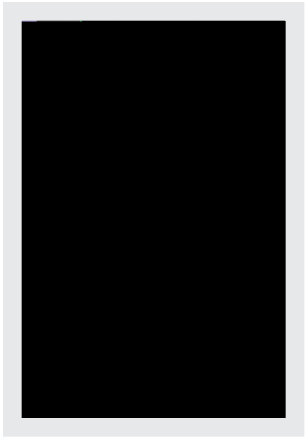

DETAILS

CONTRIBUTORS

SUGGESTED CITATION

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
Board on Children, Youth, and Families

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NOTICE: The project that is the subject of this report was approved by the Governing Board of the National Research Council, whose members are drawn from the councils of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine. The members of the committee responsible for the report were chosen for their special competences and with regard for appropriate balance.

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

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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.

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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.

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.

Development; National Institutes of Health National Institute of Diabetes and Digestive and Kidney Diseases; U.S. Department of Health and Human siirtxt (>>7.1771.656 0 Td( )TjEM410.567 0 OofiSpan <</Actualn an ipme'es

ated. We also thank Leslie Pray for technical editing and Florence Poillon for copyediting. Both the Director of the Food and Nutrition Board, Linda Meyers, and the Director of the Board on Children, Youth, and Families,

Contents

* Appendixes A through G are not printed in this book, but can be found on the CD at the

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

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

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.

The committee began its work by considering appropriate BMI cutoff points and describing trends over time in maternal prepregnancy BMI and

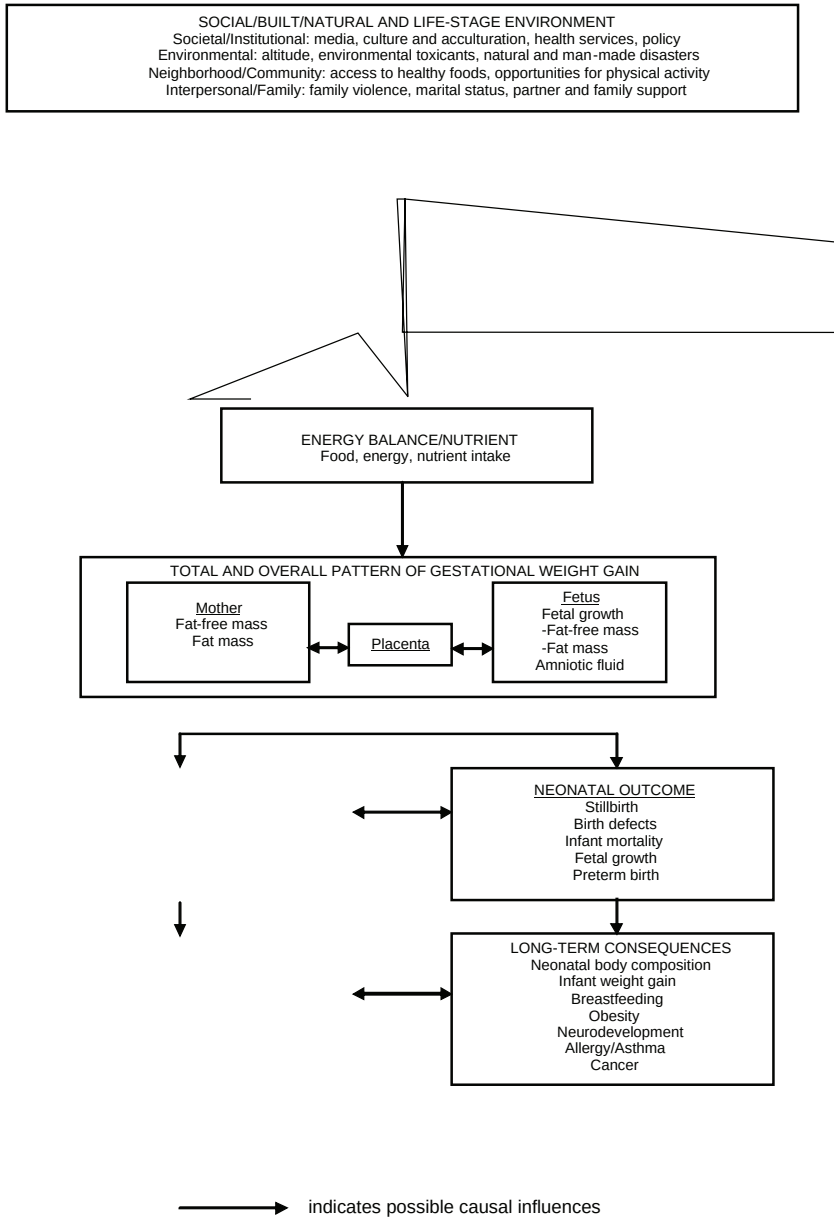

FIGURE S-1 Schematic summary of potential determinants and consequences for gestational weight gain.
SOURCE: Modified from IOM, 1990.

during pregnancy. In addition, the committee recommends that researchers should conduct studies on the effects of weight loss or low GWG, including periods of prolonged fasting and the development of ketonuria/ketonemia

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

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 health care providers in doing this, the committee has prepared charts that could be used as a basis for this discussion with the pregnant woman. These charts are

REFERENCES

Abrams B., S. Carmichael and S. Selvin. 1995. Factors associated with the pattern of maternal weight gain during pregnancy. *Obstetrics and Gynecology* 86(2): 170-176.

ACOG (American College of Obstetricians and Gynecologists). 2002. ACOG committee opinion. Exercise during pregnancy and the postpartum period. Number 267, January 2002. American College of Obstetricians and Gynecologists. *International Journal of Gynaecology and Obstetrics* 77(1): 79-81.

Carmichael S., B. Abrams and S. Selvin. 1997. The pattern of maternal weight gain in women with good pregnancy outcomes. *American Journal of Public Health* 87(12): 1984-1988.

IOM (Institute of Medicine). 1990. *Nutrition During Pregnancy.*

1

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 health care sector to provide optimal guidance to women before, during, and after pregnancy so that they can achieve healthy outcomes for both themselves and their newborns. During this time, two reports have contributed to providing this guidance.

The first 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 limiting3(a)-80(27-3(d)-)-3(c)-

groups, women of short stature, and women carrying twins; and detailed historic trends in weight gain recommendations and guidelines. The IOM (1990) recommendations for weight gain during pregnancy have been adopted by or have been influential in many countries. Observational studies have demonstrated that women who enter pregnancy at a normal BMI and gain within the recommended ranges 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; Groth, 2006).

In the years since the release of the weight gain recommendations from the IOM (1990) report, however, some dramatic shifts in the demographic and epidemiologic profile of the U.S. population have occurred. Notably, the population of U.S. women of childbearing age has become more diverse; and prepregnancy BMI and excess GWG have increased across all population groups, particularly among minority groups who are already at risk for poor maternal and child health outcomes (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 necessary.

RATIONALE FOR REVISING THE GUIDELINES

General Principles Framing the IOM (1990) Pregnancy Weight 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, the 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, 2009]) 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: An Implementation Guide* (IOM, 1992), identified specific actions practitioners could take to achieve the recommendations in working with patients. They also identified a series of

(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 goal of this group was to determine whether new research provided a basis for practitioners to change guidance for GWG and 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), *Nutrition During Lactation* (1991), and *Nutrition During Pregnancy and Lactation: An 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). Additionally, 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).

Also since publication of the IOM (1990) report and the subsequent 1991 and 1992 reports, research on GWG has demonstrated that weight patterns (underweight and overweight) and total weight gain 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 mellitus (GDM), cesarean delivery (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).

dations for (1) the health of the mother, particularly for women who are overweight, underweight, older, adolescent, or short in stature; (2) infant and child health; and (3) 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 health care 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:

- 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 health care seighlt2acaras3(h)4(l)ltaarlghpigre oalt2oeiglt2oehpinm

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

The summary report from that workshop, *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 health care 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.

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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, health care, and health systems;
    - timing and components of interventions; and
    - 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.

5. Identify gaps in knowledge and recommend research priorities.

Approach to the Task

The committee approached its task by gathering information from existing scientific literature, including a systematic review of the literature by the Agency for Healthcare Research and Quality (AHRQ) (Viswanathan

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

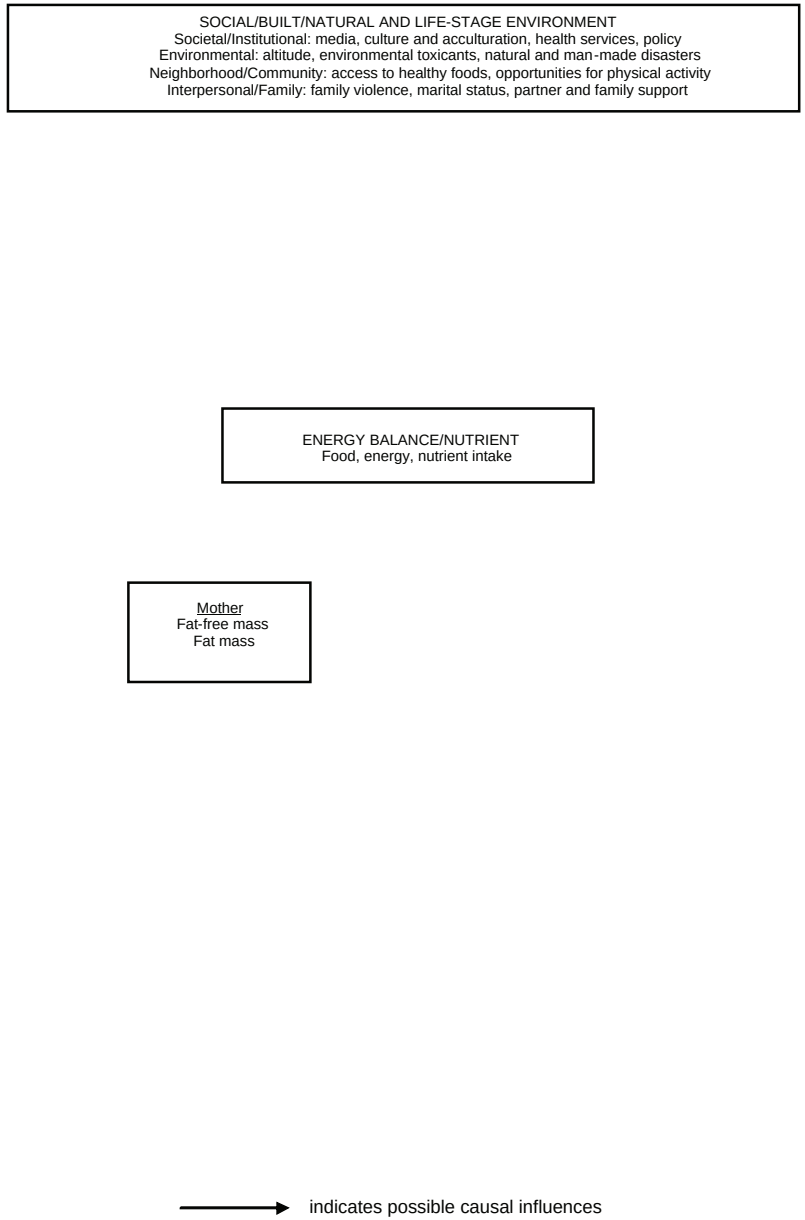

FIGURE 1-1 Schematic summary of potential determinants and consequences for gestational weight gain.
SOURCE: Modified from IOM, 1990.

below recommended levels; identifies data gaps; and makes recommendations based on the committee's findings.

The report begins, in this chapter, 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).

Trends in GWG since the time of the IOM (1990) report are considered in Chapter 2, with particular attention to weight gain in racial or ethnic subgroups of the U.S. population. The information reviewed in this chapter helped guide the committee's recommendations for assisting women in achieving the new GWG guidelines presented in Chapter 7.

The 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.

REFERENCES

Abenhaim H. A., R. A. Kinch, L. Morin, A. Benjamin and R. Usher. 2007. Effect of prepregnancy body mass index categories on obstetrical and neonatal outcomes. *Archives of Gynecology and Obstetrics* 275(1): 39-43.

Abrams B., S. L. Altman and K. E. Pickett. 2000. Pregnancy weight gain: still controversial.

Ogden C. L., M. D. Carroll, L. R. Curtin, M. A. McDowell, C. J. Tabak and K. M. Flegal. 2006. Prevalence of overweight and obesity in the United States, 1999-2004. *Journal of the American Medical Association* 295(13): 1549-1555.

Oken E., E. M. Taveras, K. P. Kleinman, J. W. Rich-Edwards and M. W. Gillman. 2007. Gestational weight gain and child adiposity at age 3 years. *American Journal of Obstetrics and Gynecology* 196(4): 322 e321-e328.

Suitor C. W. 1997. *Maternal Weight Gain: A Report of an Expert Work Group*. Arlington, VA: National Center for Education in Maternal and Child Health.

Taffel S. M., K. G. Keppel and G. K. Jones. 1993. Medical advice on maternal weight gain and actual weight gain. Results from the 1988 National Maternal and Infant Health Survey. *Annals of the New York Academy of Sciences* 678: 293-305.

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.

WHO (World Health Organization). 1995. Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. *World Health Organization Technical Report Series* 854: 1-452.

2

Descriptive Epidemiology and Trends

The committee began its reexamination of the Institute of Medicine (IOM) (1990) recommendations for weight gain during pregnancy by evaluating trends since 1990 in both prepregnancy maternal body mass index (BMI) and gestational weight gain (GWG). As described in detail in Chapter 3, prepregnancy BMI and GWG are interrelated. When evaluating trends in GWG, the committee considered whether women were gaining

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

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 ≥ 25 kg/m^2), and almost one-third were obese (BMI ≥ 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.

	1976-1980	1988-1994	1999-2004
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, Maryland, December 2, 2008.

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; among the latter, 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.

Because of these trends, 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 past decade (Kim et al., 2007) (Figure 2-1). More specifically, although the prevalence of overweight has increased only 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.

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

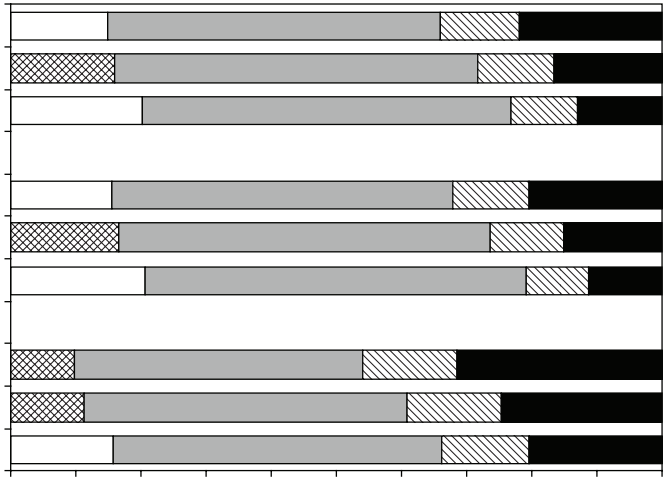

FIGURE 2-1 Trends in the distribution of BMI[a] from 1993 to 2003 among prepregnant U.S. women in the total population and by race or ethnicity.

[a]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.

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 IOM (1990) report. The WHO BMI categories also include several grades or categories of obesity (see Table 2-2).

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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

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

Required Data

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.

Weight Gain Relative to Prepregnancy BMI

Unfortunately, the standard birth certificate lacks data on maternal prepregnancy weight and height. Thus, data from this source cannot pro-

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	8 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.

vide information about GWG relative to prepregnant BMI category. Birth certificate data may yield more useful statistics for weight gain surveillance in the near future. The IOM (1990) report called for collection of maternal prepregnancy weight and height, and 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, permit identification of trends if -3(y)-3( )m3( )m3( )treitef weighft gatesf c-3( )-164(a)-3(b)-3

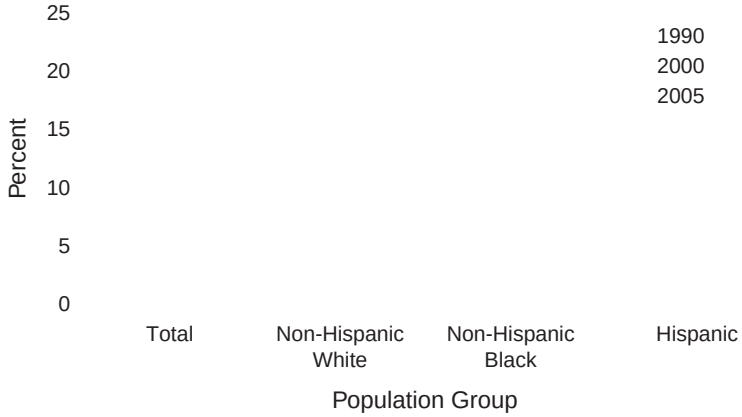


FIGURE 2-4 Percentage of women in the United States who gained more than 40 pounds during pregnancy, by race or ethnicity of the mother, 1990, 2000, and 2005.

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.

postpartum period. Currently, 37 states, New York City and the Yankton Sioux Tribe (South Dakota) participate in PRAMS (available online at

above the 15-pound recommended minimum. Similar trends were observed in 1992-1993 and 1998 (data not shown).

In 2002-2003, nearly half of underweight women represented in the PRAMS data gained within the range recommended by the 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.

Data Obtained from PNSS

The only other large U.S. data source on GWG and prepregnancy BMI, PNSS, collects data on low-income women participating in public health programs (predominantly the U.S. Department of Agriculture's [USDA's] Special Supplemental Nutrition Program for Women, Infants, and Children [WIC]) from 26 states, 5 tribal governments, and 1 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 the IOM (1990), but the data were not stratified by pregravid BMI. In this analysis, the data also were not limited to singleton, term pregnancies. Given these limitations, the data from PNSS show that from 1997 to 2007 in the total population of participating women, the proportion who gained within the range recommended by the IOM (1990) changed very little (Figure 2-8). Indeed, only about 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 women 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

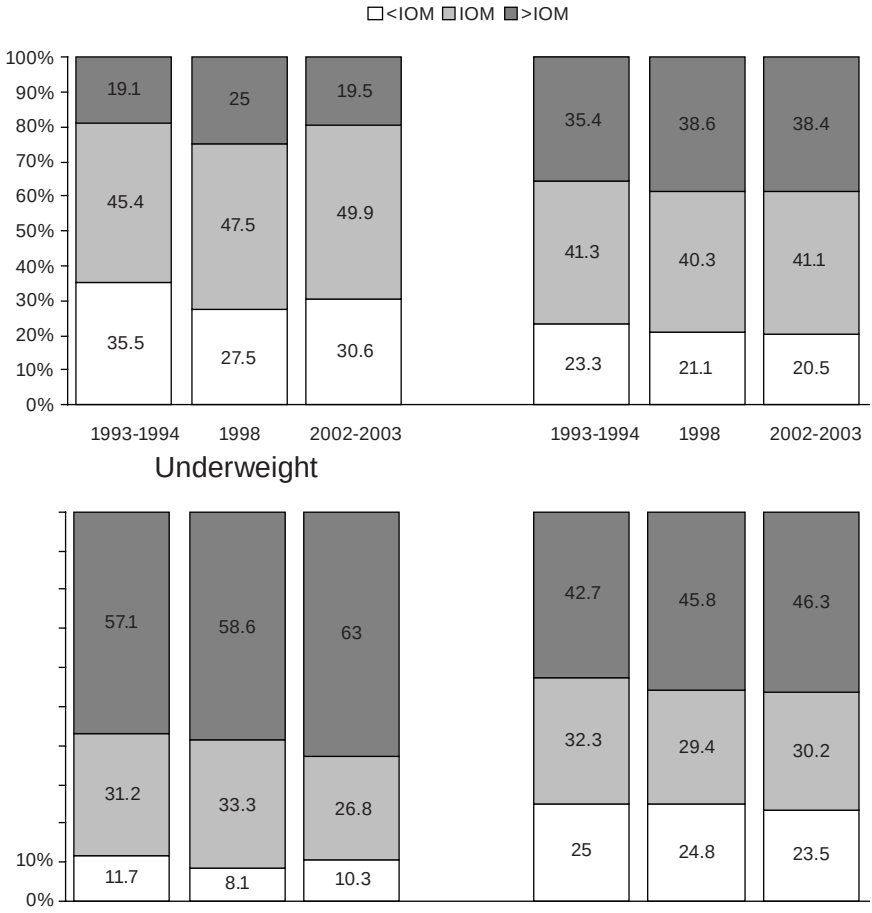
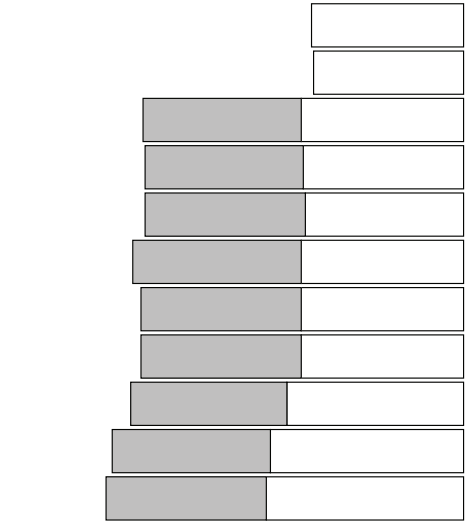


FIGURE 2-7 Distribution of gestational weight gain by prepregnancy BMI category among singleton, term deliveries from 1993 to 2003.
NOTE: IOM BMI categories were used (underweight [lean], < 19.8 kg/m^2; normal, 19.8 to 26.0 kg/m^2; overweight, 26.1 to 29.0 kg/m^2; obese, > 29.0 kg/m^2).
SOURCE: Information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



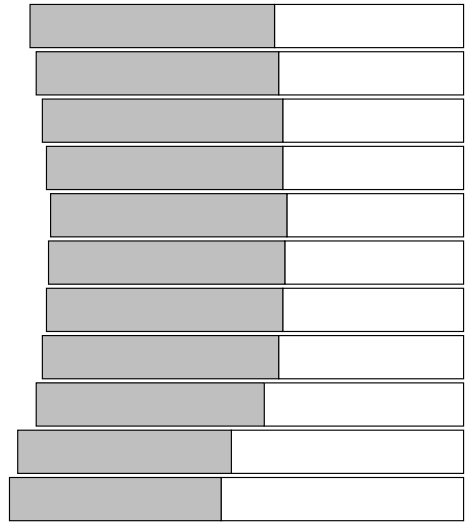


FIGURE 2-8 Distribution of gestational weight gain from 1997 to 2007 by pregravid BMI.
NOTE: BMI based on IOM categories (underweight [lean], <19.8 kg/m^2; normal, 19.8 to 26.0 kg/m^2; overweight, 26.1 to 29.0 kg/m^2; obese, >29.0 kg/m^2).
SOURCES: Personal communication, A. Sharma, CDC, Atlanta, Georgia, 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].

Concluding Remarks

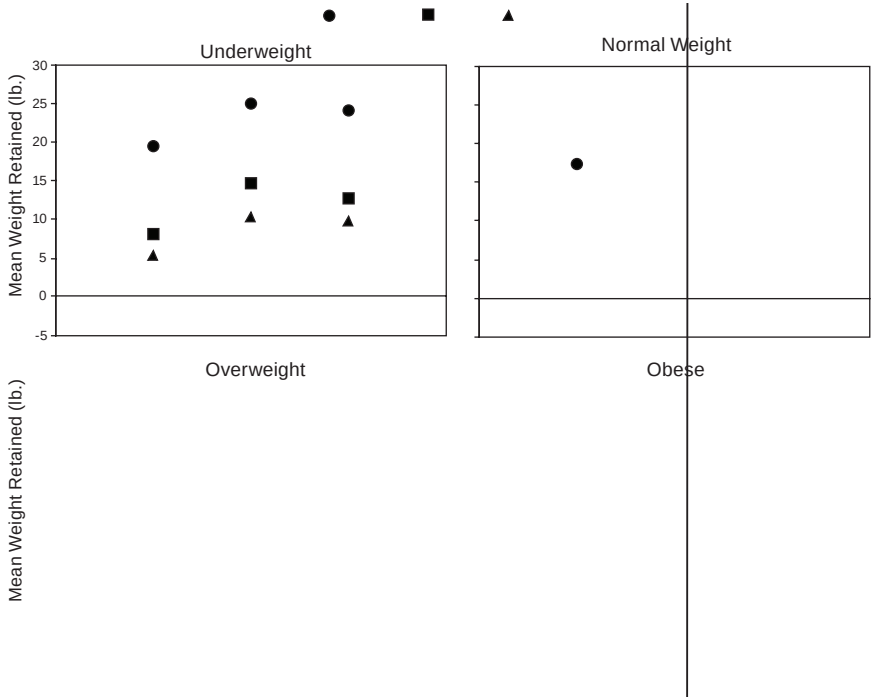
Taken together, data from PRAMS and PNSS illustrate that less than half of the women l-3(a)ss8(o)-3(f)-3( )-1p8(t)-3(h)p-3(S)-3(l-3(t)-3(e)-3(i0 0 0 10 54

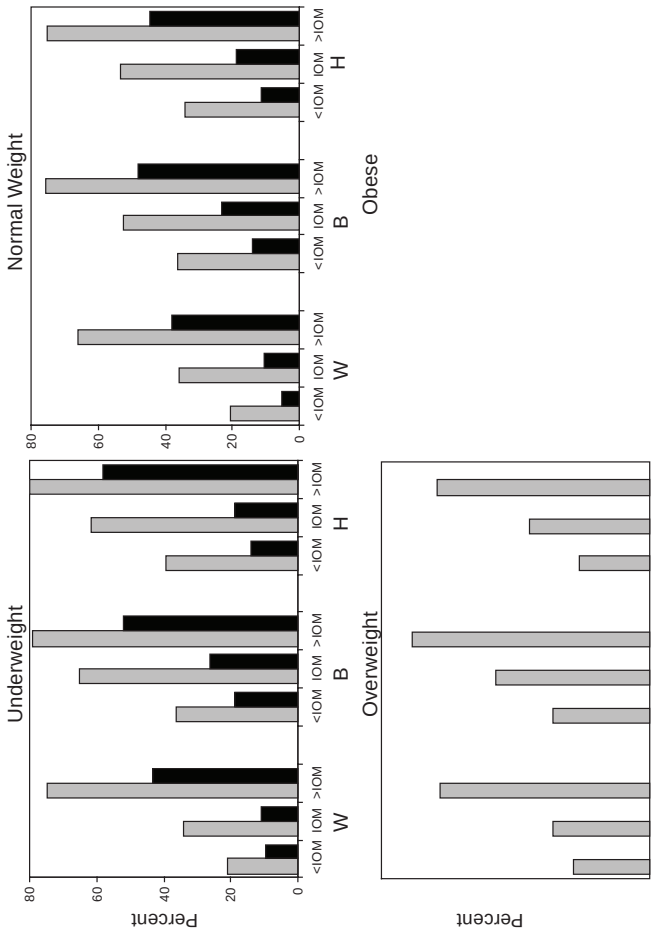
postpartum weight retention as a function of both prepregnancy body size (e.g., BMI) and adequacy of GWG.

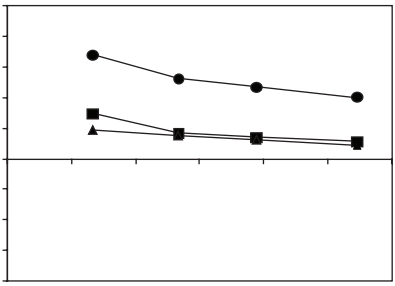
Unfortunately, data on maternal postpartum weights are not widely available, particularly for times later in the year after birth; this is different than during pregnancy, when maternal weight is monitored and routinely recorded in the clinical record. The committee used two sets of data for its evaluation of postpartum weight retention: PNSS, which was described earlier in the discussion on GWG trends, and the Infant Feeding Practices Study II (IFPS II).

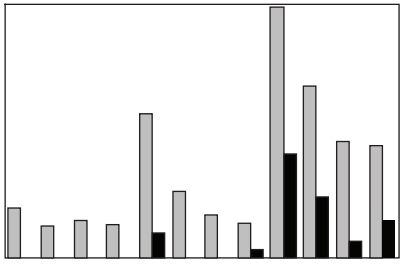
Data Obtained from PNSS

In addition to the data on GWG, PNSS 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. However, only about 49,000 of these 1.4 million records 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, Georgia, December 2008); the committee's analysis was restricted to data collected at 24 weeks' postpartum or later. Notably, PNSS data are not nationally representative, and the women with postpartum records at >









Concluding Remarks

Taken together, data from both PNSS and 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 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 1 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,

(Guenther et al., 2006) (Figure 2-14). Additionally, approximately two-thirds of women 14-50 years of age did not consume at least five servings of fruits and vegetables per day (Serdula et al., 2004; CDC; available online at http://www.cdc.gov/brfss/index.htm [accessed June 29, 2009]). 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

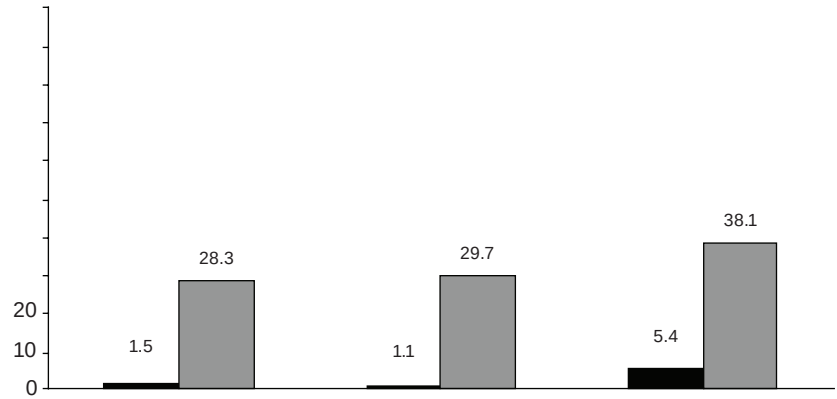


FIGURE 2-14 Percentage of U.S. childbearing-aged women who consumed the recommended number of servings of fruits and vegetables per day and five servings of fruits and vegetables per day.
NOTE: Recommended combined fruit and vegetable servings are eight servings for women aged 14-18 and 31-50 and nine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

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.

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 (HHS, 2000) and the 2008 Physical Activity Guidelines (HHS, 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

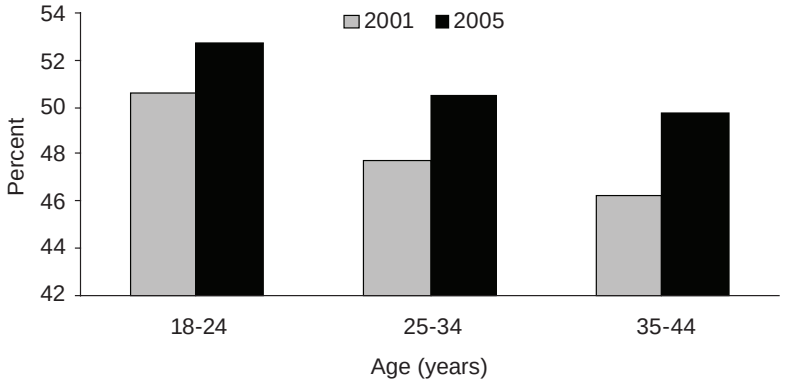


FIGURE 2-16 Trends in estimated percentage of women of childbearing age who reported meeting guidelines for regular physical activity.
NOTE: Physical activity is defined as at least 30 minutes of moderate-intensity activity per day on 5 or more days a week, or at least 20 minutes a day of vigorous-intensity activity on 3 or more days a week, or both, when not working; an exercise occurrence is defined as 10+ minutes.
SOURCE: CDC, 2007.

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

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.

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

The following describes trends since 1990 in known GWG-related

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 kg/m^2), class II (BMI 35-39.9 kg/m^2), and class III obesity (BMI ≥ 40 kg/m^2), 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 Alaska Native mothers also had rates above the national average.

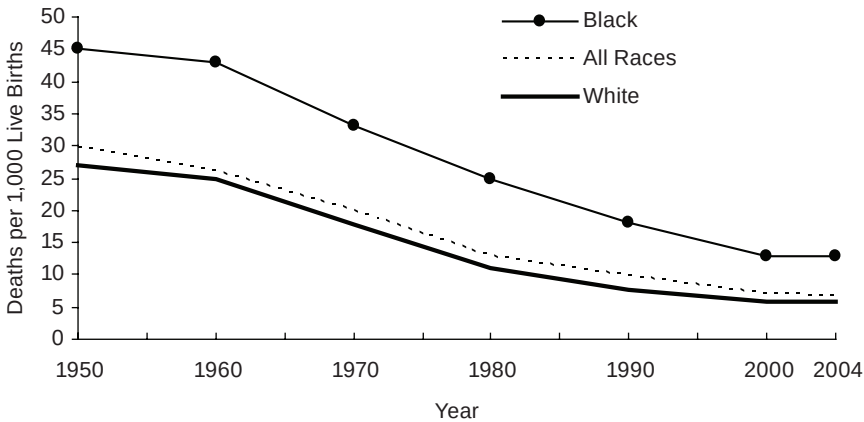


FIGURE 2-21 Infant mortality rates in the United States, 1950 through 2004, by race.
SOURCE: NCHS, 2007b.

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 mortality have decreased over time, the reverse J-shape of this relationship has not changed.

Between 1990 and 2005, the proportion of small infants increased and the proportion of large infants decreased (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.

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 Hispanic and white infants, th(,)--3(e)]TJ( )26(t)-2(h)-s(a)-2(n)-2(c)-2( )2[(a)-2(r

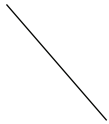


FIGURE 2-23 Percentage distribution of births by birth weight, United States, 1990 and 2005.
SOURCE: NCHS, 2007a.

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; Svens-

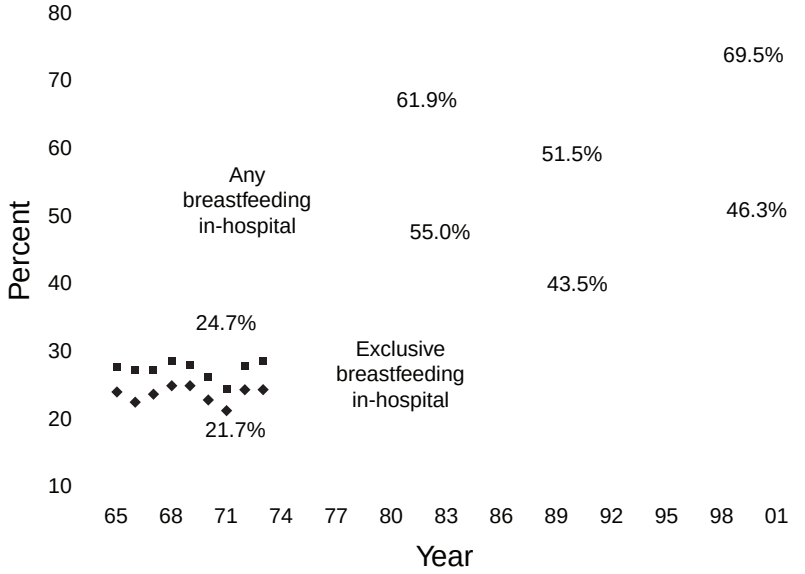


FIGURE 2-27 In-hospital breastfeeding and exclusive breastfeeding rates, 1965-2001.

SOURCE: Ryan et al., 2002. Reproduced with permission from *Pediatrics*, Vol. 110, pp. 1103-1109. Copyright © 2002 by the AAP.

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, pp. 1103-1109. Copyright © 2002 by the AAP.

FINDINGS AND RECOMMENDATIONS

Findings

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

   •

ing 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 weiern4-8(s)u8(a)-8(r)v0(w)-8(e)-8(i)l8(i)l8(i)a7(r)-8(n)c0

Bish C. L., S. Y. Chu, C. K. Shapiro-Mendoza, A. J. Sharma and H. M. Blanck. 2009. Trying to lose or maintain weight during pregnancy-United States, 2003. *Matern Child Health Journal* 13(2): 286-292.

CDC (Centers for Disease Control and Prevention). 2004. Smoking during pregnancy—United States, 1990-2002. *Morbidity and Mortality Weekly Report* 53(39): 911-915.

CDC. 2005. Trends in leisure-time physical inactivity by age, sex, and race/ethnicity—United States, 1994-2004. *Morbidity and Mortality Weekly Report* 54(39): 991-994.

CDC. 2007. Prevalence of regular physical activity among adults—United States, 2001 and 2005. *Morbidity and Mortality Weekly Report* 56(46): 1209-1212.

CDC. 2008a. *Morbidity and Mortality Weekly Report* 57(5): 127.

CDC. 2008b. *Morbidity and Mortality Weekly Report* 57(11): 281-308.

Chu S. Y., S. Y. Kim, C. H. Schmid, P. M. Dietz, W. M. Callaghan, J. Lau and K. M. Curtis. 2007. Maternal obesity and risk of cesarean delivery: a meta-analysis. *Obesity Reviews* 8(5): 385-394.

Cogswell M. E., M. K. Serdula, A. H. Mokdad and D. F. Williamson. 1996. Attempted weight

MacDorman M. F., F. Menacker and E. Declercq. 2008. Cesarean birth in the United States: epidemiology, trends, and outcomes. *Clinics in Perinatology* 35(2): 293-307.

Martin J. A., H. C. Kung, T. J. Mathews, D. L. Hoyert, D. M. Strobino, B. Guyer and S. R. Sutton. 2008. Annual summary of vital statistics: 2006. *Pediatrics* 121(4): 788-801.

Mathews T. J. and M. F. MacDorman. 2007. Infant mortality statistics from the 2004 period linked birth/infant death data set. *National Vital Statistics Reports* 55(14): 1-32.

Menacker F., E. Declercq and M. F. Macdorman. 2006. Cesarean delivery: background, trends, and epidemiology. *Seminars in Perinatology* 30(5): 235-241.

Mokdad A. H., M. K. Serdula, W. H. Dietz, B. A. Bowman, J. S. Marks and J. P. Koplan. 1999. The spread of the obesity epidemic in the United States, 1991-1998. *Journal of the American Medical Association* 282(16): 1519-1522.

NCHS (National Center for Health Statistics). 2002. Births: final data for 2001. *National Vital*

Wallis A. B., A. F. Saftlas, J. Hsia and H. K. Atrash. 2008. Secular trends in the rates of pre-eclampsia, eclampsia, and gestational hypertension, United States, 1987-2004. *American Journal of Hypertension* 21(5): 521-526.

WHO (World Health Organization). 1998. *Obesity—preventing and managing the global epidemic.* WHO Consultation on Obesity Report. Geneva.

Websites:
http://www.cdc.gov/prams/
http://www.cdc.gov/PEDNSS/pnss_tables/pdf/national_table20.pdf
http://www.cdc.gov/ifps/questionnaires.htm
http://www.cdc.gov/nchs/vitalstats.htm
http://www.cdc.gov/brfss/index.htm
http://www.ers.usda.gov/Publications/ERR49/ERR49.pdf
http://mchb.hrsa.gov/whusa08/hstat/mh/pages/237mm.html
http://www.cdc.gov/nchs/data/databriefs/db09.htm
http://www.marchofdimes.com/files/MP_Late_Preterm_Birth-Every_Week_Matters_3-24-06.pdf
http://www.cdc.gov/nccdphp/dnpa/obesity/childhood/prevalence.htm

3

Composition and Components of Gestational Weight Gain: Physiology and Metabolism

Gestational weight gain (GWG) is a unique and complex biological

cal composition and accretion rates of maternal, placental, and fetal components of GWG are presented, followed by discussions of the maternal and fetal-placental physiology underlying weight gain in pregnancy. Lastly, pathophysiologic conditions that may adversely affect GWG are reviewed to provide a foundation for understanding changes in body weight and composition during pregnancy.

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

FIGURE 3-2 Birth weight as a function of maternal weight gain and prepregnancy weight for height.
SOURCE: Modified from Abrams and Laros (1986). This article was published in the *American Journal of Obstetrics and Gynecology*

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.

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 of having a twin delivery at ≥ 36 weeks gestation and birth weight ≥ 2,500 g were significantly lower among women who gained < 35 pounds (adjusted odds ratio [AOR] 0.49, 95% confidence interval

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

[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 gestational diabetes mellitus [GDM], pregnancy-induced hypertension, preeclampsia, and anemia [AOR 1.63, 95% CI: 1.02-2.60] or cesarean delivery [AOR 1.85, 95% CI: 1.20-2.87]).

In summary, GWG in twin gestations mirrors that in singleton pregnancies, i.e., there is an inverse relationship between maternal GWG and maternal prepregnancy BMI. These results suggest that a balance is needed between optimal GWG for maternal and twin outcomes.

Triplet and Quadruplet Pregnancies

Fewer studies are available on triplet and quadruplet pregnancies (Appendix C, Table C-2). Reported GWG among mothers carrying triplets ranged fro/ apan86iT13(t)-3(h)-3(s)-3(a)-3(n)-33(p)-3(l)2F.267 0 Td5o8d5o1_1 -3(a)-3(

range (Appendix C, Tables C-1A and C-1B). For example, the pattern of GWG by maternal BMI category was examined in a large cohort of women visiting the University of California, San Francisco clinics (Abrams and Selvin, 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; and mean 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, GWG rates among 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); although the third-trimester gain was slightly lower

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

Maternal Components of Gestational Weight Gain

The committee reviewed evidence on maternal total body water (TBW) accretion, fat-free mass (FFM) accretion (i.e., protein accretion), and fat mass (FM) accretion. Each of these maternal components of GWG exhibit

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

unique patterns of accretion during pregnancy, with varying effects on outcome.

