 – 2003, *n* = 33,615

	Rate of Gestational Weight Gain*		
	Lower Tertile	**Middle Tertile**	**Upper Tertile**
Overall, unadjusted	1.3 (1.2, 1.5)	1.0	1.1 (1.0, 1.3)
By BMI, adjusted for race/ethnicity (GWG*BMI p = 0.28)			
<18.5 (underweight)	1.0 (0.6, 1.6)	1.0	0.8 (0.5, 1.2)
18.5 – < 25 (normal weight)	1.4 (1.2, 1.7)	1.0	1.1 (1.0, 1.3)
25 – <30 (overweight)	1.2 (0.9, 1.5)	1.0	1.1 (0.8, 1.4)
30+ (obese)	1.1 (0.8, 1.5)	1.0	1.2 (0.9, 1.7)
By race/ethnicity, adjusted for BMI(GWG*ethnicity p = 0.75)			
Non-Hispanic white	1.1 (0.9, 1.4)	1.0	1.1 (0.9, 1.3)
Non-Hispanic black	1.3 (1.0, 1.5)	1.0	1.2 (1.0, 1.5)
Hispanic	1.5 (1.1, 1.9)	1.0	1.0 (0.7, 1.4)
Asian	1.2 (0.9, 1.8)	1.0	1.1 (0.7, 1.7)
By race/ethnicity (n = 898,893), adjusted for pre-pregnancy weight (GWG*ethnicity p < 0.001)			
Non-Hispanic white	1.1 (1.1, 1.2)	1.0	1.1 (1.1, 1.1)
Non-Hispanic black	1.2 (1.2, 1.3)	1.0	1.0 (0.9, 1.0)
Hispanic	1.1 (1.1, 1.2)	1.0	1.0 (0.9, 1.0)
Asian	1.1 (1.0, 1.1)	1.0	1.1 (1.0, 1.1)
By BMI and race/ethnicity (GWG*BMI*ethnicity p = 0.56)			
Non-Hispanic white			
<18.5 (underweight)	0.8 (0.4, 1.7)	1.0	0.7 (0.3, 1.4)
18.5 – < 25 (normal weight)	1.3 (1.0, 1.7)	1.0	1.1 (0.9, 1.5)
25 – <30 (overweight)	1.2 (0.8, 1.9)	1.0	0.8 (0.5, 1.3)
30+ (obese)	0.9 (0.5, 1.6)	1.0	1.4 (0.8, 2.5)
Non-Hispanic black			
<18.5 (underweight)	1.0 (0.4, 2.5)	1.0	0.7 (0.3, 1.7)
18.5 – < 25 (normal weight)	1.3 (1.0, 1.8)	1.0	1.2 (0.9, 1.6)
25 – <30 (overweight)	1.3 (0.9, 1.9)	1.0	1.3 (0.9, 2.0)
30+ (obese)	1.3 (0.8, 1.9)	1.0	1.2 (0.7, 2.0)
Hispanic			
<18.5 (underweight)	1.7 (0.4, 7.2)	1.0	1.1 (0.3, 4.2)
18.5 – < 25 (normal weight)	1.7 (1.1, 2.6)	1.0	1.0 (0.6, 1.5)
25 – <30 (overweight)	1.0 (0.6, 1.9)	1.0	1.0 (0.5, 1.8)
30+ (obese)	1.2 (0.6, 2.4)	1.0	1.1 (0.5, 2.3)
Asian			
<18.5 (underweight)	0.4 (0.1, 1.9)	1.0	0.9 (0.3, 2.5)
18.5 – < 25 (normal weight)	1.6 (1.0, 2.6)	1.0	1.4 (0.9, 2.3)
25 – <30 (overweight)	0.9 (0.4, 2.1)	1.0	0.7 (0.2, 2.2)
30+ (obese)	0.3 (0.1, 1.3)	1.0	0.5 (0.1, 3.1)

*Rate of gestational weight gain equivalent for 40 weeks gestation: lower tertile = -13.6 – 12 kg gain; middle tertile = 12.1 – 16.4 kg gain; upper tertile = 16.5 – 47.6 kg gain

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TABLE 6 Odds Ratios and 95% Confidence Intervals for the Association of Rate of Gestational Weight Gain with Primary Cesarean Delivery vs. Vaginal Delivery among Singleton Births, New York City, 1995 – 2003, *n* = 29,797

	Rate of Gestational Weight Gain*		
	Lower Tertile	**Middle Tertile**	**Upper Tertile**
Overall, unadjusted	1.0 (0.9, 1.1)	1.0	1.4 (1.3, 1.5)
By BMI, adjusted for race/ethnicity (GWG*BMI p = 0.81)			
<18.5 (underweight)	0.9 (0.6, 1.4)	1.0	1.7 (1.2, 2.3)
18.5 – < 25 (normal weight)	0.8 (0.8, 0.9)	1.0	1.3 (1.2, 1.5)
25 – <30 (overweight)	0.9 (0.8, 1.0)	1.0	1.4 (1.2, 1.6)
30+ (obese)	0.9 (0.7, 1.0)	1.0	1.4 (1.1, 1.7)
By race/ethnicity, adjusted for BMI (GWG*ethnicity p = 0.04)			
Non-Hispanic white	0.8 (0.7, 0.9)	1.0	1.4 (1.3, 1.6)
Non-Hispanic black	0.9 (0.8, 1.1)	1.0	1.3 (1.1, 1.5)
Hispanic	0.8 (0.7, 1.0)	1.0	1.3 (1.1, 1.5)
Asian	0.9 (0.7, 1.1)	1.0	1.4 (1.2, 1.8)
By race/ethnicity (n = 813,272), adjusted for pre-pregnancy weight (GWG*ethnicity p < 0.001)			
Non-Hispanic white	0.8 (0.8, 0.8)	1.0	1.4 (1.4, 1.4)
Non-Hispanic black	0.9 (0.8, 0.9)	1.0	1.3 (1.2, 1.3)
Hispanic	0.8 (0.8, 0.8)	1.0	1.3 (1.3, 1.3)
Asian	0.8 (0.8, 0.9)	1.0	1.3 (1.3, 1.4)
By BMI and race/ethnicity (GWG*BMI*ethnicity p = 0.86)			
Non-Hispanic white			
<18.5 (underweight)	1.2 (0.6, 2.3)	1.0	1.9 (1.1, 3.4)
18.5 – < 25 (normal weight)	0.7 (0.6, 0.9)	1.0	1.4 (1.2, 1.5)
25 – <30 (overweight)	0.9 (0.7, 1.1)	1.0	1.5 (1.3, 1.9)
30+ (obese)	0.8 (0.6, 1.1)	1.0	1.4 (1.1, 1.9)
Non-Hispanic black			
<18.5 (underweight)	1.0 (0.4, 2.6)	1.0	2.7 (1.2, 6.1)
18.5 – < 25 (normal weight)	1.0 (0.8, 1.2)	1.0	1.3 (1.1, 1.6)
25 – <30 (overweight)	0.9 (0.7, 1.1)	1.0	1.1 (0.9, 1.5)
30+ (obese)	0.9 (0.7, 1.2)	1.0	1.4 (1.0, 1.9)
Hispanic			
<18.5 (underweight)	0.7 (0.2, 2.6)	1.0	0.9 (0.4, 2.5)
18.5 – < 25 (normal weight)	0.8 (0.6, 1.0)	1.0	1.2 (1.0, 1.5)
25 – <30 (overweight)	0.9 (0.6, 1.3)	1.0	1.2 (0.8, 1.7)
30+ (obese)	0.9 (0.6, 1.4)	1.0	1.6 (1.0, 2.7)
Asian			
<18.5 (underweight)	0.7 (0.3, 1.5)	1.0	1.5 (0.9, 2.5)
18.5 – < 25 (normal weight)	1.0 (0.7, 1.3)	1.0	1.4 (1.1, 1.9)
25 – <30 (overweight)	1.0 (0.6, 1.8)	1.0	1.9 (1.0, 3.6)
30+ (obese)	0.5 (0.2, 1.4)	1.0	0.5 (0.1, 2.1)

*Rate of gestational weight gain equivalent for 40 weeks gestation: lower tertile = -13.6 – 12 kg gain; middle tertile = 12.1 – 16.4 kg gain; upper tertile = 16.5 – 47.6 kg gain

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TABLE 7 Odds Ratios and 95% Confidence Intervals for the Association of Gestational Weight Gain with Term Small-for-Gestational Age among Singleton Births, New York City, 1995 – 2003, *n* = 31,760