Total Body Water Accretion

Total body water accretion is largely under hormonal control and is highly variable during pregnancy. 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 ECF (1,496 g) with no edema or leg edema and ECF (4,697 g) with generalized edema (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). Larciprete et al. (2003) also found that total body water accretion was positively correlated with birth weight, in agreement

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

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 (Figure 3-4; Taggart et al., 1967). This pattern of fat deposition is unique to pregnancy.

Sohlstrom and Forsum (1995) used magnetic resonance imaging to show that the majority of fat deposited during pregnancy is subcutaneous. Based on estimates of fat deposition and distribution both before and after

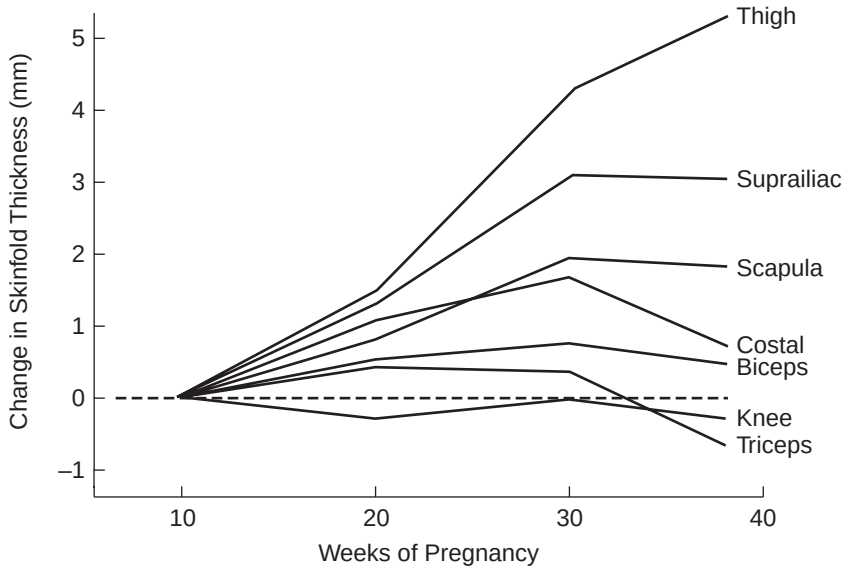


FIGURE 3-4 Longitudinal changes in skinfold thickness throughout pregnancy.
SOURCE: Taggart et al., 1967. Changes in skinfolds during pregnancy. *British Journal of Nutrition* 21(2): 439-451. Reprinted with the permission of Cambridge University Press.

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 9(d)-3(e)-d0(T)-3(h)-3(e3(0(T.3(n)-3(1(

relative changes in glycogen and fat exceeded the changes in amount (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).

Fetus

The optimal weight for a term infant is difficult to define. Not only are available methods for measuring fetal growth rate limited and prone to error, but fetal growth is impacted by a wide range of maternal physiologi

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 monochori-

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 derived from either direct collection or dye dilution techniques. At 8 weeks of gestation, amniotic volume increases at a rate of 10 mL per week, and at 13 weeks the rate 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.

MATERNAL PHYSIOLOGY

Understanding 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. Changes in many of the obligatory components of GWG (for example, TBW) are directly related to the alterations in maternal physiology that must occur for a healthy fetus and placenta to grow and develop. When the evidence permitted, the com
and hy feth-1.8ppr.u3( )-154((d)-3)-4in ip-1543(ih)-3(e)-3c154(G)-3(.)-3(-3( )sh)-3

perfusion; this results in an increase in cardiac output and a relatively small decrease in mid-gestational blood pressure. Venous blood pressure rises in the lower limbs due to mecha-3(l)-23l tt atveow-gestaatd pressure ieth rin

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 in-

secretion as a function of increased progesterone. Motility of the 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 nonpregnant values (Hytten and Chamberlain, 1991). The concentrations of binding proteins for corticosteroids, sex steroids, thyroid hormones, and vitamin D also increase.

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 nonpregnant 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 increase 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).

Alterations in maternal physiology during pregnancy are mediated by placental factors, as evidenced by the significant increase in maternal insulin sensitivity that occurs within days after delivery of the fetus and placenta (Ryan et al., 1985). 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) reported that circulating cytokines (i.e., TNF-α concentration) were inversely correlated with insulin sensitivity.

The metabolic changes in insulin sensitivity that occur 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 post-

partum 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 women (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

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.

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 dipeasens i0-3(e)-31 ng i0-3(n)-3( )-167(i)-3(n)-3i

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

The sex steroids and human placental lactogen (hPL), which best reflect the endocrine function of the placenta have been considered 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, there has been speculation that this process could contribute to weight regulation (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 toward lipid rather than glucose utilization, favoring 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 also 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 hy-

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; inflammatory mechanisms are novel potential regulatory pathways that will also have to be examined.

differences between groups, but by 16 and 18 hours, the pregnant women had substantial increases in free fatty acid (FFA) and β-hydroxybutyrate (βHA), both of which were inversely correlated with glucose levels. There was a significant difference in FFA concentrations between obese and lean pregnant women only at 16 hours of fasting. In contrast, there were no significant differences in βHA 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 that occur because of the increase in insulin resistance. As discussed previously, the accelerated starvation occurs as a result of increased insulin resistance, particularly related to lipid metabolism. 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. Using a portable capillary meter, Gin et al. (2006) measured capillary blood ketones and βHA 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 nonpregnant 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 nonpregnant 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 nonpregnant 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 nondiabetic women, only 7 percent developed ketonuria. Measurement of blood ketones was never positive if the urine measure was ≤

a. Mean GWG of normal weight women with twin births ranges from 15.5 to 21.8 kg.
b. GWG for triplets ranges 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.

3. When stratified by the World Health Organization (WHO) pre-pregnancy BMI categories, sample sizes from data on twins are insufficient to designate a range 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 elevated measures of blood βHA and acetoacetate. In women with diabetes, plasma FFA and βHA are 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 Na

Bleker O. P. and H. J. Hoogland. 1981. Short review: ultrasound in the estimation of human intrauterine placental growth. *Placenta* 2(3): 275-278.

Blickstein I. 2002. Normal and abnormal growth of multiples. *Seminars in Neonatology* 7(3): 177-185.

Brace R. A. and E. J. Wolf. 1989. Normal amniotic fluid volume changes throughout pregnancy. *American Journal of Obstetrics and Gynecology* 161(2): 382-388.

Catalano P. M., A. Thomas, L. Huston-Presley and S. B. Amini. 2007. Phenotype of infants of mothers with gestational diabetes. *Diabetes Care* 30(Suppl 2): S156-S160.

Cedergren M. 2006. Effects of gestational weight gain and body mass index on obstetric outcome in Sweden. *International Journal of Gynaecology and Obstetrics* 93(3): 269-274.

Challier J. C., S. Basu, T. Bintein, J. Minium, K. Hotmire, P. M. Catalano and S. Hauguel-de Mouzon. 2008. Obesity in pregnancy stimulates macrophage accumulation and inflammation in the placenta. *Placenta* 29(3): 274-281.

Chez R. A. and F. D. Curcio, 3rd. 1987. Ketonuria in normal pregnancy. *Obstetrics and Gynecology* 69(2): 272-274.

Gylaecollagsson I. M., G. SydsjTd[(C)-3(h)-3(e)(o)-3(d)-0133(e)-3(t)[(G)-3(y)h3(t)-3(r)l7(S)-3(y)-3(d)-3(c)-3(e),7(I)-3

Hauguel-de Mouzon S., J. Lepercq and P. Catalano. 2006. The known and unknown of leptin in pregnancy. *American Journal of Obstetrics and Gynecology* 194(6): 1537-1545.

Hediger M. L., T. O. Scholl, I. G. Ances, D. H. Belsky and R. W. Salmon. 1990. Rate and amount of weight gain during adolescent pregnancy: associations with maternal weight-for-height and birth weight. *American Journal of Clinical Nutrition* 52(5): 793-799.

Hickey C. A., S. P. Cliver, S. F. McNeal, H. J. Hoffman and R. L. Goldenberg. 1995. Prenatal weight gain patterns and spontaneous preterm birth among nonobese black and white women. *Obstetrics and Gynecology* 85(6): 909-914.

Hopkinson J. M., N. F. Butte, K. J. Ellis, W. W. Wong, M. R. Puyau and E. O. Smith. 1997. Body fat estimation in late pregnancy and early postpartum: comparison of two-, three-,

Lantz M. E., R. A. Chez, A. Rodriguez and K. B. Porter. 1996. Maternal weight gain patterns and birth weight outcome in twin gestation. *Obstetrics and Gynecology* 87(4): 551-556.

Larciprete G., H. Valensise, B. Vasapollo, F. Altomare, R. Sorge, B. Casalino, A. De Lorenzo

Sparks J. W. 1984. Human intrauterine growth and nutrient accretion. *Seminars in Perinatology* 8(2): 74-93.

Stevens-Simon C., E. R. McAnarney, K. J. Roghmann and G. B. Forbes. 1997. Composition of gestational weight gain in adolescent pregnancy. *Journal of Maternal-Fetal Medicine* 6(2): 79-86.

Stewart M. O., P. G. Whittaker, B. Persson, U. Hanson and T. Lind. 1989. A longitudinal study of circulating progesterone, oestradiol, hCG and hPL during pregnancy in type 1 diabetic mothers. *British Journal of Obstetrics and Gynaecology* 96(4): 415-423.

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 that 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 influ-

ences 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,

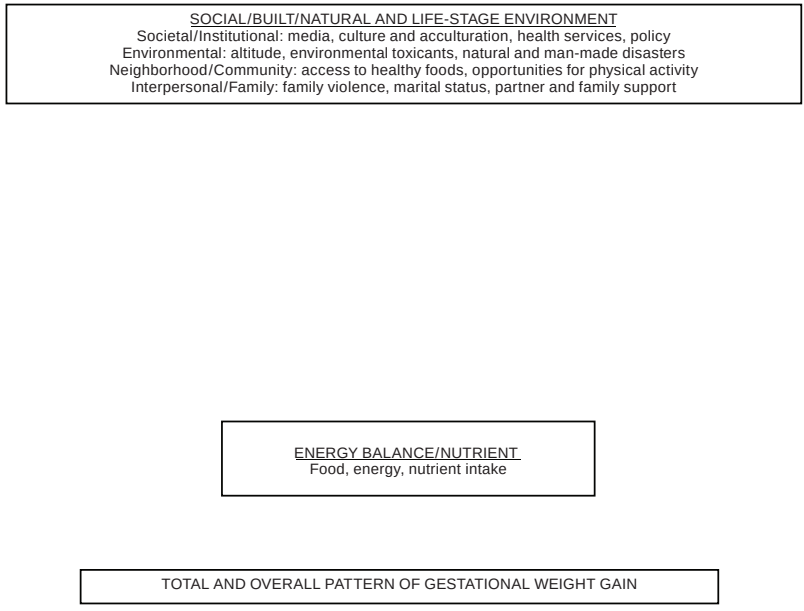


FIGURE 4-1 Schematic summary of determinants associated with GWG.

affect consumption behavior. Other studies have shown that the media

individual and social characteristics of the provider as contrasted with those of the pregnant woman, and racial/ethnic and socioeconomic disparities in

Another example of policy influencing 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

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were available. These findings were independent of income level; however, the study did not report on GWG.

Opportunities for Physical Activity

Although 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 3 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

The committee considered three types of interpersonal/family factors

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Ventura (1994), 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 the IOM (1990) guidelines, compared to 42 percent of women who were separated or divorced, and 48 percent of single women. They also found that 21 percent of married women had gained below the 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.

Partner and Family Support

The committee identified only two studies pertaining to the relationship between partner support and GWG. In the first, Dipietro et al. (2003) examined the influences of partner support on attitudes or behaviors toward GWG. In a cross-sectional study of 130 women with low-risk pregnancies, they found that partner support was associated with negative pregnancy body image, but not with attitudes or behaviors toward GWG. Siega-Riz and Hobel (1997) evaluated a clinic sample of 4,791 Hispanic women in Los Angeles and 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.

Again, the committee only identified two studies pertaining to the influence of family support on GWG. In a sample of 99 pregnant adolescents, Stevens-Simon et al. (1993b) found 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. In a study of 46 pregnant Mexican American adolescents, Gutierrez (1999) reported 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 investigators did not report on the contribution of family support to either GWG attitude or actual GWG.

MATERNAL FACTORS

as age and race/ethnicity; physiological factors, some of which are also discussed in depth elsewhere in this report, and genetic factors known to impact GWG and those that may impact GWG because of their known influence on birth weight; and developmental and epigenetic programming in the mother, which may influence how a woman responds later in life.

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 2 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

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 the 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

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.

Endres et al. (1987) evaluated nutrient intake, prepregnancy weight, and GWG in pregnant women enrolled in the WIC program who were over 35 years of age versus adolescents aged 15-18 years. The investigators calculated prepregnancy BMI for both groups and found 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 Recommended Dietary Allowances [RDAs]), but the younger women had higher mean energy intakes (

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. (1977)	12-17	Caucasian (one black woman)	26	13.4	26
	18-19		22	12.4	31
	20-24		24	11.1	17
	25-32		26	10.7	18
Frisancho et al. (1983)	12-13	Saltef	1		

In their review of birth records of 913,320 singleton births in New York City from 1995 to 2003, Stein (information contributed to the committee in consultation with Stein [see Appendix G, Part III]) 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. 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 natigeemittee

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 = 8,091)[a]		15-24 (n = 9,970)[a]		25-34 (n = 14,545)[a]		35-44 (n = 10,311)[a]		≥ 45 (n = 7,112)[a]	
Characteristic	Percent[b]	SE[b]	Percent[b]	SE[b]	Percent[b]	SE[b]	Percent[b]	SE[b]	Percent[b]	SE[b]
Age, yr (n)[a]										
14-19 (5,249)	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 (7,651)	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 (7,790)	21.7	0.7	21.1	0.6	23.9	0.7	18.2	0.6	15.1	0.6
Hispanic (7,428)	17.3	0.7	21.2	0.7	29.3	0.8	20.1	0.7	12.1	0.6
Other (7,221)	16.4	0.8	19.9	0.9	30.6	1.1	19.8	0.9	13.5	0.8
Education, y (n)										
< 12 (8,154)	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 (5,100)	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

NOTE: c^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 U.S. women delivering live births, 2004-2005: fueling future obesity. *American Journal of Obstetrics and Gynecology.* Copyright (2009), with permission from Elsevier.

Food Insecurity

Food insecurity is closely tied to socioeconomic status and is therefore discussed here 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" (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. 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-

Genetic Characteristics

The role of DNA sequence variation in the regulation of body weight is being investigated in many laboratories worldwide, but few investigators are focusing their attention on the genetics of weight gain during pregnancy. The committee was unable to identify studies dealing with the heritability of GWG. The only evidence on the genetic basis of GWG comes from a small number of reports focusing on the contribution of single nucleotide polymorphisms (SNPs) in specific genes. At present no study has considered the important issue of nutrition or physical activity interactions with genes on GWG.

Most of the SNP studies have focused on 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). Festa et al. (1999) showed that Austrian mothers who were homozygous for the 64Arg allele gained more weight from baseline to gestational weeks 20 to 31 than heterozygotes. Among pregnant women with type 2 diabetes, Yanagisawa et al. (1999) showed that 12.2 percent of those homozygous and 19.2 percent of those heterozygous for the Trp allele and 28.6 percent homozygous for the Arg allele gained more than 5 units in BMI during pregnancy. In contrast, in a study from Greece, Alevizaki et al. (2000) found no differences among the ADRβ3 genotypes 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. Similarly, Tsai et al. (2004) found no differences in weight gain at 24 to 31 weeks of gestation among genotypes in a Taiwanese population. In the largest study to date, involving 627 pregnant women from Italy, Fallucca et al. (2006) found no effect of the ADRβ3 polymorphism on GWG. In the same study, a marker in the insulin receptor substrate 1 (IRS-1) gene was also not associated with GWG.

Tok et al. (2006) examined the Pro12Ala polymorphism in the peroxisome proliferator-activated receptor gamma 2 (*PPARδ2*) in pregnant Turkish women. Among 62 women who had gestational diabetes mellitus (GDM), those with the Pro12Ala polymorphism gained more weight during pregnancy. Among 100 nondiabetic pregnant women, 294 women homozygous for the T allele with uncomplicated, singleton pregnancies who had term deliveries ranging from 37 to 40 weeks gained significantly more weight (17.4 ± 0.9 kg) than those with the C allele (15.1 ± 0.4 kg). However, the sample included women from various ethnic ancestries, which may have affected the results in an undetermined manner.

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adequate statistical power was available to identify the effects of alleles or genotypes with a small effect size.

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 large-for-gestational age (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.

The most compelling data for a role of paternal birth weight on weight of the offspring at birth also comes from a Norwegian study. A total of 67,795 father-mother-firstborn child trios 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 wntedighsiofator-mother's birth weion

Importantly, variation in birth weight is influenced by a number of other factors in addition to the genetic makeup of the newborn. Several studies have identified maternal genotype as another important factor. For example, in the large Norwegian study cited above, maternal genetic factors accounted for 22 percent of the variation in birth weight (Lunde et al., 2007).

In another study of 6,811 white singletons and their natural parents, Griffiths et al. (2007) evaluated the effect of parental height and weight on offspring length and weight at birth and observed that the effects of parental height on birth weight were similar for both. However, the influence of the mother's weight on the infant's birth weight was stronger than that of the father. Finally, in a report on parental role on the familial aggregation of SGA in 256 infants, Jaquet et al. (2005) found that both parents contributed almost equally to the risk. Specifically, 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 SGA, compared to those who were appropriate-for-gestational age (AGA). When both parents had been SGA the risk of an SGA infant was 16 times higher.

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

allele (single nucleotide polymorphism [SNP] at position -30) at the *GCK*

mutation carriers compared to nonmutated family members (Figure 4-2). Fifty-four percent of mutation carriers were macrosomic, compared with 13 percent for nonmutation family members.

In another candidate gene study, a common SNP in the fat mass and obesity associated gene (*FTO*) was investigated for its relationship to weight

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 that associate with increased risk for GWG outside recommended ranges in the context of maternal dietary and physical activity habits.

Developmental Programming

In addition to genetics, a multitude of other maternal factors could potentially influence GWG. Early developmental programming is one of them. Developmental, or in utero, programming refers to 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. These exposures and experiences may encode the functions of organs or systems that become manifest as elevated or diminished risk for disease later in life (Barker, 1998; Seckl, 1998).

The following example illustrates how developmental programming may influence maternal GWG. It has been suggested that developmental programming could influence the ability to respond to and cope with repeated exposure to stress. If this is shown to be the case, it could explain why some women may be at greater risk for excessive GWG. More specifically, ani-

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

Some types of developmental programming may be mediated through epigenetic events—that is, chemical modifications to the DNA and histone proteins that influence gene expression and manifest as phenotypic differences potentially mimicking those associated with DNA sequence variants. Epigenetic events begin accruing early after fertilization. Some occur at the embryonic stage in key tissues, often resulting in silencing (or switching off) /Span <</MCIulff)

cal factors: (1) prepregnancy BMI, (2) changes in the hormonal milieu that impact the maternal metabolic response during pregnancy, and (3) changes in basal metabolic rate (BMR) and energy expenditure (EE) during pregnancy. The following discussion summarizes this evidence. The BMI studies are, to some extent, an expansion of Chapter 2 on trends in BMI since publication of the IOM (1990) report; and the studies on hormonal milieu, BMR, and EE are, to some extent, an extension of the Chapter 3 discussion on endocrine and metabolic changes that occur during pregnancy.

Pregravid BMI

Based on epidemiological studies (e.g., those described in Chapter 2), GWG is generally inversely proportional to maternal BMI. For example, in a report of over 2.3 million deliveries in Germany from 1995-2000, Voigt et al. (2007) reported that overall, relatively short and heavy women had lower GWGs than tall and thin women. In the United States, Chu et al. (2009) used the PRAMS data from 2004-2005 to assess the amount of GWG among 52,988 underweight, normal weight, overweight, and obese U.S. women delivering full-term singleton infants (Table 4-4). They found that, overall, GWG decreased with increasing BMI. When they stratified by BMI, they found that obese women gained less weight during pregnancy than normal or overweight women; yet about one-fourth of obese women still gained 35 pounds or more. In a multivariable regression model, maternal prepregnancy obesity was the strongest predictor of low GWG (obesity correlated with lowest GWG), followed by higher parity, African American or Hispanic racial identity, and higher maternal age.

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 that can influence GWG.

Insulin, Leptin, and Hormonal Milieu, 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 TEE. The authors concluded, from finding a small range for energy savings from either minor physical

TABLE 4-4 Gestational Weight Gain (pounds) by Prepregnancy BMI Among Mothers Delivering Full-term, Singleton Births, PRAMS, 2004-2005

	≤ 14 (n = 8,442)[a]		15-24 (n = 10,583)[a]		25-34 (n = 15,477)[a]		35-44 (n = 10,942)[a]		≥ 45 (n = 7,544)[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/m^2)	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/m^2)	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/m^2)	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/m^2)	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

NOTE: c^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 U.S. women

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 andty3(e)-3(a)a,(F)-3(o)-3(o)-3(duu)-3(p)-33(,)-(Fe)-3( )-3

creased 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 f gestatple regregn a na8

components of maternal metabolism and may exert an indirect effect on GWG through their effects on maternal insulin sensitivity.

Leptin is produced in relatively large quantities by the placenta and is transferred primarily into the maternal circulation (Hauguel-de Mouzon et al., 2006), with maternal leptin concentrations increasing by 12 weeks' gestation and having a significant positive correlation with both maternal

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 6 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

dysfunctional perception of body weight and shape (Wisner et al., 2007), and both may affect GWG. Anorexia, which is defined as body weight less than 85 percent of expected weight for age and height, occurs in between 0.5-1.0 percent of women of reproductive age. Bulimia is defined as weight at the minimally normal range but where the individual employs binge eat

1998 to approximately 72,177 in 2002 (Santry et al., 2005; Davis et al., 2006). Most of the procedures during this time were performed on women; 81 percent in 1998 and 84 percent in 2002. As a result of this trend, 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), recommending that obese women who have undergone bariatric surgery receive the following counseling before and during pregnancy:

- Patients with adjustable g

Twins and Higher Order Pregnancy

As discussed in Chapter 3, the presence of multiple fetuses in a pregnancy has an influence on total GWG. In comparison to a 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

The committee evaluated whether several weight-related psychological factors—depression, stress, social support, and attitudes toward weight gain—might be determinants of GWG. The following discussion summarizes the committee's review of the evidence. Based on its review, the committee found that depression, or depressive symptoms, are associated with both low and high GWG (i.e., lower or higher than the recommended ranges) but that the evidence on whether and how the other psychological factors impact GWG is inconclusive. The discussion on depression extends the Chapter 2 summary of trends since 1990 in depression during pregnancy.

Depression

The committee identified three studies showing a positive associaDepressiia44

posits. Webb et al. (2009) found that 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. Casanueva et al. (2000) conducted a case-control study to test for associations between maternal depressive symptoms and fat deposition among Mexican pregnant adolescents. They used body weight and anthropometric measures of skinfold thickness to determine fat deposition beginning at 20 weeks gestation through 4 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 associated with negative attitudes about GWG (Stevens-Simon et al., 1993b; 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).

Not all studies have shown a positive association between depression and either high or low GWG. For example, Cameron et al. (1996) studied a biracial sample of 132 women in mid-gestation and found a positive association between GWG and depression score for white women with high self-esteem, a negative correlation with depression score and third-trimester weight among white women with low self-esteem, and no association between depression score and GWG among black women. Walker and Kim (2002) analyzed data from a longitudinal study of postpartum weight patterns in low-income women and found that depressive symptoms were not significantly associated with GWG. Collectively, however, 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

The committee found a lack of consistent evidence in support of a relationship between stress and GWG. 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; most of the available evidence is observational, and estimates of the impact of stress are confounded by the different kinds of effects that can occur depending on how an individual responds.

Picone et al. (1982) examined the influence of psychological stress as a factor in GWG and pregnancy outcome in a controlled prospective study of a group of 60 women utilizing an urban prenatal clinic. Psychological stress was assessed using a social readjustment rating scale from the Holmes-Rahe life events questionnaire. The investigators found a correlation between higher stress scores and lower GWG, independent of nutrient or caloric

intake. The finding suggests that stress did not affect food intake in these subjects, rather it impacted the utilization of calories and nutrients from the foods consumed to support pregnancy.

Based on the published regression models, either crude or adjusted, there does not appear to be a robust association between the appraisals of stress, sufficiency of coping resources, and adequacy of GWG. However, when evaluating the risk ratio differences observed between women who

1975) showed that 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) analyzed data in 156 healthy German women and reported that, while neither maternal BMI nor energy intake was related to birth weight, both were related to "net weight gain." The authors defined maternal weight gain as 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. Women in the high-BMI group (defined as > 24) had an overall lower net weight gain (4.2 kg), compared to women in the normal-BMI group (6.2 kg) and low-BMI group (5.9 kg). However, the lower weight gain was confined to the multigravid women, with primigravid women actually having a greater net weight gain. These associations did not appreciably change when adjusted for energy intake, which did not vary during the course of pregnancy.

In another study, Olson and Strawderman (2003) used a proxy measure for energy intake by questioning 622 healthy pregnant women about changes in the amount of food eaten prior to and during pregnancy. They found 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 to maintaining similar levels of food intake during and prior to pregnancy. Women who ate "much more" during rather than before their pregnancy had an adjusted odds ratio of 2.35 for excessive GWG. Lagiou et al. (2004) found that increased GWG by the end of the second trimester of pregnancy was associated, in a clinic sample of 224 pregnant women, with higher total energy intake and a higher proportion of protein and lipids of animal origin (and lower proportion of carbohydrates).

Finally, 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 the effect of GWG on consumption of different types of food as well as macronutrient and micronutrient intake. Stevens-Simon and McAnarney (1992) showed that adolescents who consumed fewer than three snacks a day had slower weight gains during pregnancy. Olson and Strawderman (2003) found that 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. More recently, Olafsdottir et al. (2006) found that the percentage of energy intake from various macronutrients is an important predictor of weight gain but only among overweight women and late in pregnancy. The investigators analyzed the relationship between dietary factors and GWG in 495 healthy Icelandic women using food frequency questionnaires; they defined optimal weight gain as 12-18 kg in normal weight women and 7-12 kg in overweight women. Eleven percent of overweight women had inadequate weight gain compared to only 2 percent of normal weight women; in contrast, 14 percent of overweight women gained > 18 kg, and 20 percent of normal weight women gained > 18 kg. The investigators found that, 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.

The committee identified two studies that examined the effects of caloric intake on GWG in relationship to glycemic load. 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 investigators speculated on several potential mechanisms that might explain the difference, including changes in: daily digestible energy requirements (i.e., metabolic efficiency), substrate utilization (glucose oxidation versus lipid oxidation), and insulin resistance and sensitivity. Deierlein et al. (2008) reported that white women with glycemic load increases were more sensitive to increased weight gain during pregnancy; the same was not true for black women.

Altogether while several studies have demonstrated a relationship between energy intake and GWG and some studies have shown that dietary intake of certain types of foods may also influence GWG, the evidence base is not substantial enough to draw any conclusions.

Physical Activity

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 (HHS, 2008) concluded that:

- Moderate-intensity leisure time physical activity is not associated with an increased risk of low birth weight, preterm delivery, or early pregnancy loss; and
- Participation in vigorous activities has been associated with small reductions in birth weight compared to less active women (Leet and Flick, 20033(r)-3(t)-3TM61_1 1 Tf10 0 (l)-3(i)-3T7icmaR2(t)2(u)2(s)2(1A
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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 average04( )-105(t)-3(h)-3(043( )-82(p)-3(r)-3(e)-3(g)-3(n)-3(a)-3(n)-3103( )-

Women Incarcerated During Pregnancy

The U.S. Department of Justice estimates that women offenders account

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, postpartum 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.

Areas for Additional Investigation

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

facilitators and barriers to adoption of GWG recommendations by health care providers in their clinical practice;
- Partner and family influences on GWG;
- Influences of genetic factors, epigenetic events, and developmental programming on GWG;
- How GWG affects 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 re-evaluate 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 or other appropriate federal agencies should:

- Track racial/ethnic and socioeconomic disparities in GWG and that the research community should conduct studies on how GWG affects 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.

REFERENCES

Abraham S., A. Taylor and J. Conti. 2001. Postnatal depression, eating, exercise, and vom

Bergmann M. M., E. W. Flagg, H. L. Miracle-McMahill and H. Boeing. 1997. Energy intake and net weight gain in pregnant women according to body mass index (BMI) status. *International Journal of Obesity and Related Metabolic Disorders* 21(11): 1010-1017.

Bjorntorp P. 1993. Visceral obesity: a "civilization syndrome." *Obesity Research* 1(3): 206-222.

Bjorntorp P. 1996. The origins and consequences of obesity. Diabetes. *Ciba Foundation Symposium* 201: 68-80; discussion 80-69, 188-193.

Bjorntorp P. and R. Rosmond. 2000. The metabolic syndrome—a neuroendocrine disorder? *British Journal of Nutrition* 83(Suppl 1): S49-S57.

Bodnar L. M., K. L. Wisner, E. Moses-Kolko, D. K. Sit and B. H. Hanusa. 2009. Prepregnancy body mass index, gestational weight gain and the likelihood of major depression during pregnancy. *Journal of Clinical Psychiatry.* Epub ahead of print.

Boy A. and H. M. Salihu. 2004. Intimate partner violence and birth outcomes: a systematic review.

Cameron R. P., C. M. Grabill, S. E. Hobfoll, J. H. Crowther, C. Ritter and J. Lavin. 1996. Weight, self-esteem, ethnicity, and depressive symptomatology during pregnancy among inner-city women. *Health Psychology* 15(4): 293-297.

Campbell D. 1983. Dietary restriction in obesity and its effect on neonatal outcome. In *Nutrition in Pregnancy. Proceedings of 10th Study Group of the RCOG*. Campbell DM and G. MDG. London: RCOG; pp. 85-98.

Campbell D. M. and I. MacGillivray. 1975. The effect of a low calorie diet or a thiazide diuretic on the incidence of pre-eclampsia and on birth weight. *British Journal of Obstetrics and Gynaecology* 82(7): 572-577.

Carmichael S. L., G. M. Shaw, D. M. Schaffer, C. Laurent and S. Selvin. 2003. Dieting behaviors and risk of neural tube defects. *American Journal of Epidemiology* 158(12): 1127-1131.

Casanueva E., D. Legarreta, M. Diaz-Barriga, Y. Soberanis, T. Cardenas, A. Iturriaga, T. Lartigue and J. Vives. 1994. Weight gain during pregnancy in adolescents: evaluation of a non-nutritional intervention. *Revista de Investigacion Clinica* 46(2): 157-161.

Casanueva E., J. Labastida, C. Sanz and F. Morales-Carmona. 2000. Depression and body fat deposition in Mexican pregnant adolescents. *Archives of Medical Research* 31(1): 48-52.

Catalano P. M., E. D. Tyzbir, R. R. Wolfe, J. Calles, N. M. Roman, S. B. Amini and E. A. Sims. 1993. Carbohydrate metabolism during pregnancy in control subjects and women with gestational diabetes. *American Journal of Physiology* 264(1 Pt 1): E60-E67.

Catalano P. M., N. M. Roman-Drago, S. B. Amini and E. A. Sims. 1998. Longitudinal changes in body composition and energy balance in lean women with normal and abnormal glucose tolerance during pregnancy. *American Journal of Obstetrics and Gynecology* 179(1): 156-165.

Catalano P. M., L. Huston, S. B. Amini 23(a)-3(t)-3(m)-3(i)-3(n)-3(-3(n)-3(-3(n)-3(-3(n)-3(-3(n)-3(-3(n)-3(-3(n)-3(-3(r

Clapp J. F., III and K. D. Little. 1995. Effect of recreational exercise on pregnancy weight gain and subcutaneous fat deposition. *Medicine and Science in Sports and Exercise* 27(2): 170-177.

Cnattingius S., M. R. Forman, H. W. Berendes and L. Isotalo. 1992. Delayed childbearing and risk of adverse perinatal outcome. A population-based study. *Journal of the American Medical Association* 268(7): 886-890.

Cnop M., P. J. Havel, K. M. Utzschneider, D. B. Carr, M. K. Sinha, E. J. Boyko, B. M. Retzlaff, R. H. Knopp, J. D. Brunzell and S. E. Kahn. 2003. Relationship of adiponectin to body fat distribution, insulin sensitivity and plasma lipoproteins: evidence for independent roles of age and sex. *Diabetologia* 46(4): 459-469.

Cogswell M. E., M. K. Serdula, A. H. Mokdad and D. F. Williamson. 1996. Attempted weight loss during pregnancy. *International Journal of Obesity and Related Metabolic Disorders* 20(4): 373-375.

Cogswell M. E., K. S. Scanlon, S. B. Fein and L. A. Schieve. 1999. Medically advised, mother's personal target, and actual weight gain during pregnancy. *Obstetrics and Gynecology* 94(4): 616-622.

Cohen J. H. and H. Kim. 2009. Sociodemographic and health characteristics associated with attempting weight loss during pregnancy. *Preventing Chronic Disease* 6(1): A07.

Copper R. L., M. B. DuBard, R. L. Goldenberg and A. I. Oweis. 1995. The relationship of maternal attitude toward weight gain to weight gain during pregnancy and low birth weight. *Obstetrics and Gynecology* 85(4): 590-595.

Cordero J. F. 1993. The epidemiology of disasters and adverse reproductive outcomes: lessons learned. *Environmental Health Perspectives* 101(Suppl 2): 131-136.

Crawford P. B., M. S. Townsend and D. L. Metz. 2004. How can Californians be overweight and hungry? *California Agriculture* 58(1): 12-17.

Cunningham F. G., J. C. Hauth, K. J. Leveno, L. Gilstrap III, S. L. Bloom and K. D. Wenstrom, Eds. 2005. *Williams Obstetrics 22nd Ed.* New York: McGraw-Hill Medical Publishing Division.

Dar E., M. S. Kanarek, H. A. Anderson and W. C. Sonzogni. 1992. Fish consumption and reproductive outcomes in Green Bay, Wisconsin. *Environmental Research* 59(1): 189-201.

Davis M. M., K. Slish, C. Chao and M. D. Cabana. 2006. National trends in bariatric surgery, 1996-2002. *Archives of Surgery* 141(1): 71-74; discussion 75.

Deierlein A. L., A. M. Siega-Riz and A. Herring. 2008. Dietary energy density but not glycemic load is associated with gestational weight gain. *American Journal of Clinical Nutrition* 88(3): 693-699.

Delpisheh A., L. Brabin, E. Attia and B. J. Brabin. 2008. Pregnancy late in life: a hospital-based study of birth outcomes. *Journal of Women's Health (Larchmt)* 17(6): 965-970.

Dipietro J. A., S. Millet, K. A. Costigan, E. Gurewitsch and L. E. Caulfield. 2003. Psychosocial influences on weight gain attitudes and behaviors during pregnancy. *Journal of the American Dietetic Association* 103(10): 1314-1319.

Dishy V., S. Gupta, R. Landau, H. G. Xie, R. B. Kim, R. M. Smiley, D. W. Byrne, A. J. Wood and C. M. Stein. 2003. G-protein beta (3) subunit 825 C/T polymorphism is associated with weight gain during pregnancy. *Pharmacogenetics* 13(4): 241-242.

Dixon J. B., M. E. Dixon and P. E. O'Brien. 2005. Birth outcomes in obese women after laparoscopic adjustable gastric banding. *Obstetrics and Gynecology* 106(5 Pt 1): 965-972.

Drewnowski A. and N. Darmon. 2005. Food choices and diet costs: an economic analysis. *Journal of Nutrition* 135(4): 900-904.

Dubois L., M. Girard, A. Girard, R. Tremblay, M. Boivin and D. Perusse. 2007. Genetic and environmental influences on body size in early childhood: a twin birth-cohort study. *Twin Research and Human Genetics* 10(3): 479-485.

Frongillo E. A., Jr., B. S. Rauschenbach, C. M. Olson, A. Kendall and A. G. Colmenares. 1997. Questionnaire-based measures are valid for the identification of rural households with hunger and food insecurity. *Journal of Nutrition* 127(5): 699-705.

Fuentes-Afflick E. and N. A. Hessol. 2008. Acculturation and body mass among Latina women. *Journal of Women's Health (Larchmt)* 17(1): 67-73.

Fuentes-Afflick E. and P. Lurie. 1997. Low birth weight and Latino ethnicity. Examining the epidemiologic paradox. *Archives of Pediatrics and Adolescent Medicine* 151(7): 665-674.

Furneaux E. C., A. J. Langley-Evans and S. C. Langley-Evans. 2001. Nausea and vomiting of pregnancy: endocrine basis and contribution to pregnancy outcome. *Obstetrical and Gynecological Survey* 56(12): 775-782.

Furuno J. P., L. Gallicchio and M. Sexton. 2004. Cigarette smoking and low maternal weight gain in Medicaid-eligible pregnant women. *Journal of Women's Health (Larchmt)* 13(7): 770-777.

Garn S. M., K. Hoff and K. D. McCabe. 1979. Is there nutritional mediation of the "smoking effect" on the fetus. *American Journal of Clinical Nutrition* 32(6): 1181-1184.

Gavard J. A. and R. Artal. 2008. Effect of exercise on pregnancy outcome. *Clinical Obstetrics and Gynecology* 51(2): 467-480.

Gigante D. P., K. M. Rasmussen and C. G. Victora. 2005. Pregnancy increases BMI in adolescents of a population-based birth cohort. *Journal of Nutrition* 135(1): 74-80.

Glynn L. M., P. D. Wadhwa, C. Dunkel-Schetter, A. Chicz-Demet and C. A. Sandman. 2001. When stress happens matters: effects of earthquake timing on stress responsivity in pregnancy. *American Journal of Obstetrics and Gynecology* 184(4): 637-642.

Goldberg G. R., A. M. Prentice, W. A. Coward, H. L. Davies, P. R. Murgatroyd, C. Wensing, A. E. Black, M. Harding and M. Sawyer. 1993. Longitudinal assessment of energy expenditure in pregnancy by the doubly labeled water method. *American Journal of Clinical Nutrition* 57(4): 494-505.

Goodwin T. M. 2002. Nausea and vomiting of pregnancy: an obstetric syndrome. *American Journal of Obstetrics and Gynecology* 186(5 Suppl Understanding): S184-S189.

Goodwin T. M., M. Montoro and J. H. Mestman. 1992. Transient hyperthyroidism and hyperemesis gravidarum: clinical aspects. *American Journal of Obstetrics and Gynecology* 167(3): 648-652.

Gortmaker S. L., A. Must, A. M. Sobol, K. Peterson, G. A. Colditz and W. H. Dietz. 1996. Television viewing as a cause of increasing obesity among children in the United States, 1986-1990. *Archives of Pediatrics and Adolescent Medicine* 150(4): 356-362.

Gortmaker S. L., K. Peterson, J. Wiecha, A. M. Sobol, S. Dixit, M. K. Fox and N. Laird. 1999. Reducing obesity via a school-based interdisciplinary intervention among youth: Planet Health. *Archives of Pediatrics and Adolescent Medicine* 153(4): 409-418.

Graham K., A. Feigenbaum, A. Pastuszak, I. Nulman, R. Weksberg, T. Einarson, S. Goldberg, S. Ashby and G. Koren. 1992. Pregnancy outcome and infant development following gestational cocaine use by social cocaine users in Toronto, Canada. *Clinical and Investigative Medicine. Medicine Clinique et Experimentale* 15(4): 384-394.

Griffiths L. J., C. Dezateux and T. J. Cole. 2007. Differential parental weight and height contributions to offspring birthweight and weight gain in infancy. *I92 >>BDC 2.25m(g)-3(a)-3(t)-3(i)]TJ*

Groth S. W. 2008. The long-term impact of adolescent gestational weight gain. *Research in Nursing and Health* 31(2): 108-118.

Gunderson E. P., R. Striegel-Moore, G. Schreiber, M. Hudes, F. Biro, S. Daniels and P. B. Crawford. 2009. Longitudinal study of growth and adiposity in parous compared with nulligravid adolescents. *Archives of Pediatrics and Adolescent Medicine* 163(4): 349-356.

Gurewitsch E. D., M. Smith-Levitin and J. Mack. 1996. Pregnancy following gastric bypass surgery for morbid obesity. *Obstetrics and Gynecology* 88(4 Pt 2): 658-661.

Gutierrez Y. M. 1999. Cultural factors affecting diet and pregnancy outcome of Mexican American adolescents. *Journal of Adolescent Health* 25(3): 227-237.

Haakstad L. A., N. Voldner, T. Henriksen and K. Bo. 2007. Physical activity level and weight gain in a cohort of pregnant Norwegian women. *Acta Obstetricia et Gynecologica Scandinavica* 86(5): 559-564.

Haas D. M(-)-3(5)-3(6)-3(-3(g)-3(i53(9)-3(-)-25 Td[(H)-3(s)-3(i)-3(c1-3(r)-3( )-31(m.0-3(i)-3(c)(s)-3(EMC /Sp

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Hickey C. A., S. P. Cliver, R. L. Goldenberg, S. F. McNeal and H. J. Hoffman. 1995. Relationship of psychosocial status to low prenatal weight gain among nonobese black and white women delivering at term. *Obstetrics and Gynecology* 86(2): 177-183.

Hickey C. A., S. P. Cliver, R. L. Goldenberg, S. F. McNeal and H. J. Hoffman. 1997. Low prenatal weight gain among low-income women: what are the risk factors? *Birth* 24(2): 102-108.

Highman T. J., J. E. Friedman, L. P. Huston, W. W. Wong and P. M. Catalano. 1998. Longitudinal changes in maternal serum leptin concentrations, body composition, and resting metabolic rate in pregnancy. *American Journal of Obstetrics and Gynecology* 178(5): 1010-1015.

Hill J. O., W. H. Saris and J. A. Levine. 2004. Energy Expenditure in Physical Activity. In *Handbook of Obesity: Etiology and Pathophysiology, 2nd Ed*. G. A. Bray and C. Bouchard. New York: Marcel Dekker Inc.

Hinton P. S. and C. M. Olson. 2001. Predictors of pregnancy-associated change in physical tEMC /Span <</þi8n(m) n <</MCID 9121 >>B1 >>B1 >>B1 >>B1 >>BJEMC /Span 0 0 8 Tf8 0 0 8 30</

Jones M. E. and M. L. Bond. 1999. Predictors of birth outcome among Hispanic immigrant women. *Journal of Prof-3(m)-3(H)-6(T)-5(23(f)-3( )-3yo.79 nfi-3(m)-3(ey<</MCID 9196 >>BDC /T1̲0̲ 17*

Lederman S. A., V. Rauh, L. Weiss, J. L. Stein, L. A. Hoepner, M. Becker and F. P. Perera.

McFarlane J., B. Parker and K. Soeken. 1996. Abuse during pregnancy: associations with maternal health and infant birth weight. *Nursing Research* 45(1): 37-42.

Meserole L. P., B. S. Worthington-Roberts, J. M. Rees and L. S. Wright. 1984. Prenatal weight gain and postpartum weight loss patterns in adolescents. *Journal of Adolescent Health Care* 5(1): 21-27.

Moraes C. L., A. R. Amorim and M. E. Reichenheim. 2006. Gestational weight gain differentials in the presence of intimate partner violence. *International Journal of Gynaecology and Obstetrics* 95(3): 254-260.

Morris D. L., A. B. Berenson, J. Lawson and C. M. Wiemann. 1993. Comparison of adolescent pregnancy outcomes by prenatal care source. *Journal of Reproductive Medicine* 38(5): 375-380.

Morris S. N. and N. R. Johnson. 2005. Exercise during pregnancy: a critical appraisal of the literature. *Journal of Reproductive Medicine* 50(3): 181-188.

Muscati S., M. Mackey and B. Newsome. 1988. The influence of smoking and stress on prenatal weight gain and infant birth weight of teenage mothers. *Journal of Nutrition Education* 20: 299-302.

Nielsen J. N., K. O. O'Brien, F. R. Witter, S. C. Chang, J. Mancini, M. S. Nathanson and L. E. Caulfield. 2006. High gestational weight gain does not improve birth weight in a cohort of African American adolescents. *American Journal of Clinical Nutrition* 84(1): 183-189.

O'Boyle A. L., E. F. Magann, R. E. Ricks, Jr., M. Doyle and J. C. Morrison. 2005. Depression screening in the pregnant soldier wellness program. *Southern M3(r)-3(n) wogcreeni., ., 4(1):*

Orstead C., D. Arrington, S. K. Kamath, R. Olson and M. B. Kohrs. 1985. Efficacy of prenatal nutrition counseling: weight gain, infant birth weight, and cost-effectiveness. *Journal of the American Dietetic Association* 85(1): 40-45.

Palmer J. L., G. E. Jennings and L. Massey. 1985. Development of an assessment form: attitude toward weight gain during pregnancy. *Journal of the American Dietetic Association* 85(8): 946-9498(9)-3(4)-</MCID 94 -1.x807T1_1 1 Tf1MCI/MCID 94 -1.x8CID 9470 >o03VD 94 -1.x

Rumbaut R. G. and J. R. Weeks. 1996. Unraveling a public health enigma: why do immigrants experience superior perinatal health outcomes? *Research in the Sociology of Health Care* 13: 335-388.

Rush D. 1974. Examination of the relationship between birthweight, cigarette smoking during pregnancy and maternal weight gain. *Journal of Obstetrics and Gynaecology of the British Commonwealth* 81(10): 746-752.

Rush D. 1981. Nutritional services during pregnancy and birthweight: a retrospective matched pair analysis. *Canadian Medical Association Journal* 125(6): 567-576.

Rush D., D. G. Horvitz, W. B. Seaver, J. Leighton, N. L. Sloan, S. S. Johnson, R. A. Kulka, J. W. Devore, M. Holt, J. T. Lynch and et al. 1988. The National WIC Evaluation: evaluation of the Special Supplemental Food Program for Women, Infants, and Children. IV. Study methodology and sample characteristics in the longitudinal study of pregnant women, the study of children, and the food expenditures study. *American Journal of Clinical Nutrition* 48(2 Suppl): 429-438.

Safyer S. M. and L. Richmond. 1995. Pregnancy behind bars. *Seminars in Perinatology* 19(4): 314-322.

Santry H. P., D. L. Gillen and D. S. Lauderdale. 2005. Trends in bariatric surgical procedures. *Journal of the American Medical Association* 294(15): 1909-1917.tAtr43(.)-3.tr43M.r43(.)-3( )13(1)-3(

Shl oSssoTras.

Singh G. K. and S. M. Yu. 1996. Adverse pregnancy outcomes: differences between US- and foreign-born women in major US racial and ethnic groups. *American Journal of Public Health* 86(6): 837-843.

Skull A. J., G. H. Slater, J. E. Duncombe and G. A. Fielding. 2004. Laparoscopic adjustable banding in pregnancy: safety, patient tolerance and effect on obesity-related pregnancy outcomes. *Obesity Surgery* 14(2): 230-235.

Smith L. M., L. L. LaGasse, C. Derauf, P. Grant, R. Shah, A. Arria, M. Huestis, W. Haning, A. Strauss, S. Della Grotta, J. Liu and B. M. Lester. 2006. The infant development, environment, and lifestyle study: effects of prenatal methamphetamine exposure, polydrug exposure, and poverty on intrauterine growth. *Pediatrics* 118(3): 1149-1156.

Sollid C. P., K. Wisborg, J. Hjort and N. J. Secher. 2004. Eating disorder that was diagnosed before pregnancy and pregnancy outcome. *American Journal of Obstetrics and Gynecology* 190(1): 204

Vilming B. and B. I. Nesheim. 2000. Hyperemesis gravidarum in a contemporary population in Oslo. *Acta Obstetricia et Gynecologica Scandinavica* 79(8): 640-643.

Vlietinck R., R. Derom, M. C. Neale, H. Maes, H. van Loon, C. Derom and M. Thiery. 1989. Genetic and environmental variation in the birth weight of twins. *Behavior Genetics* 19(1): 151-161.

Voigt M., S. Straube, P. Schmidt, S. Pildner von Steinburg and K. T. Schneider. 2007. [Standard values for the weight gain in pregnancy according to maternal height and weight]. *Zeitschrift fur Geburtshilfe und Neonatologie* 211(5): 191-203.

Wagner C. L., L. D. Katikaneni, T. H. Cox and R. M. Ryan. 1998. The impact of prenatal drug exposure on the neonate. *Obstetrics and Gynecology Clinics of North America* 25(1): 169-194.

Walker L. O. and M. Kim. 2002. Psychosocial thriving during late pregnancy: relationship to ethnicity, gestational weight gain, and birth weight. *Journal of Obstetric, Gynecologic, and Neonatal Nursing* 31(3): 263-274.

Waterland R. A. and R. L. Jirtle. 2003. Transposable elements: targets for early nutritional effects on epigenetic gene regulation. *Molecular and Cellular Biology* 23(15): 5293-5300.

Waterland R. A., M. Travisano, K. G. Tahiliani, M. T. Rached and S. Mirza. 2008. Methyl donor [illegible] gestational amplification of obesity.

Wolff M. S., S. Engel, G. Berkowitz, S. Teitelbaum, J. Siskind, D. B. Barr and J. Wetmur. 2007. Prenatal pesticide and PCB exposures and birth outcomes. *Pediatric Research* 61(2): 243-250.

Yanagisawa K., N. Iwasaki, M. Sanaka, S. Minei, M. Kanamori, Y. Omori and Y. Iwamoto. 1999. Polymorphism of the beta3-adrenergic receptor gene and weight gain in pregnant diabetic women. *Diabetes Research and Clinical Practice* 44(1): 41-47.

Websites:
http://www.ojp.usdoj.gov/bjs/prisons.htm
http://cdc.gov/yrbss

(2008) AHRQ evidence-based review on outcomes of gestational weight gain that are related to the mother during pregnancy, at delivery, and postpartum periods. Studies were rated "good," "fair," or "poor" based on a scoring algorithm developed by the AHRQ study reviewers using previously published guidelines (Downs and Black, 1998; Deeks et al., 2003). The methodological approach and system of rating articles used in the AHRQ review is provided in Appendiq((i)-(g)-3(i)-3(q((i)-(g)-c013(t)-3(i)3( )-51(u)si)-(g)-c03( )-

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.

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 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 most-studied prenatal maternal outcomes resulting from excessive GWG are pregnancy-associated hypertension (including preeclampsia and eclampsia) and the risk of complications in labor and delivery. In the postpartum period, potential consequences include weight retention and lactation performance. Weight retention can lead to higher weight status in subsequent pregnancies predisposing the woman to more adverse reproductive outcomes (creating a cycle of risk) and other long-term maternal health consequences such as increased risk for type 2 diabetes, cancer, cardiovascular disease, and mental health issues. Therefore these outcomes are also included in the model. Unfortunately the literature in this area does not allow inference of causality since it is based solely

Gestational Diabetes and Impaired Glucose Tolerance

Although pregnancy is frequently accompanied by a pronounced physiological decrease in peripheral insulin sensitivity (reviewed in Chapter 3), the combination of decreased peripheral insulin sensitivity and beta-cell dysfunction can lead to the development of abnormal glucose tolerance during pregnancy, or GDM. 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( )-47(t)-2u()-3(m)-3(a.-3(e)-3(n)-3(,)-3( )-66(2)-3(07-3(0-73(3).-3

Outside the AHRQ review, Catalano et al. (1993) reported that weight gain in women with GDM was less than in a normal glucose tolerance group primarily because of greater pregravid weight. However, when GWG was assessed separately for early, mid- and late gestation, there was a significant decreased rate of weight gain in overweight women with GDM 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.

Hypertensive Disorders

Hypertensive disorders during pregnancy include pregnancy-induced

GWG and hypertensive disorders. Five of these studies (two rated fair and the rest rated poor) examined pregnancy-induced hypertension (Edwards et al., 1996; Bianco et al., 1998; Thorsdottir et al., 2002; Brennand et al., 2005; Jensen et al., 2005). Only two of the studies reported an association between higher GWG and pregnancy-induced hypertension (Thorsdottir et al., 2002; Jensen et al., 2005). The five studies differed in their control

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.

CONSEQUENCES AT DELIVERY

The IOM (1990) report examined the link between GWG and complications during labor and delivery but only because such complications were viewed as being consequences of the delivery of a large-for-gestational age (LGA) infant. That report concluded that the contribution of GWG to delivery outcomes was quite small. Since then, the literature has grown and the outcomes related to delivery have been subdivided to better understand the process of labor. The discussion below addresses recent evidence for an association between GWG and each of these delivery outcomes. 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.

Induction of Labor

The AHRQ review (Viswanathan et al., 2008) included five studies 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. Although statistically significant increases in the outcomes associated with high GWG were reported in all five studies, comparisons across studies were not meaningful because of

differences in the definition of high GWG and a lack of control for confounding factors.

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). Although two of the three studies found a significant increase in the length of labor with higher weight gains, both lacked control for confounding factors (Ekblad and Grenman, 1992; Purfield and Morin, 1995). As a result, the evidence was rated as weak for an association between higher GWG and longer duration of labor.

Mode of Delivery

Substantial research has been conducted since the IOM (1990) report on the association between GWG and mode of delivery, with the AHRQ review (Viswanathan et al., 2008) examining a total of 21 studies 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; BT*[(M)-3[(M)-3f3(e)-3(r)-3( )-1(e)-3(t)-3( )-1(a)-3(l)-

dence among obese and morbidly obese women was rated as weak (Parker

2002; Li et al., 2003; Hilson et al., 2006; Baker et al., 2007). Although three of the studies 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), the evidence for any association between GWG and duration of exclusive or any breastfeeding was rated weak; evidence that low weight gain is associated with decreased initiation of breastfeeding was rated moderate. Since the AHRQ review, the committee identified one other study, a cross-sectional study done in Greece reporting 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 (Manios et al., 2008).

Postpartum Weight Retention

Postpartum weight is a woman's weight immediately after delivery of the fetus, placenta, and amniotic fluid. 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

GESTATIONAL WEIGHT GAIN FOR THE MOTHER

Postpartum Depression

As with depression during pregnancy, 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

Other Adverse Health Outcomes

Mental Health

As previously discussed, 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 for a connection between postpartum weight retention up to 1 year post-delivery and self-esteem/depression. These studies did not control for prepregnancy BMI.

Cancer

The committee found weak evidence for an association of GWG and risk of breast cancer. Specifically, a 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. Among premenopausal women in the population, weight gains of > 16 kg

woman's life. It is well established, however, that obesity is associated with

Research Recommendation 5-1: The committee recommends that the National Institutes of Health and other relevant agencies should provide support to researchers to conduct observational and experimental studies 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 disease 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 ma-

Baker J. L., M. Gamborg, B. L. Heitmann, L. Lissner, T. I. Sorensen and K. M. Rasmussen. 2008. Breastfeeding reduces postpartum weight retention. *American Journal of Clinical Nutrition* 88(6): 1543-1551.

Bellamy L., J. P. Casas, A. D. Hingorani and D. J. Williams. 2007. Pre-eclampsia and risk of cardiovascular disease and cancer in later life: systematic review and meta-analysis. *British Medical Journal* 335(7627): 974.

Berg A. H. and P. E. Scherer. 2005. Adipose tissue, inflammation, and cardiovascular disease. *Circulation Research* 96(9): 939-949.

Bianco A. T., S. W. Smilen, Y. Davis, S. Lopez, R. Lapinski and C. J. Lockwood. 1998. Pregnancy outcome and weight gain recommendations for the morbidly obese woman. *Obstetrics and Gynecology* 91(1): 97-102.

Bodnar L. M., J. M. Catov, M. A. Klebanoff, R. B. Ness and J. M. Roberts. 2007. Prepregnancy body mass index and the occurrence of severe hypertensive disorders of pregnancy. *Epidemiology* 18(2): 234-239.

Brennand E. A., D. Dannenbaum and N. D. Willows. 2005. Pregnancy outcomes of First Nations women in relation to pregravid weight and pregnancy weight gain. *Journal of Obstetrics and Gynaecology Canada* 27(10): 936-944.

Butte N. F., C. Garza, J. E. Stuff, E. O. Smith and B. L. Nichols. 1984. Effect of maternal diet and body composition on lactational performance. *American Journal of Clinical Nutrition* 39(2): 296-306.

Butte N. F., K. J. Ellis, W. W. Wong, J. M. Hopkinson and E. O. Smith. 2003. Composition of gestational weight gain impacts maternal fat retention and infant birth weight. *American Journal of Obstetrics and Gynecology* 189(5): 1423-1432.

Callaway L. K., H. D. McIntyre, M. O'Callaghan, G. M. Williams, J. M. Najman and D. A. Lawlor. 2007. The association of hypertensive disorders of pregnancy with weight gain over the subsequent 21 years: findings from a prospective cohort study. *American Journal of Epidemiology* 166(4): 421-428.

Calle E. E., C. Rodriguez, K. Walker-Thurmond and M. J. Thun. 2003. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. *New England Journal of Medicine* 348(17): 1625-1638.

Catalano P. M. 2007. Increasing maternal obesity and weight gain during pregnancy: the obstetric problems of plentitude. *Obstetrics and Gynecology* 110(4): 743-744.

Catalano P. M., N. M. Roman, E. D. Tyzbir, A. O. Merritt, P. Driscoll and S. B. Amini. 1993. Weight gain in women with gestational diabetes. *Obstetrics and Gynecology* 81(4): 523-528.

Cedergren M. 2006. Effects of gestational weight gain and body mass index on obstetric outcome in Sweden. *International Journal of Gynaecology and Obstetrics* 93(3): 269-274.

Chen G., S. Uryasev and T. K. Young. 2004. On prediction2-41(S)-3(.)-3(3(t)]TJEM.(T)108(.)-3(.(2-41(S)-3(

Jain N. J., C. E. Denk, L. K. Kruse and V. Dandolu. 2007. Maternal obesity: can pregnancy weight gain modify risk of selected adverse pregnancy outcomes? *American Journal of Perinatology* 24(5): 291-298.

Jenkin W. and M. Tiggemann. 1997. Psychological effects of weight retained after pregnancy. *Women & Health* 25(1): 89-98.

Jensen D. M., P. Ovesen, H. Beck-Nielsen, L. Molsted-Pedersen, B. Sorensen, C. Vinter and P. Damm. 2005. Gestational weight gain and pregnancy outcomes in 481 obese glucose-tolerant women. *Diabetes Care* 28(9): 2118-2122.

Johnson J. W., J. A. Longmate and B. Frentzen. 1992. Excessive maternal weight and pregnancy outcome. *American Journal of Obstetrics and Gynecology* 167(2): 353-370; discussion 370-352.

Joseph K. S., D. C. Young, L. Dodds, C. M. O'Connell, V. M. Allen, S. Chandra and A. C. Allen. 2003. Changes in maternal characteristics and obstetric practice and recent increases in primary cesarean delivery. *Obstetrics and Gynecology* 102(4): 791-800.

Kabiru W. and B. D. Raynor. 2004. Obstetric outcomes associated with increase in BMI category during pregnancy. *American Journal of Obstetrics and Gynecology* 191(3): 928-932.

Keppel K. G. and S. M. Taffel. 1993. Pregnancy-related weight gain and retention: implications of the 1990 Institute of Medicine guidelines. *American Journal of Public Health* 83(8): 1100-1103.

Kieffer E. C., W. J. Carman, B. W. Gillespie, G. H. Nolan, S. E. Worley and J. R. Guzman. 2001. Obesity and gestational diabetes among African-American women and Latinas in Detroit: implications for disparities in women's health. *Journal -women'. . .ies in 3(t)-3(c)-3(o)-3(m)-*

Linne Y., L. Dye, B. Barkeling and S. Rossner. 2004. Long-term weight development in women: a 15-year follow-up of the effects of pregnancy. *Obesity Research* 12(7): 1166-1178.

Luke B., M. L. Hediger and T. O. Scholl. 1996. Point of diminishing returns: when does gestational weight gain cease benefiting birthweight and begin adding to maternal obesity? *Journal of Maternal-Fetal Medicine* 5(4): 168-173.

Madlon-Kay D. J. 1993. Striae gravidarum. Folklore and fact. *Archives of Family Medicine* 2(5): 507-511.

Manios Y., E. Grammatikaki, K. Kondaki, E. Ioannou, A. Anastasiadou and M. Birbilis. 2008. The effect of maternal obesity on initiation and duration of breast-feeding in Greece: the GENESIS study. *Public Health Nutrition* 1-8.

Marrero J. M., P. M. Goggin, J. S. de Caestecker, J. M. Pearce and J. D. Maxwell. 1992. Determinants of pregnancy heartburn. *British Journal of Obstetrics and Gynaecology* 99(9): 731-734.

Murakami M., M. Ohmichi, T. Takahashi, A. Shibata, A. Fukao, N. Morisaki and H. Kurachi. 2005. Prepregnancy body mass index as an important predictor of perinatal outcomes in Japanese. *Archives of Gynecology and Obstetrics* 271(49or 2s in

6

Consequences of Gestational Weight Gain for the Child

The emphasis of the report *Nutrition During Pregnancy* (IOM, 1990)

factors ranging from the social/built/natural environment (macro) through behavior, physiology, and genetics (micro) (see Chapter 4) and interacting with each other over the life course, with different determinants being more or less important at different life stages. The other paradigm—the "developmental origins of health and disease" paradigm—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 and can have long-lasting, sometimes irreversible, health consequences. The pe-

differ from other animal species in duration of pregnancy, placentation, and other important factors, the importance of the findings from animal studies

comes include stillbirth and birth defects. In contrast, neonatal body com-

EFFECTS ON NEONATAL MORBIDITY AND MORTALITY

There is a substantial literature on prepregnancy BMI and neonatal morbidity and mortality; maternal prepregnancy BMI is strongly associated with infant mortality and a number of other clinically important outcomes, including stillbirth and tillbi(b)-F(t)-3(i)-gT*[(i)-3(r)-3(a)-3(t)-36(a)--(a)-2(a)-)(a)-.(a)-

to confounding. These component relationships (prepregnancy BMI and

Birth Defects

The authoring committee of the IOM (1990) report did not identify any studies on the association between GWG and birth defects. Since the etiologic period for congenital defects is so early in pregnancy, GWG is not likely to be causally relevant. .n953(.)-3( )17(.n3(r)-3(e)h3(l)-3(y)-3( )h3( )17(b)-3(e)C T

showing a diminishing effect of low GWG with increasing BMI. In the highest GWG group, 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, showing the exact opposite tendency—excessive GWG was more strongly associated with neonatal death with increasing prepregnancy 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 postneonatal deaths but was less pronounced (see 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

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.

importance of the outcome but also because of the implications for the more voluminous literature on fetal growth and preterm birth. Although

immutable, however, because health disparities are strongly influenced by social and behavioral factors.

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

had infants that weighed 93 g less on average than controls. Fewer of the treated number developed GDM or had cesarean deliveries.

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 lower 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. There are several possi(h)-6(e)-6ossi ns(i)(r)-6(a)-6(l)-3182(n)-3scs(r)-6(a)-11t3r6(a)-1

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 an 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 g). 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 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 duration of gestation, studies that separate these two components are more informative.

First, 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*

categories for underweight women, and a 3- to 4-fold gradient in risk for women in the other BMI categories (see Table 6-2). 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.

Summary of the Evidence on an Association Between GWG and Fetal Growth

In summary, the evidence that GWG is related to birth weight for gestational age based on observational studies 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.

It is not yet clear, however, whether the associations between GWG and birth weight for gestational age is impacted by fpac6213(i)-3(s)-144(n)-3(o)-3(

ity, smoking status, or other maternal attributes has been sparse, and the few studies summarized in the AHRQ review inconsistent. In addition to prepregnancy BMI, the only other factor that appears to imp1(t)-3(o)-61(a)-3(p)-3(p(e

a consistently increased risk of preterm birth among women 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 those studies that provided relative risks comparing higher and lower GWG to the middle range, 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.

Effect modification by prepregnancy BMI (Siega-Riz et al., 1996;

The committee found no studies that directly link 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, 2004; Kumarasamy et al., 2005).

Again, the committee also found no studies directly linking GWG to amniochorionic-decidual or systemic inflammation. 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 are known to 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 the amino acids needed for synthesis of antioxidant defense enzymes, such as glutathione and albumin (reactive oxygen species scavengers); and many micronutrients themselves are antioxidants. Increases 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.

Summary of the Evidence on an Association Between GWG and Preterm Birth

In summary, there is strong evidence for a U-shaped 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. 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 birth are stronger among underweight women.

The committee was unable to infer 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, suggesting that a direct causal relationship is biologically 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.

LONG-TERM CONSEQUENCES

The IOM (1990) report recommendations for GWG focused largely on avoiding inadequate GWG and the short-term consequences of low fetal growth and prematurity (see Chapter 1). Since that time, the emergence of epidemic obesity in the U.S. population has raised the possibility that excessive weight gain may also be harmful. A small number of recent studies have addressed the relationship between GWG and adiposity at birth, markers of childhood obesity and cardiometabolic sequelae of childhood obesity. The following discussion summarizes the committee's review of the evidence for associations between GWG and neonatal body composition, infant weight gain, breastfeeding initiation, and other long-term outcomes.

Neonatal Body Composition

As previously explained (see Fetal Growth section in this chapter), GWG is directly associated with fetal growth as measured by birth weight for gestational age. For long-term adiposity-related outcomes, however, it is important to measure not only weight (and length) at birth but also body composition. As mentioned in the chapter introduction, it has been hypothesized that relative amounts of adiposity and lean mass in fetal and neonatal life are important in setting long-term cardio-metabolic trajectories. Catalano and colleagues performed a series of studies examining the relationships between various maternal characteristics and neonatal body composition as 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, weight gain was higher among lean/average (mean 15.2 kg) than overweight/obese (13.8 kg) women. Among the overweight/obese women, stepwise regression analyses that included pregravid weight as a covariate revealed that the higher the GWG, the more the newborn fat mass. The authors did not report a correlation among the lean women, presumably because the associated p-value exceeded 0.05. In another study, which combined data from diabetic and nondiabetic pregnant women (total n = 415), GWG was directly associated with both lean and fat mass at birth (Catalano and Ehrenberg, 2006). The latter results are consistent with those of Udal et al. (1978), who found a direct association between GWG and the sum of 8 neonatal skinfold measurements among 109 nondiabetic mothers

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 vs. I	per 5 lb I/E vs. A	per 1 kg I/E vs. A
Child BMI Outcome	OR for overweight	OR for overweight	OR for overweight	OR for overweight

10,000 7-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), 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

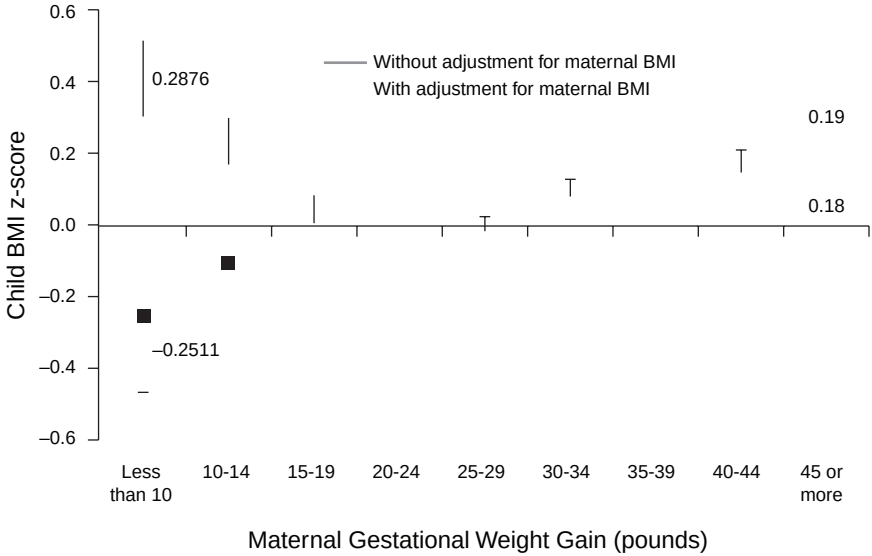

FIGURE 6-3 Associations of maternal gestational weight gain with child BMI z-score at ages 9-14 years, with and without adjustment for maternal prepregnancy BMI. All estimates are adjusted for maternal age, race/ethnicity, marital status, household income, paternal education, child sex, gestation length, 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.

and female 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

Long-term neurodevelopment in preterm SGA In preterm SGA infants, the majority of longitudinal studies reviewed by the committee 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, 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 sleep 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.

Effect size was again assessed for cognitive measures. Of 19 studies reviewed, 13 reported cognitive scores by SGA status; of these, 1 reported

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), socioeconomic, 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, 2005; Hack et al., 1998; Brandt et al., 2003; Kilbride et al., 2004; 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). Because of these study design limitations, 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.

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 The Apgar score (see Glossary in Appendix A) assessments are usually conducted 1 and 5 minutes after birth, and scores can range from 0 to 10. However, Apgar scores in term infants, even at 5 minutes, have important limitations, as they are not adequate predictors of longer term morbidity and mortality and do not correlate well with neurological outcomes (ACOG, 2006) although very low scores (0-3) associated with low birth weight do predict neonatal mortality. The AHRQ review (Viswanthan et al., 2008) identified five studies examining the influence of GWG on a newborn's Apgar score (Stevens-Simon and McAnarney, 1992; Nixon et al., 1998; Cedergren et al., 2006, Stotland et al., 2006; Wataba et al., 2006). Taken together, 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 7(t)-1(56(n)-3(e)-3(u)-n(6)-TJEM

in mobilization of maternal adipose tissue and possibly lean body mass.

to controls with adequate GWG. In that study, 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 Two lines of evidence link GWG to cancer: First, a recent meta-analysis (Hjalgrim et al., 2003) estimated that d lha-3( )-76oe7e7e7e7e7

levels, providing some support to the estrogen hypothesis; however, higher GWG was associated with 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 offspring of 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 nrestivt whigat gait ait epregnancyecsoldlraldtct rvhadee

weight gain predicts SGA. Both LGA and SGA are themselves markers of neonatal morbidity. The literature on preterm birth is more ambiguous because of a less-extensive body of epidemiologic evidence, a nonlinear (U-shape) relationship between GWG and preterm birth that is modest in magnitude, and uncertainty about biologic mechanisms. Even when GWG is measured in a way that takes into account the shortened duration of pregnancy associated with GWG with preterm births, results are 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, postpartum weight retention, and childhood

LGA. Despite a limited number of randomized controlled trials, biological plausibility from animal models is strong. Relative risks for GWG and SGA appear to be higher among women with lower prepregnancy BMI.

4. The evidence for a relationship between GWG and preterm birth,

GESTATIONAL WEIGHT GAIN FOR THE CHILD

Gillman M. W. 2008. The first months of life: a critical period for development of obesity. *American Journal of Clinical Nutrition* 87(6): 1587-1589.

Gluckman P. D. and M. A. Hanson. 2006a. Changing times: the evolution of puberty. *Molecular and Cellular Endocrinology* 254-255: 26-31.

Gluckman P. D. and M. A. Hanson. 2006b. Evolution, development and timing of puberty. *Trends in Endocrinology and Metabolism* 17(1): 7-12.

IOM (Institute of Medicine). 1990. *Nutrition During Pregnancy.* Washington, DC: National Academy Press.

IOM. 2007. *Preterm Birth: Causes, Consequences, and Prevention.* Washington, DC: The National Academies Press.

Kiel D. W., E. A. Dodson, R. Artal, T. K. Boehmer and T. L. Leet. 2007. Gestational weight gain and pregnancy outcomes in obese women: how much is enough? *Obstetrics and Gynecology* 110(4): 752-758.

Kilbride H. W., K. Thorstad and D. K. Daily. 2004. Preschool outcome of less than 801-gram preterm infants compared with full-term siblings. *Pediatrics* 113(4): 742-747.

King J. C. 2006. Maternal obesity, metabolism, and pregnancy outcomes. *Annual Review of Nutrition* 26: 271-291.

Kinnunen T. I., R. Luoto, M. Gissler, E. Hemminki and L. Hilakivi-Clarke. 2004. Pregnancy weight gain and breast cancer risk. *BMC Women's Health* 4(1): 7.

Kono Y., J. Mishina, T. Takamura, H. Hara, I. Sakuma, S. Kusuda and H. Nishida. 2007. Impact of being small-for-gestational age on survival and long-term outcome of extremely premature infants born at 23-27 weeks' gestation. *Journal of Perinatal Medicine* 35(5): 447-454.

Kramer M. S. and R. Kakuma. 2003. Energy and protein intake in pregnancy. *Cochrane Database of Systematic Reviews* (4): CD000032.

Kuh D. and Y. Ben-Shlomo. 2004. *A Life Course Approach to Chronic Disease Epidemiology: Tracing the Origins of Ill-Health from Early to Adult Life.* London: Oxford University Press.

Kulseng S., A. Jennekens-Schinkel, P. Naess, P. Romundstad, M. Indredavik, T. Vik and A.

Maffeis C., R. Micciolo, A. Must, M. Zaffanello and L. Pinelli. 1994. Parental and perinatal factors associated with childhood obesity in north-east Italy. *International Journal of Obesity and Related Metabolic Disorders* 18(5): 301-305.

McCarton C. M., I. F. Wallace, M. Divon and H. G. Vaughan, Jr. 1996. Cognitive and neu-

Ong K. K. and R. J. Loos. 2006. Rapid infancy weight gain and subsequent obesity: systematic reviews and hopeful suggestions. *Acta Paediatrica* 95(8): 904-908.

Ong K. K., M. L. Ahmed, P. M. Emmett, M. A. Preece and D. B. Dunger. 2000. Association between postnatal catch-up growth and obesity in childhood: prospective cohort study. *British Medical Journal* 320(7240): 967-971.

Paavonen E. J., S. Strang-Karlsson, K. Raikkonen, K. Heinonen, A. K. Pesonen, P. Hovi, S. Andersson, A. L. Jarvenpaa, J. G. Eriksson and E. Kajantie. 2007. Very low birth weight increases risk for sleep-disordered breathing in young adulthood: the Helsinki Study of Very Low Birth Weight Adults. *Pediatrics* 120(4): 778-784.

Paz I., R. Gale, A. Laor, Y. L. Danon, D. K. Stevenson and D. S. Seidman. 1995. The cognitive outcome of full-term small for gestational age infants at late adolescence. *Obstetrics and Gynecology* 85(3): 452-456.

Paz I., A. Laor, R. Gale, S. Harlap, D. K. Stevenson and D. S. Seidman. 2001. Term infants with fetal growth restriction are not at increased risk for low intelligence scores at age 17 years. *Journal of Pediatrics* 138(1): 87-91.

Peng Y., B. Huang, F. Biro, L. Feng, Z. Guo and G. Slap( )-32(S)-3(Z)-3(.)-3( )-31. Gutcom. o2 lo2 Biwtw-3(

Samuelsson A. M., P. A. Matthews, M. Argenton, M. R. Christie, J. M. McConnell, E. H. Jansen, A. H. Piersma, S. E. Ozanne, D. F. Twinn, C. Remacle, A. Rowlerson, L. Poston and P. D. Taylor. 2008. Diet-induced obesity in female mice leads to offspring hyperphagia, adiposity, hypertension, and insulin resistance: a novel murine model of developmental programming. *Hypertension* 51(2): 383-392.

Schieve L. A., M. E. Cogswell and K. S. Scanlon. 1999. Maternal weight gain and preterm delivery: differential effects by body mass index. *Epidemiology* 10(2): 141-147.

Segal P., J. K. Hamilton, M. Sermer, P. W. Connelly, A. J. Hanley, B. Zinman and R. Retnakaran. 2008. Maternal obesity and familial history of diabetes have opposing effects on infant birth weight in women with mild glucose intolerance in pregnancy. *Journal of Maternal-Fetal & Neonatal Medicine* 21(1): 73-79.

Sewell M. F., L. Huston-Presley, D. M. Super and P. Catalano. 2006. Increased neonatal fat mass, not lean body mass, is associated with maternal obesity. *American Journal of Obstetrics and Gynecology* 195(4): 1100-1103.

Shaw G. M., K. Todoroff, S. L. Carmichael, D. M. Schaffer and S. Selvin. 2001. Lowered weight gain during pregnancy and risk of neural tube defects among offspring. *International Journal of Epidemiology* 30(1): 60-65.

Siega-Riz A. M., L. S. Adair and C. J. Hobel. 1996. Maternal underweight status and inadequate rate of weight gain during the third trimester of pregnancy increases the risk of preterm delivery. *Journal of Nutrition* 126(1): 146-153.

Silva P. A., R. McGee and S. Williams. 1984. A longitudinal study of the intelligence and behavior of preterm and small for gestational age children. *Journal of Developmental and Behavioral Pediatrics* 5(1): 1-5.

Silverman B. L., T. Rizzo, O. C. Green, N. H. Cho, R. J. Winter, E. S. Ogata, G. E. Richards and B. E. Metzger. 1991. Long-term prospective evaluation of offspring of diabetic mothers. *Diabetes* 40(Suppl 2): 121-125.

Simmons R. A. 2007. Developmental origins of diabetes: the role of epigenetic mechanisms. *Current Opinion in Endocrinology, Diabetes, and Obesity* 14(1): 13-16.

Sinclair K. D., R. G. Lea, W. D. Rees and L. E. Young. 2007. The developmental origins of health and disease: current theories and epigenetic mechanisms. *Society of Reproduction and Fertility Supplement* 64: 425-443.

Sommerfelt K., H. W. Andersson, K. Sonnander, G. Ahlsten, B. Ellertsen, T. Markestad, G. Jacobsen, H. J. Hoffman and L. Bakketeig. 2000. Cognitive development of term small

Strang-Karlsson S., K. Raikkonen, E. Kajantie, S. Andersson, P. Hovi, K. Heinonen, A. K. Pesonen, A. L. Jarvenpaa, J. G. Eriksson and E. J. Paavonen. 2008a. Sleep quality in young adults with very low birth weight—the Helsinki study of very low birth weight adults. *Journal of Pediatric Psychology* 33(4): 387-395.

Strang-Karlsson S., K. Raikkonen, A. K. Pesonen, E. Kajantie, E. J. Paavonen, J. Lahti, P. Hovi, K. Heinonen, A. L. Jarvenpaa, J. G. Eriksson and S. Andersson. 2008b. Very low birth weight and behavioral symptoms of attention deficit hyperactivity disorder in young adulthood: the Helsinki study of very-low-birth-weight adults. *American Journal of Psychiatry* 165(10): 1345-1353.

Strauss R. S. 2000. Adult functional outcome of those born small for gestational age: twenty-six-year follow-up of the 1970 British Birth Cohort. *Journal of the American Medical Association* 283(5): 625-632.

Sullivan M. C., K. Hawes, S. B. Winchester and R. J. Miller. 2008. Developmental origins theory from prematurity to adult disease. *Journal of Obstetric, Gynecologic, and Neonatal Nursing* 37(2): 158-164.

Susser M. 1991. Maternal weight gain, infant birth weight, and diet: causal sequences. *American Journal of Clinical Nutrition* 53(6): 1384-1396.

Taveras E. M., S. L. Rifas-Shiman, M. B. Belfort, K. P. Kleinman, E. Oken and M. W. Gillman. 2009. Weight status in the first 6 months of life and obesity at 3 years of age. *Pediatrics* 123(4): 1177-1183.

Tavris D. R. and J. A. Read. 1982. Effect of maternal weight gain on fetal, infant, and childhood death and on cognitive development. *Obstetrics and Gynecology* 60(6): 689-694.

Trichopoulos D. 1990. Hypothesis: does breast cancer originate in utero? *Lancet* 335(8695): 939-940.

Udal J. N., G. G. Harrison, Y. Vaucher, P. D. Walson and G. Morrow, 3rd. 1978. Interaction of maternal and neonatal obesity. *Pediatrics* 62(1): 17-21.

Van Assche F. A., L. Aerts and W. Gepts. 1979. Morphological changes in the endocrine pancreas in pregnant rats with experimental diabetes. *Journal of Endocrinology* 80(2): 175-179.

Watt J. and K. T. Strongman. 1985. Mother-infant interactions at 2 and 3 months in preterm, small-for-gestational-age, and full-term infants; their relationship with cognitive development at 4 months. *Early Human Development* 11(3-4)1_1 1 Tf8 0 0 8 235.1331 557.824 Tm[( )-3-13 <

outcomes and their frequency in the population. To develop estimates of risk and frequency, the committee used data from the published literature and from additional, commissioned analyses (see below).

The committee considered the incidences, long-term sequelae, and baseline risks of several potential outcomes associated with GWG (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 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. 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.

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). At the other extreme is the single target approach. For example, in the United States, the 1970 report *Maternal Nutrition and the*

analysis be undertaken "in which probabilities and utilities (values) are assigned to each potential outcome" to assist in balancing the risks and benefits of any recommendation.

for Cedergren's analysis (2007), none of these investigators considered the

variety of maternal and neonatal outcomes associated with prepregnant BMI and GWG and their interaction. For those outcomes 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), the researchers calculated the absolute risk 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) detected a dramatic increase in postpartum weight retention ≥ 5 kg with increasing GWG in all categories of prepregnant BMI. In addition, they calculated the proportion of women who had changed from one BMI category to another at 6 months postpartum according to GWG. They found that only 0.4 percent of underweight women became 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." For normal weight, overweight, and obese women, however, the trade-off 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. As was the case for the other studies, Nohr et al. (2008) 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 vary widely (Table 7-1). This is particularly striking for underweight and normal weight women but is also the case for overweight women. The differences in conclusions may have resulted from the different mix of outcomes consideresion utsi oted

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	—	—	—		

of Health (NHLBI, 1998). These categories have been widely adopted in the United States and internationally and, if used in formulating recommen-

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

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 be over 20 percent instead of being about 5 percent. Thus, the weight gain

hood 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 either 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 on nutrition and physical activity) or other interventions that might be deemed necessary to achieve the recommended levels of weight gain. Thus, the committee recognizes that full implementation of these guidelines may entail additional medical expg tional

1. Before conception, use consistent and reliable procedures to measure and record in the medical record the woman's weight and height without shoes.
2. Determine the woman's prepregnancy BMI.
3. Carefully measure the woman's height without shoes and weight in light clothing at the first prenatal visit using procedures that have been rigorously standardized at the site of prenatal care. 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 and together with the pregnant woman, set a weight-gain goal based on prepregnant BMI and other relevant considerations and explain to the woman 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, together with the woman try to determine the cause and then develop and implement corrective actions.