	Gestational Weight Gain			
	0 – 9 kg	**10 – 14 kg**	**15 – 19 kg**	**20+ kg**
Overall, unadjusted	1.3 (1.2, 1.4)	1.0	0.7 (0.7, 0.8)	0.5 (0.4, 0.6)
By BMI, adjusted for race/ethnicity (GWG*BMI p = 0.43)				
<18.5 (underweight)	1.5 (1.0, 2.3)	1.0	0.7 (0.5, 1.0)	0.4 (0.2, 0.6)
18.5 – < 25 (normal weight)	1.5 (1.3, 1.7)	1.0	0.7 (0.6, 0.8)	0.5 (0.4, 0.6)
25 – <30 (overweight)	1.4 (1.1, 1.7)	1.0	0.6 (0.5, 0.8)	0.6 (0.5, 0.9)
30+ (obese)	1.7 (1.3, 2.4)	1.0	0.9 (0.6, 1.4)	0.9 (0.5, 1.4)
By race/ethnicity, adjusted for BMI (GWG*ethnicity p = 0.50)				
Non-Hispanic white	1.5 (1.3, 1.7)	1.0	0.7 (0.6, 0.8)	0.5 (0.4, 0.6)
Non-Hispanic black	1.3 (1.1, 1.7)	1.0	0.8 (0.7, 1.1)	0.6 (0.5, 0.8)
Hispanic	1.7 (1.3, 2.1)	1.0	0.7 (0.5, 0.9)	0.6 (0.4, 0.8)
Asian	1.6 (1.2, 2.0)	1.0	0.8 (0.6, 1.0)	0.5 (0.3, 0.7)
By race/ethnicity (n = 848,426), adjusted for pre-pregnancy weight (GWG*ethnicity p < 0.001)				
Non-Hispanic white	1.7 (1.6, 1.7)	1.0	0.7 (0.6, 0.7)	0.5 (0.5, 0.5)
Non-Hispanic black	1.5 (1.5, 1.6)	1.0	0.7 (0.7, 0.7)	0.6 (0.5, 0.6)
Hispanic	1.5 (1.5, 1.5)	1.0	0.7 (0.7, 0.8)	0.6 (0.6, 0.6)
Asian	1.7 (1.6, 1.7)	1.0	0.7 (0.7, 0.7)	0.6 (0.6, 0.7)
By BMI and race/ethnicity (GWG*BMI*ethnicity p=0.41)				
Non-Hispanic white				
<18.5 (underweight)	1.0 (0.5, 1.9)	1.0	0.5 (0.3, 1.0)	0.5 (0.3, 1.1)
18.5 – < 25 (normal weight)	1.6 (1.3, 1.9)	1.0	0.7 (0.6, 0.9)	0.5 (0.4, 0.6)
25 – <30 (overweight)	1.3 (0.9, 1.7)	1.0	0.4 (0.3, 0.7)	0.3 (0.2, 0.6)
30+ (obese)	1.8 (1.1, 2.8)	1.0	0.6 (0.3, 1.3)	0.8 (0.4, 1.7)
Non-Hispanic black				
<18.5 (underweight)	2.2 (0.9, 5.8)	1.0	0.9 (0.4, 2.5)	0.2 (0.02, 1.4)
18.5 – < 25 (normal weight)	1.2 (0.9, 1.7)	1.0	0.7 (0.5, 1.0)	0.4 (0.3, 0.6)
25 – <30 (overweight)	1.2 (0.8, 1.9)	1.0	0.9 (0.6, 1.5)	1.1 (0.6, 1.9)
30+ (obese)	2.1 (1.2, 3.7)	1.0	1.5 (0.7, 3.1)	1.4 (0.6, 3.3)
Hispanic				
<18.5 (underweight)	4.8 (1.2, 18.4)	1.0	1.6 (0.5, 5.6)	1.1 (0.3, 4.8)
18.5 – < 25 (normal weight)	1.4 (1.0, 1.9)	1.0	0.6 (0.4, 0.8)	0.5 (0.3, 0.7)
25 – <30 (overweight)	1.6 (1.0, 2.6)	1.0	0.7 (0.4, 1.3)	0.8 (0.4, 1.5)
30+ (obese)	2.1 (1.0, 4.2)	1.0	1.1 (0.5, 2.9)	0.6 (0.2, 2.4)
Asian				
<18.5 (underweight)	1.4 (0.6, 3.1)	1.0	0.7 (0.4, 1.3)	0.1 (0.01, 0.5)
18.5 – < 25 (normal weight)	1.7 (1.3, 2.3)	1.0	0.8 (0.6, 1.0)	0.7 (0.4, 1.1)
25 – <30 (overweight)	1.6 (0.9, 3.0)	1.0	0.9 (0.4, 2.0)	0.3 (0.04, 2.2)
30+ (obese)	0.1 (0.01, 1.2)	1.0	0.4 (0.04, 3.7)	n/c*

*n/c = not calculable

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TABLE 8 Odds Ratios and 95% Confidence Intervals for the Association of Gestational Weight Gain with Term Large-for-Gestational Age among Singleton Births, New York City, 1995 – 2003, *n* = 31,760

	Gestational Weight Gain			
	0 – 9 kg	**10 – 14 kg**	**15 – 19 kg**	**20+ kg**
Overall, unadjusted	0.9 (0.8, 1.0)	1.0	1.4 (1.3, 1.5)	2.5 (2.3, 2.7)
By BMI, adjusted for race/ethnicity (GWG*BMI p = 0.01)				
<18.5 (underweight)	0.4 (0.1, 1.9)	1.0	1.6 (0.8, 3.2)	4.9 (2.6, 9.4)
18.5 – < 25 (normal weight)	0.7 (0.5, 0.8)	1.0	1.5 (1.3, 1.7)	2.8 (2.5, 3.3)
25 – <30 (overweight)	0.6 (0.5, 0.7)	1.0	1.3 (1.1, 1.6)	2.2 (1.8, 2.6)
30+ (obese)	0.7 (0.6, 0.9)	1.0	1.5 (1.2, 1.9)	1.9 (1.5, 2.5)
By race/ethnicity, adjusted for BMI (GWG*ethnicity p = 0.93)				
Non-Hispanic white	0.7 (0.6, 0.9)	1.0	1.5 (1.3, 1.7)	2.6 (2.3, 3.0)
Non-Hispanic black	0.6 (0.5, 0.7)	1.0	1.4 (1.2, 1.7)	2.2 (1.8, 2.6)
Hispanic	0.6 (0.4, 0.9)	1.0	1.4 (1.1, 1.9)	2.7 (2.0, 3.6)
Asian	0.7 (0.4, 1.1)	1.0	1.5 (1.0, 2.2)	3.1 (2.0, 4.9)
By race/ethnicity (n = 848,426), adjusted for pre-pregnancy weight (GWG*ethnicity p < 0.001)				
Non-Hispanic white	0.6 (0.6, 0.7)	1.0	1.6 (1.5, 1.6)	2.5 (2.4, 2.6)
Non-Hispanic black	0.7 (0.7, 0.7)	1.0	1.5 (1.4, 1.5)	2.2 (2.2, 2.3)
Hispanic	0.7 (0.6, 0.7)	1.0	1.5 (1.4, 1.5)	2.4 (2.3, 2.5)
Asian	0.6 (0.6, 0.7)	1.0	1.6 (1.5, 1.7)	2.7 (2.4, 2.9)
By BMI and race/ethnicity (GWG*BMI*ethnicity p = 0.21)				
Non-Hispanic white				
<18.5 (underweight)	n/c*	1.0	1.1 (0.4, 3.1)	4.3 (1.7, 10.6)
18.5 – < 25 (normal weight)	0.9 (0.7, 1.1)	1.0	1.5 (1.2, 1.8)	3.0 (2.5, 3.7)
25 – <30 (overweight)	0.5 (0.4, 0.8)	1.0	1.5 (1.2, 2.0)	2.3 (1.8, 3.1)
30+ (obese)	0.7 (0.5, 1.0)	1.0	1.5 (1.1, 2.1)	1.8 (1.3, 2.6)
Non-Hispanic black				
<18.5 (underweight)	0.5 (0.1, 4.5)	1.0	1.2 (0.3, 4.5)	4.8 (1.5, 15.3)
18.5 – < 25 (normal weight)	0.5 (0.3, 0.8)	1.0	1.5 (1.2, 2.0)	2.3 (1.8, 3.0)
25 – <30 (overweight)	0.6 (0.4, 0.8)	1.0	1.1 (0.9, 1.5)	2.3 (1.7, 3.1)
30+ (obese)	0.7 (0.5, 1.0)	1.0	1.5 (1.1, 2.2)	1.4 (0.9, 2.1)
Hispanic				
<18.5 (underweight)	n/c*	1.0	n/c*	n/c*
18.5 – < 25 (normal weight)	0.4 (0.2, 0.9)	1.0	1.6 (1.1, 2.4)	3.1 (2.1, 4.7)
25 – <30 (overweight)	0.8 (0.4, 1.4)	1.0	1.3 (0.7, 2.2)	1.4 (0.8, 2.5)
30+ (obese)	0.7 (0.3, 1.3)	1.0	1.1 (0.5, 2.4)	4.2 (2.1, 8.2)
Asian				
<18.5 (underweight)	n/c*	1.0	3.6 (0.7, 18.3)	5.7 (0.9, 35.0)
18.5 – < 25 (normal weight)	0.7 (0.3, 1.4)	1.0	1.5 (0.9, 2.3)	3.1 (1.8, 5.3)
25 – <30 (overweight)	0.7 (0.3, 1.5)	1.0	0.7 (0.3, 2.0)	1.8 (0.5, 5.8)
30+ (obese)	0.6 (0.1, 3.7)	1.0	4.4 (0.9, 22.7)	13.3 (2.2, 81.9)

*n/c = not calculable

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TABLE 9a Adjusted* Odds Ratios and 95% Confidence Intervals for the Association of Rate of Gestational Weight Gain with Preterm Birth < 37 Weeks among Singleton Births, New York City, 1995 – 2003, *n* = 34,307

	Rate of Gestational Weight Gain**		
	Lower Tertile	**Middle Tertile**	**Upper Tertile**
Overall	1.3 (1.1, 1.4)	1.0	1.1 (1.0, 1.3)
By BMI (GWG*BMI p = 0.12)			
<18.5 (underweight)	1.1 (0.7, 1.6)	1.0	0.8 (0.5, 1.2)
18.5 – < 25 (normal weight)	1.4 (1.2, 1.6)	1.0	1.2 (1.0, 1.3)
25 – <30 (overweight)	1.1 (0.9, 1.4)	1.0	1.1 (0.9, 1.3)
30+ (obese)	1.1 (0.8, 1.4)	1.0	1.2 (0.9, 1.6)
By race/ethnicity (GWG*ethnicity p = 0.53)			
Non-Hispanic white	1.1 (1.0, 1.4)	1.0	1.1 (0.9, 1.3)
Non-Hispanic black	1.2 (1.0, 1.4)	1.0	1.2 (1.0, 1.4)
Hispanic	1.3 (1.0, 1.7)	1.0	1.0 (0.8, 1.3)
Asian	1.4 (1.0, 1.9)	1.0	1.1 (0.7, 1.5)
By BMI and race/ethnicity (GWG*BMI*ethnicity p = 0.79)			
Non-Hispanic white			
<18.5 (underweight)	1.0 (0.5, 1.9)	1.0	0.7 (0.4, 1.4)
18.5 – < 25 (normal weight)	1.3 (1.1, 1.7)	1.0	1.2 (0.9, 1.4)
25 – <30 (overweight)	1.1 (0.8, 1.6)	1.0	1.0 (0.7, 1.5)
30+ (obese)	1.0 (0.6, 1.5)	1.0	1.3 (0.8, 2.2)
Non-Hispanic black			
<18.5 (underweight)	1.3 (0.6, 2.7)	1.0	0.8 (0.3, 1.7)
18.5 – < 25 (normal weight)	1.3 (0.9, 1.6)	1.0	1.1 (0.9, 1.5)
25 – <30 (overweight)	1.1 (0.8, 1.6)	1.0	1.2 (0.9, 1.7)
30+ (obese)	1.3 (0.9, 1.8)	1.0	1.5 (0.9, 2.2)
Hispanic			
<18.5 (underweight)	1.1 (0.3, 4.2)	1.0	0.7 (0.2, 2.5)
18.5 – < 25 (normal weight)	1.5 (1.0, 2.2)	1.0	1.1 (0.8, 1.6)
25 – <30 (overweight)	1.1 (0.7, 1.9)	1.0	0.9 (0.5, 1.6)
30+ (obese)	1.0 (0.6, 1.8)	1.0	0.9 (0.5, 1.8)
Asian			
<18.5 (underweight)	0.3 (0.1, 1.4)	1.0	0.9 (0.4, 2.1)
18.5 – < 25 (normal weight)	1.8 (1.2, 2.7)	1.0	1.3 (0.8, 2.1)
25 – <30 (overweight)	1.0 (0.5, 2.3)	1.0	0.8 (0.3, 2.3)
30+ (obese)	0.5 (0.1, 1.4)	1.0	0.4 (0.1, 2.3)

*Adjusted for maternal age, parity, education, and smoking

** Rate of gestational weight gain equivalent for 40 weeks gestation: lower tertile = -13.6 – 12 kg gain; middle tertile = 12.1 – 16.4 kg gain; upper tertile = 16.5 – 47.6 kg gain

***n/c = not calculable

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TABLE 9b Adjusted* Odds Ratios and 95% Confidence Intervals for the Association of Rate of Gestational Weight Gain with Spontaneous Preterm Birth < 37 Weeks vs. Term Birth ≥ 37 Weeks among Singleton Births, New York City, 1995 – 2003, *n* = 33,615

	Rate of Gestational Weight Gain**		
	Lower Tertile	**Middle Tertile**	**Upper Tertile**
Overall	1.3 (1.1, 1.4)	1.0	1.1 (1.0, 1.3)
By BMI (GWG*BMI p = 0.27)			
<18.5 (underweight)	1.0 (0.6, 1.6)	1.0	0.7 (0.5, 1.2)
18.5 – < 25 (normal weight)	1.4 (1.2, 1.7)	1.0	1.1 (1.0, 1.4)
25 – <30 (overweight)	1.2 (0.9, 1.5)	1.0	1.1 (0.8, 1.4)
30+ (obese)	1.1 (0.8, 1.4)	1.0	1.2 (0.9, 1.7)
By race/ethnicity (GWG*ethnicity p = 0.77)			
Non-Hispanic white	1.1 (0.9, 1.4)	1.0	1.0 (0.9, 1.3)
Non-Hispanic black	1.3 (1.0, 1.5)	1.0	1.2 (1.0, 1.5)
Hispanic	1.4 (1.1, 1.9)	1.0	1.0 (0.7, 1.4)
Asian	1.2 (0.8, 1.8)	1.0	1.1 (0.7, 1.7)
By BMI and race/ethnicity (GWG*BMI*ethnicity p = 0.50)			
Non-Hispanic white			
<18.5 (underweight)	0.8 (0.4, 1.7)	1.0	0.7 (0.3, 1.4)
18.5 – < 25 (normal weight)	1.3 (1.0, 1.7)	1.0	1.1 (0.9, 1.4)
25 – <30 (overweight)	1.2 (0.8, 1.9)	1.0	0.8 (0.5, 1.3)
30+ (obese)	0.9 (0.5, 1.5)	1.0	1.4 (0.8, 2.5)
Non-Hispanic black			
<18.5 (underweight)	1.0 (0.4, 2.4)	1.0	0.7 (0.3, 1.7)
18.5 – < 25 (normal weight)	1.3 (1.0, 1.8)	1.0	1.2 (0.9, 1.6)
25 – <30 (overweight)	1.3 (0.9, 1.9)	1.0	1.4 (0.9, 2.0)
30+ (obese)	1.3 (0.8, 2.0)	1.0	1.2 (0.7, 2.0)
Hispanic			
<18.5 (underweight)	1.6 (0.4, 7.0)	1.0	1.1 (0.3, 4.2)
18.5 – < 25 (normal weight)	1.7 (1.1, 2.6)	1.0	1.0 (0.6, 1.5)
25 – <30 (overweight)	1.0 (0.6, 1.8)	1.0	1.0 (0.5, 1.8)
30+ (obese)	1.2 (0.6, 2.3)	1.0	1.0 (0.5, 2.3)
Asian			
<18.5 (underweight)	0.4 (0.1, 1.9)	1.0	0.9 (0.3, 2.5)
18.5 – < 25 (normal weight)	1.6 (1.0, 2.6)	1.0	1.4 (0.9, 2.3)
25 – <30 (overweight)	0.9 (0.4, 2.1)	1.0	0.7 (0.2, 2.2)
30+ (obese)	0.3 (0.1, 1.3)	1.0	0.6 (0.1, 3.4)

*Adjusted for maternal age, parity, education, and smoking

** Rate of gestational weight gain equivalent for 40 weeks gestation: lower tertile = -13.6 – 12 kg gain; middle tertile = 12.1 – 16.4 kg gain; upper tertile = 16.5 – 47.6 kg gain

***n/c = not calculable

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TABLE 9c Adjusted* Odds Ratios and 95% Confidence Intervals for the Association of Rate of Gestational Weight Gain with Primary Cesarean Delivery vs. Vaginal Delivery among Singleton Births, New York City, 1995 – 2003, *n* = 29,797