DISCUSSION OF THE NEW GUIDELINES

These new guidelines differ from those issued in 1990 in two important

gained < 5 kg. It is possible, based on the data collected in these investigations and compared to higher gains, that weight gains < 5 kg may be associated with a more favorable trade-off among outcomes. However, the

women, with increased risk of LGA and its consequences. 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

veloped from published and commissioned research data needed to support a more complete and persuasive analysis were unavailable. In particular, more information is needed on associations between GWG and longer term maternal outcomes, such as postpartum weight retention and later reproductive function and health, and child health outcomes such as fetal growth restriction, child neurocognitive outcomes, and o -3(e)-34(o)-3le suc lucsue hgrowth

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 relevant federal agencies, private voluntary organizations, and medical and public health organizations should 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

Straube S., M. Voigt, V. Briese and K. T. Schneider. 2008. Weight gain in pregnancy according to maternal height and weight. *Journal of Perinatal Medicine* 36(5): 405-412.

Susser M. 1991. Maternal weight gain, infant birth weight, and diet: causal sequences. *American Journal of Clinical Nutrition* 53(6): 1384-1396.

Tanner J. M. and A. M. Thomson. 1970. Standards for birthweight as gestation periods from 32 to 42 weeks, allowing for maternal height and weight. *Archives of Disease in Childhood* 45(242): 566-569.

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 Internat9iETEMC BTteniETv9(r)-9(n)-9

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) as well as interventions that have been conducted to improve GWG in response to the Institute of Medicine (IOM) 1990 guidelines. In addition, the committee 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 the committee's work, this chapter provides a framework for developing such a plan.

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heavier than in the past challenge them to meet the previous guidelines (IOM, 1990) and will continue to make it difficult for women to meet the

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.CID 15116 >>BDC /T1_2 1 Tf0.55215 >>BDC /C0_0 (n = 1 , 2) (n = 2)
erweight (2 .0-2 .9 kg/m						

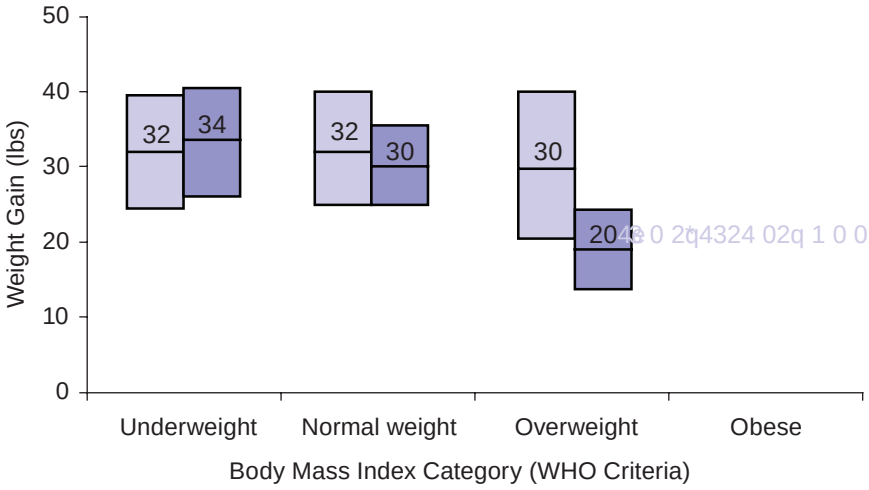

FIGURE 8-1 Comparison of weight gain by BMI category between data reported in the Pregnancy Risk Assessment Monitoring System (PRAMS), 2002-2003, and weight gain as recommended in the new guidelines.

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. Although such services are not uniformly available today and may not be covered by medical insurance plans, 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 omeCOons

Most recent studies have focused on various ways to help women to limit their weight gain during pregnancy. None of four trials conducted in North American populations was completely successful in helping women limit GWG and adhere to the IOM (1990) guidelines. First, in a study of Cree women from Quebec, Gr(a)-3(y)-3/Span <<1v1.MCID 1537o(p)-3(l)-3(o)-3(v1.M

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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 1-hour dietary consultations that were designed to help them restrict their GWG to 6-7 kg 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 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 BMI. The 49 women in the intervention group received 5 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.

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 kg. Achieving this goal required a substantial investment in individual dietary or motivational coualo32.067 -1.gl -170(a)-3(n)-3(,)-3(l)-3( )-16(i)-3(n)-3(-170(S)-3(w)-3(e)-3(d)-
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IMPLEMENTATION STRATEGIES FOR NEW GUIDELINES

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, here 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, 1992a).

To meet the recommendations of this report fully, two different challenges must be met. First, a higher proportion of American women must conceive at a weight within the range of normal BMI values. 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.

Conceiving at a Normal BMI Value

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

Counseling is already an integral part of the preconception recommendations from the Centers for Disease Control and Prevention (CDC) (Johnson et al., 2006), which 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. The IOM report *Nu-*

trition During Pregnancy and Lactation: An Implementation Guide (IOM, 1992a) also includes practical guidelines for preconceptional care.

It is noteworthy that few intervention studies have evaluated ways to

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 health care 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 typically only some weight gain usually occurs in the first trimester and that weight gain is greater and 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 to women the intended messages 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 and for 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) that might be causing them to have

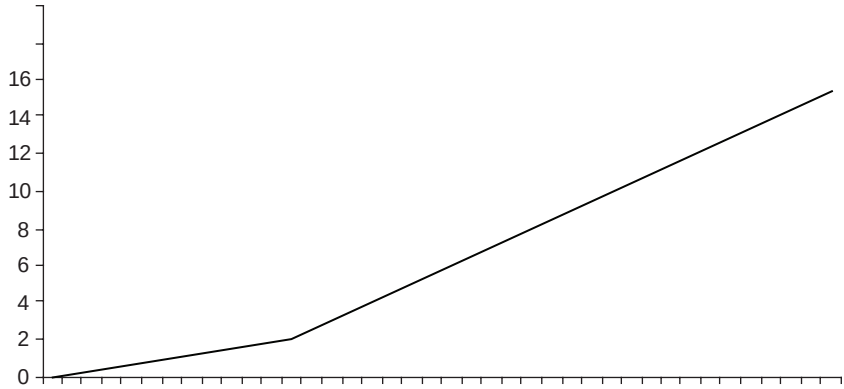
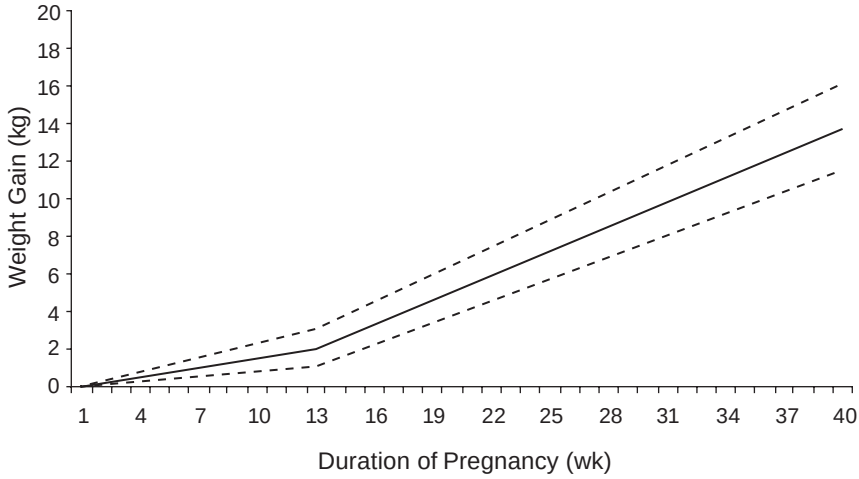


FIGURE 8-2 Recommended weight gain by week of pregnancy for underweight (BMI: < 18.5 kg/m^2) women (dashed lines represent range of weight gain).
NOTE: First trimester gains were determined using three sources (Siega-Riz et al., 1994; Abrams et al., 1995; Carmichael et al., 1997).



excessively high or low gains before any corrective action is recommended. The committee endorses this approach.

In addition to being made aware of their weight gain as pregnancy progresses through the use of weight-gain charts, 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 support necessary for returning to their prepregnant weight within the first year and for achieving normal BMI values before 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 [available online at 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 the type of trained practitioners that work in many health clubs and community-based ( )-26(t)-(t)-3(h)-3( )-26(c)-3(l)-3(MT06-(t)-tr)-3(a)-3(c)-3(i)-3(l)-3(i)-

cal activity is available on the Internet (see, for example, MyPyramid.gov [available online at http://mypyramid.gov/pyramid/physical_activity_tips.html, accessed February 18, 2009]), including advice specifically designed for pregnant women (available online at http://www.acog.or3(l)-3(u)-3(d)-3(i)-3(n)-3(g)

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 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). The community has a particularly important role to play in fostering appropriate GWG in low-income women. Approaches to consider range from social marketing (Siegel and Lotenberg, 2007) to improving the cultural skills of the health care providers that communicate GWG recommendations at an individual level (Haughton and George, 2008).

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 of women of childbearing age. In particular, the committee recognizes that full implementation of these guidelines would mean:

- Offering services, such as counseling on diet and physical activity, as well as access to contraception, to all overweight women, not just those seeking prenatal services, with the aim of improving

the current environment. However, the reduction in future health problems among both women and their children that could 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 studies must have adequate statistical power to determine not only if a given intervention helps women to gain within the recommended range but also if it improves the maternal and infant 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. It will also be important 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 avoiding postpartum weight retention.

Recommendations for Action

Action Recommendation 8-1: The committee recommends that a83(e)-3(r)- th

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 should 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.

REFERENCES

Abrams B., S. Carmichael and S. Selvin. 1995. Factors associated with the pattern of maternal weight gain during pregnancy. *Obstetrics and Gynecology* 86(2): 1 1A3(n)58230(14-3(89(.)-3(89(.)v)-3
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Haughton B. and A. George. 2008. The Public Health Nutrition workforce and its future challenges: the US experience. *Public Health Nutrition* 11(8): 782-791.
HHS (U.S. Department of Health and Human Services). 2008. *Physical Activ*

McFadden A. and S. King. 2008. *The Effectiveness of Public Health Interventions to Promote Nutrition of Pre-Conceptional Women*. York, UK: NICE Maternal and Child Nutrition Programme.

Olson C. M. 2008. Achieving a healthy weight gain during pregnancy. *Annual Review of Nutrition* 28: 411-423.

Olson C. M., M. S. Strawderman and R. G. Reed. 2004. Efficacy of an intervention to prevent excessive gestational weight gain. *American Journal of Obstetrics and Gynecology* 191(2): 530-536.

Parker J. D. and B. Abrams. 1992. Prenatal weight gain advice: an examination of the recent prenatal weight gain recommendations of the Institute of Medicine. *zE53(b)-3(s)-3(t)-3(e)-4(t)-3(r)Med*

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
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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 p.m. Break for Lunch

SESSION 3: GESTATIONAL WEIGHT GAIN AND PREGNANCY OUTCOMES

1:00 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. Nohr, 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

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Committee Member Biographical Sketches

KATHLEEN M. RASMUSSEN, Sc.D. (*Chair*

for Nutrition, the Society for Epidemiologic Research, and the Society for Pediatric and Perinatal Epidemiologic Research.

CLAUDE BOUCHARD, Ph.D.,

(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, BurlinTa s-121(D)-333(y)89(.)-3( )-333(sU)-3(n)-3(i)-3(c)-3(e)-3(i)-3(v)333(e)-3(d)-3( )-

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 health care 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 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

Women's Hospital. She also served a postdoctoral fellowship in cardiology

primary research activities and interests are in reproductive, environmental, and cancer epidemiology. Dr. 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 Depart

appendix A

Acronyms and Abbreviations, Glossary, and Supplemental Information

ACRONYMS AND ABBREVIATIONS

ACTH	adrenocorticotrophic hormone
ADHD	

ECF	extracellular fluid
FFA	free fatty acid
FFM	fat-free mass
FM	fat mass
GDM	ge(e)-3((e)(a)-3(t)-3( )-31(m)-3ETEoETEn)-3( )-31lmadm(e)(te)-3((e)(a)-3(

PNSS	Pregnancy Nutrition Surveillance System
PRAMS	Pregnancy Risk Assessment Monitoring System
PROM	premature rupture of membranes
PTB	preterm birth
QALY	quality-adjusted life-years
SD	standard deviation
SGA	small-for-gestational age
SNP	single nucleotide polymorphism
STBM	syncytiotrophoblast microparticles
TBK	total body potassium
TBN	total body nitrogen
TBW	total body water
TEE	total energy expenditure
TNF-α	tumor necrosis factor-alpha
VLBW	very low birth wel-3(cb-3(p)-3(h)-3(a)]TJETEMC /Span <</MCID 16633

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.

Adiponectin

Also called adipocyte complement-related protein of 30 kDa (Acrp30).

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.

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 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

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.

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.

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 having a heavy atomic weight (2.014).

Dyspnea

Difficulty in breathing, often associated with lung or heart disease, and resulting in shortness of breath.

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.

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.

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Fetus
: The developing organism in the human uterus after the second month of gestation.

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 abnor

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 gonadotropin (HCG)
: A hormone that is secreted by the placenta during early pregnancy to maintain corpus luteum function and stimulate placental progesterone production; is commonly tested for as an indicator of pregnancy.

Hydrodensitometry
: Method of body composition measurement applying Archimedes' principle by submerging subject in water.

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).

Hypoxia
: Insufficient levels of oxygen in blood or tissue.

In utero
: In the uterus.

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.

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.

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.

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.

Placenta previa
: A complication of pregnancy in which the placenta grows in the lowest part of the womb (uterus) and covers all or part of the opening to the cervix.

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.

systematic inflammatory response of both normal and preeclamptic pregnancies.

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)

T2.4 The daily amount of energy used by an individual for maintenance, metabolic (energy

TABLE A-1 Description and Comparison of Public Health Surveys of Pregnant Women, Infants, and Children

Survey	Objectives	Population/Data Collection
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.
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. 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.

Data Source	Available Data	Strengths/Limitations

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- xcT-3a25 TlT-3(u-3(ds)-31ctualv 3(t)-3(p1iO /yN(h) e3(d)dT /LNET)-31t(n)-( )-

Data Source	Available Data	Strengths/Limitations

-3(a)-3(g)-3(t)-3(e)]TJEMC /Artifact <</O /Layout >>BDC pcoecy and ection

REFERENCE

Fein S. B., J. Labiner-Wolfe, K. R. Shealy, R. Li, J. Chen and L. M. Grummer-Strawn. 2008. Infant Feeding Practices Study II: study methods. *Pediatrics* 122(Suppl 2): S28-35.

Websites:
http://www.cdc.gov/ifps/
http://www.cdc.gov/pednss/index.htm
http://www.cdc.gov/prams/

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

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	—
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

continued

TABLE B-2 Continued

Nutrient	EAR[a]	RDA[b]/AI[c]	UL[d]
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			
14-18 y	—	—	1.0 mg/day
19-30 y	—	—	1.0 mg/day
31-50 y	—	—	1.0 mg/day

Nutrient	EAR[a]	RDA[b]/AI[c]	UL[d]
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
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 Allowad3(r)-3(t)/138 2 65.75 -1.906 Tm[(65.75 -1.906 T9t2/T1 1 Tf1 0 0 1 265.75 -1.906 T9t2AI) n rdinary t-type n Col umn 3.

[a]ET9t2AR = E265.7timated Average Requirement. An ET9t2AR is the average daily nutrient intake level e265.7timated 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 6 T9t29T9t27-T9t29T9t28 percent) healthy indivi in a group. It is calculated from an ET9t2AR.

[c]AI = dequate ntake. f ufficient cientific vidence s not vailable o e265.7tablish n R, and thus calculate and RDT9t2A, an AI s usually developed. The AI or rea265.7t-fed nfants s the mean intake. The AI for other life-265.7tage groups and gender groups 6 T9t2except healthy brea265.7t-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 o pecify with confidence the percentage

[f] The ULs for magnesium represent intake from pharmacological agents only and do not include intake from food and water.

[g]

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*	Limit to no more than 25% of total energy

*Not a recommended intake. A daily intake of added sugars that individuals should aim for

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 1977-1978 and 1994-1996.

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, sweet-

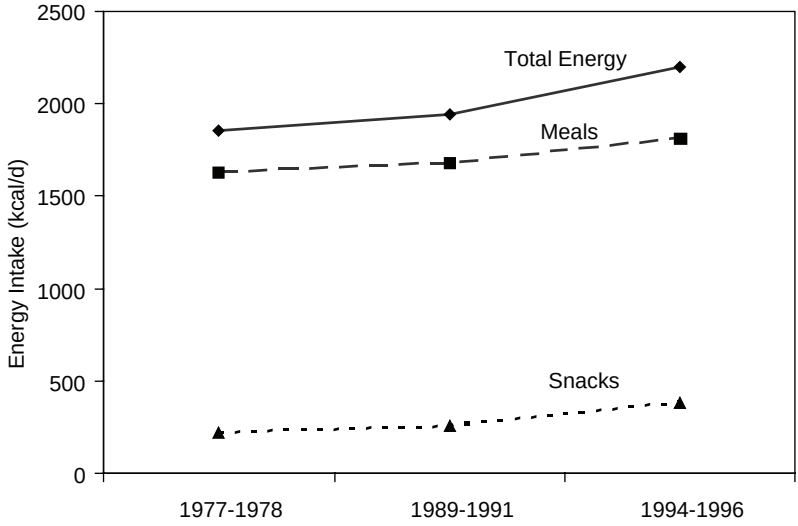

FIGURE B-1 Trends in energy intake and meal pattern type, U.S. adults aged 19-39 years.
SOURCE: Nielsen et al., 2002.

ened beverage intake increased and milk consumption decreased between survey years 1977-1978 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]),

ined 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.

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 (HHS/USDA, 2005). The HEI-2005 has 12 components, as seen in Table B-5 (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 *so*lid *f*ats, *a*lcoholic beverages, and *a*dded 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 1994-1996 to 2001-2002, 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

FIGURE B-3 Mean daily intakes of vegetables by subtype among U.S. females 19-30 years of age.
SOURCE: Guenther et al., 2006.

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 1994-1996 to 2001-2002 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.

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 1977-1978 to 65 percent from 1994-1996 (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.

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

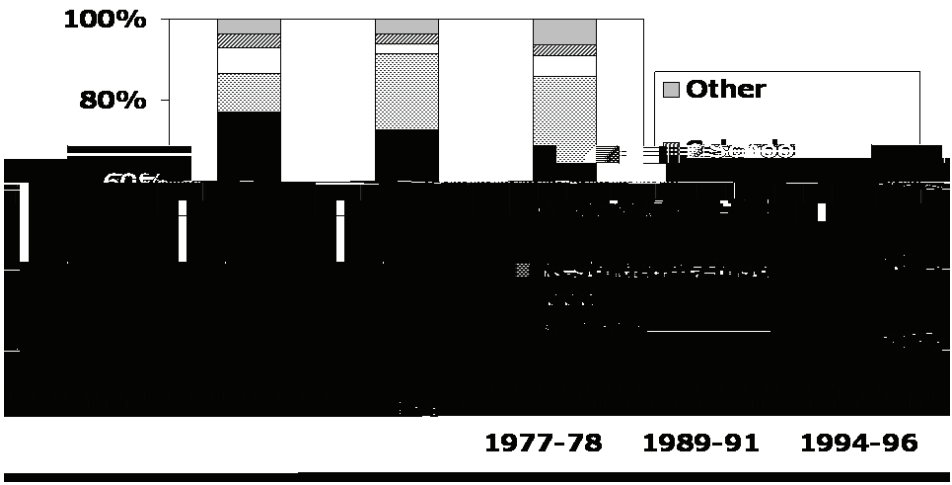

FIGURE B-4 Trends in energy intake (percent energy) by location, Americans aged 2+ years.
SOURCE: Nielsen et al., 2002.

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

REFERENCES

Guenther P. M., K. W. Dodd, J. Reedy and S. M. Krebs-Smith. 2006. Most Americans eat much less than recommended amounts of fruits and vegetables. *Journal of the American Dietetic Association* 106(9): 1371-1379.

HHS/USDA (U.S. Department of Health and Human Services/United States Department of Agriculture). 2005. *Dietary Guidelines for Americans, 2005. 6th Edition*. Washington, DC: Government Printing Office.

IOM (Institute of Medicine). 2006. *Dietary Reference Intakes: The Essential Guide to Nutrient Requirement*. Washington, DC: The National Academies Press.

Nielsen S. J. and B. M. Popkin. 2004. Changes in beverage intake between 1977 and 2001. *American Journal of Preventive Medicine* 27(3): 205-210.

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*.

TABLE C-1A Continued

Study Description	Population Characteristics	Study Criteria	Weight Gain by Trimester			Total Weight Gain
			1st	2nd	3rd	
Author, year: Catalano et al., 1993	*Total Study N:* 390	Women with GDM (n = 78)				12.6 kg
		Controls (n = 312)				15.0 kg
Country: USA		Underweight				
		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: Cedergren, 2006*	*Total Study N:* 245,526	BMI < 20				13.5 kg
		BMI 20-24.9				13.8 kg
		BMI 25-29.9				13.2 kg
		BMI 30-34.9				11.1 kg
Country: 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:
						7.5 kg
						9.8 kg
Country: Sweden						

Study Description	Population Characteristics	Study Criteria	Weight Gain by Trimester			Total Weight Gain
			1st	2nd	3rd	
Author, year: Johnston et al., 1992	*Total Study N:* 272	Adolescents (n = 123)				15.4 kg
		Adults (n = 149)				14.4 kg
	Group Description:					*(39.6 wks gestation)*
Country: USA	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
	Group Description:					
Country: Finland	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:	Time of delivery:				
	Mostly Canadian-born, Caucasians	≥ 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						

Study Description	Population Characteristics	Study Criteria	Weight Gain by Trimester
Weight Gain Descriptian			

Study Description	Population Characteristics	Study Criteria	Weight Gain by Trimester			Total Weight Gain
			1st	2nd	3rd	
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	
Country: USA	*Group Description:* 80% Hispanic	Term deliveries (n = 7,072)	1.2 kg	0.53 kg/wk	0.53 kg/wk	
Author, year: Stevens-Simon and McAnarney, 1992*	*Total Study N:* 141	Weight Gain:				
	Group Description: Teens, Black	Slow, < 0.28 kg/wk (n = 23)			0.16 kg/wk	6.50 kg
		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
Country: UK		Obese (n = 25)				9.7 kg

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: Cogswell et al., 1995	*Total Study N:* 53,541		*Gained < 6.8 kg:*
		BMI 19.8-26	6%
		BMI > 26-29	11%
		BMI > 29	25%
	Group Description: Low income women		*Gained 6.8-8.6 kg:*
		BMI 19.8-26	6%
		BMI > 26-29	8%
		BMI > 29	10%
	BMI 19.8-26 (n = 33,809)		*Gained 9.1-10.9 kg:*
		BMI 19.8-26	11%
	BMI > 26-29 (n = 7,661)	BMI > 26-29	13%
		BMI > 29	13%
	BMI > 29 (n = 12,071)		*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: Hickey et al., 1993*	*Total Study N:* 1,168		*Gained within IOM recommendations:*
		Black women, pregravid BMI:	
		Low, < 19.8 (n = 221)	37.1%
		Normal, 19.8-26.0 (n = 350)	30.9%
	Group Description: Low income, high risk women	High, > 26.0-29.0 (n = 84)	27.4%
		White women, pregravid BMI:	
	Black (n = 803)	Low, < 19.8 (n = 118)	37.3%
	White (n = 365)	Normal, 19.8-26.0 (n = 168)	35.7%
		High, > 26.0-29.0 (n = 29)	20.7%

TABLE C-1B Continued

Study Description	Population Characteristics	Weight/BMI Category	Weight Gain (percent of n)
Author, year: Kiel et al., 2007*	*Total Study N:* 120,170		*Gain of less than 0.9 kg:*
		BMI 30-34.9	3%
		BMI 35-39.9	8%
		BMI ≥ 40	15%
	Group Description:		*Gain of 0.9-6.4 kg:*
Country: USA	Obese women	BMI 30-34.9	15%
	BMI 30-34.9 (n = 70,536)	BMI 35-39.9	22%
	BMI 35-39.9 (n = 30,609)	BMI ≥ 40	25%
	BMI ≥ 40 (n = 19,025)		*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: Nohr et al., 2007*	*Total Study N:* 62,167		*Gain of 0.28 kg/wk:*
		BMI < 18.5	15.3%
		BMI 18.5-24.9	11.5%
		BMI 25-29.9	19.6%
		BMI 30+	36.1%
Country: Sweden			*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%
		BMI 25-29.9	18.3%
		BMI 30+	14.2%
Author, year: Schieve et al., 2000*	*Total Study N:* 3,511		*Gained < 0.23 kg/wk:*
		Low BMI	4%
		Average BMI	5%
		High BMI	23%
Country: 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%

continued

TABLE C-1B Continued

Study Description	Population Characteristics	Weight/BMI Category	Weight Gain (percent of n)
Author, year: Stotland et al., 2006* *Country:* USA	*Total Study N:* 15,101	Low and Normal BMIs	*Gain of < 0.27 kg/wk:* 11% *Gain of 0.27-0.52 kg/wk:* 68.2% *Gain of > 0.27 kg/w:* 21.1%
Author, year: Taffel et al., 1993 *Country:* USA	*Total Study N:* 1,707 *Group Description:* BMI < 19.8 (n = 379) BMI 19.8-26 (n = 1,024) BMI > 26 (n = 304)	BMI < 19.8 BMI 19.8-26 BMI > 26 BMI < 19.8 BMI 19.8-26 BMI > 26 BMI < 19.8 BMI 19.8-26 BMI > 26	*Actual Gain < 10 kg:* 13% 16% 38% *Actual Gain 10-12.3 kg:* 21% 19% 19% *Actual Gain > 12.3 kg:* 66% 64% 42%
Author, year: Wen et al., 1990* *Country:* USA	*Total Study N:* 17,149 *Group Description:* Black and White indigent women	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%

*Indicates that study is included in the systematic literature review conducted by Viswanathan et al., 2008.

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				
Country: USA	*Group Description:* Twins (n = 1,984) Singletons (n = 201,784)	Underweight				17.9 kg
		Normal weight				16.9 kg
		Overweight				17.0 kg
		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:				
Country: Canada	*Group Description:* Normal weight women (BMI = 19-28), aged 20-35	> 3 kg			15.50 kg	
		Intermediate			13.37 kg	
		SGA			14.66 kg	

continued

Author (Year)	Population Characteristics	Study Criteria	Weight Gain by Trimester			Total Weight Gain
			1st	2nd	3rd	
Triplets						
Author, year:	*Total Study N:*	Prepregnancy BMI:				
Luke (1995)	23 (8)	Underweight (<19.8)	2.2 (1.8)	0.5 (1.4)	0.5 (1.4)	3.2 (2.5)
		Normal (19.8-26.0)	1.1 (1.4)	0.2 (1.4)	0.2 (1.4)	1.5 (2.0)
Country:	*Group Description:*	Overweight (26.1-29.0)	0.5 (1.4)	0.2 (1.4)	0.2 (1.4)	0.9 (2.0)
USA	Triplet gestations	Obese (>29.0)	0.5 (1.4)	0.2 (1.4)	0.2 (1.4)	0.9 (2.0)

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 >

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

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 -3(3(n)-3(a)-a)-3ou-3(i)-3(m)-3(r)-3(i)-3(a)-506mmmwwM06mmmm-3(i)-,3()- wA-3(-3(i)-n3()-)3(wA-3(-3(a)

TABLE C-4 Maternal Weight Gain and Body Composition

Author (Year)	Population/Study Characteristics	Study Criteria	Body Composition Measurements (FFM, FM, TBW)
Author, year: Bronstein et al., 1996 *Country:* USA	*Total Study N:* 33 *Group Description:* Non-pregnant and pregnant women [BC by densitometry}	Pregnant women (n = 16) Non-pregnant women (n = 17)	FFM at 31-35 weeks: 55.5 kg FM at 31-35 weeks: 32.8 kg

Pregravid Weight/ Body Comp	Weight Gain/Body Composition by Trimester			Total Weight Gain	Postpartum Weight/Body Composition
	1st	2nd	3rd		

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Author (Year)	Population/Study Characteristics	Study Criteria	Body Composition Measurements (FFM, FM, TBW)
Author, year: Catalano et al., 1998 *Country:* USA	*Total Study N:* 16 *Group Description:* Women with normal and abnormal glucose tolerance (Ab GT); Ab GT (n = 6), Controls (n = 10) [BC by densitometry]	Ab GT (n = 6) Controls (n = 10)	Pregravid measurements *FFM:* 46.4 kg 46.3 kg *FM:* 12.8 kg 10.2 kg *Sum of 7 site skinfolds:* 88.7 mm 74.0 mm

Pregravid Weight/ Body Comp	Weight Gain/Body Composition by Trimester			Total Weight Gain	Postpartum Weight/Body Composition
	1st	2nd	3rd		
	12-14 wks		34-36 wks		
	Weight:		*Weight:*		
59.2 kg	60.5 kg		71.6 kg		
56.5 kg	59.9 kg		70.0 kg		
	FFM:		*FFM:*		
	46.4 kg		53.4 kg		
	46.8 kg		53.6 kg		
	FM:		*FM:*		
	14.1 kg		18.2 kg		
	13.2 kg		16.5 kg		
	Sum of 7 site skinfolds:		*Sum of 7 site skinfold:*		
	93.5 mm		108.9 mm		
	89.0 mm		109.8mm		
					FM gain
56.2 kg				12.3 kg	4.7 kg
67.6 kg				13.0 kg	4.2 kg
69.7 kg				12.0 kg	3.2 kg
66.3 kg				13.7 kg	4.8 kg
			32 wks:		*2 wks pp:*
0 ko fat					

Author (Year)	Population/Study Characteristics	Study Criteria	Body Composition Measurements (FFM, FM, TBW)
Author, year: Kopp-Hoolihan et al., 1999 *Country:* USA	*Total Study N:* 9 *Group Description:* Healthy, non-smokers planning a pregnancy		

Pregravid Weight/ Body Comp	Weight Gain/Body Composition by Trimester			Total Weight Gain	Postpartum Weight/Body Composition
	1st	2nd	3rd		
	8-10 wks	*24-26 wks*	*34-36 wks*		4-6 wks
64.7 kg	64.9 kg	72.1 kg	75.9 kg		68.0 kg
33.5 kg	33.9 kg	36.5 kg	39.1 kg		33.8 kg
0.72	0.73	0.74	0.74		0.72
2525 g	—	—	—		2463 g
46.3 kg	46.7 kg	49.7 kg	52.8 kg		46.7 kg
20.2 kg	20.3 kg	24.4 kg	24.3 kg		22.0 kg
—	0.19 kg	7.23 kg	3.76 kg	11.2 kg	—
—	0.10 kg	4.10 kg	-0.10 kg	4.20 kg	—
—	53.00	57.00	-3.00	—	—

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Author (Year)	Population/Study Characteristics	Study Criteria	Body Composition Measurements (FFM, FM, TBW)
Author, year:			

Pregravid Weight/ Body Comp	Weight Gain/Body Composition by Trimester			Total Weight Gain	Postpartum Weight/Body Composition
	1st	2nd	3rd		
	1212				

TABLE C-5 Mean Weights and Percentiles for Placentas (singletons, twins, and triplets)

Gestational age (weeks)	90th Percentile			Mean Placental Weight			10th Percentile			Cases (*n*)		
	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										

TABLE C-6 DNA, Glycogen, and Lipid Content in Placentas from Normal and Diabetic Human Pregnancies

REFERENCES

Abrams B. and S. Selvin. 1995. Maternal weight gain pattern and birth weight. *Obstetrics and Gynecology* 86(2): 163-169.

Abrams B., V. Newman, T. Key and J. Parker. 1989. Maternal weight gain and preterm delivery. *Obstetrics and Gynecology* 74(4): 577-583.

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Luke B., J. Minogue and H. Abbey. 1992. The association between maternal weight gain and the birth weight of twins. *The Journal of Maternal-Fetal Medicine* 1: 267-276.

Luke B., E. Bryan, C. Sweetland, S. Leurgans and L. Keith. 1995. Prenatal weight gain and the birthweight of triplets. *Acta Geneticae Medicae et Gemellologiae* 44(2): 93-101.

Luke B., M. L. Hediger, C. Nugent, R. B. Newman, J. G. Mauldin, F. R. Witter and M. J. O'Sullivan. 2003. Body mass index—specific weight gains associated with optimal birth weights in twin pregnancies. *Journal of Reproductive Medicine* 48(4): 217-224.

Muscati S. K., K. Gray-Donald and K. G. Koski. 1996. Timing of weight gain during pregnancy: promoting fetal growth and minimizing maternal weight retention. *International Journal of Obesity and Related Metabolic Disorders* 20(6): 526-532.

Nohr E. A., M. Vaeth, B. H. Bech, T. B. Hr . GM...Hr tiong fei3(n)-3(a)s-3(r)-3( )-3( )-31(R)-3(e)-3( )J65(H

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*.

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 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.	The impact of the IOM guidelines on actual gestational weight gain may be limited in	

Determinants	Findings/Interpretations	Comments	References
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, b(.)-3(,)-7(w)-3deruc93T-3(t)-3(,)-3(3( )-31(l3(n)-3(d)-t8)-31(w)-3(o)-3(	

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 explored the association of food insecurity with adherence to		

Medical Factors: Anorexia Nervosa and Bulimia	Sollid et al. (2004) found increased preterm delivery & SGA among women with eating disorders but did not obtain information on gestational weight gain. Kouba et al. (2005) 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 et al., 2009
Psychological Factors: Stress	The committee found no robust association between stress and gestational weight gain.		Picone et al., 1982 Orr et al., 1996 Rondo et al., 2003 Brawarksy et al., 2005 Dominguez et al., 2005
Psychological Factors:			

TABLE D-1 Continued

Determinants	Findings/Interpretations	Comments	References
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

REFERENCES

Abraham S., A. Taylor and J. Conti. 2001. Postnatal depression, eating, exercise, and vomiting before and during pregnancy. *International Journal of Eating Disorders* 29(4): 482-487.

Abrams B., S. L. Altman and K. E. Pickett. 2000. Pregnancy weight gain: still controversial. *American Journal of Clinical Nutrition* 71(5 Suppl): 1233S-1241S.

ACOG (American College of Obstetricians and Gynecologists). 2005. Committee Opinion number 315, September 2005. Obesity in pregnancy. *Obstetrics and Gynecology* 106(3): 671-675.

Adams E. J., L. Grummer-Strawn and G. Chavez. 2003. Food insecurity is associated with increased risk of obesity in California women. *Journal of Nutrition* 133(4): 1070-1074.

Adriaanse H. P., J. A. Knottnerus, L. R. Delgado, H. H. Cox and G. G. Essed. 1996. Smoking in Dutch pregnant women and birth weight. *Patient Education and Counseling* 28(1): 25-30.

Alevizaki M., L. Thalassinou, S. I. Grigorakis, G. Philippou, K. Lili, A. Souvatzoglou and E. Anastasiou. 2000. Study of the Trp64Arg polymorphism of the beta3-adrenergic receptor in Greek women with gestational diabetes. *Diabetes Care* 23(8): 1079-1083.

Anderson S. A. 1990. Core Indicators of Nutritional State for Difficult-to-Sample Populations. *Journal of Nutrition* 120(11 Suppl): 1555-1600.

Artal R., R. B. Catanzaro, J. A. Gavard, D. J. Mostello and J. C. Friganza. 2007. A lifestyle intervention of weight-gain restriction: diet and exercise in obese women with gestational diabetes mellitus. *Applied Physiology, Nutrition, and Metabolism*

Span <</MCID 22876 >>BDC -3(e)-3(t)-3b03(0)-3-108EMC EMC CID 2-MC CID 2

Butte N. F., W. W. Wong, M. S. Treuth, K. J. Ellis and E. O'Brian Smith. 2004. Energy requirements during pregnancy based on total energy expenditure and energy deposition. *American Journal of Clinical Nutrition* 79(6): 1078-1087.

Cabral H., L. E. Fried, S. Levenson, H. Amaro and B. Zuckerman. 1990. Foreign-born and US-born black women: differences in health behaviors and birth outcomes. *American Journal of Public Health* 80(1): 70-72.

Callister L. C. and A. Birkhead. 2002. Acculturation and perinatal outcomes in Mexican immigrant childbearing women: an integrative review. *Journal of Perinatal and Neonatal Nursing* 16(3): 22-38.

Cameron R. P., C. M. Grabill, S. E. Hobfoll, J. H. Crowther, C. Ritter and J. Lavin. 1996. Weight, self-esteem, ethnicity, and depressive symptomatology during pregnancy among inner-city women. *Health Psychology* 15(4): 293-297.

Campbell D. 1983. Dietary restriction in obesity and its effect on neonatal outcome. In *Nutrition in pregnancy. Proceedings of 10th Study Group of the RCOG*. Campbell DM and G. MDG. London, RCOG; pp. 85-98.

Campbell D. M. and I. MacGillivray. 1975. The effect of a low calorie diet or a thiazide diuretic on the incidence of pre-eclampsia and on birth weight. *British Journal of Obstetnes.*

Fallucca F., M. G. Dalfra, E. Sciullo, M. Masin, A. M. Buongiorno, A. Napoli, D. Fedele and A. Lapolla. 2006. Polymorphisms of insulin receptor substrate 1 and beta3-adrenergic receptor genes in gestational diabetes and normal pregnancy. *Metabolism* 55(11): 1451-1456.

Festa A., W. Krugluger, N. Shnawa, P. Hopmeier, S. M. Haffner and G. Schernthaner. 1999. Trp64Arg polymorphism of the beta3-adrenergic receptor gene in pregnancy: association with mild gestational diabetes mellitus. *Journal of Clinical Endocrinology and Metabolism* 84(5): 1695-1699.

Fonager K., H. T. Sorensen, J. Olsen, J. F. Dahlerup and S. N. Rasmussen. 1998. Pregnancy outcome for women with Crohn's disease: a follow-up study based on linkage between national registries. *American Journal of Gastroenterology* 93(12): 2426-2430.

Forsum E., A. Sadurskis and J. Wager. 1985. Energy maintenance cost during pregnancy in healthy Swedish women. *Lancet* 1(8420): 107-108.

Fraga M. F., E. Ballestar, M. F. Paz, S. Ropero, F. Setien, M. L. Ballestar, D. Heine-Suner, J. C. Cigudosa, M. Urioste, J. Benitez, M. Boix-Chornet, A. Sanchez-Aguilera, C. Ling, E. Carlsson, P. Poulsen, A. Vaag, Z. Stephan, T. D. Spector, Y. Z. Wu, C. Plass and M. Esteller. 2005. Epigenetic differences arise during the lifetime of monozygotic twins. *Proceedings of the National Academy of Sciences of the United States of America* 102(30): 10604-10609.

Fretts R. C. 2005. Etiology and prevention of stillbirth. *American Journal of Obstetrics and Gynecology* 193(6): 1923-1935.

Frongillo E. A., Jr., B. S. Rauschenbach, C. M. Olson, A. Kendall and A. G. Colmenares. 1997. Questionnaire-based measures are valid for the identification of rural households with hunger and food insecurity. *Journal of Nutrition* 127(5): 699-705.

Fuentes-Afflick E. and P. Lurie. 1997. Low birth weight and Latino ethnicity. Examining the epidemiologic paradox. *Archives of Pediatrics and Adolescent Medicine* 151(7): 665-674.

Fuentes-Afflick E. and N. A. Hessol. 2008. Acculturation and Body Mass among Latina Women. *Journal of Women's Health (Larchmt)* 17(1): 67-73.

Furneaux E. C., A. J. Langley-Evans and S. C. Langley-Evans. 2001. Nausea and vomiting of pregnancy: endocrine basis and contribution to pregnancy outcome. *Obstetrical and Gynecological Survey* 56(12): 775-782.

Furuno J. P., L. Gallicchio and M. Sexton. 2004. Cigarette smoking and low maternal weight gain in Medicaid-eligible pregnant women. *Journal of Women's Health (Larchmt)* 13(7): 770-777.

Garn S. M., K. Hoff and K. D. McCabe. 1979. Is there nutritional mediation of the "smoking effect" on the fetus. *American Journal of Clinical Nutrition* 32(6): 1181-1184.

Glynn L. M., P. D. Wadhwa, C. Dunkel-Schetter, A. Chicz-Demet and C. A. Sandman. 2001. When stress happens matters: effects of earthquake timing on stress responsivity in pregnancy. *American Journal of Obstetrics and Gynecology* 184(4): 637-642.

Goldberg G. R., A. M. Prentice, W. A. Coward, H. L. Davies, P. R. Murgatroyd, C. Wensing, A. E. Black, M. Harding and M. Sawyer. 1993. Longitudinal assessment of energy expenditure in pregnancy by the doubly labeled water method. *American Journal of Clinical Nutrition* 57(4): 494-505.

Goodwin T. M. 2002. Nausea and vomiting of pregnancy: an obstetric syndrome. *American Journal of Obstetrics and Gynecology* 186(5 Suppl Understanding): S184-S189.

Goodwin l 1993. t-3(.)-3(y)-3(n)-3(9)-3(3)-3(a)-3( )-47(a)-39.065 -993H993.ast36.315 0 a 993108(.)-3( )-59

Gortmaker S. L., K. Peterson, J. Wiecha, A. M. Sobol, S. Dixit, M. K. Fox and N. Laird. 1999. Reducing obesity via a school-based interdisciplinary intervention among youth: Planet Health. *Archives of Pediatrics and Adolescent Medicine* 153(4): 409-418.

Graham K., A. Feigenbaum, A. Pastuszak, I. Nulman, R. Weksberg, T. Einarson, S. Goldberg, S. Ashby and G. Koren. 1992. Pregnancy outcome and infant development following gestational cocaine use by social cocaine users in Toronto, Canada. *Clinical and Investigative Medicine. Medecine Clinique et Experimentale* 15(4): 384-394.

Gross S., C. Librach and A. Cecutti. 1989. Maternal weight loss associated with hyperemesis gravidarum: a predictor of fetal outcome. *American Journal of Obstetrics and Gynecology* 160(4): 906-909.

Gross T., R. J. Sokol and K. C. King. 1980. Obesity in pregnancy: risks and outcome. *Obstetrics and Gynecology* 56(4): 446-450.

Groth S. 2007. Are the Institute of Medicine recommendations for gestational weight gain appropriate for adolescents? *Journal of Obstetric, Gynecologic, and Neonatal Nursing* 36(1): 21-27.

Gurewitsch E. D., M. Smith-Levitin and J. Mack. 1996. Pregnancy following gastric bypass surgery for morbid obesity. *Obstetrics and Gynecology* 88(4 Pt 2): 658-661.

Gutierrez Y. M. 1999. Cultural factors affecting diet and pregnancy outcome of Mexican American adolescents. *Journal of Adolescent Health* 25(3): 227-237.

Haakstad L. A., N. Voldner, T. Henriksen and K. Bo. 2007. Physical activity level and weight gain in a cohort of pregnant Norwegian women. *Acta Obstetricia et Gynecologica Scandinavica* 86(5): 559-564.

Haas D. M. and L. A. Pazdernik. 2006. A cross-sectional survey of stressors for postpartum women during wartime in a military medical facility. *Military Medicine* 171(10): 1020-1023.

Hanson J. W., A. P. Streissguth and D. W. Smith. 1978. The effects of moderate alcohol consumption during pregnancy on fetal growth and morphogenesis. *Journal of Pediatrics*

Lane S. D., R. H. Keefe, R. Rubinstein, B. A. Levandowski, N. Webster, D. A. Cibula, A. K. Boahene, O. Dele-Michael, D. Carter, T. Jones, M. Wojtowycz and J. Brill. 2008. Structural violence, urban retail food markets, and low birth weight. *Health & Place* 14(3): 415-423.

Laraia B. A., A. M. Siega-Riz, J. S. Kaufman and S. J. Jones. 2004. Proximity of supermarkets is positively associated with diet quality index for pregnancy. *Preventive Medicine* 39(5): 869-875.

Laraia B., L. Messer, K. Evenson and J. S. Kaufman. 2007. Neighborhood factors associated with physical activity and adequacy of weight gain during pregnancy. *Journal of Urban Health* 84(6): 793-806.

Lawrence M., J. Singh, F. Lawrence and R. G. Whitehead. 1985. The energy cost of common daily activities in African women: increased expenditure in pregnancy? *American Journal of Clinical Nutrition* 42(5): 753-763.

Lederman S. A., V. Rauh, L. Weiss, J. L. Stein, L. A. Hoepner, M. Becker and F. P. Perera. 2004. The effects of the World Trade Center event on birth outcomes among term deliveries at three lower Manhattan hospitals. *Environmental Health 3(r)-3(e)-3(r)-eEn*

Polivy J. 1996. Psychological consequences of food restriction. *Journal of the American Dietetic Association* 96(6): 589-592; quiz 593-594.

Power M. L., M. E. Cogswell and J. Schulkin. 2006. Obesity prevention and treatment practices of U.S. obstetrician-gynecologists. *Obstetrics and Gynecology* 108(4): 961-968.

Siega-Riz A. M., K. R. Evenson and N. Dole. 2004. Pregnancy-related weight gain—a link to obesity? *Nutrition Reviews* 62(7 Pt 2): S105-S111.

Singh G. K. and S. M. Yu. 1996. Adverse pregnancy outcomes: differences between US- and foreign-born women in major US racial and ethnic groups. *American Journal of Public Health* 86(6): 837-843.

Skull A. J., G. H. Slater, J. E. Duncombe and G. A. Fielding. 2004. Laparoscopic adjustable banding in pregnancy: safety, patient tolerance and effect on obesity-related pregnancy outcomes. *Obesity Surgery* 14(2): 230-235.

Smith L. M., L. L. LaGasse, C. Derauf, P. Grant, R. Shah, A. Arria, M. Huestis, W. Haning, A. Strauss, S. Della Grotta, J. Liu and B. M. Lester. 2006. The infant development, environment, and lifestyle study: effects of prenatal methamphetamine exposure, polydrug exposure, and poverty on intrauterine growth. *Pediatrics* 118(3): 1149-1156.

Sollid C. P., K. Wisborg, J. Hjort and N. J. Secher. 2004. Eating disorder that was diagnosed before pregnancy and pregnancy outcome. *American Journal of Obstetrics and Gynecology* 190(1): 206-210.

Sternfeld B., C. P. Quesenberry, Jr., B. Eskenazi and L. A. Newman. 1995. Exercise during pregnancy and pregnancy outcome. *Medicine and Science in Sports and Exercise* 27(5): 634-640.

Stevenson L. 1997. Exercise in pregnancy. Part 1: Update on pathophysiology. *Canadian Family Physician* 43: 97-104.

Stevens-Simon C. and E. R. McAnarney. 1992. Determinants of weight gain in pregnant adolescents. *Journal of the American Dietetic Association* 92(11): 1348-1351.

Stevens-Simon C., E. R. McAnarney and K. J. Roghmo35n andhm5n anlescenlciatir(s)-3(.)-3( )]TJEMC /S
gnanNe5 5Je5Se5Hegc5P5B

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Wagner C. L., L. D. Katikaneni, T. H. Cox and R. M. Ryan. 1998. The impact of prenatal drug exposure on the neonate. *Obstetrics and Gynecology Clinics of North America* 25(1): 169-194.

Walker L. O. and M. Kim. 2002. Psychosocial thriving during late pregnancy: relationship to ethnicity, gestational weight gain, and birth weight. *Journal of Obstetric, Gynecologic, and Neonatal Nursing* 31(3): 263-274.

Weissman A., E. Siegler, R. Neiger, P. Jakobi and E. Z. Zimmer. 1989. The influence of increased seismic activity on pregnancy outcome. *European Journal of Obstetrics, Gynecology, and Reproductive Biology* 31(3): 233-236.

Wells C. S., R. Schwalberg, G. Noonan and V. Gabor. 2006. Factors influencing inadequate

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) guide-

* Appendixes and evidence tables cited in this report are provided electronically at http://www.ahrq.gov/downloads/pub/evidence/pdf/admaternal/admaternalapp.pdf.

lines 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 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

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 aa c t23(e0 >>BD

APPPP

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.

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 (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,

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	
• Poor on source of information for exposure	
•	

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. sal. 3(i)-3(e(n)-3(d)-c)-3(u)-3(s)-3(t)-3(o)-3(

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

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 ar-

marks ($P < .05$) 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

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
Murakami et al., 2005[52]	Pregravid weight:	G1: < 8.5 kg gain	AOR (95% CI) gestational diabetes	Maternal age, parity, smoking, weight gain, gestational weeks; pregravid BMI
Japan hospital data	Self-report on first visit	G2: 8.5-12.5 kg gain	G1: 5.14 (0.97-27.20)	
633	Total weight gain: Measured on admission for birth	G3: > 12.5 kg gain	G2: Reference	
All BMIs			G3: 3.91 (0.61-24.73)	
Fair				
Thorsdottir, et al., 2002[53]	Pregravid weight:	G1: < 11.5 kg gain	Incidence gestational diabetes	Age, parity, height, gestational age
Iceland University Hospital	Self-report	G2: 11.5-16.0 kg gain	G1: 2.9%	
615	Total weight gain: Maternity records (no specifics offered)	G3: 16.1-20.0 kg gain	G2: 0	
BMI: 19.5-25.5		G4: > 20 kg gain	G3: 0	
Fair			G4: 0	
			(P = .015)	
Bianco, et al., 1998[54]				
US New York				
Medical Center Database				
11,840				
Nonobese (BMI 19-27) and Morbidly obese (BMI > 35)				

Self8103(d)-3(l)-3(y)-3( )-3(r)-3(t)]TJEMC /Span <</MCID 24776 >>BDC 0 -1.875 TD[(T)95(o)-3(t)-3(a)-381al Cght gain:
Mate8123(d)-3(l)-3(y)-3( )-3C >>BDC 0m24776 >>Bu(b)-3(a)-3(s)-TJEMC /SpfC 0 -1.8DC 0m/Span <<( )-30(Y)1 oe as1un <</MCID 2477TJ

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,[32] 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 3(a)-3(l)-3(137)-3(c)--3(137)-3

range of weight gain included in the reference category 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 BMIbtTJEMC /Span <<73.3981 531>>BDC 7 0 0 7 298.4736 543.61 Tm[543.61 Tm7

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

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight
	SEf3-5(I)-6Rc
	BItc >>(e)-3(u)-3(d3-5(I)-6R)-31(W)71(n)-3(e)-3((W)71(A)-3(e)-3((M)-ti)-3(y
	BFBMetrPregravid Wwgght
	Cetlf3-5(-)-3(e)-3(g)-3(p)-3(r)108r CC(e)-3(d)-35531(M)-titywgght
	W2t /5P2[(W)-(2)C 19.07t1 EM(0303()].3(-]0f-l)23(-]6f-l)-3(t(B)-J(MMC /Sp

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)

Results	Confounders and Effect Modifiers Included in Analysis
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
Incidence preeclampsia G1: 1.9% G2: 3.2% G3: 1.6% (*P* = .203) G4: 2.8% G5: 3.7% G6: 3.7% (*P* = .002) No clear trends for preeclampsia or severe preeclampsia by pregravid weight status and kg/week weight gains. AOR generally crossed 1.0 or had wide confidence intervals.	Pregravid weight

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]

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,(v)-3tn womed fr09

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 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

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

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 gesta

One 13(s)-3(t)-3(r)-3(a)-3(t)-3(i)-3(fi)-3(e)-3(d)-3(13(i)-3(t)-3se)-3(13(s)-3(a)-3(m)-
weight 253(a)23(n)23(d)23(253(w)23(e)23(i)23(g)23(h)23(t)23(253(g)23ae)23(i)23

sociation 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 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]

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

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 agcity,ypesex, mateb(gc)-3(i)6325 >>BDCE

TABLE 7. Continued

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])	Labor pattern—I stage (minutes ± SD) G1: 333 ± 208 G2: 374 ± 208 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	NA

BMI, body mass index; G, group; SD, standard deviation.

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 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

TABLE 8.

Results		Confounders and Effect Modifiers Included in Analysis
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) 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 < 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)	Maternal age, parity, smoking in early pregnancy, and year of birth
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 > 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)	
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 birthweightghtghtgeract <</

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 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

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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
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

Results	Confounders and Effect Modifiers Included in Analysis
G1: Proportional Gain: Adjusted Relative Risk (95% CI) G2: Absolute Gain: Adjusted RR (95% CI) U-3(e)-3(i)23(d)-3(j)-3im32-3( )-31(AC /Artifact <</O /LayoutEMC /Arti <</O /La3(n)-3(a)-3(%)-3( )-</O 01 in	

Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	G1:wiG1:G1:10 GeeG1:11G1:1sG1:1sG1:14 Definition of Groups
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: 1(e)]TJEMC3.5 12.5 Td[(G)-3(1)-3(:)4 G3: 1(e)]TJEMC3.5 12.5 Td[(G)-3(1)-3(:)5

Results	Confounders and Effect Modifiers Included in Analysis
Cesarean % G1: 25.8% G2: 26.8% G3: 28.8% G4: 35.0% G5: 33.8% (P = NS)	NA

Results	Confounders and Effect Modifiers Included in Analysis
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 cli-3( )-31(a)-3(n)-3(d)2g,

TABLE 8. Continued

Author, Date Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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
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

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.

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 cesar-

Results	Confounders and Effect Modifiers Included in Analysis
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Modi.6o5.6o/Ar9.1339 508.414( )- 68BDC BT	
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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]

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 17826 >>BDC -11.21 -1.2 Td[(

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

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).

O

Birth Outcomes

Preterm birth

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] d d d studies 31-3(x)-3(a)-3(

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
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
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 trimes Tf0.551 0(e)-3(s)-sl.,

Results	Confounders and Effect Modifiers Included in Analysis
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
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
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 spontaneous preterm birth/kg increase in total weight gain: 0.84 (0.82-0.87)	BMI, maternal age, infant sex cigarettes per day maternal height, parity, race, pattern of gain derived from quadratic curves
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

continued

TABLE 10. Continued

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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 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
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

Results	Confounders and Effect Modifiers Included in Analysis

Author, Year
Country, Setting
Sample Size
Baseline BMI

Results	Confounders and Effect Modifiers Included in Analysis
Reference category of rate of weight gain: 0.35-< 0.46 kg/wk	None
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 among women of low BMI	
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
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 preterm89(,)-3( )-31(i)-3(n)- age 8.75 Td[(P)-3(r)-3(e)-3(g)-3(r)-3(a)-3(

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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 ≤

Results	Confounders and Effect Modifiers Included in Analysis
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) *P* = 0.21 for interaction between G3 and G4	Pregravid BMI, pregravid weight, height, age, parity, smoking, social class education, infant sex

Regression analysis with gestational age (weeks) as the
dependent variable and net gestational weight onalt usginuaB3(g)-3(e)-=gh3(6)-3(5)- >>BDC T*d[( )-31(=i)-3
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est 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 (" f6a1s ofth" studied

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

teen 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,
3(5)-3(5)-3(,)-3(5)-3(9)-3(,)-3(7)-42-3(-)-3(9)-3(3)-3(,)-3(9)-3(3)93(,)-3(9)-3(7)-3(-)9,-3(7)-3(-)43(1)-3(0)-3(3)-3(,)-3(1)-3(0)-d

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: <		

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
Ekblad and Grenman, 1992[68] Finland, hospital 357 Prepregnancy weight 20% over or under ideal body 0 -1.25 TD[(o)TDI[(i)-3(g)-3(h)-3474 >>cy weigca9T*[(0 -1.25 mu)-3(n)-3(d)-3(e)-3()]TJEMC /Span <</MCID 2943(e)-3(i)-3(g)-4/Span <</M93(t)-3(a)-3(l)]TJEMC				

born to women who gained < 35 lbs. One study among morbidly obese women (BMI > 35) found a similar trend, although it was inconsistent at

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
Aras et al., 1				
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 ale statinal 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
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		

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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 Modifi7JEMC /S(d[(D)-3(e) yutine retay-3(t)-3(a)-3(n)]TJEMC eysG suu-3rneaseain G
				ti ti

TABLE 12. Continued

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
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] Italy, diabetes clinic 180 All weights/BMI Poor	Pregravid weight: Not reported Total weight gain: Collected by study investigators		F statistic = 3.16, $P = 0.08$	Pregravid BMI, maternal triglycerides, plasma glucose
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

APPENDIX E

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.,				

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]

TABLE 14. Net and Proportional Gestational Weight Gain and Infant Birthweight

Author, Year												

APPENDIX E

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
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

Results	Confounders and Effect Modifiers Included in Analysis
G1: 38.2 G2: 61.8 G3: 22.1 G4: 77.9	N/A
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
G1: 1.15 (0.78-1.67)	Gestational age, adolescence, pregravid underweight, number of Healthiest Baby Possible visits
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

continued

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
Kirchengast and		

TABLE 15. Continued

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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
Lasker et al., 2005[109] USA, hospital 5,528 All weights/BMI Poor	Pregravid weight: Not stated Total weight gain: Routine prenatal care or maternity records	OR and 95% CI, for LBW G1: GWG < 10 lbs G2: GWG > 30 lbs G3: GWG 21-30 lbs (Reference)

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.

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]

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

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
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 LBW G1: Net WG < G : Net WG 0rr. . l : Net WG 0r l : Net WG G1: . (Referene) : . (. .) : . (. .) : . (. .)Maternal ra pregravid weight, n oacoalcoorg ue, pre inant eepar, 19

Results	Confounders and Effect Modifiers Included in Analysis
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
G1: 3.5% G2: 7.4% G3: 2.1% G4: 2.8% G5: 2.1% G6: 4.6%	N/A
G1: 5% G2: 32% G3: 3.1% G4: 2.7%	N/A

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]

TABLE 17. Gestational Weight Gain and Macrosomia > 4,500 g

Author, Year	Pregravid Weight (How Measured)		
Country, Setting			
Sample Size	Total Weight Gain (How Measured)		
Baseline BMI			
Quality		Definition of Groups	

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight

Zhou and Olsen, 1997[108]	Denmark, two communities	7,122

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 U.S. study reported significant associations between weight gain and macrosomia only Womeud.SCID 3fmenonnd me nde ep3(d)omntsly-3( )-161((m)-3(e)-3(-3(,)-3( )-1

TABLE 18. Gestational Weight Gain and Macrosomia > 4,000g

Author, Year		
Country, Setting	Pregravid Weight	
Sample Size		
Baseline BMI		
Quality		
	(s-(u)-3(d)-3G(-)-2(l)-:(-)-3(g)-3(h-)-G(-)-3(0)-31(t)- ET-(B)-1(l)-6(M)-3(r)0-k CID 31842>>BDC T*[(()-66l[(C)-3(o)-3(u)-3(n)-3G(-)-3(	
	(alirBeB Gra	

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 ge3( )24(f)-EMC /Spa p

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 3(d)-3(d)-3(s)-3( )-175(o3(a)-5(i)-e)-3( )-107(3(d)-3(d)-175(c

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
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

Results	Confounders and Effect Modifiers Included in Analysis

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups	Relative0ris1s ly,tm9(,)t >>BDC 9. records
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 pan <</MCps	Total weight gain: Routine prenatal care or maternity records

<

Results	Confounders and Effect Modifiers Included in Analysis
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
G1: 12.0 G2: 11.8 G3: 18.8 G4: 25.8 G5: 23.8 ($P < 0.01$)	N/A

TD[(T)95(o)-3(t)-3(a-31(f)-3(o)-3(r)-3( )-31(G)-3(W)-3(G)-3(:)]TJEMC /Act <</O /Layout >>BDC -3.147 -1.875 Td[(P)-3(o)-

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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
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

Results	Confounders and Effect Modifiers Included in Analysis
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
G1: $c^2 = 8.2$ ($P < 0.005$)	N/A

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 qual

TABLE 20. Gestational Weight Gain and LGA by BMI Status

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
Caulfield et al., 1998[116]	Pregravid weight: Self-report	ORs and 95% CIs for LGA per 50g/wk increase in rate of weight gain by BMI
USA, hospital obstetric database 3,870 All weights/BMI Good	Total weight gain: Routine prenatal care or maternity records	G1: Underweight G2: Normal weight G3: Overweight
Cedergren, 2006[58]	Pregravid weight: Self-report	ORs and 95% CIs for LGA (> 2 SD above the mean)
Sweden, Medical Birth Registry 245,526 All weights/BMI Fair	Total weight gain: Routine prenatal care or maternity records	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)
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 > 25 lbs G2: OR of LGA for weight gain < 15 lbs G3: Reference weight gain 15-25 lbs

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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 LGA 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)

BMI, body mass index; CI, confidence interval; g, grams; g/wk, gram per week; kg/wk, kilo-

TABLE 21. Gestational Weight Gain and SGA

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight

Results	Confounders and Effect Modifiers Included in Analysis
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
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
G1: 3.0 (2.5-3.5) G2: 1.9 (1.MC /Arti(I)f3(o)-3(f)-3(5-3(1)-3(.c)-3( )-31(.)-3(5)-625 Td[(G)-3(1)-3(:)-3( )-31(3)-3(.)-3(0)-3( )-30 care or.f 167,750 All weights/BMI Fair	PregravidMfact <</Oa3(n)su/Layout >>BDC JEMC /Art(f)-3( )-3ayout >037/ Self-report Total weight gain: Routine prenatal care or maternity

TABLE 21. Continued

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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 > 25 lbs G2: OR of SGA for weight gain < 15 lbs G3: Reference Weight gain 15-25 lbs
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 SGA for each 5 kg decrease in net gestational WG
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
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

Results	Confounders and Effect Modifiers Included in Analysis
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
G1: 1.32 (1.20-1.44)	Pregravid weight, infant sex, smoking, parity, maternal diabetes, height, previous LBW infant, severe pregnancy-induced hypertension

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight

Results	Confounders and Effect Modifiers Included in Analysis

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(o)-3(r)-3/C01.28 Tm1.929/O /La<001E>t >>BDC 8 0 0 8 -378 490.75 Tm[(J)-3m[(A)-3(u) 5151tegTot27.27 73(t)-3G(e)-JE

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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 Infant weight percentile for mothers with normal prepregnancy weight and normal weight gain 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%
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

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
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
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)
Cheng et al., 2004[124] USA, birth certificate registry 14,114 All weights/BMI Fair	Pregravid weight: Self-report Total weight gain: Not stated	95% CIs of SGA for low weight gain (< 0.2 kg/wk) by BMI G1: Underweight G2: Normal weight G3: Overweight G4: Obese

Results	Confounders and Effect Modifiers Included in Analysis
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
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
G1: (1.2-2.4) G2: (1.9-2.7) G3: (1.6-2.9) G4: (1.4-2.1)	Maternal age, education, Medicaid status, pregravid BMI, smoking, previous SGA, adequacy of prenatal care, maternal cardiac disease, preeclampsia, year of birth of second infant

continued

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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 T*[(m)-3(a)-3(t)-ords 01(a)-3(n)-3((D 33674 >>B13(t)-)-3(o0CA6 68 Weight gain

Results	Confounders and Effect Modifiers Included in Analysis
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
G1: 19.5% G2: 13.5% G3: 9.5% G4: 14.2% G5: 9.9% G6: 11.5%	N/A

continued

Results	Confounders and Effect Modifiers Included in Analysis

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]

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-CID 33919 ii3(u)-3(l)y3(o)-3(m)-33( )-99(s)-3(t

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

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
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

Results	Confounders and Effect Modifiers Included in Analysis
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
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
Gestational weight gain was not a predictor of Apgar scores < 7	Age, parity, BMI
AOR for 1 min Apgar scores < 4 for nulliparous women with low BMI, weekly weight gain < 15 kgr , compared with women gaining 0.25-0.3 kgr : 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 kgr compared with women	

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.

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]

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 ofty(b)-3(u)-3ioma weight gaid und tht outcomttct not8ma low quandec3(r)-3(i)-3(a)-3(t)-3(i)-3(o)-3(t)-3( )-38(o)

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	

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Cetlegpra(a)-3(n)-3( )-30(()-3(3(n)-3(r3(o)-3(w)a)-3((a)-3(p)-3(e)-3(g)-3(a)
TGE-311M0 TGE-312M0r R0 Td(M)-3(0)-31. 9.401C3.7[(P)-3(N)-31

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[12] Antaa				

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.

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 hy3(r)-3(t)-(.)-3(3)- tI 25w ⁄AMCID 325.1.se1.tiric1. l c1sults

the following complications: macrosomia (birthweight > 4,500 g), hypoglycemia (at least one plasma glucose <

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

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.

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]

Detailed results One good-quality study analyzed the relationship between weight gain (total surproiled res etween 43(i-pl34742 is)-4( )ta14(1oEM

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
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

Results	Confounders and Effect Modifiers Included in Analysis
G1: $\beta = 0.21$ ($P < 0.0003$)	

TABLE 25. Continued

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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
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

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.

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 cireiofer - ènei length, BMI, PI, and irthweighthed cireioferenei gestational weighs gain wasseiatedwith core

Results

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 k477m744leglth 5thngli clu5tly uu 5tlgl5 All t17th All

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

O-47.75 -1d0-15.625 Td C-3( )-c-30(i)-3(n)-3(c)-3(o)-3(m)-3(an <</MCI03JEMC /Sp00)-3(l)-3(i)-3(t)-3(y)]TJ]TJEMC /Span.625 Td[(O( )-5.306 133.509 Tm)-3(-)-3

Author, Year Country, Setting Sample Size Baseline BMI Quality		

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 (IRRs) 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]

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 postpar-

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
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

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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 (va7 >>BDC 8 -3(y)]TJEMC /Span <</MCID 35629 >>BDC	

Confounders and Effect Modifier(How

Results

TABLE 27. Continued

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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
Ohlin et al., 1990[145] Sweden, maternity clinics 1423 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Maternity records	Gestational weight gain as a continuous variable (kg)

Results	Confounders and Effect Modifiers Included in Analysis

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

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tum[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 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.

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.

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

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

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
Thorsdottir, 2002[53] Iceland, hospital records 614 Normal weight/BMI 19.5-5-5-7.tW3(I)]TJEMC /Span <</MCID136458 >>BDC 0 -1.875 FmtI				

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

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

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
Kiel et al., 2007[4] USA, Hospital				

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	

Author, Year	
Country, Setting	
Sample Size	
Baseline BMI	
Quality	
Pregravid Weight	
(How Measured88b8p3(d)-3( )-33 36994tMCID 36992 >>Bravid rBWeight6	
(How Measured	

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) Fair	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, the association between cesarean delivery and weight gain was 1.45 (1.21-1.73)	Age, race, parity, pregravid BMI, height, maternal high and low weight gain, smoking, gestational age, birthweight
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./Artif-3(2)-3(-Arti)	

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
Thorsdottir et al., 2002				

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]

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

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
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 (Underwt & < 0.38 kg/wk or normal wt & < 0.37 kg/wk)

Results	Confounders and Effect Modifiers Included in Analysis

34.)-3(-)]TJE (BMS(t)-3(a)-2(l)-3( )-31(r)-3(e)-3(c)-3(o)-3(r)-3(d)-3(s)]TJEMC /Artifact <</O /Layout >>BDC 9.125 4.375 Td[

TABLE 31. Continued

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
Stotland et al., 2006[85] USA, academic medical center 15,101 Underweight BMI < 19.8 and normal weight BMI 19.8-26 Fair	Pregravid weight: Self-reported Total weight gain: prenatal records	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

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; PTB, pre-term birth; USA, United States of America; wk, week; wt, weight.

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,

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

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 ≤ 29[20,156] 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.

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

(s)]TJEMC0( )-31(T)-3(h)-7n)-3(l)-3(y)]TJEMC /Artifact <</O /Layout >>fact <</O teasur(s)]TJEMC0( )-31(T)-3(h)-7nlydht

TABLE 32. Continued

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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
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
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
Ogunyemi et al., 1998[60] USA, Hospital 582 All weight/BMIs (using IOM definitions) Fair	Pregravid weight: Self-reported Total gestational weight gain: Prenatal records, measured	G1: Low < IOM G2: Normal = IOM G3: High > IOM

TABLE 32. Continued

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
Scholl et al., 1995[158] USA Camden Study 274 Normal weight BMI 19.8-26 air	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
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
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

&, and; AOR, adjusted odds ratio; birthwt, birthweight; BMI, body mass index; g, gram; G, group; GDM, 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.

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.

Results	Confounders and Effect Modifiers Included in Analysis
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
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
G1: 3,302 g G2: 3,192 g G3: 3,337 g G4: 3,506 g G5: 3,453 g $P = < 0.05$	NR

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

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
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	

Author, Year Country, Setting Sample Size Baseline BMI Quality

Results	Confounders and Effect Modifiers Included in Analysis
Within every BMI-race 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 m3(e)-3(r)-3(e)-31(w)-3(i-31(f)-3(o)-3(r)-3( )-31(a)-3(l)-3(l)]TJEMC /Artifact <</O /Layout >>BDC T*[	

TABLE 33. Continued

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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

Results

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)

Results	Confounders and Effect Modifiers Included in Analysis
G1:12.8% G2: 8.9% G3: 7.9% G4: 6.8% G5: 8.7% *P* (for G1-G5) = 0.405	Age, parity, pregravid BMI, GDM, pregnancy-induced hypertension, prenatal adequacy, alcohol use, drug use, smoking, gestational age
G6: 8.5% G7: 5.6% G8: 8.9% *P* (for G6-G8) = 0.183	
AOR (95% CI) for birthweight < 2,500 g among obese women (BMI > 29.0): G3: 1.0 (reference) G1: 4.2 (0.9-19.6)	
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

continued

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
Ogunyemi et al., 1999[60] USA, Hospital 582 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories: G1: < IOM G2: Within IOM G3: > IOM BMI IOM
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
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 BMSSd trim0.2 BMSSd trim : 1 a BM0 N3.0SSd trim N : 1 G12 Low 1st B(27.0: N : 1 G1: Low 1stght 280.2

Results	Confounders and Effect Modifiers Included in Analysis
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, tobacco use, previous fetal death, hypertension, asthma, previous LBW, vomiting, NICU
Distribution of LBW, %: G1: 21.4 G2: 10.6 G3: 4.3 *P* = NS	Not applicable
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

continued

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
Bianco et al., 1998[54] USA, medical center 11,926 /Span <</MCID 39045	PregraviwTd[(P)-3(W)71(e)-3(i)-3(g)-3:T-3(e)-3(d)]TJEMC /Span 52/MCID 3 >>BDC 0LN1.25 Td[(C)-3 Td[b27 0 Td[(R)-3(i)-3(n)-3(>BDC e)-3( )-31(B)-3(TotawTd[(P)-3(W)71(e)-3(i)-3(g)-3(a)-3(gT)95(o)-3>BDC T*[:T-3(e)-3(d)]TJE	
avnaonb27 0 Td[(R)-3(i)-3(n)-3(>BDC e)-3( )-31(B)-3(.-3(e)-3(d)]TJEMC /Span <</MCID 39/C0)>>BDC 9.5		

Results	Confounders and Effect Modifiers Included in Analysis
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
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)	Ethnicity, intendedness of pregnancy, Medicaid status, WIC status, prenatal care, diabetes, hypertension
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)	

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.

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.

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.

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]).

Detailed results for LGA infant weight One study reported the risk of LGA among women of all weight status groups

TABLE 34. Weight Change Relative to IOM Thresholds and Large-for-Gestational-Age Infant Weight

Results	Confounders and Effect Modifiers Included in Analysis
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,3( )]TJEMC /Artifact <</C

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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: G1: < 25 lbs G2: 25-35 lbs G3: > 35 lbs
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
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
Stevens-Simon and McAnarney, 1992[15]		

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups

Stotland et al.,-3(s)]TJEMC /Span <</MCID s

Confounders and Effect Modifiers

Results

Results	Confounders and Effect Modifiers Included in Analysis
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 61(G)-3(6)13( )y rG6ng a >	

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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

Results	Confounders and Effect Modifiers Included in Analysis
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
Birthweight >4,500g,% G1:120e G2: 1205 G3: G3G5 G4: 13G6 G5: 23G6E>Tj EMC /Artifact <</O /Layout >>BDC /T1_1 1 Tf 6.411 0 Td [( )-31(()-f(N)-3(o)-3(r)-[( )-3[(G) G7: 6.6 G8: 1616	

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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

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 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

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 (sm (sght lig slTnT/Span <MCID 40456 >BDC -BT/T1201 Tf00 0 0 10 27

TABLE 35. Weight Change Relative to IOM Thresholds and Small-for-Gestational-Age

Results	Confounders and Effect Modifiers Included in Analysis

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(f)-25 TD25 TdA-3(I)-3( )]TJEMC /Artifact <</O /Layout >>BDC 0 -1.-3(e)-3(a)s( )-31(N)c3(e)-3(a)-3(r)-3(i)e3(c)d3(c)-3(k0-

TABLE 35. Continued

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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
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
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 19.8-26 G6: < 11.5 G7: 11.5-16 G8: > 16

Results	Confounders and Effect Modifiers Included in Analysis
SGA,%: G1: 22.3 G2: 15.6 G3: 11.5 $P < 0.01$ for G1-G3	Parity, pregravid BMI, time between last weight measure and delivery, height
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	
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

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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
Parker and Abrams, 1992[118] USA, hospital (California) 6,690 All weight/BMI Fair	Pregravid weight: Self-report Total weight gain: Measured	Maternal weight gain categories: G1: < IOM G2: Within IOM G3: > IOM BMI IOM

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
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
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
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

BMI, body mass index; G, group; IOM, Institute of Medicine; kg/wk, kilogram per week; NS, not significant; USA, United States of America.

Overview of results Three fair studies provide insufficient evidence to support an association between weight gain and low Apgar scores.

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

Results	Confounders and Effect Modifiers Included in Analysis
Maternal weight gain by IOM guidelines was not not a signigicant predictor of Apgar scores (details—none reported)	None

Distribution of 1-minute Apgar score ≤ 4,%:
G1: 25.0
G2: 4.5
G3: 14.9
P =4,92 for G1 v-3(2(o)-3(r)-ryout >>BDC /T1_2 1 Tf0.551 0 Td[( )-5(0)-3(.)-3/Artifact <(r)-3( )-31(s)-3(c)-3
GN/Artifact <</Oe/O /p-3(/O-3(3(e)-3(g)-3<0062(t)]TJEMC 75out >-13. T*[(GSct <</O //ArtifactlTf0.614

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 US study of a hospital database examined perinatal mortality (Evidence Table 47).

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 yi(b)-9.0088 420d-184(yi(b)-EMC /Spa(yi(b)-(f)-3(e)-3c-3( )-27(w)-(b)-3(i)-3

(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 (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 Nu

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] Denmark-National Birth 37,459 All wt/BMI Under wt BMI < 18.5 Normal BMI 18.5-24.9 Overweight BMI 25-29 Obese BMI ≥ 30 Fair	Pregravid weight: Prepregnant weight Self reported Total weight gain: 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. Unadjusted RR full BF G1: 1.13 (95% 1.08-1.18) G3: 1.05 (1.03-1.08). Any BF G1: RR 1.16 (1.11-1.22) G3: 1.05 (1.03-1.08) GWG not a predictor of full or any when BMI was in the model.	BMI
Li et al., 2003[167] USA WIC clinics				

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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.

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 U.S. study.[167]

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]

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

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

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]	Pregravid weight:	Maternal weight gain	Mean (SEM) retained weight (defined as 2-day	
USA, clinic	Self-report	< IOM recommendations:	postpartum weight minus pregravid weight, kg):	
487	Total weight gain:	G1: BMI < 19.8	G1: 3.2 (0.5) $P < 0.05$ compared to G4	
All weight/BMI	Measured	G2: BMI 19.8-26.0	G2: 0.8 (0.4) $P < 0.05$ compared to G5	
Fair		G3: BMI > 26.0	G3: - 5.0 (0.7) $P < 0.05$ compared to G6	
		Maternal weight gain within IOM recommendations:	G4: 8.2 (0.7)	
		G4: BMI < 19.8	G5: 7.0 (0.4)	
		G5: BMI 19.8-26.0	G6: 1.4 (0.8)	
		G6: BMI > 26.0		
		Maternal weight gain > IOM recommendations:		
		G7: BMI < 19.8		
		G8: BMI 19.8-26.0		
		G9: BMI > 26.0		

TABLE 39. Weight Change Relative to IOM Thresholds and Weight Retention During the First Year Postpartum

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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
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 BMI IOM

Author, Year Country, Setting Sample Size Baseline BMI Quality	Pregravid Weight (How Measured) Total Weight Gain (How Measured)	Definition of Groups
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
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

Results	Confounders and Effect Modifiers Included in Analysis
Mean weight retention at 6 months postpartum, lbs: G1: 0.4 G2: 3.7 G3: 13.5 $P < 0.001$	Mode of delivery, infant sex, breastfeeding, infant birthweight, pregravid BMI
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 18 months postpartum: r = 0.49, $P < 0.001$	
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)	

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the United States, and one was from Sweden.

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 Fair	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 baseline and 15 years postpartum: G1: 0.43 (-1.87-2.73) G2: reference	

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 (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

In the other U.S. study, the incidence of overweight at the second preg-

7. AA Hedley, CL Ogden, CL Johnson, MD Carroll, LR Curtin, and KM Flegal. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999-2002 J Am Med Assoc 2004. Jun 16. 291: (23) 2847-50.
8. CL Ogden, MD Carroll, LR Curtin, MA McDowell, CJ Tabak, and KM Flegal. Prevalence of overweight and obesity in the United States, 1999-2004 J Am Med Assoc 2006. Apr 5. 295: (13) 1549-55.
9. Institute of Medicine. Influence of Pregnancy Weight on Maternal and Child Health: Workshop Report Committee on the Impact of Pregnancy Weight on Maternal and Child Health. Washington, DC: National Academy Press 2007.
10. MS Kramer, I Morin, H Yang, RW Platt, R Usher, and H McNamara, et al. Why are babies getting bigger? Temporal trends in fetal growth and its determinants J Pediatr 2002. Oct. 141: (4) 538-42. (PubMed)
11. Martin JA, Hamilton BE, Menacker F, Sutton PD, Mathews TJ. Preliminary births for 2004: Infant and maternal health. Health E-stats. Hyattsville, MD: National Center for Health Statistics. Released November 15, 2005.
12. NJ Eastman and E Jackson. Weight relationships in pregnancy. I. The bearing of maternal weight gain and pre-pregnancy weight on birth weight in full term pregnancies Obstet Gynecol Surv 1968. Nov. 23: (11) 1003-25. (PubMed)
13. B Abrams. Prenatal weight gain and postpartum weight retention: a delicate balance Am J Pub Health 1993. 83: (8) 1082-4. (PubMed) (Full Text in PMC)
14. Suitor CW. Maternal weight gain: a report of an expert work group PDF Full Text. Rockville, MD: United States Department of Health and Human Services Public Health Service 1997.
15. LA Schieve, ME Cogswell, and KS Scanlon. Trends in pregnancy weight gain within and outside ranges recommended by the Institute of Medicine in a WIC population Matern Child Health J 1998. Jun. 2: (2) 111-6. (PubMed)
16. SA Lederman, A Paxton, SB Heymsfield, J Wang, J Thornton, and RN Pierson Jr. Body fat and water changes during pregnancy in women with different body weight and weight gain Obstet Gynecol 1997. Oct. 90: (4 Pt 1) 483-8. (PubMed)
17. S Carmichael, B Abrams, and S Selvin. The pattern of maternal weight gain in women with good pregnancy outcomes Am J Public Health 1997. Dec. 87: (12) 1984-8. (PubMed) (Full Text in PMC)
18. LE Caulfield, FR Witter, and RJ Stoltzfus. Determinants of gestational weight gain outside the recommended ranges among black and white women Obstet Gynecol 1996. May. 87: (5 Pt 1) 760-6. (PubMed)
19.

25.

42. L Tulman, KH Morin, and J Fawcett. Prepregnant weight and weight gain during

60. D Ogunyemi, S Hullett, J Leeper, and A Risk. Prepregnancy body mass index, weight gain during pregnancy, and perinatal outcome in a rural black population J Matern Fetal Med 1998. Jul-Aug. 7: (4) 190-3. (PubMed)
61. K Wataba, T Mizutani, K Wasada, M Morine, T Sugiyama, and N Suehara. Impact of prepregnant body mass index and maternal weight gain on the risk of pregnancy complications in Japanese women Acta Obstet Gynecol Scand 2006. 85: (3) 269-76. (PubMed)
62. CW Ko. Risk factors for gallstone-related hospitalization during pregnancy and the postpartum Am J Gastroenterol 2006. Oct. 101: (10) 2263-8. (PubMed)
63. G Lindseth and MY Bird-Baker. Risk factors for cholelithiasis in pregnancy Res Nurs Health 2004. 27: (6) 382-91. (PubMed)
64. CA Gosselink, EE Ekwo, RF Woolson, A Moawad, and CR Long. Dietary habits, prepregnancy weight, and weight gain during pregnancy. Risk of pre term rupture of amniotic sac membranes Acta Obstet Gynecol Scand 1992. Aug. 71: (6) 425-38. (PubMed)
65. EA Nohr, BH Bech, M Vaeth, KM Rasmussen, TB Henriksen, and J Olsen. Obesity, gestational weight gain and preterm birth: a study within the Danish National Birth Cohort Paediatr Perinat Epidemiol 2007. Jan. 21: (1) 5-14. (PubMed)
66. JM Lang, E Lieberman, and A Cohen. A comparison of risk factors for preterm labor and term small-for-gestational-age birth Epidemiology 1996. Jul. 7: (4) 369-76. (PubMed)
67. BW Graves, SA DeJoy, A Heath, and P Pekow. Maternal body mass index, delivery route, and induction of labor in a midwifery caseload J Midwifery Womens Health 2006. Jul-Aug. 51: (4) 254-9. (PubMed)
68. U Ekblad and S Grenman. Maternal weight, weight gain during pregnancy and pregnancy outcome Int J Gynaecol Obstet 1992. Dec. 39: (4) 277-83. (PubMed)
69. P Purfield and K Morin. Excessive weight gain in primigravidas with low-risk pregnancy: selected obstetric consequences J Obstet Gynecol Neonatal Nurs 1995. Jun. 24: (5) 434-9.
70. JW Johnson, JA Longmate, and B Frentzen. Excessive maternal weight and pregnancy outcome Am J Obstet Gynecol 1992. Aug. 167: (2) 353-70. discussion 70-2 (PubMed)
71. TJ Rosenberg, S Garbers, H Lipkind, and MA Chiasson. Maternal obesity and diabetes as risk factors for adverse pregnancy outcomes: differences among 4 racial/ethnic groups Am J Public Health 2005. Sep. 95: (9) 1545-51. (PubMed) (Full Text in PMC)
72. KS Joseph, DC Young, L Dodds, CM O'Connell, VM Allen, and S Chandra, et al. Changes in maternal characteristics and obstetric practice and recent increases in primary cesarean delivery Obstet Gynecol 2003. Oct. 102: (4) 791-800. (PubMed)
73. G Chen, S Uryasev, and TK Young. On prediction of the cesarean delivery risk in a large private practice Am J Obstet Gynecol 2004. Aug. 191: (2) 616-24. discussion 24-5 (PubMed)
74. TK Young and B Woodmansee. Factors that are associated with cesarean delivery in a large private practice: the importance of prepregnancy body mass index and weight gain Am J Obstet Gynecol 2002. Aug. 187: (2) 312-8. discussion 8-20 (PubMed)
75. MJ Shepard, AF Saftlas, L Leo-Summers, and MB Bracken. Maternal anthropometric factors and risk of primary cesarean delivery Am J Public Health 1998. Oct. 88: (10) 1534-8. (PubMed) (Full Text in PMC)
76. FR Witter, LE Caulfield, and RJ Stoltzfus. Influence of maternal anthropometric status and birth weight on the risk of cesarean delivery Obstet Gynecol 1995. Jun. 85: (6) 947-51. (PubMed)
77.