	Rate of Gestational Weight Gain**		
	Lower Tertile	**Middle Tertile**	**Upper Tertile**
Overall	1.0 (0.9, 1.1)	1.0	1.3 (1.2, 1.4)
By BMI (GWG*BMI p = 0.62)			
<18.5 (underweight)	0.9 (0.6, 1.3)	1.0	1.7 (1.2, 2.3)
18.5 – < 25 (normal weight)	0.9 (0.8, 1.0)	1.0	1.3 (1.2, 1.4)
25 – <30 (overweight)	1.0 (0.8, 1.1)	1.0	1.3 (1.1, 1.5)
30+ (obese)	0.9 (0.7, 1.0)	1.0	1.2 (1.0, 1.5)
By race/ethnicity (GWG*ethnicity p = 0.15)			
Non-Hispanic white	0.9 (0.8, 1.0)	1.0	1.3 (1.2, 1.5)
Non-Hispanic black	0.9 (0.8, 1.1)	1.0	1.3 (1.1, 1.5)
Hispanic	0.9 (0.7, 1.1)	1.0	1.2 (1.0, 1.5)
Asian	1.0 (0.8, 1.2)	1.0	1.3 (1.1, 1.6)
By BMI and race/ethnicity (GWG*BMI*ethnicity p = 0.64)			
Non-Hispanic white			
<18.5 (underweight)	1.2 (0.6, 2.3)	1.0	1.9 (1.1, 3.3)
18.5 – < 25 (normal weight)	0.8 (0.7, 0.9)	1.0	1.3 (1.1, 1.4)
25 – <30 (overweight)	1.0 (0.7, 1.2)	1.0	1.4 (1.1, 1.8)
30+ (obese)	0.9 (0.7, 1.2)	1.0	1.3 (0.9, 1.7)
Non-Hispanic black			
<18.5 (underweight)	0.8 (0.3, 2.3)	1.0	2.6 (1.2, 6.0)
18.5 – < 25 (normal weight)	1.1 (0.9, 1.3)	1.0	1.3 (1.1, 1.6)
25 – <30 (overweight)	0.9 (0.7, 1.2)	1.0	1.1 (0.9, 1.4)
30+ (obese)	0.8 (0.6, 1.1)	1.0	1.1 (0.8, 1.6)
Hispanic			
<18.5 (underweight)	1.0 (0.3, 3.7)	1.0	1.1 (0.4, 2.9)
18.5 – < 25 (normal weight)	0.9 (0.7, 1.1)	1.0	1.2 (1.0, 1.5)
25 – <30 (overweight)	1.0 (0.7, 1.4)	1.0	1.1 (0.7, 1.6)
30+ (obese)	1.0 (0.6, 1.7)	1.0	1.7 (1.0, 2.9)
Asian			
<18.5 (underweight)	0.7 (0.3, 1.4)	1.0	1.4 (0.8, 2.5)
18.5 – < 25 (normal weight)	1.1 (0.8, 1.5)	1.0	1.3 (1.0, 1.7)
25 – <30 (overweight)	1.0 (0.6, 1.9)	1.0	1.6 (0.8, 3.0)
30+ (obese)	0.4 (0.1, 1.2)	1.0	0.4 (0.1, 1.7)

*Adjusted for maternal age, parity, education, and smoking

** Rate of gestational weight gain equivalent for 40 weeks gestation: lower tertile = -13.6 – 12 kg gain; middle tertile = 12.1 – 16.4 kg gain; upper tertile = 16.5 – 47.6 kg gain

***n/c = not calculable

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TABLE 9d Adjusted* Odds Ratios and 95% Confidence Intervals for the Association of Gestational Weight Gain with Term Small-for-Gestational Age among Singleton Births, New York City, 1995 – 2003, *n* = 31,760

	Gestational Weight Gain			
	0 – 9 kg	**10 – 14 kg**	**15 – 19 kg**	**20+ kg**
Overall	1.3 (1.2, 1.4)	1.0	0.7 (0.7, 0.8)	0.5 (0.4, 0.6)
By BMI (GWG*BMI p = 0.42)				
<18.5 (underweight)	1.5 (1.0, 2.3)	1.0	0.7 (0.5, 1.0)	0.3 (0.2, 0.6)
18.5 – < 25 (normal weight)	1.5 (1.3, 1.7)	1.0	0.7 (0.6, 0.8)	0.5 (0.4, 0.6)
25 – <30 (overweight)	1.4 (1.1, 1.7)	1.0	0.6 (0.5, 0.8)	0.6 (0.4, 0.8)
30+ (obese)	1.8 (1.3, 2.4)	1.0	0.9 (0.6, 1.4)	0.9 (0.5, 1.4)
By race/ethnicity (GWG*ethnicity p = 0.52)				
Non-Hispanic white	1.5 (1.3, 1.7)	1.0	0.7 (0.6, 0.8)	0.5 (0.4, 0.6)
Non-Hispanic black	1.3 (1.1, 1.7)	1.0	0.9 (0.7, 1.1)	0.6 (0.4, 0.8)
Hispanic	1.6 (1.3, 2.1)	1.0	0.7 (0.5, 0.9)	0.6 (0.4, 0.8)
Asian	1.6 (1.2, 2.0)	1.0	0.8 (0.6, 1.0)	0.5 (0.3, 0.7)
By BMI and race/ethnicity (GWG*BMI*ethnicity p = 0.42)				
Non-Hispanic white				
<18.5 (underweight)	1.1 (0.6, 2.0)	1.0	0.5 (0.3, 0.9)	0.5 (0.2, 1.0)
18.5 – < 25 (normal weight)	1.5 (1.3, 1.9)	1.0	0.7 (0.6, 0.9)	0.5 (0.4, 0.6)
25 – <30 (overweight)	1.2 (0.9, 1.7)	1.0	0.4 (0.3, 0.7)	0.3 (0.2, 0.6)
30+ (obese)	1.8 (1.1, 2.8)	1.0	0.6 (0.3, 1.2)	0.8 (0.4, 1.7)
Non-Hispanic black				
<18.5 (underweight)	2.3 (0.9, 5.9)	1.0	1.0 (0.4, 2.5)	0.2 (0.02, 1.4)
18.5 – < 25 (normal weight)	1.2 (0.9, 1.7)	1.0	0.7 (0.5, 1.0)	0.4 (0.3, 0.6)
25 – <30 (overweight)	1.2 (0.8, 1.9)	1.0	0.9 (0.6, 1.6)	1.1 (0.6, 1.9)
30+ (obese)	2.1 (1.2, 3.8)	1.0	1.5 (0.7, 3.1)	1.4 (0.6, 3.4)
Hispanic				
<18.5 (underweight)	4.3 (1.1, 16.8)	1.0	1.7 (0.5, 5.9)	1.1 (0.3, 4.6)
18.5 – < 25 (normal weight)	1.3 (1.0, 1.9)	1.0	0.6 (0.4, 0.8)	0.5 (0.3, 0.7)
25 – <30 (overweight)	1.5 (1.0, 2.5)	1.0	0.7 (0.4, 1.3)	0.8 (0.4, 1.4)
30+ (obese)	2.1 (1.0, 4.1)	1.0	1.2 (0.5, 2.9)	0.6 (0.2, 2.2)
Asian				
<18.5 (underweight)	1.4 (0.6, 3.1)	1.0	0.7 (0.4, 1.3)	0.1 (0.01, 0.5)
18.5 – < 25 (normal weight)	1.7 (1.3, 2.3)	1.0	0.8 (0.6, 1.0)	0.7 (0.4, 1.1)
25 – <30 (overweight)	1.6 (0.9, 3.0)	1.0	0.9 (0.4, 2.0)	0.3 (0.04, 2.2)
30+ (obese)	0.1 (0.01, 1.2)	1.0	0.4 (0.05, 3.8)	n/c*

*Adjusted for maternal age, education, and smoking
**n/c = not calculable

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TABLE 9e Adjusted* Odds Ratios and 95% Confidence Intervals for the Association of Gestational Weight Gain with Term Large-for-Gestational Age among Singleton Births, New York City, 1995 – 2003, *n* = 31,760