78.

97. NF Butte, KJ Ellis, WW Wong, JM Hopkinson, and EO Smith. Composition of gestational weight gain impacts maternal fat retention and infant birth weight Am J Obstet Gynecol 2003. Nov. 189: (5) 1423-32. (PubMed)
98. JE Brown, MA Murtaugh, DR Jacobs Jr, and HC Margellos. Variation in newborn size according to pregnancy weight change by trimester Am J Clin Nutr 2002. Jul. 76: (1) 205-9. (PubMed)
99. B Zaren, S Cnattingius, and G Lindmark. Fetal growth impairment from smoking—is it influenced by maternal anthropometry? Acta Obstet Gynecol Scand Suppl 1997. 165: 30-4. (PubMed)
100. A Pezzarossa, N Orlandi, V Baggi, D Dazzi, E Ricciarelli, and F Coppola. Effects of maternal weight variations and gestational diabetes mellitus on neonatal birth weight J Diabetes Complications 1996. Mar-Apr. 10: (2) 78-83. (PubMed)
101. B Abrams and S Selvin. Maternal weight gain pattern and birth weight Obstet Gynecol 1995. Aug. 86: (2) 163-9. (PubMed)
102. ML Hediger, TO Scholl, JI Schall, MF Healey, and RL Fischer. Changes in maternal upper arm fat stores are predictors of variation in infant birth weight J Nutr 1994. Jan. 124: (1) 24-30. (PubMed)
103. JY Groff, PD Mullen, M Mongoven, and K Burau. Prenatal weight gain patterns and infant birthweight associated with maternal smoking Birth: Issues in Perinatal Care 1997. 24: (4) 234-9.
104. B Luke, ML Hediger, and TO Scholl. Point of diminishing returns: when does gestational weight gain cease benefiting birthweight and begin adding to maternal obesity? J Maternal-Fetal Med 1996. 5: (4) 168-73.
105. SK Muscati, K Gray-Donald, and KG Koski. Timing of weight gain during pregnancy: promoting fetal growth and minimizing maternal weight retention Int J Obes Relat Metab Disord 1996. Jun. 20: (6) 526-32. (PubMed)
106. CA Hickey, R Uauy, LM Rodriguez, and LW Jennings. Maternal weight gain in low-income black and Hispanic women: evaluation by use of weight-for-height near term Am J Clin Nutr 1990. Nov. 52: (5) 938-43. (PubMed)
107. E Desjardins and D Hardwick. How many visits by health professionals are needed to make a difference in low birthweight? A dose-response study of the Toronto Healthiest Babies Possible program Can J Public Health 1999. Jul-Aug. 90: (4) 224-8. (PubMed)
108. W Zhou and J Olsen. Gestational weight gain as a predictor of birth and placenta weight according to pre-pregnancy body mass index Acta Obstet Gynecol Scand 1997. Apr. 76: (4) 300-7. (PubMed)
109. JN Lasker, B Coyle, K Li, and M Ortynsky. Assessment of risk factors for low birth weight deliveries Health Care for Women International 2005. 26: (3) 262-80. (PubMed)
110. MM Hedderson, NS Weiss, DA Sacks, DJ Pettitt, JV Selby, and CP Quesenberry, et al. Pregnancy weight gain and risk of neonatal complications: macrosomia, hypoglycemia, and hyperbilirubinemia Obstet Gynecol 2006. Nov. 108: (5) 1153-61. (PubMed)
111. H Takimoto, T Sugiyama, H Fukuoka, N Kato, and N Yoshiike. Maternal weight gain ranges for optimal fetal growth in Japanese women Int J Gynaecol Obstet 2006. Mar. 92: (3) 272-8. (PubMed)
112. RL Bergmann, R Richter, KE Bergmann, A Plagemann, M Brauer, and JW Dudenhausen. Secular trends in neonatal macrosomia in Berlin: influences of potential determinants Paediatr Perinat Epidemiol 2003. Jul. 17: (3) 244-9. (PubMed)
113. T Clausen, TK Burski, N Oyen, K Godang, J Bollerslev, and T Henriksen. Maternal anthropometric and metabolic factors in the first half of pregnancy and risk of neonatal macrosomia in term pregnancies. A prospective study Eur J Endocrinol 2005. Dec. 153: (6) 887-94. (PubMed)

132. NA Sowan and ML Stember. Parental risk factors for infant obesity Am J Matern Child Nurs 2000. 25: (5) 234-41.
133. KK Ong, ML Ahmed, PM Emmett, MA Preece, and DB Dunger. Association between postnatal catch-up growth and obesity in childhood: prospective cohort study Br Med J 2000. Apr 8. 320: (7240) 967-71. (PubMed) (Full Text in PMC)
134. C Li, MI Goran, H Kaur, N Nollen, and JS Ahluwalia. Developmental trajectories of overweight during childhood: role of early life factors Obesity (Silver Spring) 2007. Mar. 15: (3) 760-71. (PubMed)
135. W Yuan, O Basso, HT Sorensen, and J Olsen. Maternal prenatal lifestyle factors and infectious disease in early childhood: a follow-up study of hospitalization within a Danish birth cohort Pediatrics 2001. Feb. 107: (2) 357-62. (PubMed)
136. Y Linne, L Dye, B Barkeling, and S Rossner. Weight development over time in parous women—the SPAWN study—15 years follow-up Int J Obes Relat Metab Disord 2003. Dec. 27: (12) 1516-22. (PubMed)
137. HE Harris, GT Ellison, and M Holliday. Is there an independent association between parity and maternal weight gain? Ann Hum Biol 1997. Nov-Dec. 24: (6) 507-19. (PubMed)
138. HE Harris, GT Ellison, M Holliday, and E Lucassen. The impact of pregnancy on the long-term weight gain of primiparous women in England Int J Obes Relat Metab Disord 1997. Sep. 21: (9) 747-55. (PubMed)
139. SC Hunt, MM Daines, TD Adams, EM Heath, and RR Williams. Pregnancy weight retention in morbid obesity Obes Res 1995. Mar. 3: (2) 121-30. (PubMed)
140. ES Parham, MF Astrom, and SH King. The association of pregnancy weight gain with the mother‘s postpartum weight J Am Diet Assoc 1990. Apr. 90: (4) 550-4. (PubMed)
141. L Walker, JH Freeland-Graves, T Milani, G George, H Hanss-Nuss, and M Kim, et al. Weight and behavioral and psychosocial factors among ethnically diverse, low-income women after childbirth: II. Trends and correlates Women Health 2004. 40: (2) 19-34.
142. Y Linne, L Dye, B Barkeling, and S p(2rk-3(m34(S)-3( )o)-3(s)-3(t)-3(p)-3(a)-3(r)-3(t)-34(k)-3(s)-3(n)-3(

150. PS Kaiser and RS Kirby. Obesity as a risk factor for cesarean in a low-risk population Obstet Gynecol 2001. Jan. 97: (1) 39-43. (PubMed)
151. CA Hickey, SP Cliver, SF McNeal, HJ Hoffman, and RL Goldenberg. Prenatal weight gain patterns and spontaneous preterm birth among nonobese black and white women Obstet Gynecol 1995. Jun. 85: (6) 909-14. (PubMed)
152. LA Schieve, ME Cogswell, KS Scanlon, G Perry, C Ferre, and C Blackmore-Prince, et al. Prepregnancy body mass index and pregnancy weight gain: associations with preterm delivery. The NMIHS Collaborative Study Group Obstet Gynecol 2000. Aug. 96: (2) 194-200. (PubMed)
153. JN Nielsen, KO O'Brien, FR Witter, SC Chang, J Mancini, and MS Nathanson, et al. High gestational weight gain does not improve birth weight in a cohort of African American adolescents Am J Clin Nutr 2006. Jul. 84: (1)enn en((a)-3(t)-3113()-3(P)-3(u)-3(b)-3(M)-3(e)-3(d)-

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 Institute of Medicine (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.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 and for the child, as discussed in Chapter 5, *Consequences of Gestational Weight Gain for the Mother*, and Chapter 6, *Consequences*

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

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),

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics
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 *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	Pregnancy weight gain = Tn <</MCID /T1_1 1 TE(a)

Outcomes/Results/Confounders		
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	*Maternal confounders/effect modifiers:* • Race • Parity • Maternal age • Number of cigarettes smoked/day • Prepregnancy weight/height • SES

continued

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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
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 *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	*Demographics:* G1: Married: 64% Race: White, 65.7% Black, 29.1% Hispanic, 4.0% Mean age: 24.9 G2: Married: 77.3% Race: White, 72.8% Black, 22.7% Hispanic, 3.2% Mean age: 26.2

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), aSpan <</MHC T*[(T)asteria[(P9066(E)-5Ar)-3(e)-3

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics
Author, year: Butte et al., 1984		
Country/Setting: USA		
Enrollment period: NR		
Study Objective: To examine		

Outcomes/Results/Confounders

TABLE F-1 Continued

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics
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 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	Low weight gain = < 0.16 kg/wk

Outcomes/Results/Confounders		
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 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)

continued

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics
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 outcome among co/SpanJEMC /Sp-3(fl)-3 /Sp3(l)-3(l)-3(m)-3(e)-3(n)-3(t)4131(o)-3(u)-3(Gm)-32Tm[:t25		

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics
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
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/Results/Confounders		
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:* • Gender • Birth weight/length
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:* • NR

continued

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics
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:*	

Outcomes/Results/Confounders		
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 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

continued

Outcomes/Results/Confounders

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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
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 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	Study population was further divided into eight categories to represent 10-lb gain increments. Prepregnancy BMI: height and weight rep(e)-2ac >>BDC 8 0 0 8 248 515.75EMC /S

Outcomes/Results/Confounders

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 < 5 lbs
G5: GWG 6-14 lbs
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

Maternal confounders/effect

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics
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:*	

Outcomes/Description	Results/Comments	Confounders
• Gestation(l)-3l weight gain • Infant birth weight	*Resulls:* BMI and GWG, but not pre-plegnancy physic(l)-3l activity level, were linked to gestation(l)-3l weeks 12 and 33 was clrrellled with ellv(l)-3ted birth weight.	*M(l)-3tern(l)-3l clnfounders/effect modifiers:* • P(l)-3rity • Smoking status • Pregnancy physic(l)-3l activity level *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
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)
Author, year: Mitchell and Lerner, 1989 *Country/Setting:* USA *Enrollment period:* NR *Study Objective:* To compare pregnancy outcome in overweight and normal weight women.	*Design:* • Cohort *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	Initial weight/BMI: recorded at first prenatal visit Gestational weight gain: difference between weight at first prenatal visit (initial weight) and weight recorded at final antepartum visit (≤ 5 days before delivery).

Outcomes/Results/Confounders		
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
Outcomes description: • Brith weight • Gestational age • 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

continued

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics
Author, year: Naeye, 1981 *Country/Setting:* USA *Enrollment period:* 1959-1966 *Study Objective:* To determine whether the growth needs of young mothers compete with the growth needs of their fetuses for available nutrients.	*Design:* • Cohort *Total Study N:* 13,830 *Inclusion criteria:* • Black • Singleton infants • Maternal age of 10-32 years *Exclusion criteria:* • Maternal diabetes mellitus • Placenta previa • Hydramnios • Oligohydramnios • Congenital malformations of the infant	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 neonate and the placenta from the maternal weight at the end of the pregnancy.
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 •	

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics
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:* • Liveborn, singleton	

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria

Outcomes/Results/Confounders

TABLE F-1 Continued

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics
Author, year: Rodriquez et al., 2008 *Country/Setting:* Sweden and Denmark *Enrollment period:* 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	*Design:* • Follow-up of prospective cohort *Total Study N:* 12,556 *Group Description:* School-aged children *Inclusion criteria:* • Live born, singleton infants *Exclusion criteria:* NR	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 subtracted from prepregnancy weight to obtain GWG Average weekly gain: divided weight gain by the number of completed gestational weight

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics
Author, year: Rudra et al., 2008 *Country/Setting:* USA (Washington State) *Enrollment period:* 1996-2005 *Study Objective:* To examine the relationship between prepregnancy weight and gestational weight gain and preterm delivery.	*Design:* • Cohort • Prospective *Total Study N:* 2,468 *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	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 Weight gain rate in early pregnancy = [(weight at 18-22 wks - prepregnancy weight)/weeks' gestation at weight meauBDC 0.503 0 Td[( )-2.C 0 -1

Outcomes/Results/Confounders

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics
Author, year: Segal et al., 2008	*Design:* • Cross-sectional	
Country/Setting: Canada	*Total Study N:* 86	
Enrollment period: NR	*Inclusion criteria:* •	
Study Objective: To examine the maternal factors that determine infant birth weight.		

Study Description

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics
Author, year: Tenovuo et al., 1988		
Country/Setting: Finland		
Enrollment period: 1985		
Study Objectiv		

Study Description	Protocol Including: Study Design/ Patient Population/ Inclusion-Exclusion Criteria

Outcomes/Results/Confounders

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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
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/Results/Confounders		
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 • 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

continued

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured), and Baseline Characteristics
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 G5: 252 G6: 154 *Inclusion criteria:* • Seen in antenatal clinic during first trimester *Exclusion criteria:* • Diabetes • Multiple pregnancy • Polyhydramnios • Gastrointestinal disorders	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/Results/Confounders

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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
Author, year: Voldner et al., 2008 *Country/Setting:* Norway *Enrollment period:* 2002-2005 *Study Objective:* To examine the modifiable determinants of fetal macrosomia, specifically lifestyle-related factors.	*Design:* • Cohort • Prospective *Total Study N:* 553 *Inclusion criteria:* • Healthy women • Singleton pregnancy	Dd md midderci, Dd md midderci, Dd md midderci, Dd md midderci, riddmid(l)- icdte(t)-3(e)-x<</MC76 47058 3(l)-3(l)-3,5lchyohcrti, , ctcon teria:

Outcomes/Results/Confounders	
Outcomes description: • Macrosomic infant (≥ 4,200 g)	*Results:* 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 g(8)-3(2)-3( )-31(k)-3(g)]TJ EMC /Artifact <</(i)-3(n)-3 O8-3(2)-3( )-31(k)-3(g)]TJ EMC /Artifact <</-31(=)-3( )-31(1)-3(.)-3(79)Lay(

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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
Author, year: Wolff et al., 2008 *Country/Setting:* Denmark *Enrollment period:* NR *Study Objective:* To examine the effects of dietary counseling on GWG and glucose metabolism in obese pregnant women.	*Design:* • Randomized control trial *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	Weight, height, blood pressure and heart rate were measured at inclusion and at 27 and 36 weeks' gestation. 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).

Study Description	Study Design/ Patient Population/ Inclusion-Exclusion Criteria	Protocol Including: Pregravid Weight (how measured), Total Weight Gain (how measured),

Outcomes/Results/Confounders		
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 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

REFERENCES

Abrams B. F. and R. K. Laros, Jr. 1986. Prepregnancy weight, weight gain, and birth weight. *American Journal of Obstetrics and Gynecology* 154(3): 503-509.

Abrams B., V. Newman, T. Key and J. Parker. 1989. Maternal weight gain and preterm delivery. *Obstetrics and Gynecology* 74(4): 577-583.

Berkowitz G. S. 1981. An epidemiologic study of preterm delivery. *American Journal of Epidemiology* 113(1): 81-92.

Billewicz W. C. and A. M. Thomson. 1957. Clinical significance of weight trends during pregnancy. *British Medical Journal* 1(5013): 243-247.

Butte N. F., C. Garza, J. E. Stuff, E. O. Smith and B. L. Nichols. 1984. Effect of maternal diet and body composition on lactational performance. *American Journal of Clinical Nutrition* 39(2): 296-306.

Chen X. K., S. W. Wen, N. Fleming, Q. Yang and M. C. Walker. 2008. Increased risks of neonatal and postneonatal mortality associated with teenage pregnancy had different explanations. *Journal of Clinical Epidemiology* 61(7): 688-694.

Frentzen B. H., D. L. Dimperio and A. C. Cruz. 1988. Maternal weight gain: effect on infant birth weight among overweight and average-weight low-income women. *American Journal of Obstetrics and Gynecology* 159(5): 1114-1117.

Geelhoed J. J., V. A. N. O.-G. L, B. O. Verburg, E. A. Steegers, A. Hofman, W. Helbing, J. C. -

Nohr E. A., M. Vaeth, J. L. Baker, T. Sorensen, J. Olsen and K. M. Rasmussen. 2008. Combined associations of prepregnancy body mass index and gestational weight gain with the outcome of pregnancy. *American Journal of Clinical Nutrition* 87(6): 1750-1759.

Papiernik E. and M. Kaminski. 1974. Multifactorial study of the risk of prematurity at 32 weeks of gestation. I. A study of the frequency of 30 predictive characteristics. *Journal of Perinatal Medicine* 2(1): 30-36.

Rodriguez A., J. Miettunen, T. B. Henriksen, J. Olsen, C. Obel, A. Taanila, H. Ebeling, K. M. Linnet, I. Moilanen and M. R. Jarvelin. 2008. Maternal adiposity prior to pregnancy is associated with ADHD symptoms in offspring: evidence from three prospective pregnancy cohorts.

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

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) (Nohr et al., 2008) was presented to the Committee to Reexamine IOM Pregnancy Weight Guidelines in June 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 two telephone interviews during pregnancy at approximately 16 and 30 weeks of

gestation, and two 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).

Independent Variables

The main exposures were prepregnancy BMI and GWG. In the first pregnancy interview, the women reported their prepregnancy weight and

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 two variables defined as postpartum weight loss (loss ≥ 2 kg) and postpartum weight retention (gain of ≥

liveries, all women with cesarean deliveries were excluded. In all adjusted models, Wald's test with nine 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.

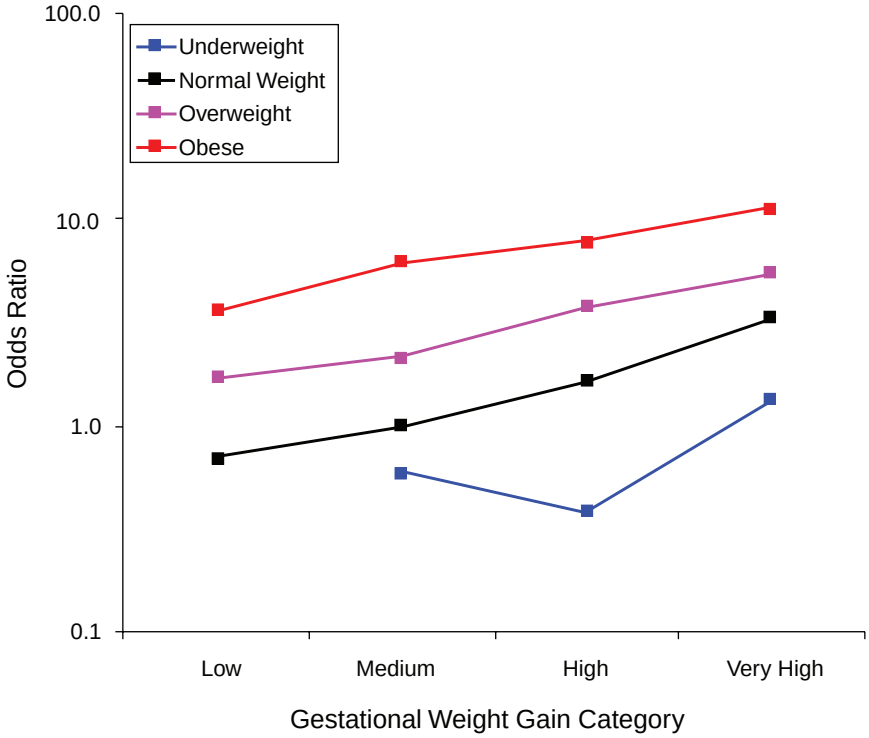

FIGURE G-1A Preeclampsia.
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE G-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

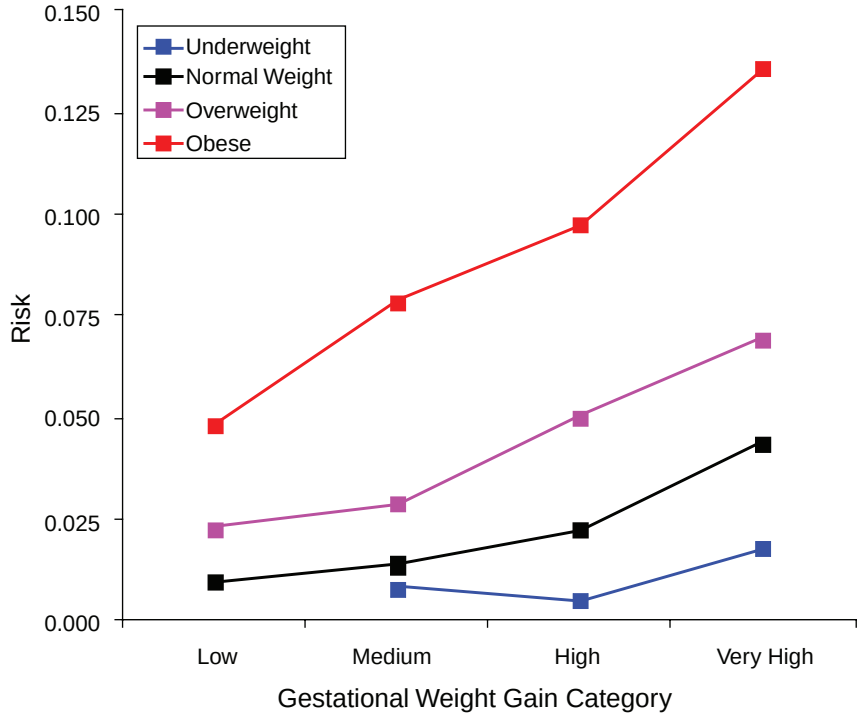

FIGURE G-1B Preeclampsia.
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

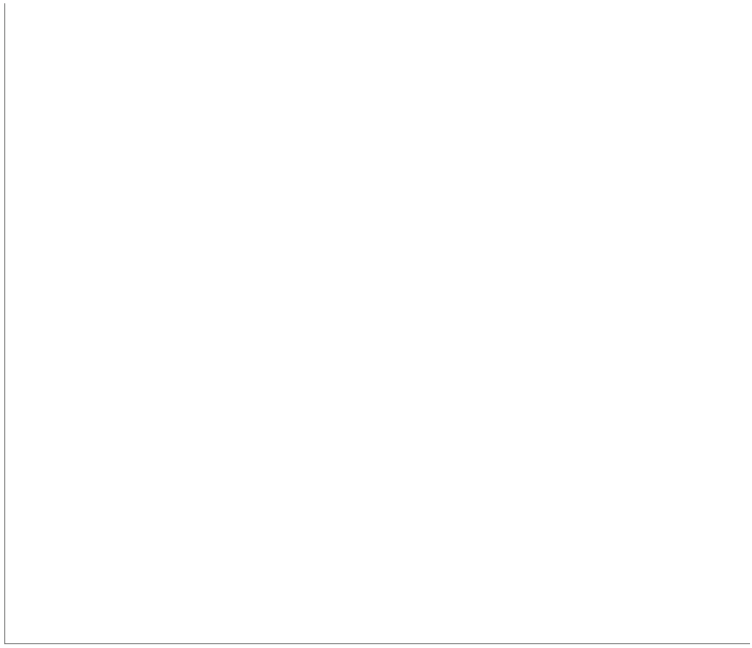

FIGURE G-2A Hypertensive disorders.
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

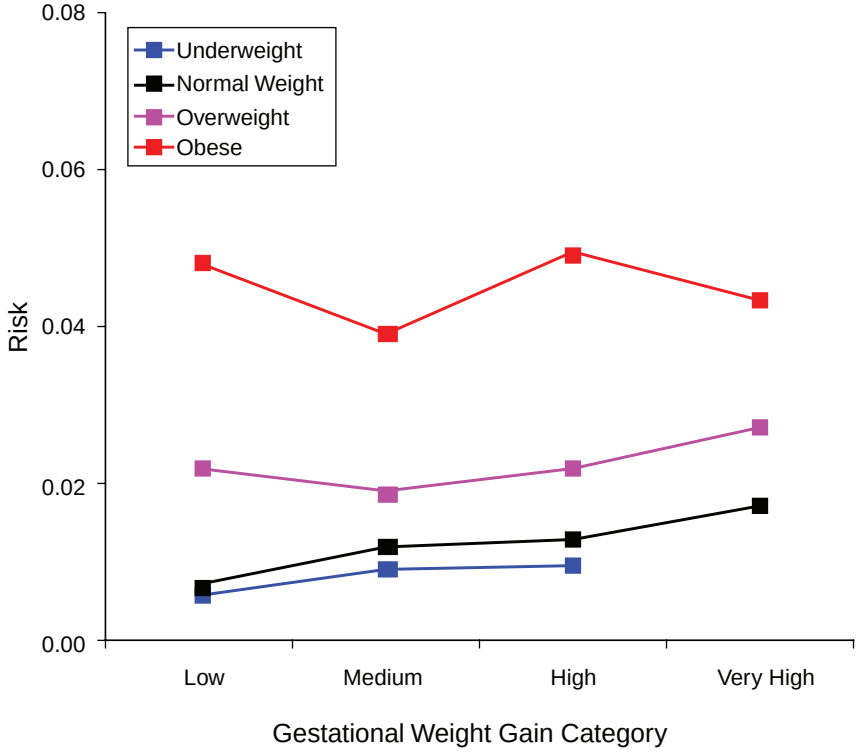

FIGURE G-2B Hypertensive disorders.
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE G-2B

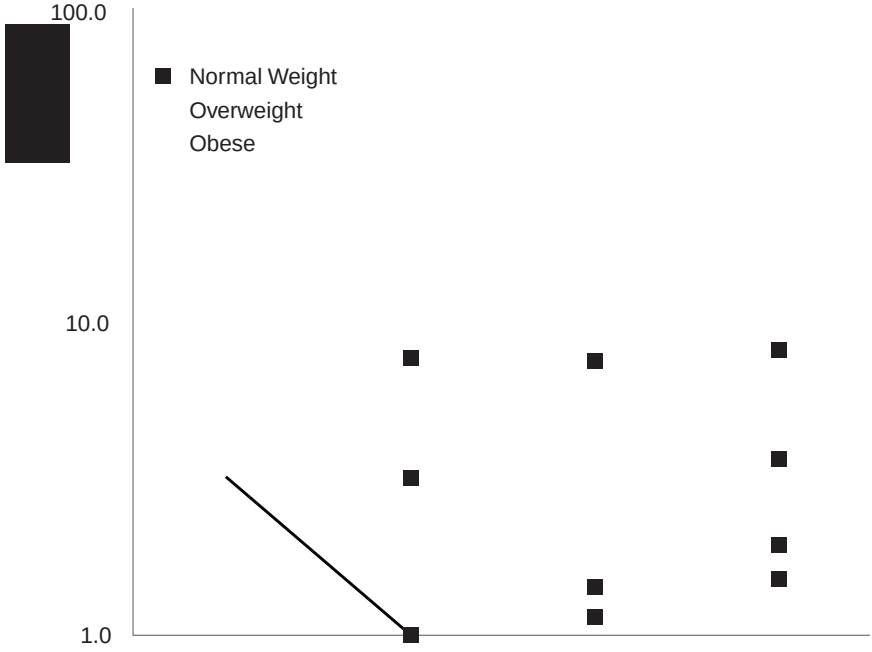

FIGURE G-3A Gestational diabetes.
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE G-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

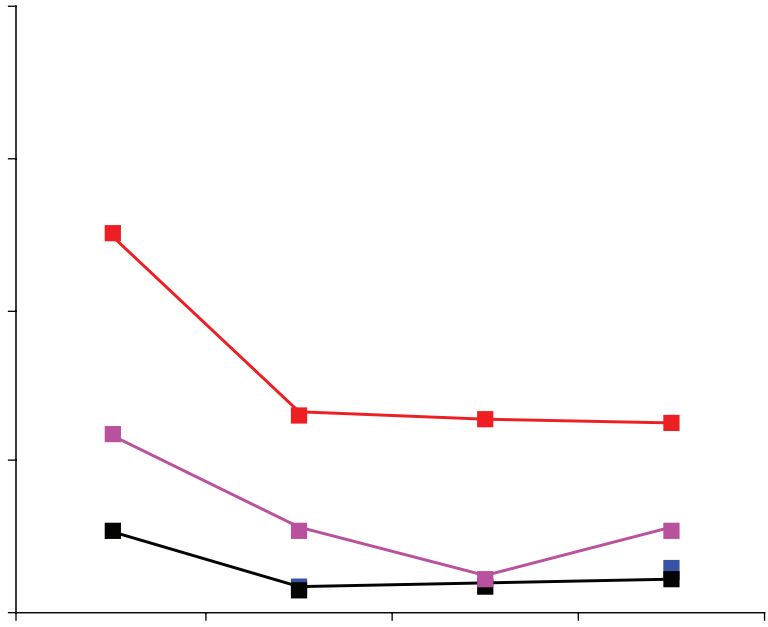

FIGURE G-3B Gestational diabetes.
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

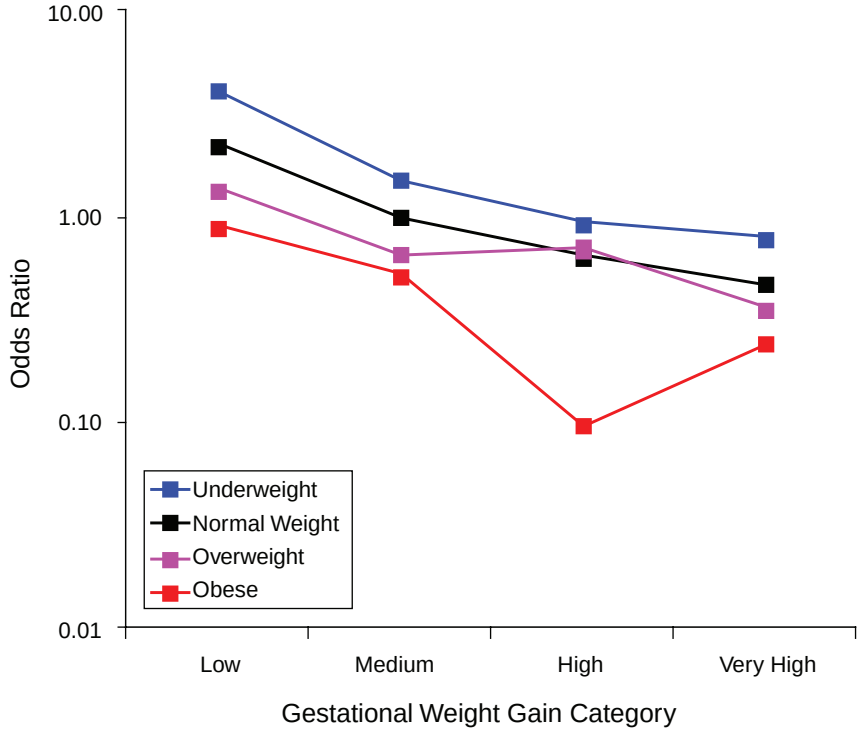

FIGURE G-4A Small-for-gestational-age infant (< 2.5 percent).
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE G-4A Small-for-Gestational-Age Infant (< 2.5 percent), Adjusted Odds Ratios (gestational weight gain by BMI)

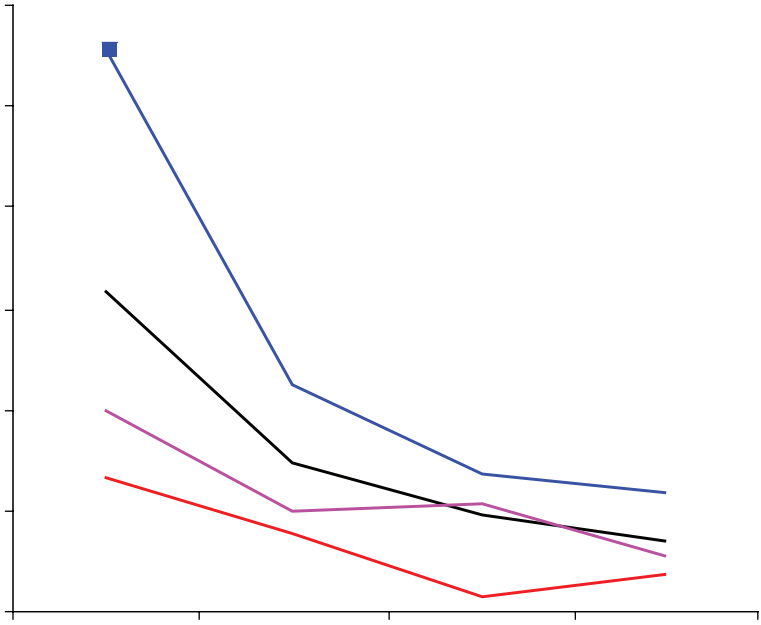

FIGURE G-4B Small-for-gestational-age infant (< 2.5 percent).
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

FIGURE G-5A

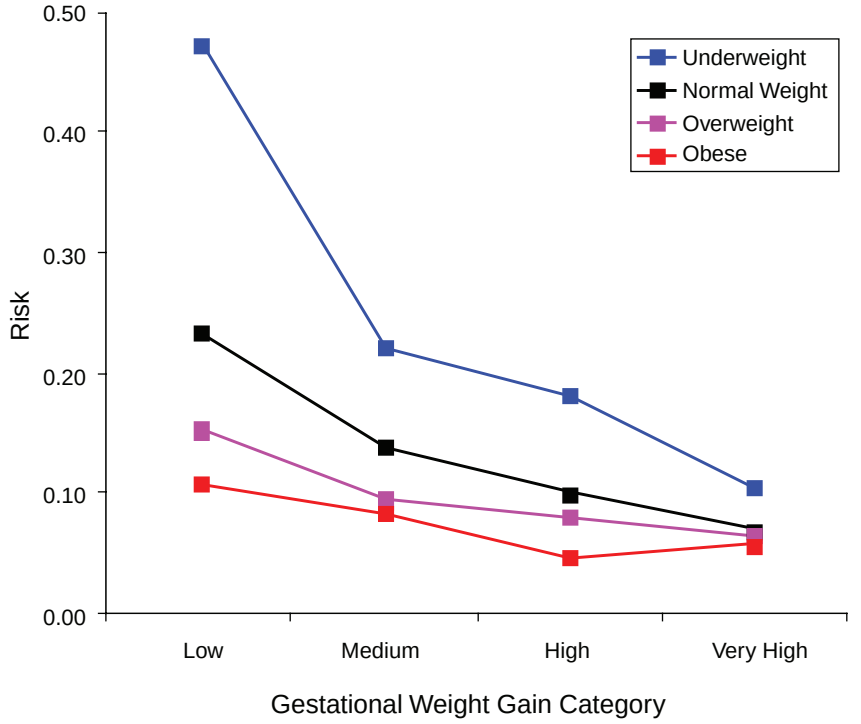

FIGURE G-5B Small-for-gestational-age infant (< 10 percent).
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE G-5B

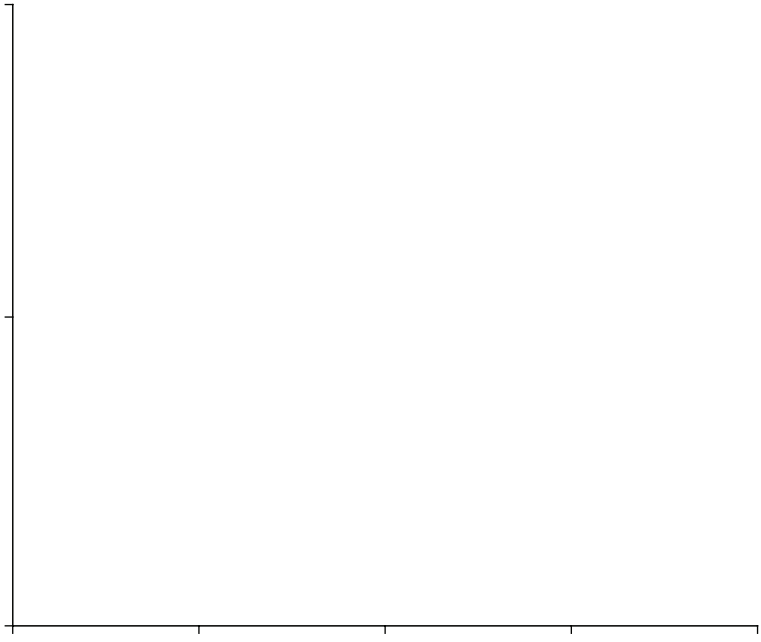

FIGURE G-6A Large-for-gestational-age infant (> 90 percent).
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

FIGURE G-6B Large-for-gestational-age infant (> 90 percent).
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous

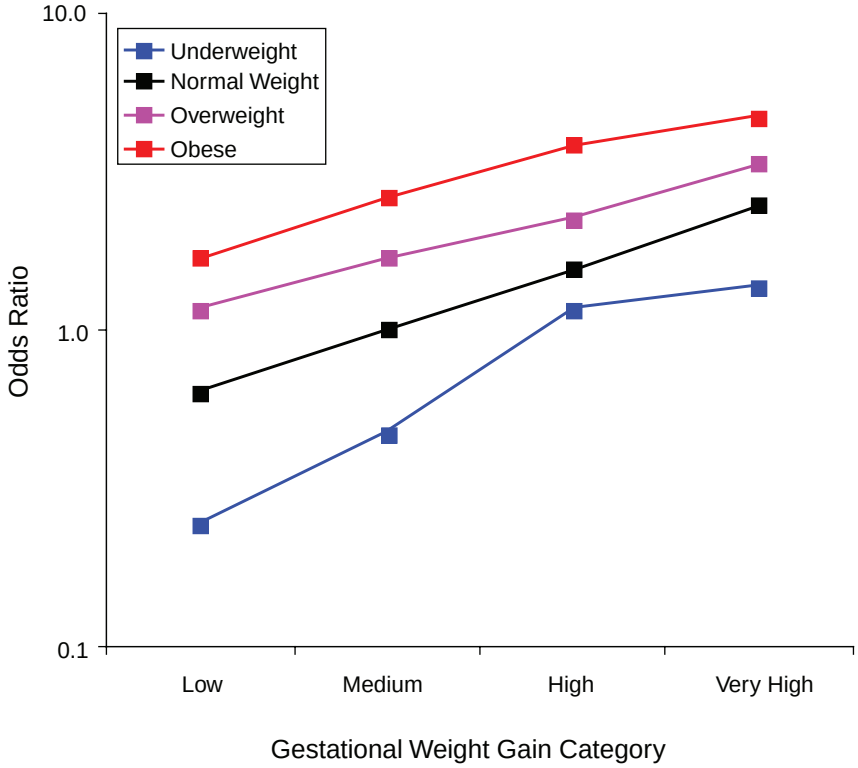

FIGURE G-7A Birth weight > 4,000 g.
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE G-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

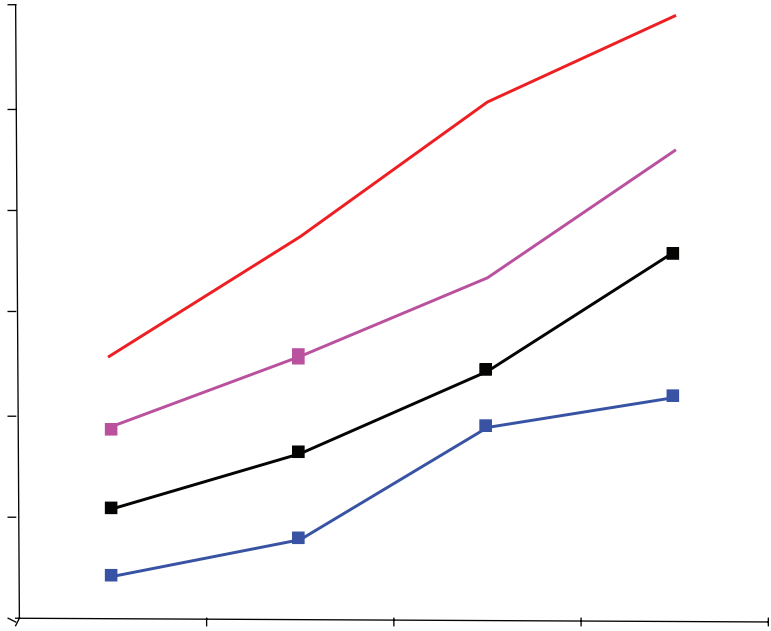

FIGURE G-7B Birth weight > 4,000 g.
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE G-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

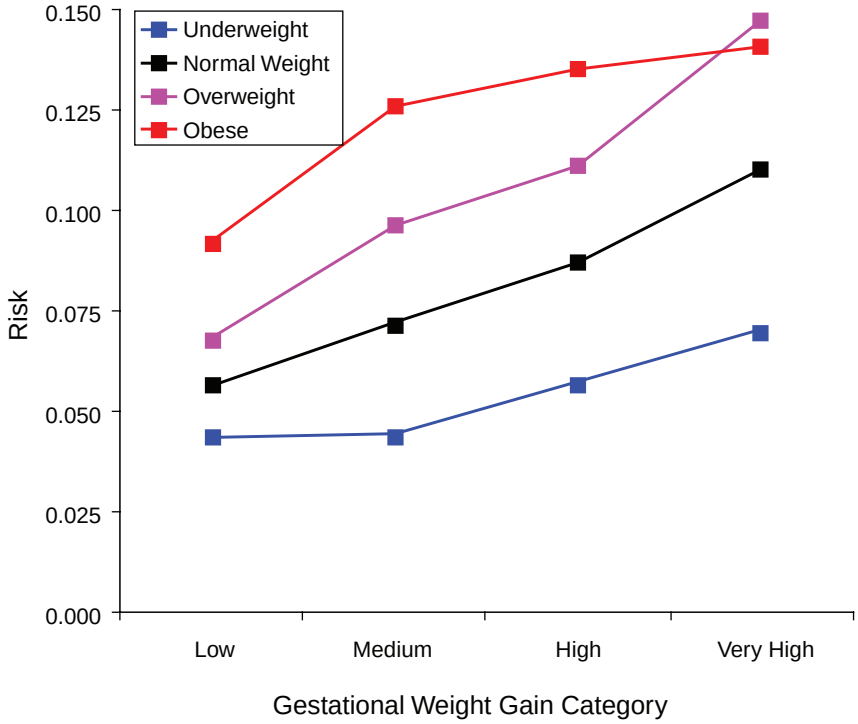

FIGURE G-8B High Ponderal Index (> 90 percent).
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE G-8B High Ponderal Index (> 90 percent), Adjusted Risks (gestational weight gain by BMI)

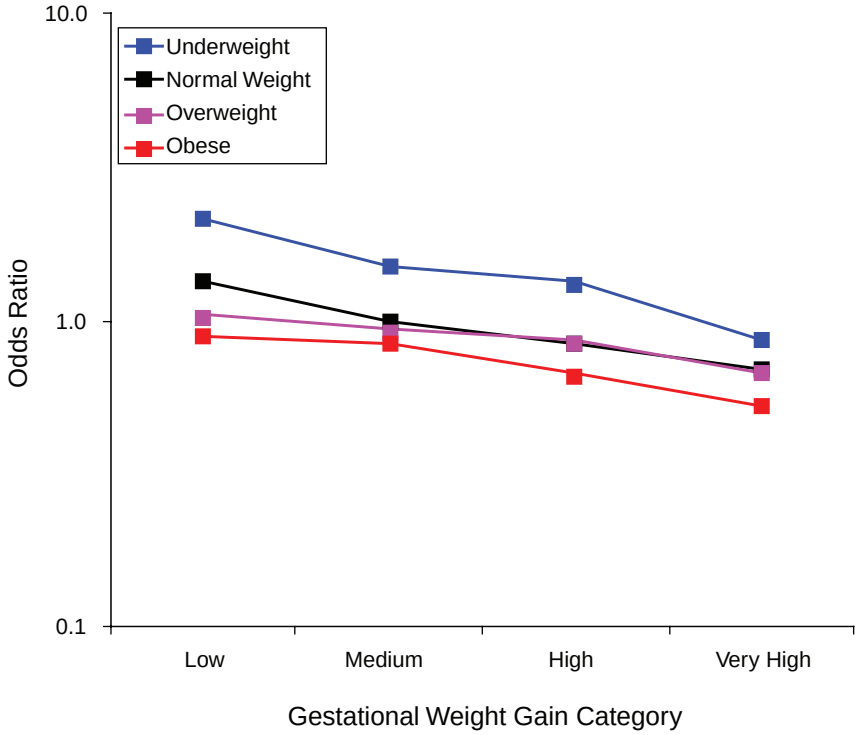

FIGURE G-9A Low Ponderal Index (< 10 percent).
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE G-9A Low Ponderal Index (<

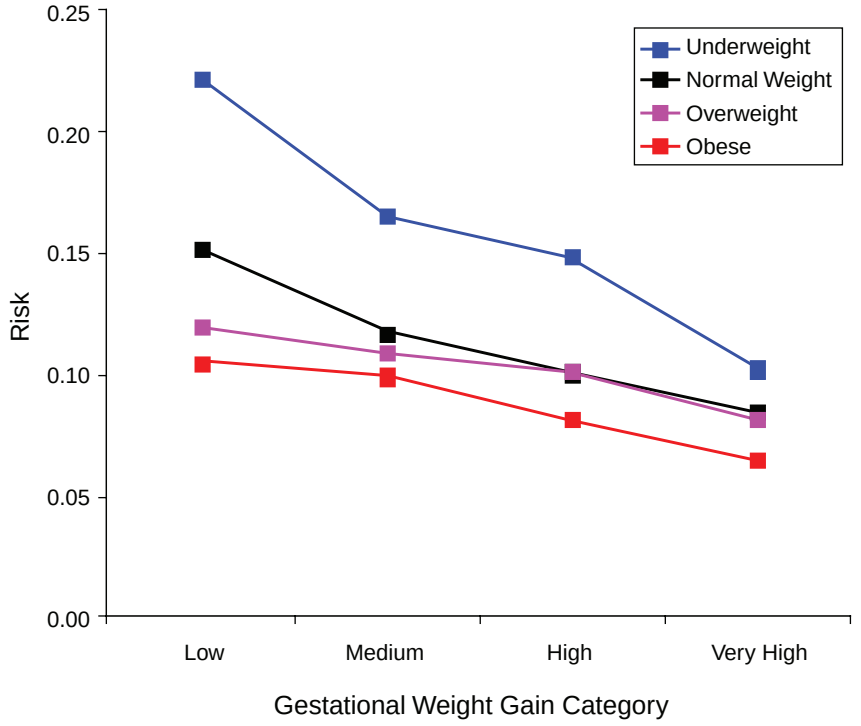

FIGURE G-9B Low Ponderal Index (< 10 percent).
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE G-9B

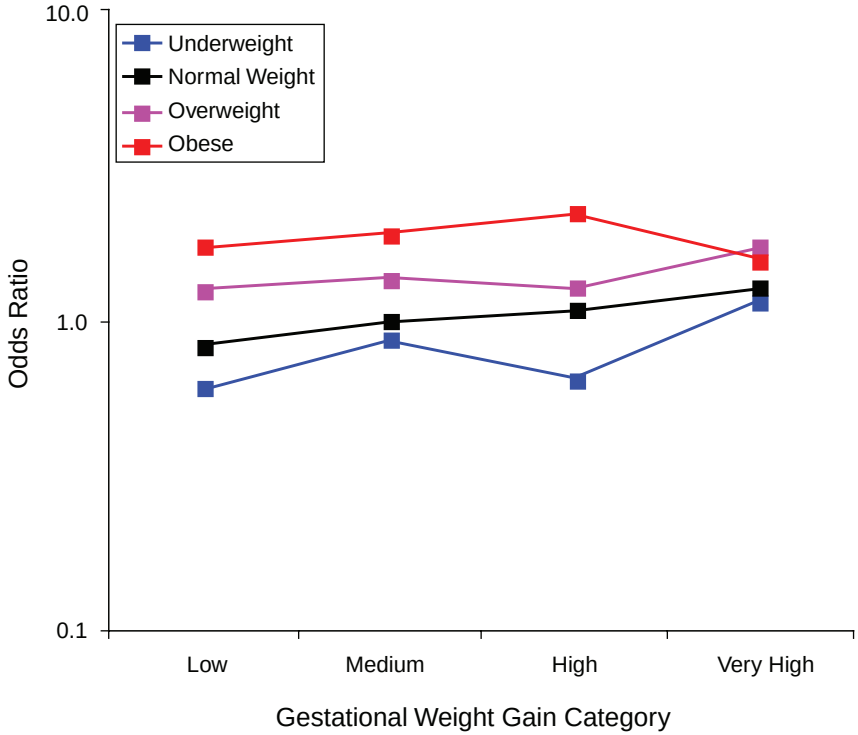

FIGURE G-10A Cesarean delivery before labor.
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE G-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

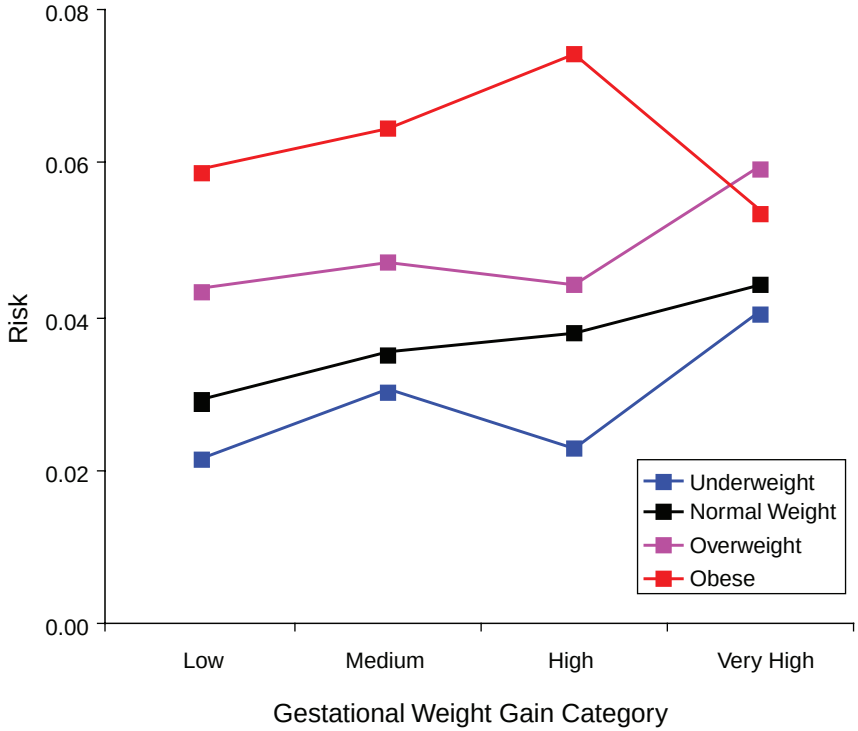

FIGURE G-10B Cesarean delivery before labor.
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE G-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%

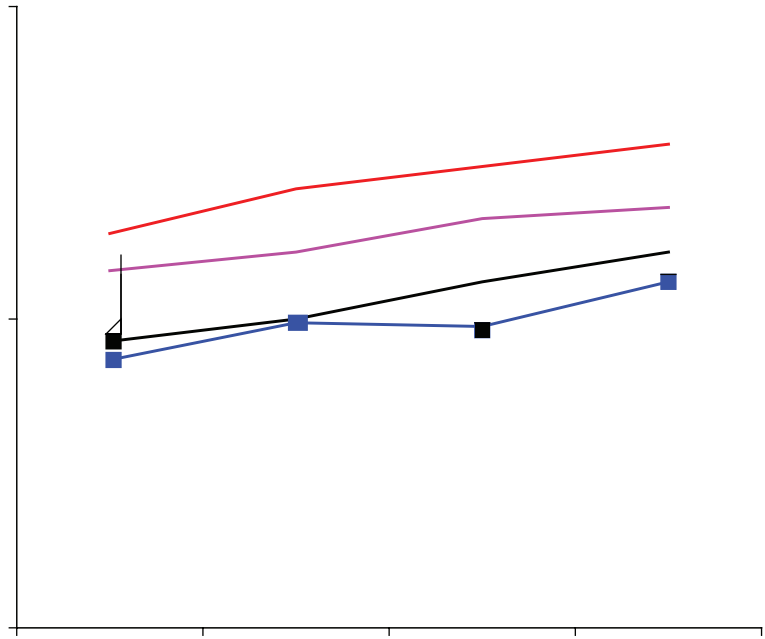

FIGURE G-11A Cesarean delivery during labor.
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

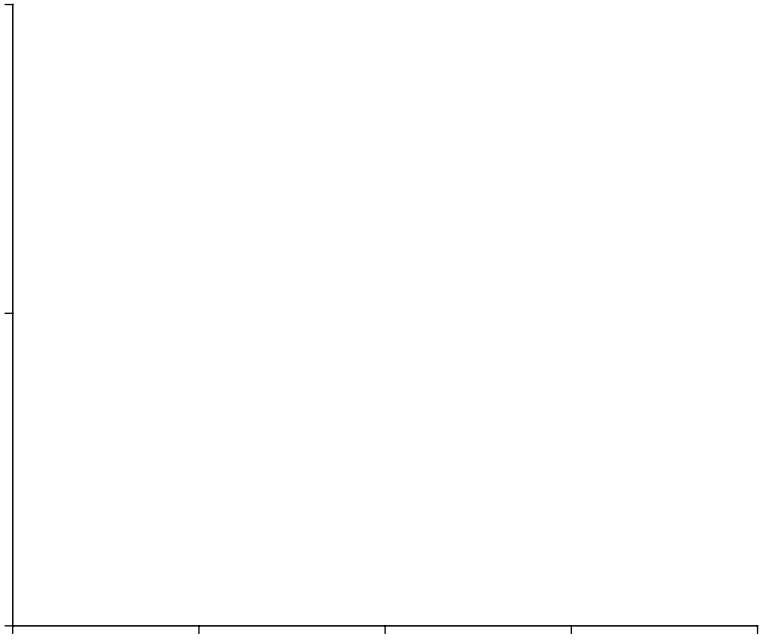

FIGURE G-12A Instrumental deliveries.
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

FIGURE G-12B Instrumental deliveries.

TABLE G-13A Low Apgar Score (<

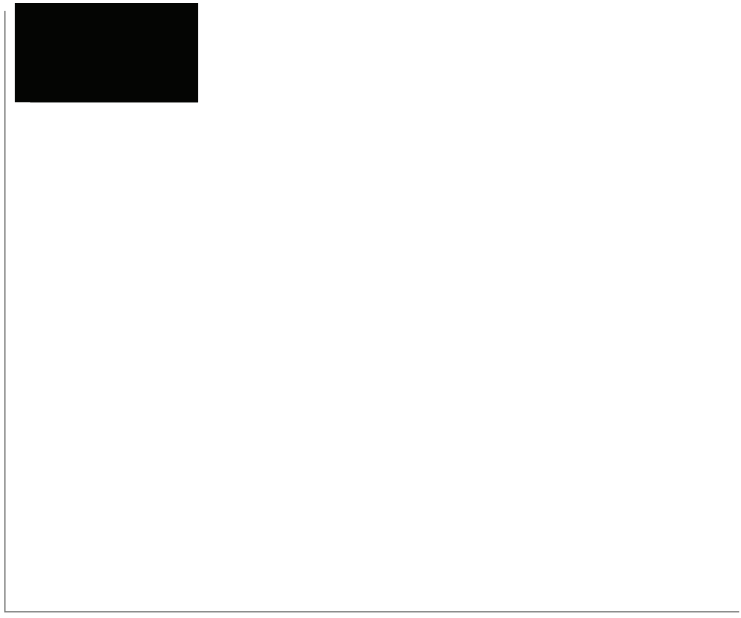

TABLE G-13B Low Apgar Score (< 8 after 5 minutes), Adjusted Risks (gestational weight gain by BMI)

	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%

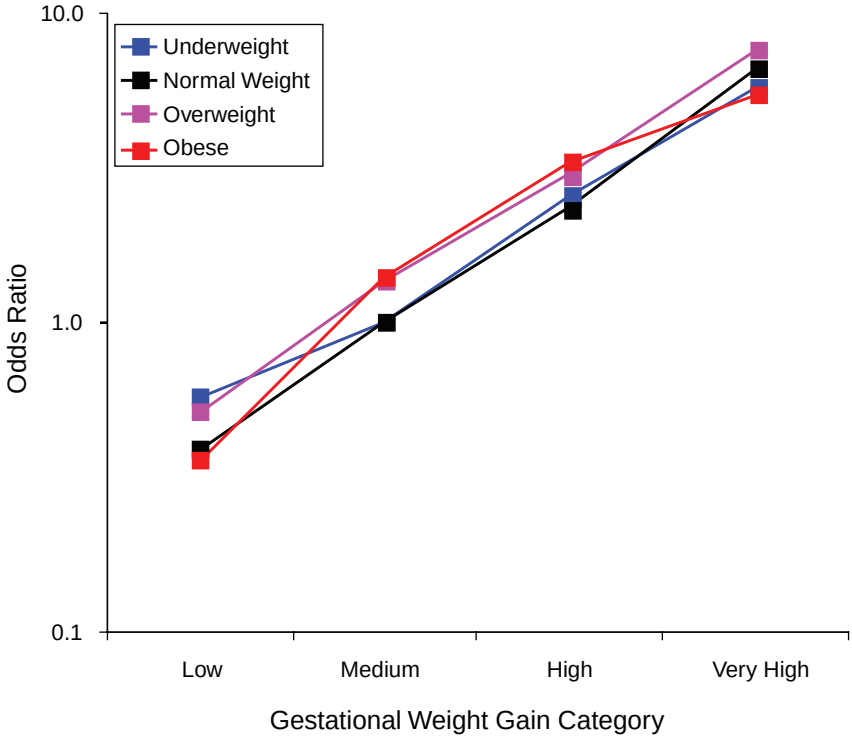

FIGURE G-14A Post partum weight retention ≥ 5 kg at 6 months.
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

TABLE G-14A Post Partum Weight Retention ≥ 5 kg at 6 Months, Adx31(R)-3(ee)-3(t)-3(e)-3d6 ee Ratio kgeetatioa (i)-3(g)-3(h)-3(t)-3( )-31gtan BM

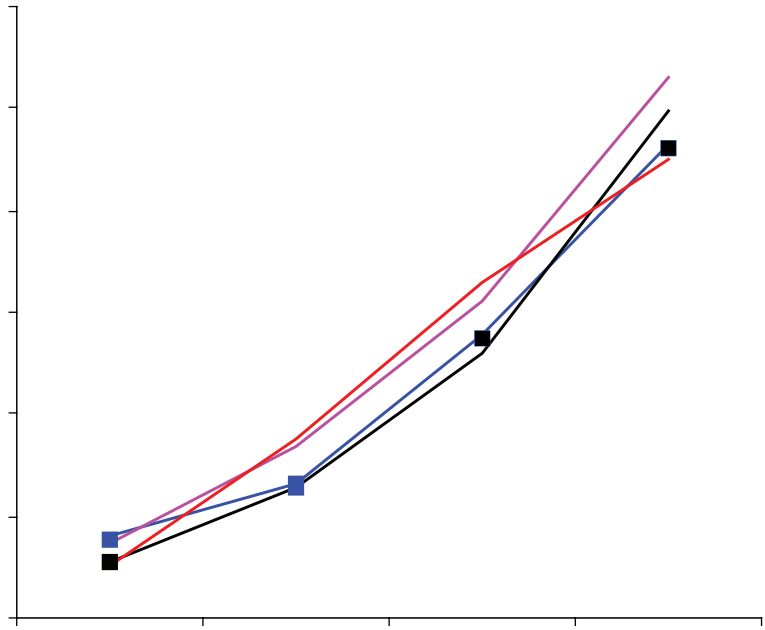

FIGURE G-14B Post partum weight retention ≥ 5 kg at 6 months.
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

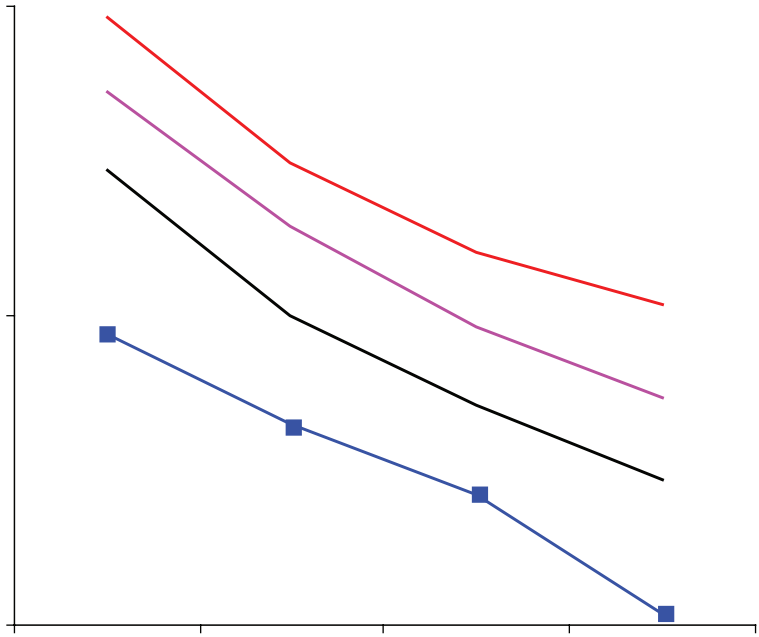

FIGURE G-15A Post partum weight loss ≥ 2 kg at 6 months.
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

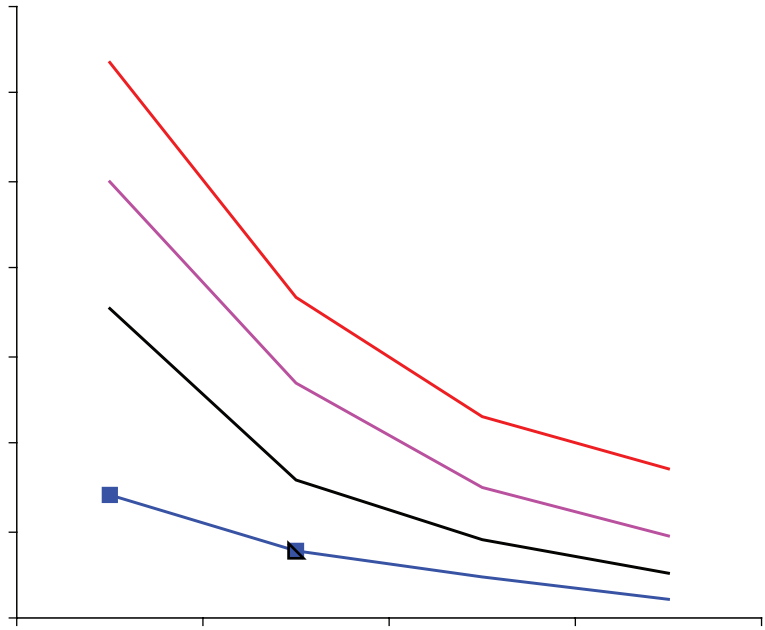

FIGURE G-15B Post partum weight loss ≥ 2 kg at 6 months.
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

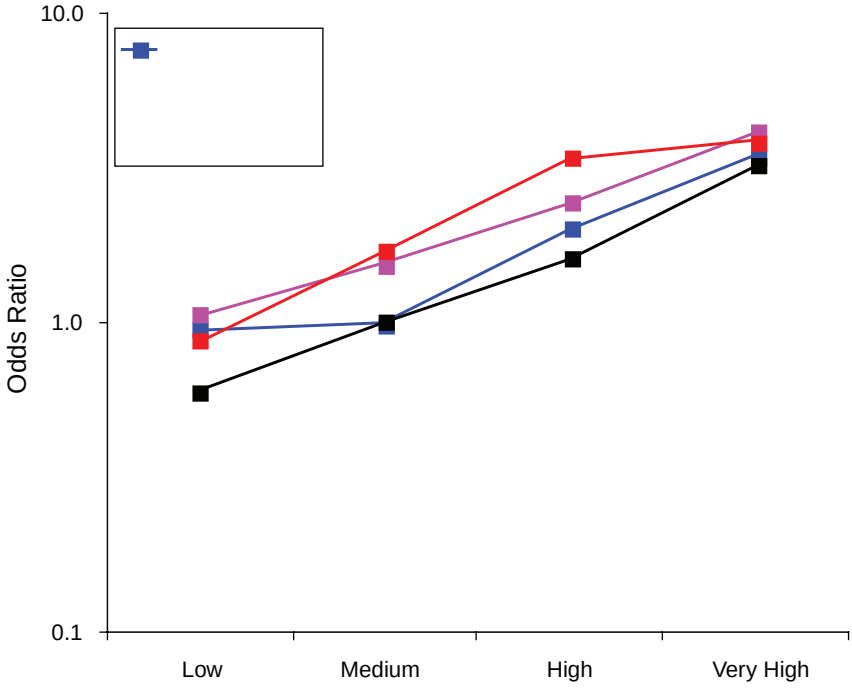

FIGURE G-16A Post partum weight retention ≥ 5 kg at 18 months.
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

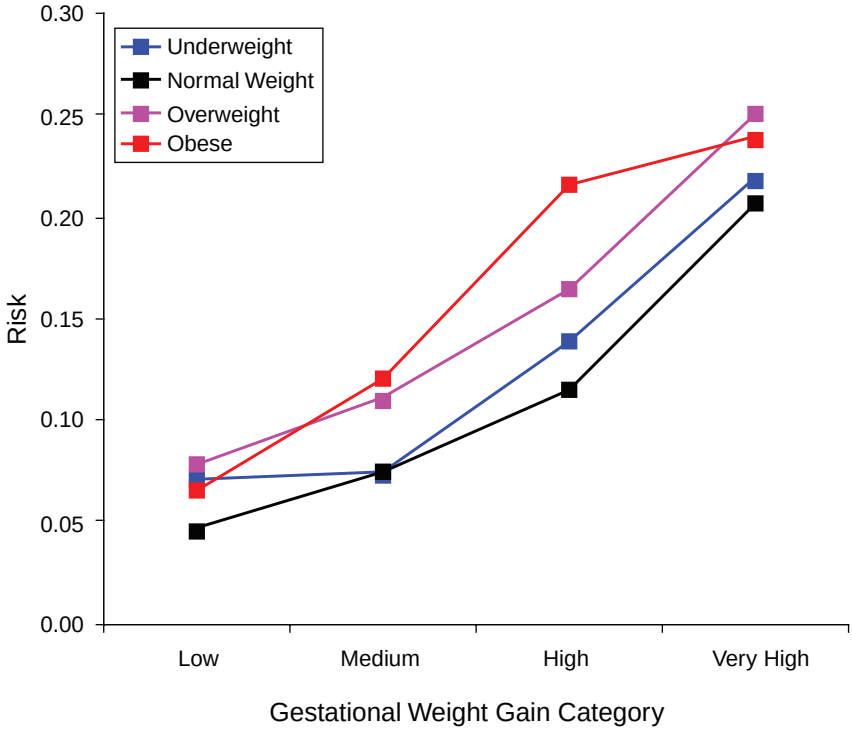

FIGURE G-16B Post partum weight retention ≥ 5 kg at 18 months.
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE G-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%

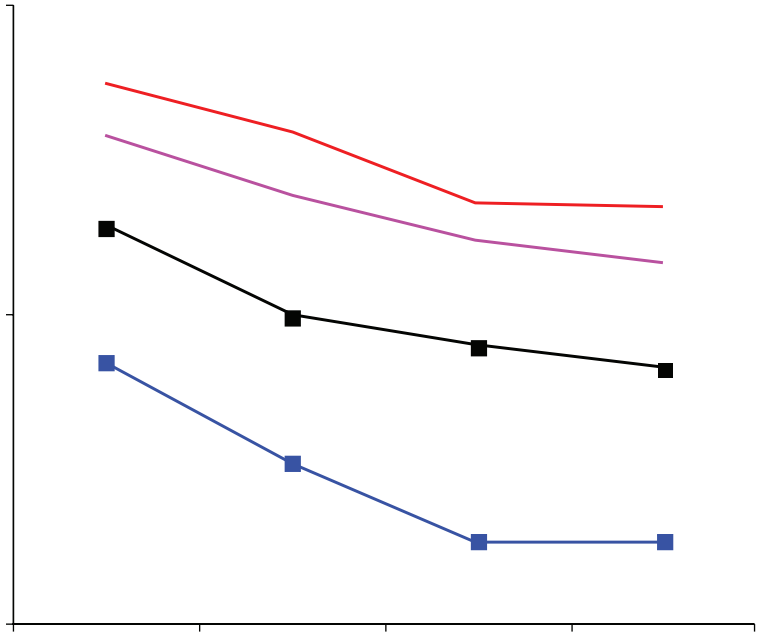

FIGURE G-17A Post partum weight loss ≥ 2 kg at 18 months.
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, social status, exercise, gestational age (days).

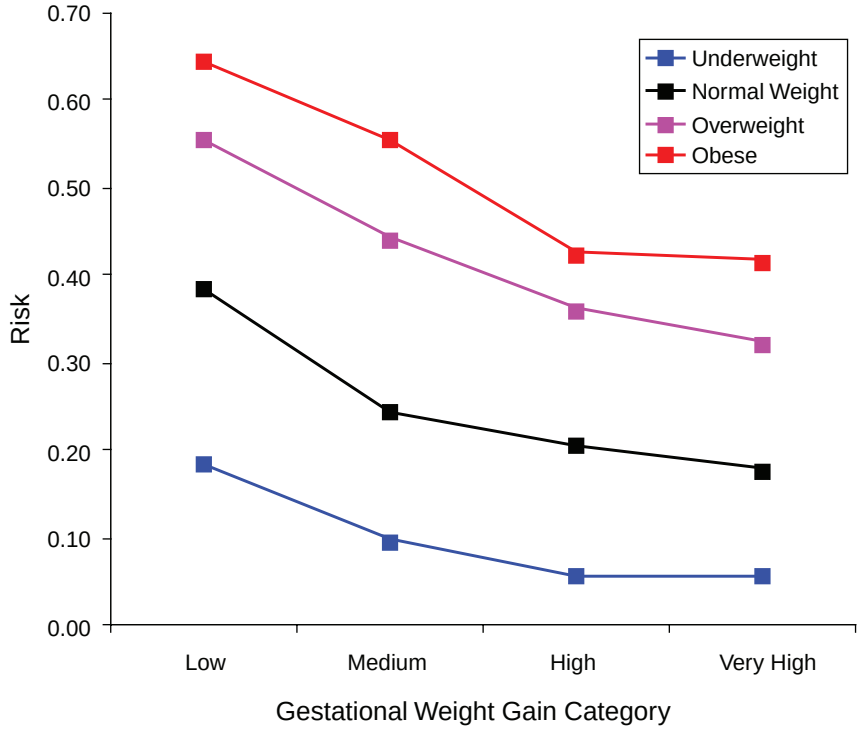

FIGURE G-17B Post partum weight loss ≥ 2 kg at 18 months.
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

TABLE G-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%

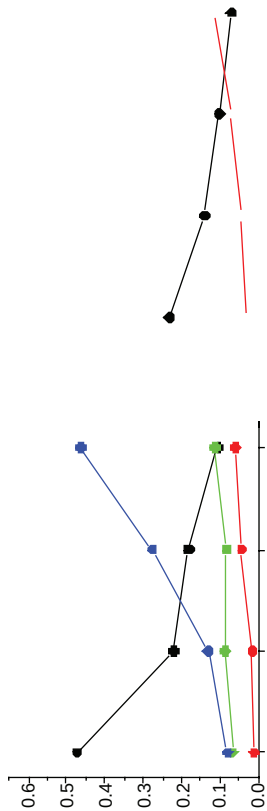

FIGURE G-18 GWG-specific absolute risks for SGA, LGA, emergency cesarean delivery and postpartum weight retention within each group.
NOTE: Points present risks of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise and 280 days of gestation. For PPWR, she breastfed < 14 weeks.

TABLE G-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: CD = cesarean delivery; LGA = large-for-gestational age; PPWR = postpartum weight retention; SGA = small-for-gestational age.

TABLE G-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: CD = cesarean delivery; LGA = large-for-gestational age; PPWR = postpartum weight retention; SGA = small-for-gestational age.

TABLE G-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: CD = cesarean delivery; LGA = large-for-gestational age; PPWR = postpartum weight retention; SGA = small-for-gestational age.

TABLE G-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: CD = cesarean delivery; LGA = large-for-gestational age; PPWR = postpartum weight retention; SGA = small-for-gestational age.

Second DNBC Report

At the IOM workshop in Washington, DC, in June 2003, the IOM committee found the additive approach with presentation of absolute a

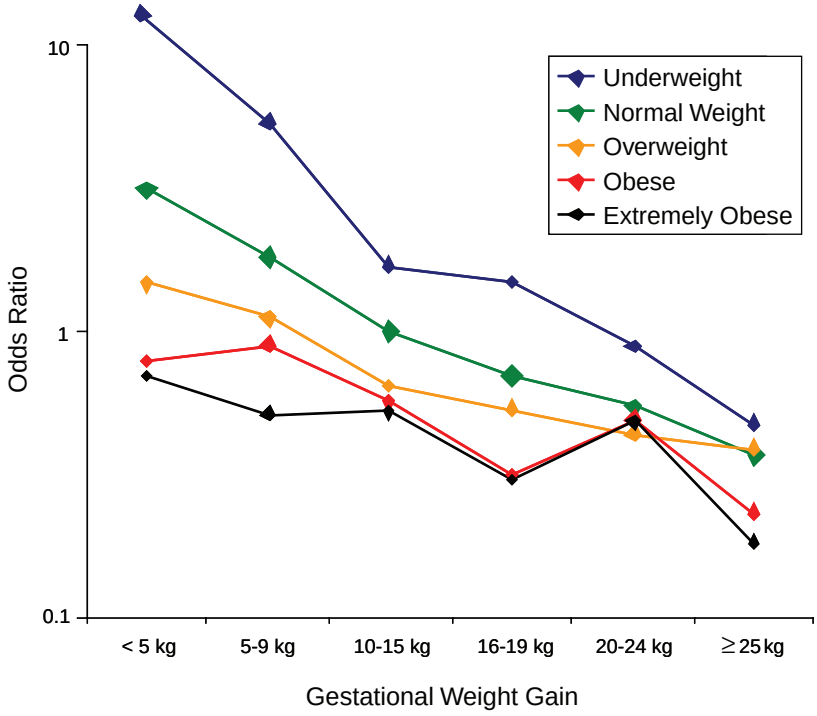

FIGURE G-19A Small-for-gestational-age infant (< 10 percentile).
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, exercise, social status, gestational age in days (p = 0.0001 [Wald's test]).

TABLE G-19A Small-for-Gestational-Age Infant, 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 (3.9; 39.8)	5.4 (3.9; 7.5)	1.7 (1.4; 2.0)	1.5 (1.1; 1.9)	0.9 (0.6; 1.3)	0.5 (0.2; 0.9)
18.5-24.9	3.1 (2.2; 4.5)	1.8 (1.6; 2.0)	1.0 (ref)	0.7 (0.6; 0.8)	0.5 (0.5; 0.6)	0.4 (0.3; 0.4)
25.0-29.9	1.5 (1.0; 2.0)	1.1 (0.9; 1.3)	0.6 (0.6; 0.7)	0.5 (0.4; 0.7)	0.4 (0.3; 0.6)	0.4 (0.3; 0.5)
30-34.9	0.8 (0.5; 1.1)	0.9 (0.7; 1.2)				
		(0.4; 0.7)	0.5 (0.3; 0.9)			
		(0.2; 0.6)	(1.3; 0.5)			

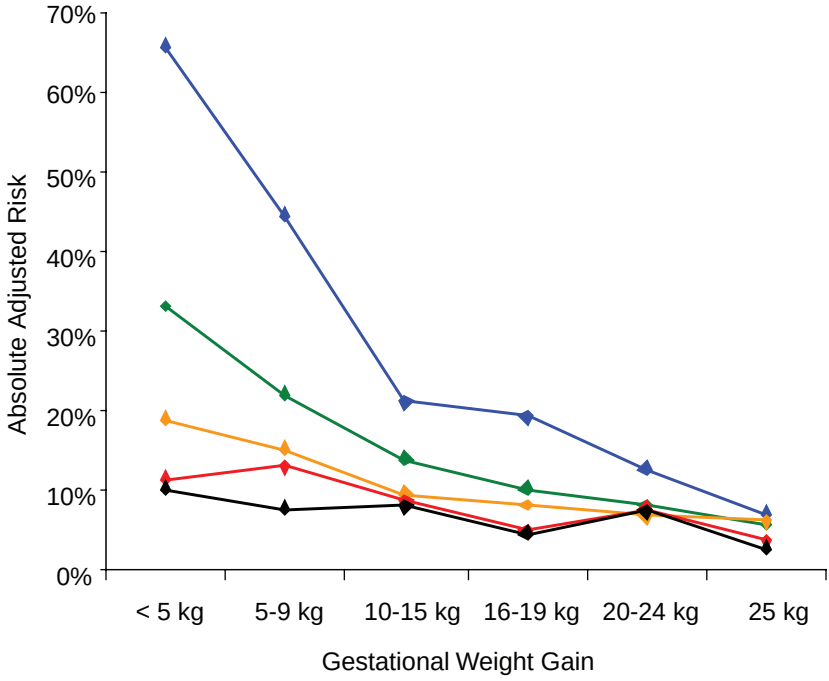

FIGURE G-19B Small-for-gestational-age infant (< 10 percentile).
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

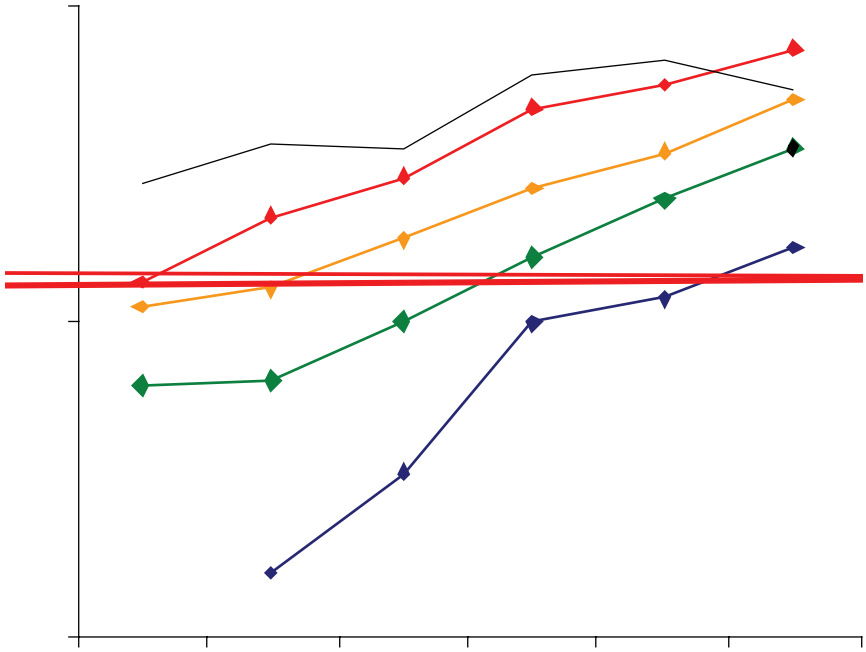

FIGURE G-20A Large-for-gestational-age infant (> 90 percentile).
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, exercise, social status, gestational age in days (p = 0.0001 [Wald's test]).