	Gestational Weight Gain			
	0 – 9 kg	**10 – 14 kg**	**15 – 19 kg**	**20+ kg**
Overall	0.9 (0.8, 1.0)	1.0	1.4 (1.3, 1.5)	2.6 (2.3, 2.9)
By BMI (GWG*BMI p = 0.02)				
<18.5 (underweight)	0.4 (0.1, 1.8)	1.0	1.6 (0.8, 3.2)	5.1 (2.7, 9.8)
18.5 – < 25 (normal weight)	0.7 (0.6, 0.9)	1.0	1.5 (1.3, 1.7)	2.9 (2.5, 3.3)
25 – <30 (overweight)	0.6 (0.5, 0.7)	1.0	1.3 (1.1, 1.6)	2.3 (1.9, 2.7)
30+ (obese)	0.7 (0.6, 0.9)	1.0	1.5 (1.2, 1.9)	2.0 (1.5, 2.5)
By race/ethnicity (GWG*ethnicity p = 0.89)				
Non-Hispanic white	0.8 (0.6, 0.9)	1.0	1.5 (1.3, 1.7)	2.7 (2.3, 3.1)
Non-Hispanic black	0.6 (0.5, 0.7)	1.0	1.4 (1.2, 1.7)	2.2 (1.9, 2.7)
Hispanic	0.7 (0.5, 0.9)	1.0	1.4 (1.1, 1.9)	2.8 (2.1, 3.8)
Asian	0.7 (0.4, 1.1)	1.0	1.5 (1.0, 2.2)	3.2 (2.0, 5.0)
By BMI and race/ethnicity (GWG*BMI*ethnicity p = 0.16)				
Non-Hispanic white				
<18.5 (underweight)	n/c*	1.0	1.1 (0.4, 3.2)	4.5 (1.8, 11.4)
18.5 – < 25 (normal weight)	0.9 (0.7, 1.2)	1.0	1.5 (1.3, 1.8)	3.1 (2.6, 3.7)
25 – <30 (overweight)	0.5 (0.4, 0.8)	1.0	1.5 (1.2, 2.0)	2.4 (1.9, 3.2)
30+ (obese)	0.7 (0.5, 1.0)	1.0	1.6 (1.1, 2.2)	1.9 (1.3, 2.7)
Non-Hispanic black				
<18.5 (underweight)	0.5 (0.1, 4.3)	1.0	1.2 (0.3, 4.4)	4.8 (1.5, 15.4)
18.5 – < 25 (normal weight)	0.5 (0.3, 0.8)	1.0	1.5 (1.2, 2.0)	2.3 (1.8, 3.1)
25 – <30 (overweight)	0.6 (0.4, 0.8)	1.0	1.1 (0.9, 1.5)	2.4 (1.8, 3.3)
30+ (obese)	0.7 (0.5, 1.0)	1.0	1.5 (1.1, 2.2)	1.4 (0.9, 2.1)
Hispanic				
<18.5 (underweight)	n/c*	1.0	n/c*	n/c*
18.5 – < 25 (normal weight)	0.4 (0.2, 0.9)	1.0	1.6 (1.0, 2.4)	3.2 (2.1, 4.8)
25 – <30 (overweight)	0.8 (0.4, 1.4)	1.0	1.3 (0.7, 2.2)	1.5 (0.8, 2.6)
30+ (obese)	0.7 (0.3, 1.3)	1.0	1.1 (0.5, 2.4)	4.7 (2.4, 9.2)
Asian				
<18.5 (underweight)	n/c*	1.0	3.6 (0.7, 18.2)	5.8 (0.9, 35.9)
18.5 – < 25 (normal weight)	0.7 (0.3, 1.4)	1.0	1.5 (0.9, 2.4)	3.1 (1.8, 5.4)
25 – <30 (overweight)	0.6 (0.3, 1.5)	1.0	0.7 (0.3, 2.0)	1.9 (0.6, 6.1)
30+ (obese)	0.6 (0.1, 3.6)	1.0	4.3 (0.8, 21.9)	13.6 (2.2, 83.6)

*Adjusted for maternal age, education, and smoking
**n/c = not calculable

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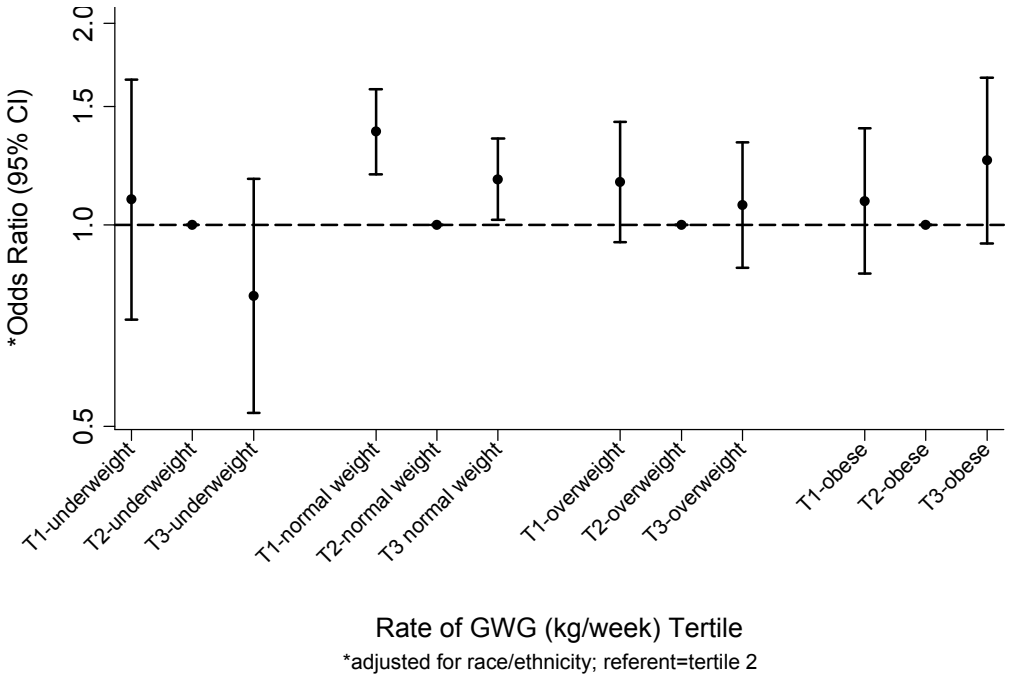

FIGURE 1 GWG and Preterm Birth, < 37 weeks by BMI

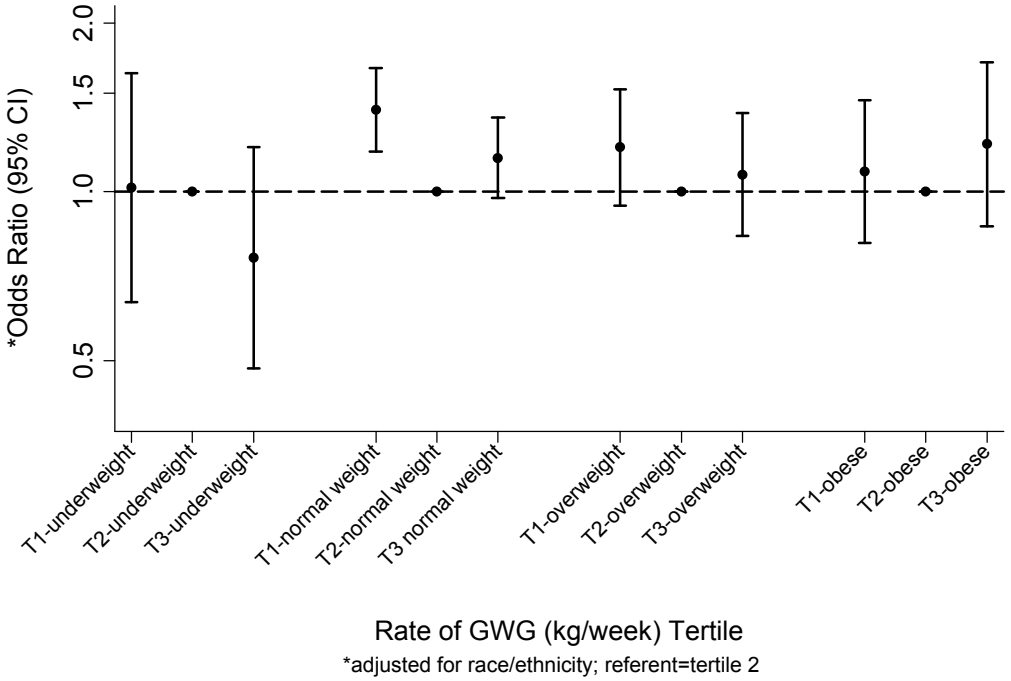

FIGURE 2 GWG and Spontaneous Preterm Birth, < 37 Weeks by BMI

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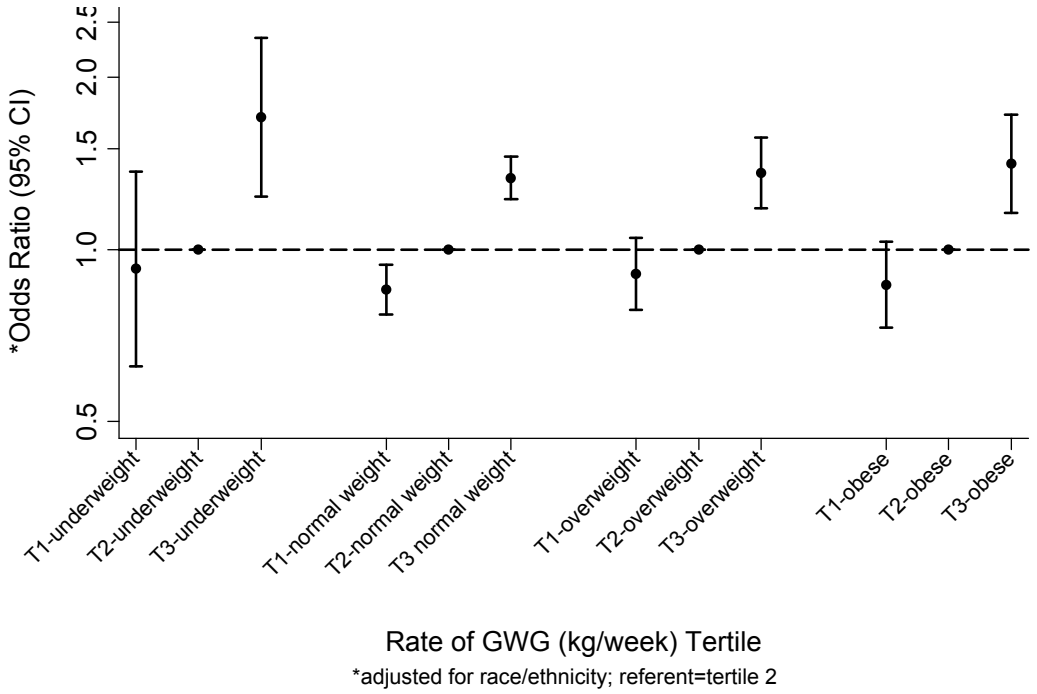

FIGURE 3 GWG and Cesarean Section by BMI

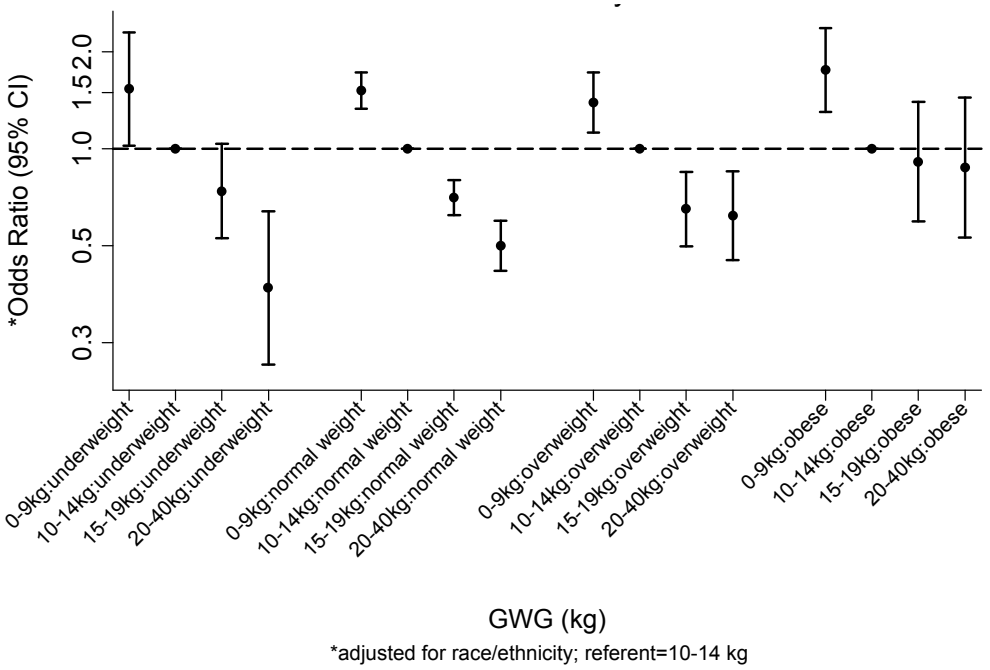

FIGURE 4 GWG and Term SGA by BMI

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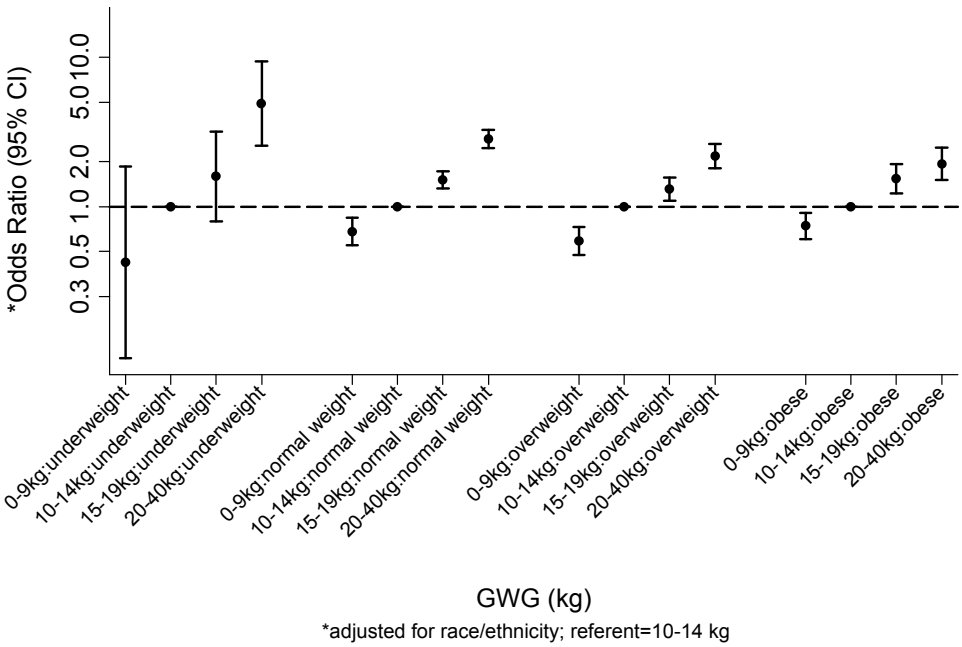

FIGURE 5 GWG and Term LGA by BMI

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PART IV: ANALYSES FROM DR. HAMMITT

OPTIMAL GESTATIONAL WEIGHT GAIN: RISK TRADEOFF CALCULATIONS

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The risks of multiple adverse pregnancy outcomes to mother and child are associated with the mother's gestational weight gain (GWG) and pregravid body mass index (BMI). The prevalence of some outcomes (e.g., prematurity, small-for-gestational age [SGA]) are more strongly associated with small GWG while others (e.g., large-for-gestational age [LGA]), childhood obesity, post-partum weight retention) are more strongly associated with large GWG. In formulating guidance about appropriate GWG, it is necessary to consider how to balance increasing risks of some outcomes against decreasing risks of others. To assist this consideration, a quantitative analysis of risk tradeoffs was performed.

Based on discussion with the Committee to Reexamine IOM Pregnancy Weight Guidelines, three outcomes were considered: infant mortality, postpartum weight retention (PPWR), and childhood obesity. These endpoints were selected because they were believed to be quantitatively important and to be reasonably estimable with available data. (In this context, quantitative importance requires that the occurrence of each outcome has significant effects on health and the probability of occurrence varies significantly with GWG.) Other outcomes (e.g., SGA, LGA) were not quantified in part because estimating the effect of these outcomes on health (i.e., ensuing morbidity and mortality) was judged to be too difficult or speculative given available data and resources.

The analysis was framed by estimating how the probability of each outcome varies with GWG controlling for pregravid BMI category (using the WHO categories: underweight < 18.5, normal 18.5–24.9, overweight 25–29.9, and obese ≥ 30 kg/m^2). These estimates are obtained from observational epidemiological data and assume that the observed associations are causal.

For each endpoint, the expected number of quality-adjusted life years (QALYs) lost over the lifetime of the mother and child was estimated. QALYs are a standard measure of health that combined length of life and quality of health. They are defined as the sum of the time spent in each health state weighted by the health-related quality of life (HRQL) associated with that state. HRQL is a measure of the quality or utility associated with a health state, normalized so that perfect health takes a value of one and a health state equivalent to dead has a value of zero (health states that are viewed as worse than dead may be assigned values smaller than zero). Summing across endpoints (weighted by their probabilities of occurrence) yields an estimate of the total expected number of QALYs lost from these three outcomes. The use of this metric implies that the health impairments of different outcomes, occurring to mothers and children, are appropriately judged by comparing the corresponding expected losses in QALYs. The use of expected QALYs to evaluate health effects within and among individuals is common in health economics and public health, but not without controversy (see, e.g., IOM, 2006).

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The following subsections describe the data used to estimate the probabilities and QALYs lost for each outcome. The final section reports the results of summing the estimated health losses across outcomes.

Infant Mortality

Infant mortality was chosen as an outcome measure that aggregates many of the pathways through which inadequate or excessive GWG may lead to fatal outcomes. Its use is convenient because infant death is clearly more significant than many other birth outcomes and by aggregating across pathways one avoids the necessity of detailed modeling associated with how various outcomes (e.g., SGA) lead to infant fatality.

Prevalence

Two estimates of the prevalence of infant mortality as a function of GWG and pregravid BMI are available: one by Chen et al. (2008) and a second conducted for the Committee by Amy Herring. Both estimates use data from the 1988 U.S. National Maternal and Infant Health Survey (NMIHS). Within BMI class, Chen et al. (2008) estimate total infant mortality prevalence among live births for each of four classes of GWG gain (< 0.15, 0.15–0.29, 0.30–0.45, ≥ 0.45 kg/wk). These were converted to full-term GWG gain by multiplying by 40, yielding the following classes: < 6, 6–12, 12–18, ≥ 18 kg. At the Committee's request, Herring estimated infant mortality rate excluding congenital defects (that are believed to be unrelated to GWG) and restricting the NMIHS sample to term births. She estimated prevalence using the four BMI classes and seven GWG classes (< 0, 0–5, 5–10, 10–15, 15–20, 20–25, ≥ 25 kg). The overall infant mortality rate in the NMIHS data is 1.0/100 live births, substantially larger than the current US value of 0.64/100. To convert to current values, all of the estimates of prevalence were multiplied by 0.64, which assumes a constant proportional improvement in infant mortality rate across BMI/GWG classes.

The Chen et al. (2008) and Herring estimates of infant mortality by GWG classes were converted to continuous functions of GWG by fitting polynomial functions to the estimated prevalence for the midpoints of the GWG categories (for open intervals a typical value was assumed). The polynomial functions are saturated, including as many terms as are estimable from the categorical estimates (i.e., third order for the Chen et al. estimates, sixth order for the Herring estimates). As a consequence, these polynomial functions exactly reproduce the observations to which they are fit. (These polynomial functions are best viewed as smoothed curves fit to the underlying categorical estimates rather than as statistical models of the relationship between infant mortality and GWG). The categorical estimates are reported in Tables 1a and 1b. Note that the Herring analysis shows that infant mortality is lower at the two extreme points than at the adjacent GWG categories (i.e., for the smallest weight gain category among underweight women and for the largest weight gain category among obese women). These departures from the anticipated J- or U-shaped relationship between GWG and infant mortality seem implausible and may reflect limited data at the extreme points or artifacts of model estimation.

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TABLE 1A Infant Mortality (Chen et al., 2008)

BMI	GWG Rate (kg/wk)	GWG (kg in 40 wk)	Prevalence (%)
Underweight	< 0.15	< 6	1.98
(< 18.5)	0.15–0.29	6–12	0.86
	0.30–0.45	12–18	0.66
	≥ 0.45	≥ 18	0.53
Normal	< 0.15	< 6	1.28
(18.5–24.9)	0.15–0.29	6–12	0.64
	0.30–0.45	12–18	0.50
	≥ 0.45	≥ 18	0.54
Overweight	< 0.15	< 6	0.88
(25.0–29.9)	0.15–0.29	6–12	0.59
	0.30–0.45	12–18	0.63
	≥ 0.45	≥ 18	0.72
Obese	< 0.15	< 6	0.87
(≥ 30.0)	0.15–0.29	6–12	0.71
	0.30–0.45	12–18	0.79
	≥ 0.45	≥ 18	1.41

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TABLE 1B Infant Mortality (Herring)

BMI	GWG (kg)	Prevalence (%)
Underweight	< 0	2.60
(< 18.5)	0 – 4.9	3.12
	5 – 9.9	1.15
	10 – 14.9	0.46
	15 – 19.9	0.44
	20 – 24.9	0.27
	≥ 25	0.61
Normal	< 0	1.66
(18.5 – 24.9)	0 – 4.9	1.40
	5 – 9.9	0.80
	10 – 14.9	0.45
	15 – 19.9	0.39
	20 – 24.9	0.39
	≥ 25	0.44
Overweight	< 0	1.30
(25 – 29.9)	0 – 4.9	0.83
	5 – 9.9	0.67
	10 – 14.9	0.56
	15 – 19.9	0.56
	20 – 24.9	0.44
	≥ 25	0.47
Obese	< 0	1.15
(≥ 30)	0 – 4.9	0.93
	5 – 9.9	0.83
	10 – 14.9	0.54
	15 – 19.9	0.65
	20 – 24.9	1.02
	≥ 25	0.50

QALYs Lost

Infant mortality implies the child's entire lifetime is lost. A value of 80 QALYs is assumed, consistent with current life expectancy at birth. In principle, one could adjust this figure downward to recognize that not all years of life are lived in perfect health (especially at older ages), but adjustment for this factor is viewed as negligible in comparison with other uncertainties and approximations in the risk tradeoff calculations. The figure might also be adjusted downward if it is considered appropriate to discount the value of future life years.

Post-partum Weight Retention (PPWR)

Prevalence

Prevalence estimates were provided by Ellen Nohr using data from the Danish National Birth Cohort (Nohr et al., 2008). For this analysis, PPWR is defined as retention of at least 5 kg body

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mass 6 months after birth. Prevalence estimates were provided for four GWG classes (< 10, 10 – 15, 16 – 19, ≥ 20 kg), as reported in Table 2. Third order polynomial functions were fit to these estimates.

TABLE 2 Post-partum Weight Retention (Nohr)

BMI	GWG (kg)	Prevalence (%)
Underweight	< 10	7.9
(< 18.5)	10-15	13.1
	16-19	27.6
	≥ 20	46.5
Normal	< 10	5.6
(18.5 – 24.9)	10-15	13.0
	16-19	26.1
	≥ 20	49.7
Overweight	< 10	7.2
(25 – 29.9)	10-15	16.9
	16-19	31.1
	≥ 20	53.2
Obese	< 10	5.1
(≥ 30)	10-15	17.5
	16-19	33.0
	≥ 20	45.0

QALYs Lost

The effects of PPWR on morbidity and mortality are estimated on the assumption that weight retained post-partum is retained for the rest of a woman's life and using estimates of how mortality and health-related quality of life vary with BMI. First, average retained weight conditional on retaining at least 5 kg at 6 months post-partum is estimated as 10 kg (based in part on data from committee member Barbara Abrams suggesting that roughly half of women who retain at least 5 kg retain at least 10 kg). The incremental effect on BMI of a 10 kg weight increase is 3.7, calculated using a nominal average height (5 foot 5 inches).

Mortality The effect of increased BMI on mortality is calculated using estimates from Peeters et al. (2003) cited by Hu (2008). Using data from the Framingham heart study, they estimated that an average 40 year old female nonsmoker loses 3.3 years of life if overweight and 7.1 years if obese. Using midpoint values of BMI for normal, overweight, and obese (assumed value = 33), a 1 point increment to BMI is associated with about 0.6 life years lost, and so the effect of a 3.7 point BMI increment is estimated as 2.2 years (this is the average of the slopes estimated by comparing overweight and obese with normal weight, 2.1 and 2.3, respectively). This effect is applied only to women with pregravid BMI in the overweight and obese categories. No account is taken of any possible beneficial effect of weight gain on mortality of underweight women.

Morbidity Jia and Lubetkin (2005) used data from the U.S. Medical Expenditure Panel Survey (MEPS) to estimate how HRQL varies with BMI class. The MEPS includes two measures of individual's current HRQL obtained using the EQ-5D and EQ-VAS. The EQ-5D is a standard instrument used to estimate HRQL based on classification of health into one of three levels (no

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problem, some problem, severe problem) on each of five dimensions or attributes (mobility, self care, usual activities, pain/discomfort, anxiety/depression). The EQ-VAS is an example of a visual analog scale, another common instrument on which respondents mark a point on a visual scale (or report a number on the scale) that they associate with their health state. Jia and Lubetkin (2005) report regression estimates of the partial effect of BMI class on each measure of HRQL, controlling for age, income, race/ethnicity, physical activity, presence of each of several diseases (asthma, hypertension, diabetes, heart disease, stroke, emphysema), and other factors. Compared with normal BMI, the estimated loss in HRQL is 0.013 (EQ-5D) and 0.0052 (VAS) for overweight, 0.033(EQ-5D) and 0.0323 (VAS) for obesity class I, and 0.073 (EQ-5D) and 0.0494 (VAS) for obesity class II (note: class I and II obesity are distinguished by BMI < 30 and ≥ 30). The total effect of higher BMI on HRQL is presumably larger than these estimates because some of the diseases for which Jia and Lubetkin control in their regression models are likely consequences of higher BMI; to adjust for this bias, the partial effects are multiplied by two.

Assuming these HRQL increments persist for the remainder of the woman's life (estimated as 50 years) and using midpoint values of BMI within BMI class (assumed value = 37 for obese class II) suggests QALY losses associated with a BMI increment of 5.7 equal 0.9 and 2.0 for overweight and obese women, respectively (the value for obese women is an average of the values for obese class I and obese class II, 1.7 and 2.3, respectively).

Summing the estimates for morbidity and mortality implies that each case of PPWR is associated with expected values of 3.1 and 4.2 QALYs lost for overweight and obese women, respectively.

Childhood Obesity

Prevalence

The relative risk of childhood obesity was estimated by committee member Matt Gillman as 1.2 per 5 kg increment in GWG for all maternal BMI groups. This result is based primarily on the Oken et al. (2008) GUTS analysis, supported by results from Wrotniak (2008) and Monteiro (2007). This estimate is for childhood obesity defined as BMI above the 95th percentile compared with below the 85th percentile for age, observed at ages 9 to 14 years. Prevalence of childhood obesity by maternal pregravid BMI category for the Oken et al. (2008) analysis is 1.9, 5.2, 12.7, and 24.6 percent for underweight, normal, overweight, and obese, respectively. The probability of childhood obesity by GWG conditional on BMI was calculated using the estimated relative risk, the prevalence by BMI category, and information on the joint distribution of GWG and BMI from Chen (supplemental material Table 1b assuming a common ratio of deaths to controls across BMI/GWG classes). (Note that the resulting population prevalence of 7.3 percent exceeds Oken's reported population prevalence of 6.5 percent.) Third order polynomials were fit to these estimates (reported in Table 3).

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TABLE 3 Childhood Obesity

BMI	GWG Rate (kg/wk)	GWG (kg in 40 wk)	Prevalence (%)
Underweight	< 0.15	< 6	1.46
(< 18.5)	0.15 – 0.29	6 – 12	1.52
	0.30 – 0.45	12 – 18	1.89
	≥ 0.45	≥ 18	2.34
Normal	< 0.15	< 6	4.06
(18.5 – 24.9)	0.15 – 0.29	6 – 12	4.23
	0.30 – 0.45	12 – 18	5.21
	≥ 0.45	≥ 18	6.40
Overweight	< 0.15	< 6	10.4
(25 – 29.9)	0.15 – 0.29	6 – 12	10.8
	0.30 – 0.45	12 – 18	13.2
	≥ 0.45	≥ 18	15.9
Obese	< 0.15	< 6	22.3
(≥ 30)	0.15 – 0.29	6 – 12	23.0
	0.30 – 0.45	12 – 18	27.1
	≥ 0.45	≥ 18	31.7

QALYs lost

Mortality Engeland et al (2003) analyzed Norwegian data on mortality as a function of adolescent obesity (at ages 14 to 19 years). With average follow-up exceeding 30 years, they estimate that adult mortality rates from about age 30 onward are 80 percent larger for males and 100 percent larger for females whose adolescent BMI exceeded the 95th percentile of a US reference population compared with those whose adolescent BMI was less than the 85th percentile. Adjusting a current US life table to increase age-specific mortality rates by 90 percent for all ages from 30 onward suggests about 7 years of life lost (i.e., life expectancy at birth falls from about 77 to 70 years). Hence childhood obesity is estimated to lead to 7 QALYs lost to mortality (implicitly assuming that BMI above the 95th percentile at ages 9 to 14 years persists to ages 14 to 19 years).

Morbidity QALYs lost to morbidity are estimated using the results for morbidity associated with PPWR above. Childhood obesity defined as BMI above the 95th percentile is assumed to persist as adult obesity (BMI ≥ 30) and to persist for 70 years. Adjusting the estimated value of 2.0 QALYs lost for morbidity associated with PPWR among obese women for the difference in duration (i.e., multiplying by 70/50) yields 2.8 QALYs.

Summing the estimates of mortality and morbidity effects yields an expected value of 9.8 QALYs lost per case of childhood obesity.

Results

The expected QALYs lost due to infant mortality and the mortality and morbidity consequences of post-partum weight retention and childhood obesity for each maternal BMI category and value of GWG are estimated by multiplying the estimated prevalence of each endpoint by the associated expected value of QALYs lost. Results are summarized in Figure 1

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using the Chen et al. (2008) estimates of infant mortality and in Figure 2 using the Herring estimates.

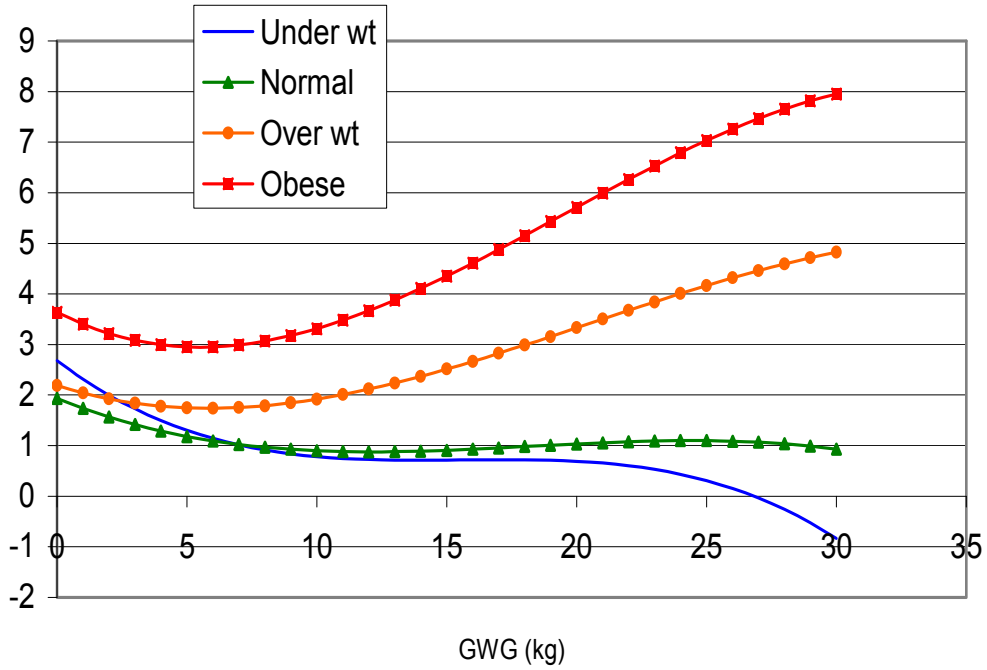

FIGURE 1 Total expected QALYs lost (Chen et al. [2008] mortality estimates)

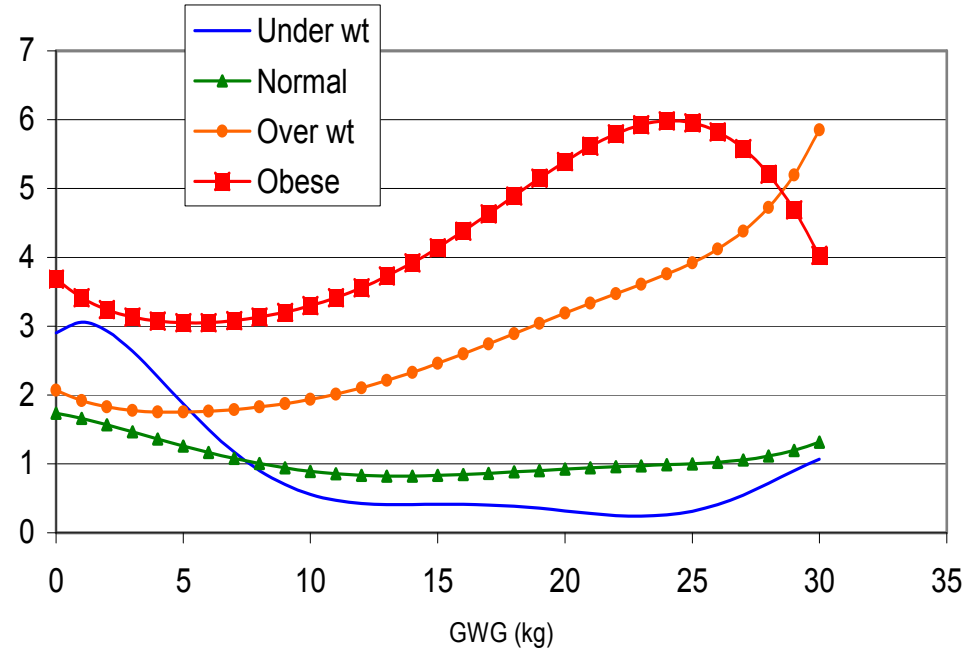

FIGURE 2 Total expected QALYs lost (Herring infant mortality estimates)

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The conclusions are similar using both sets of infant mortality estimates. For overweight and obese women, the estimated total mortality and morbidity consequences for mother and child of the endpoints included in this analysis are minimized for GWG less than about 10 to 15 kg. For normal and underweight women, estimated mortality and morbidity consequences are minimized for GWG greater than about 10 to 15 kg. Within these ranges, estimated total QALY losses are not very sensitive to GWG. In Figure 2, the prominent departure from a trend for obese women at high GWG, and the less prominent departure from a trend for underweight women at low GWG reflect the surprisingly low estimates of infant mortality prevalence for these categories shown in Table 1b. As noted above, these departures from the trend toward increasing infant mortality with very low or very high GWG may reflect limited data for these categories or modeling artifacts. Similarly, the trend toward negative QALY losses for high GWG among underweight women shown in Figure 1 is also likely to reflect limited data and possible model artifacts associated with extrapolation beyond the range of observations.

The vertical scale suggests that the expected loss of quality-adjusted life years per live birth varies from near zero for normal weight and underweight women who experience adequate gestational weight gain to five or more for overweight and obese women who experience substantial gestational weight gain. These values suggest the scale of the public health problem associated with overweight women and excessive gestational weight gain – the average loss may be on the order of 5 to 10 percent of the total lifetime QALYs experienced per birth.

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