FIGURE G-20B Large-for-gestational-age infant (> 90 percentile).
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high

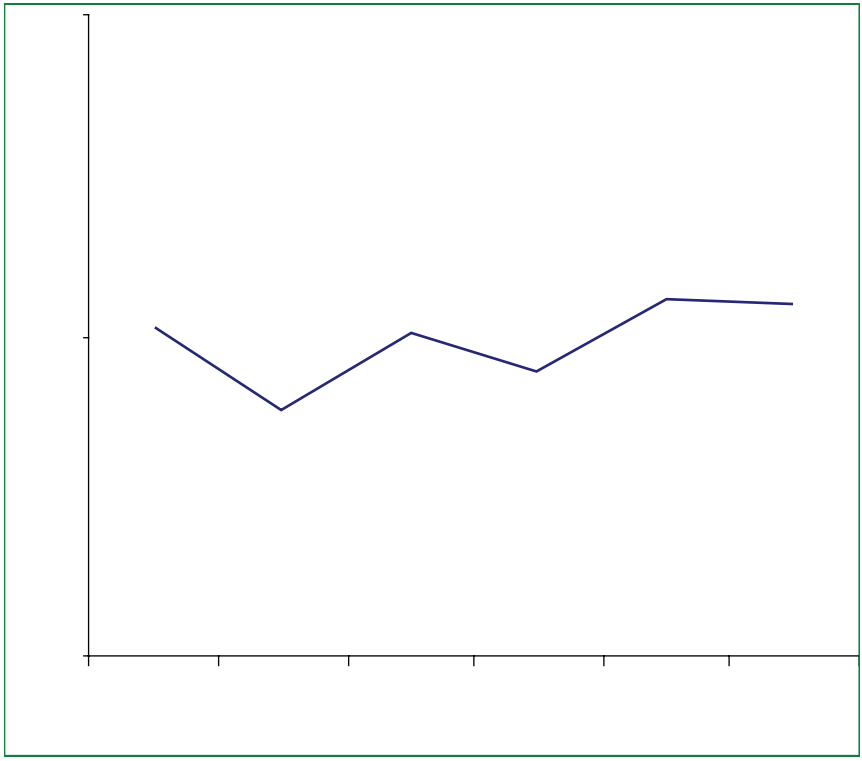

FIGURE G-21A Emergency cesarean deliveries.
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, exercise, social status, gestational age in days ($p = 0.23$ [Wald's test]).

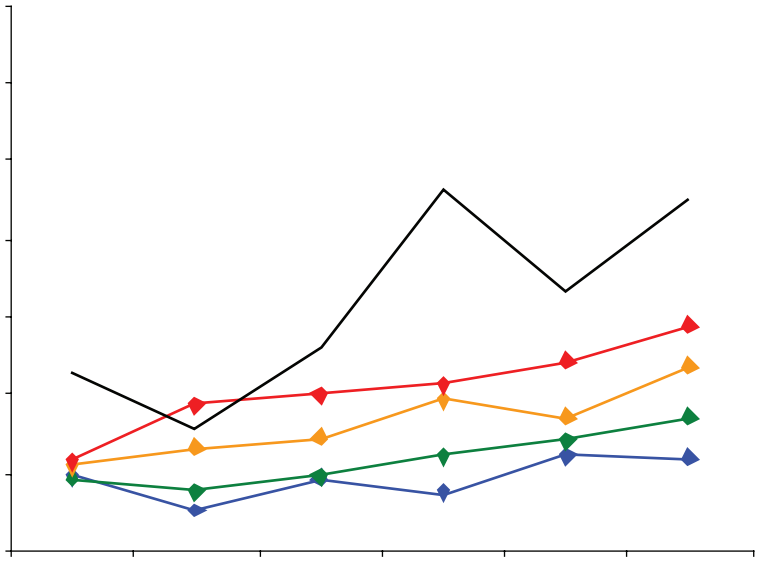

FIGURE G-21B Emergency cesarean deliveries.
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

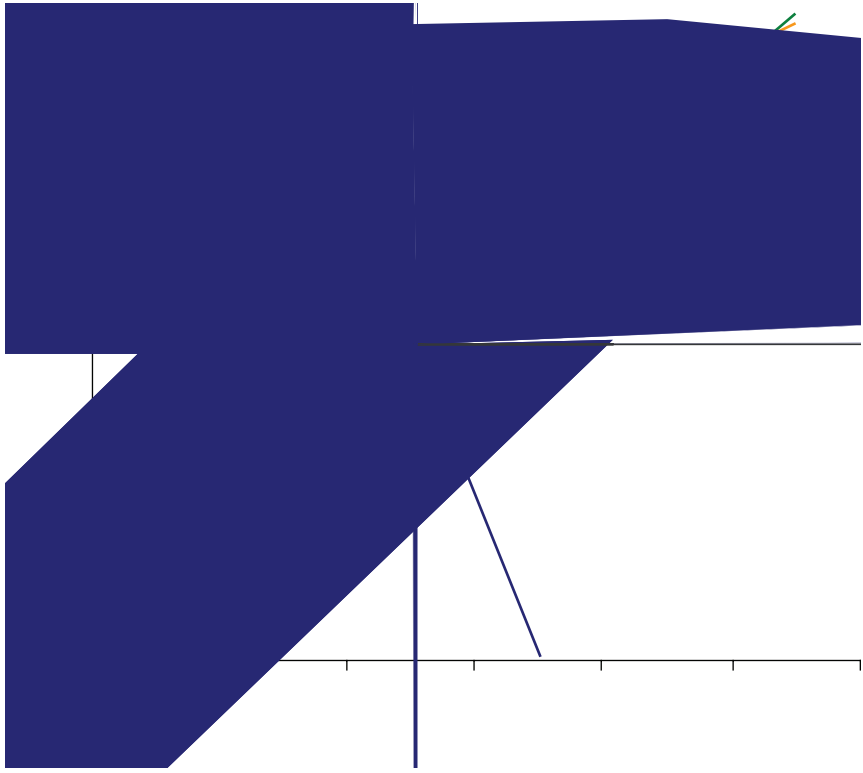

FIGURE G-22A Postpartum weight retention ≥ 5 kg at 6 months.
NOTE: Full model. Odds ratios adjusted for age, parity, height, smoking, alcohol consumption, exercise, social status, gestational age in days (p = 0.001 [Wald's test]).

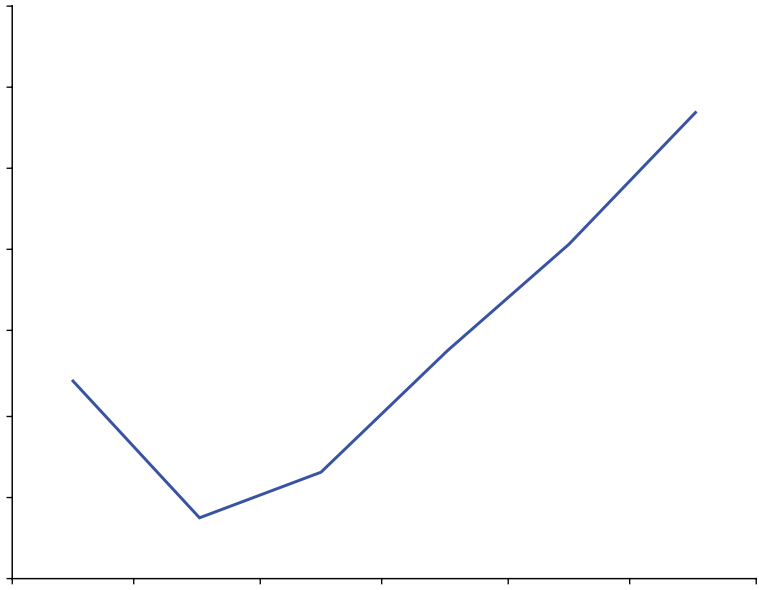

FIGURE G-22B Postpartum weight retention ≥ 5 kg at 6 months.
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, nonsmoker, no alcohol consumption, high social status, no exercise, 280 days of gestation.

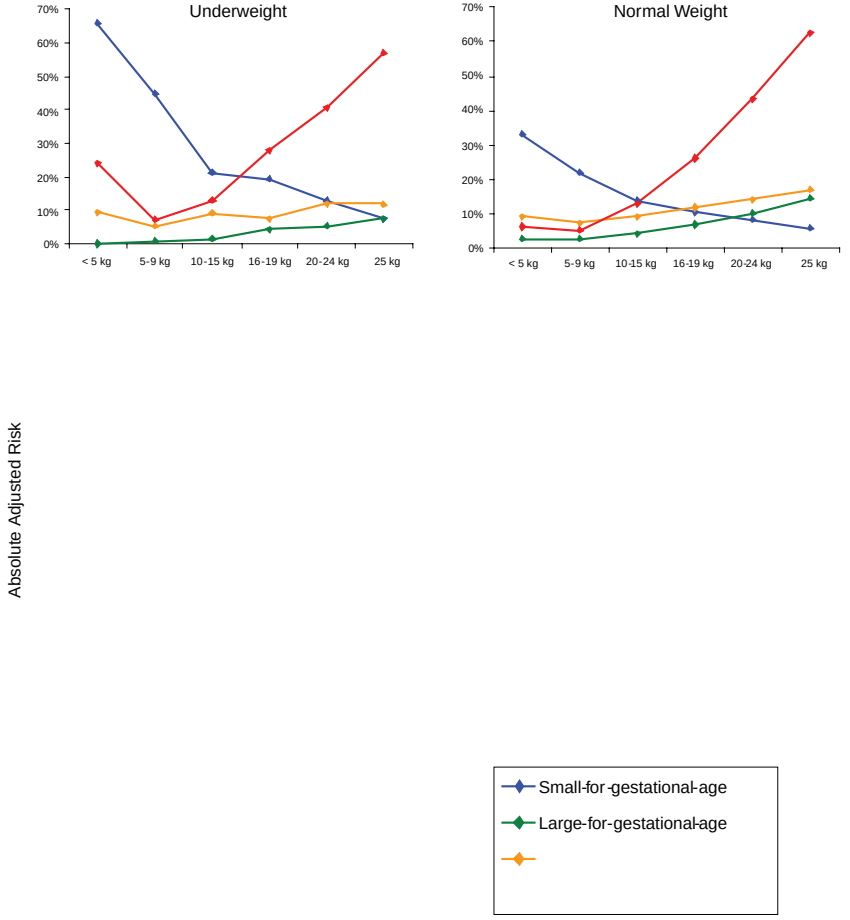

FIGURE G-23 GWG-specific absolute risks for SGA, LGA, emergency cesarean delivery and postpartum weight retention within each BMI group.
NOTE: Absolute risks derived from odds ratios. Presents risk of a primiparous woman, age 25-29, height 1.60-1.69, non smoker, no alcohol consumption, high social status, no exercise, 280 days of gestation. For PPWR, she is breastfeeding less than 14 weeks.

- 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

tion, 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 seven 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:

- Figure G-24 (Table G-23): Unexposed primiparae, GWG-specific risks of pregnancy outcomes

FIGURE G-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.

TABLE G-23 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 (0.36; 0.56)	0.20 (0.17; 0.24)	0.14 (0.10; 0.19)	0.11 (0.08; 0.17)	0.06 (0.03; 0.11)
LGA		0.02 (0.00; 0.07)	0.01 (0.01; 0.03)	0.06 (0.04; 0.10)	0.04 (0.02; 0.08)	0.08 (0.04; 0.16)
Emergency CS		0.09 (0.04; 0.18)	0.08 (0.05; 0.10)	0.09 (0.06; 0.13)	0.11 (0.07; 0.17)	0.12 (0.06; 0.21)
PPWR		0.04 (0.02; 0.10)	0.09 (0.07; 0.12)	0.21 (0.17; 0.26)	0.32 (0.26; 0.40)	0.48 (0.38; 0.59)
Normal weight						

SGA hl hhh.1.SBT/Span <</MCID 50 ET/Span63(30-3(;)-3(;)-3(;)-3(;)-3(;8-3(;)-33)-3(0)-3(4)]TJEMC /Span <</MCID 50195 >>BDC T23[(()-3(0)-3(.)-3(0)-3(0)-

	< 5 kg	< 10 kg	10-15 kg	16-19 kg	20-24 kg	≥ 25 kg
Overweight						
SGA	0.16 (0.11; 0.24)	0.16 (0.13; 0.20)	0.10 (0.08; 0.11)			

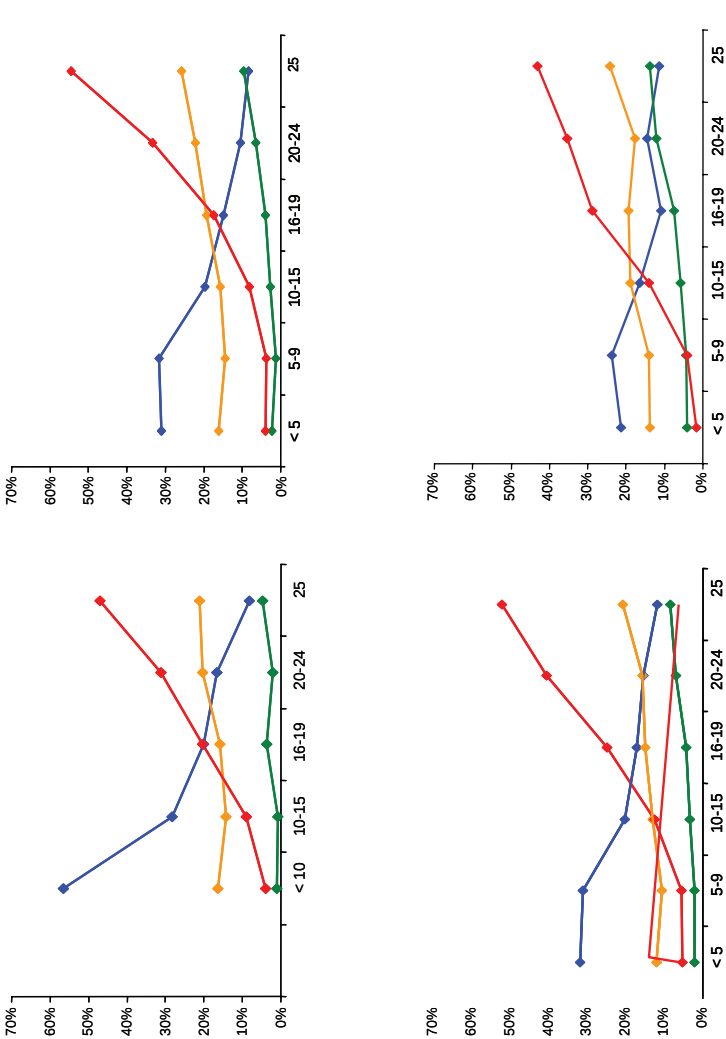

FIGURE G-25 Short primiparae, GWG–specific risks of pregnancy outcomes.
NOTE: Age 25-29, height < 160 cm, high social status. In pregnancy: no smoking, no alcohol, moderate exercise. For PPWR, she breastfed > 14 weeks.

TABLE G-24

TABLE G-25 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 (0.57; 0.76)	0.38 (0.32; 0.44)	0.28 (0.22; 036)	0.24 (0.17; 0.32)	0.12 (0.06; 0.23)
LGA		0.01 (0.00; 0.03)	0.01 (0.00; 0.01)	0.03 (0.02; 0.04)	0.02 (0.01; 0.03)	0.03 (0.01; 0.07)
Emergency CS		0.08 (0.04; 0.17)	0.07 (0.05; 0.10)	0.08 (0.05; 0.12)	0.11 (0.07; 0.17)	0.11 (0.06; 0.20)
PPWR		0.04 (0.02; 0.10)	0.10 (0.07; 0.12)	0.21 (0.17; 0.27)	0.33 (0.26; 0.40)	0.49 (0.38; 0.59)
Normal weight						
SGA	0.41 (0.29; 0.55)	0.42 (0.38; 0.46)	0.28 (0.25; 0.30)	0.21 (0.19; 0.24)	0.15 (0.13; 0.18)	0.13 (0.10; 0.15)
LGA	0.02 (0.00; 0.05)	0.01 (0.01; 0.01)	0.02 (0.01; 0.02)	0.03 (0.02; 0.04)	0.05 (0.04; 0.06)	0.07 (0.06; 0.09)
Emergency CS	0.08 (0.03; 0.18)	0.07 (0.06; 0.09)	0.08 (0.07; 0.09)	0.10 (0.09; 0.12)	0.11 (0.10; 0.13)	0.14 (0.11; 0.16)
PPWR	0.04 (0.02; 0.11)	0.04 (0.03; 0.05)	0.09 (0.08; 0.10)	0.18 (0.17; 0.21)	0.35 (0.32; 0.87)	0.56 (0.53; 0.60)

continued

	< 5 kg	< 10 kg	10-15 kg	16-19 kg	20-24 kg	≥ 25 kg
Overweight						
SGA	0.32 (0.31; 0.33)	0.23 (0.26; 0.37)	0.20 (0.17; 0.23)	0.17 (0.14; 0.21)	0.15 (0.12; 0.19)	0.12 (0.09; 0.16)
LGA	0.02 (0.01; 0.05)	0.02 (0.01; 0.03)	0.03 (0.03; 0.04)	0.04 (0.03; 0.06)	0.07 (0.05; 0.09)	0.08 (0.06; 0.11)
Emergency CS	0.12 (0.07; 0.20)	0.11 (0.08; 0.14)	0.13 (0.11; 0.15)	0.15 (0.12; 0.18)	0.16 (0.13; 0.19)	0.20 (0.16; 0.25)
PPWR	0.05 (0.03; 0.10)	0.06 (0.04; 0.08)	0.12 (0.11; 0.14)	0.25 (0.21; 0.2 (0.54; 0.0	0.16 (0.13; 0.04)	0.1 (0.12; 0.1
	0.12					

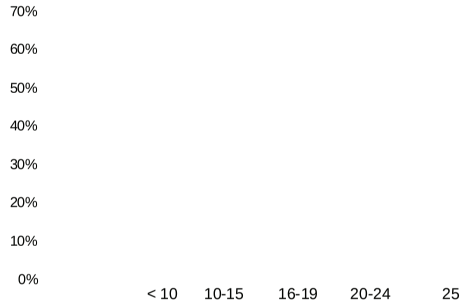

FIGURE G-27 Young primiparae, GWG–specific risks of pregnancy outcomes.
NOTE: Age < 20, height 160-169 cm, high social status. In pregnancy: no smoking, no alcohol, moderate exercise. For PPWR, she breastfed > 14 weeks.

TABLE G-26 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 (0.22; 0.46)	0.13 (0.09; 0.18)	0.09 (0.06; 0.14)	0.07 (0.04; 0.12)	0.03 (0.01; 0.07)
LGA		0.01 (0.00; 0.06)	0.01 (0.00; 0.02)	0.05 (0.03; 0.09)	0.03 (0.01; 0.07)	0.06 (0.03; 0.14)
Emergency CS		0.06 (0.03; 0.14)	0.05 (0.03; 0.09)	0.06 (0.03; 0.11)	0.08 (0.05; 0.14)	0.09 (0.04; 0.17)
PPWR		0.06 (0.02; 0.13)	0.12 (0.09; 0.17)	0.27 (0.20; 0.35)	0.39 (0.30; 0.49)	0.56 (0.44; 0.67)
Normal weight						
SGA	0.15 (0.08; 0.24)	0.15 (0.11; 0.21)	0.09 (0.06; 0.12)	0.06 (0.04; 0.09)	0.04 (0.03; 0.06)	0.03 (0.02; 0.05)
LGA	0.03 (0.01; 0.10)	0.02 (0.01; 0.03)	0.03 (0.02; 0.05)	0.05 (0.03; 0.08)	0.08 (0.05; 0.13)	0.12 (0.08; 0.19)
Emergency CS	0.06 (0.02; 0.15)	0.06 (0.04; 0.89)	0.06 (0.04; 0.09)	0.08 (0.05; 0.11)	0.09 (0.06; 0.13)	0.11 (0.07; 0.15)
PPWR	0.05 (0.02; 0.14)	0.05 (0.04; 0.07)	0.11 (0.09; 0.14)	0.23 (0.19; 0.28)	0.41 (0.35; 0.48)	0.63 (0.56; 0.69)

Overweight							
SGA	0.10 (0.06; 0.17)	0.10 (0.07; 0.15)	0.06 (0.04; 0.08)	0.05 (0.03; 0.07)	0.04 (0.03; 0.07)	0.03 (0.02; 0.05)	
LGA	0.04 (0.02; 0.09)	0.04 (0.02; 0.07)	0.06 (0.04; 0.10)	0.08 (0.05; 0.12)	0.13 (0.08; 0.19)	0.15 (0.10; 0.23)	
Emergency CS	0.09 (0.05; 0.17)	0.08 (0.05; 0.13)	0.10 (0.07; 0.14)	0.12 (0.08; 0.17)	0.13 (0.09; 0.18)	0.16 (0.11; 0.23)	
PPWR	0.07 (0.03; 0.13)	0.07 (0.05; 0.11)	0.16 (0.12; 0.20)	0.30 (0.24; 0.37)	0.47 (0.40; 0.54)	0.59 (0.51; 0.66)	
Obese							
SGA	0.06 (0.04; 0.10)	0.07 (0.05; 0.11)	0.05 (0.03; 0.07)	0.03 (0.01; 0.05)	0.04 (0.02; 0.07)	0.03 (0.01; 0.06)	
LGA	0.08 (0.04; 0.13)	0.08 (0.05; 0.13)	0.11 (0.07; 0.17)				

70%
60%
50%
40%
30%
20%
10%
0%

FIGURE G-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.

TABLE G-27 Unexposed Multiparae, GWG–Specific Risks of Pregnancy Outcomes

	< 5 kg	< 10 kg	10-15 kg	16-19 kg	20-24 kg	≥ 25 kg
Underweight						

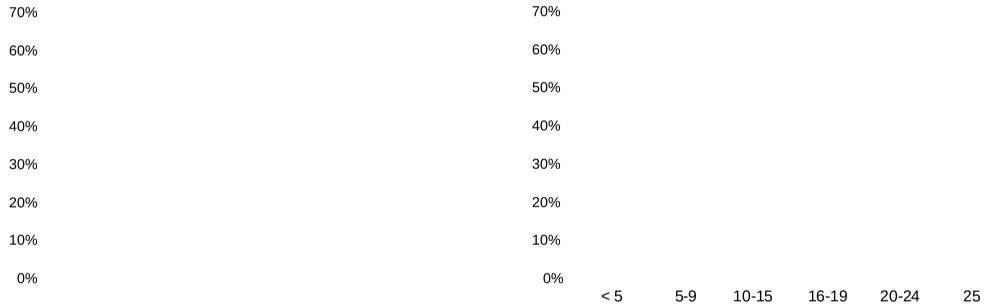

FIGURE G-29 Short multiparae, GWG–specific risks of pregnancy outcomes.
NOTE: Age 25-29, height < 160 cm, high social status. In pregnancy: no smoking, no alcohol, moderate exercise. For PPWR, she breastfed > 14 weeks.

TABLE G-28 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 (0.30; 0.52)	0.18 (0.14; 0.23)	0.18 (0.13; 0.24)	0.12 (0.07; 0.19)	0.07 (0.03; 0.17)
LGA		0.01 (0.00; 0.03)	0.02 (0.01; 0.03)	0.05 (0.03; 0.07)	0.07 (0.05; 0.11)	0.10 (0.05; 0.19)
Emergency CS		0.01 (0.00; 0.09)	0.06 (0.04; 0.09)	0.05 (0.02; 0.09)	0.06 (0.03; 0.14)	0.08 (0.03; 0.22)
PPWR		0.09 (0.05; 0.16)	0.13 (0.10; 0.16)	0.26 (0.20; 0.32)	0.39 (0.32; 0.48)	0.57 (0.45; 0.68)
Normal weight						
SGA	0.30 (0.21; 0.41)	0.17 (0.14; 0.21)	0.11 (0.09; 0.13)	0.07 (0.06; 0.09)	0.07 (0.05; 0.09)	0.04 (0.03; 0.06)
LGA	0.04 (0.02; 0.09)	0.04 (0.03; 0.05)	0.06 (0.05; 0.07)	0.09 (0.07; 0.11)	0.12 (0.10; 0.15)	0.17 (0.14; 0.21)
Emergency CS	0.04 (0.01; 0.12)	0.04 (0.03; 0.06)	0.06 (0.04; 0.07)	0.07 (0.05; 0.09)	0.09 (0.07; 0.11)	0.10 (0.07; 0.14)
PPWR	0.07 (0.03; 0.13)	0.05 (0.04; 0.07)	0.13 (0.11; 0.15)	0.26 (0.23; 0.29)	0.42 (0.38; 0.46)	0.58 (0.53; 0.62)

Overweight						
SGA	0.15 (0.10; 0.21)	0.10 (0.08; 0.13)	0.07 (0.05; 0.09)	0.06 (0.04; 0.09)	0.04 (0.03; 0.07)	0.05 (0.03; 0.09)
LGA	0.06 (0.04; 0.09)	0.07 (0.06; 0.09)	0.10 (0.08; 0.12)	0.14 (0.11; 0.17)	0.17 (0.14; 0.21)	0.23 (0.19; 0.29)
Emergency CS	0.04 (0.02; 0.09)	0.08 (0.06; 0.12)	0.08 (0.06; 0.10)	0.12 (0.09; 0.16)	0.08 (0.05; 0.12)	0.13 (0.09; 0.19)
PPWR	0.05 (0.03; 0.09)	0.08 (0.06; 0.10)	0.17 (0.14; 0.19)	0.30 (0.26; 0.34)	0.49 (0.44; 0.54)	0.57 (0.51; 0.63)
Obese						
SGA	0.09 (0.06; 0.13)	0.07 (0.05; 0.10)	0.07 (0.05; 0.10)	0.04 (0.02; 0.09)	0.07 (0.03; 0.14)	0.01 (0.00; 0.09)
LGA	0.09 (0.07; 0.12)	0.13 (0.10; 0.17)	0.15 (0.12; 0.18)	0.24 (0.18; 0.31)	0.21 (0.15; 0.29)	0.22 (0.15; 0.31)
Emergency CS	0.08 (0.05; 0.13)	0.11 (0.08; 0.15)	0.13 (0.09; 0.17)	0.18 (0.11; 0.27)	0.21 (0.13; 0.32)	0.26 (0.15; 0.40)
PPWR	0.04 (0.02; 0.06)	0.07 (0.05; 0.10)	0.18 (0.14; 0.21)	0.30 (0.23; 0.37)	0.40 (0.31; 0.48)	0.52 (0.42; 0.62)

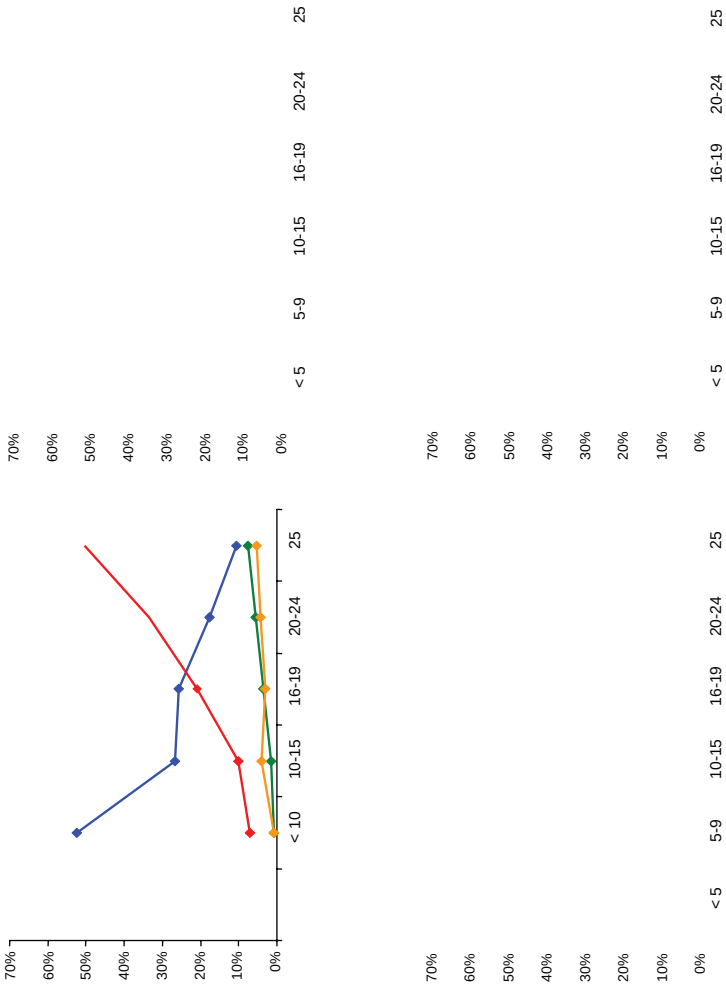

FIGURE G-30 Smoking multiparae, GWG–specific risks of pregnancy outcomes.
NOTE: Age 25-29, height 160-169 cm, high social status. Smoked during pregnancy, no alcohol, moderate exercise. For PPWR, she breastfed > 14 weeks.

TABLE G-29 Continued

	< 5 kg	< 10 kg	10-15 kg	16-19 kg	20-24 kg	≥ 25 kg
Overweight						
SGA	0.22 (0.16; 0.30)	0.16 (0.12; 0.20)	0.11 (0.09; 0.13)	0.10 (0.07; 0.13)	0.07 (0.04; 0.10)	0.09 (0.05; 0.14)
LGA	0.04 (0.03; 0.07)	0.05 (0.04; 0.07)	0.08 (0.07; 0.09)	0.11 (0.09; 0.13)	0.13 (0.11; 0.16)	0.18 (0.15; 0.22)
Emergency CS	0.03 (0.01; 0.06)	0.05 (0.04; 0.08)	0.05 (0.04; 0.7)	0.08 (0.06; 0.11)	0.05 (0.03; 0.08)	0.09 (0.06; 0.13)
PPWR	0.04 (0.02; 0.07)	0.06 (0.05; 0.08)	0.13 (0.12; 0.15)	0.25 (0.22; 0.28)	0.43 (0.38; 0.47)	0.51 (0.45; 0.56)
Obese						
SGA	0.13 (0.09; 0.19)	0.12 (0.08; 0.16)	0.11 (0.08; 0.15)	0.06 (0.03; 0.14)	0.11 (0.05; 0.21)	0.02 (0.00; 0.25)
LGA	0.07 (0.05; 0.09)	0.10 (0.08; 0.13)	0.11 (0.09; 0.14)	0.19 (0.14; 0.24)	0.17 (0.12; 0.23)	0.17 (0.12; 0.31)
Emergency CS	0.05 (0.03; 0.09)	0.07 (0.05; 0.10)	0.08 (0.06; 0.11)	0.12 (0.07; 0.19)	0.14 (0.09; 0.23)	0.18 (0.10; 0.29)
PPWR	0.03 (0.02; 0.05)	0.06 (0.04; 0.08)	0.14 (0.12; 0.17)	0.25 (0.19; 0.31)	0.33 (0.26; 0.42)	0.45 (0.36; 0.56)

FIGURE G-31 GWG-specific risk of pregnancy outcomes in subtypes of underweight women.

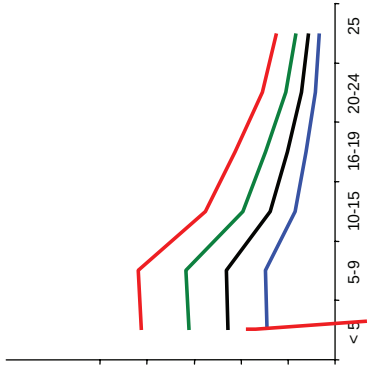

FIGURE G-32 GWG-specific risk of pregnancy outcomes in subtypes of normal weight women.0%10%

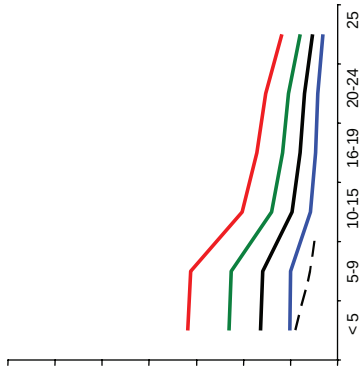

FIGURE G-33 GWG-specific risk of pregnancy outcomes in subtypes of overweight women.

FIGURE G-34 GWG-specific risk of pregnancy outcomes in subtypes of obese women.

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 infant. These results are presented in:

- Figure G-35 (Tables G-30A through G-30D): Underweight women, risks before and after adjustment for birth weight
- Figure G-36 (Tables G-31A through G-31D): Normal weight

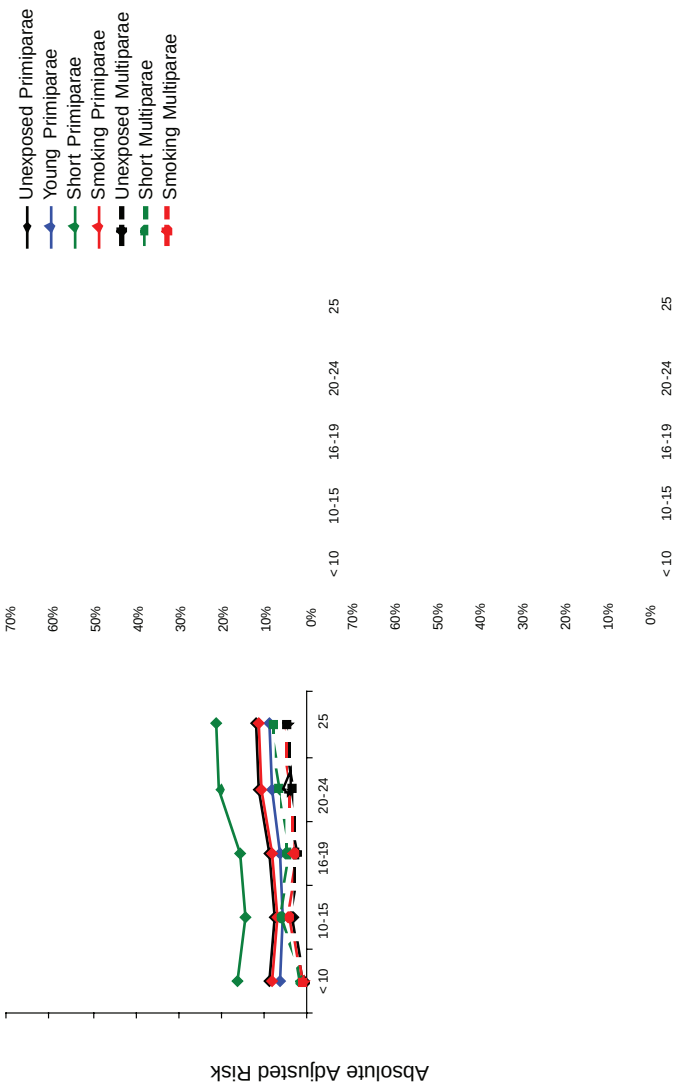

FIGURE G-35 Underweight women, emergency cesarean delivery (CS) and postpartum weight retention (PPWR) with and without adjustment for birth weight.
NOTE: Absolute risks in different types of underweight women. When adjusted for birth weight, the presented risk is that of a women giving birth to a 3,500-3,999 g infant.

TABLE G-30A 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

TABLE G-30B 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					

TABLE G-30C 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 G-30D 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

TABLE G-31A 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		
0.08	0.11		0.16	0.15	0.06	0.06	0.08

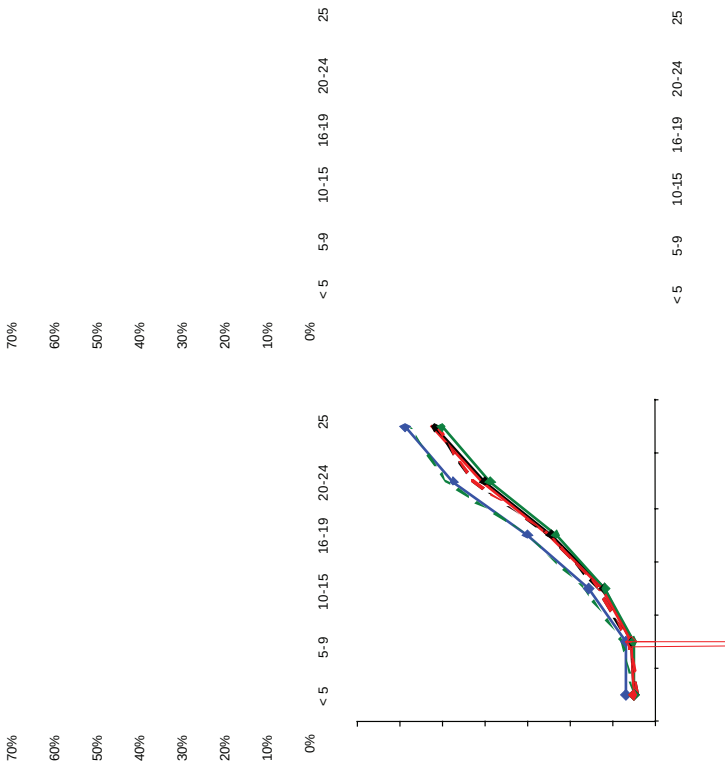

FIGURE G-37 Overweight women, emergency cesarean delivery (CS) and postpartum weight retention (PPWR) with and without adjustment for birth weight.
NOTE: Absolute risks in different types of overweight women. When adjusted for birth weight, the presented risk is that of a women giving birth to a 3,500-3,999 g infant.

TABLE G-32A 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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Unexposedtttotipara

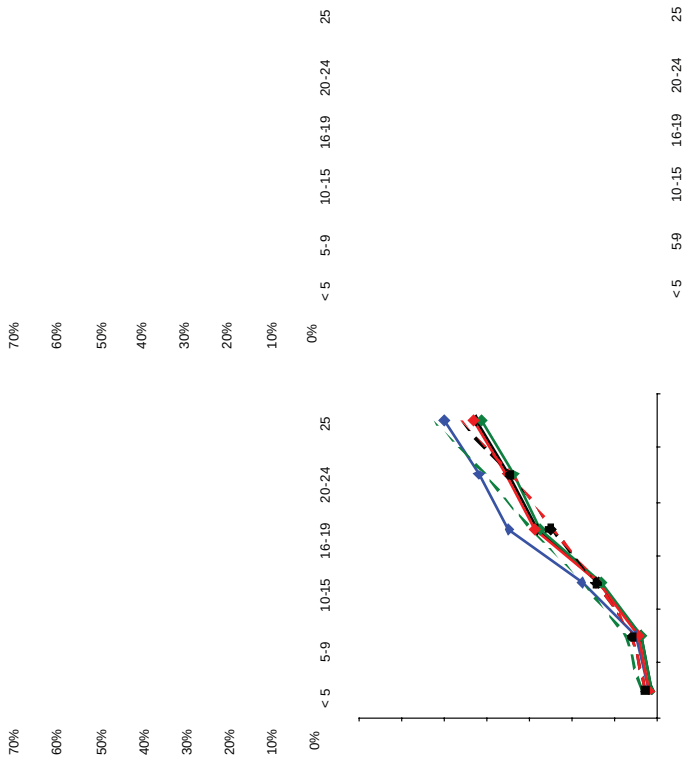


FIGURE G-38 Obese women, emergency cesarean delivery (CS) and postpartum weight retention (PPWR) with and without adjustment for birth weight.
NOTE: Absolute risks in different types of obese women. When adjusted for birth weight, the presented risk is that of a women giving birth to a 3500-3999 g infant.

TABLE G-33A 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

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,

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 te6ttnay,t(r)-3(s)-6D5tnsd -3(-3(c)-3(a)-3(t))-3(s)-6D5d-6D5tnsil3(t)-35t child3-3(with e6tttEMC i4b3(c)H489(w)-3(i)-3(t)-3(h)-3( )-892ttere cin T*[(w)(h)-3/MCID -letP

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 (Figure G-39). 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 G-39; 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 World Health Organization (WHO) cutoffs for BMI.

Weight gain adequacy was related to pregravid BMI category, as described below in Table G-34. 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,

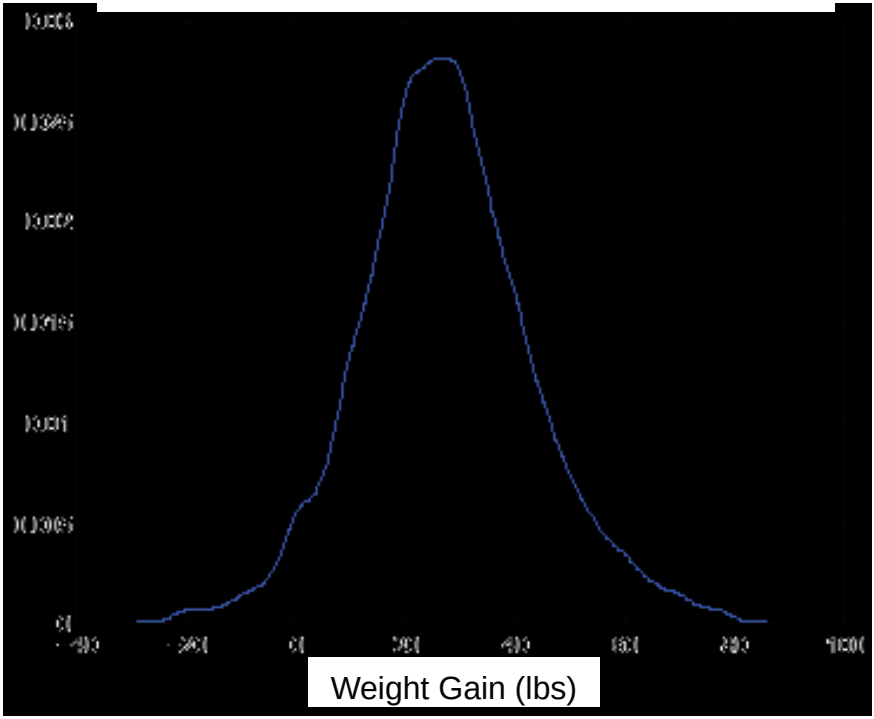


FIGURE G-39 Empirical distribution of weight gain in NMIHS.

TABLE G-34 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

based on the IOM (1990) recommended first trimester gain, extrapolating

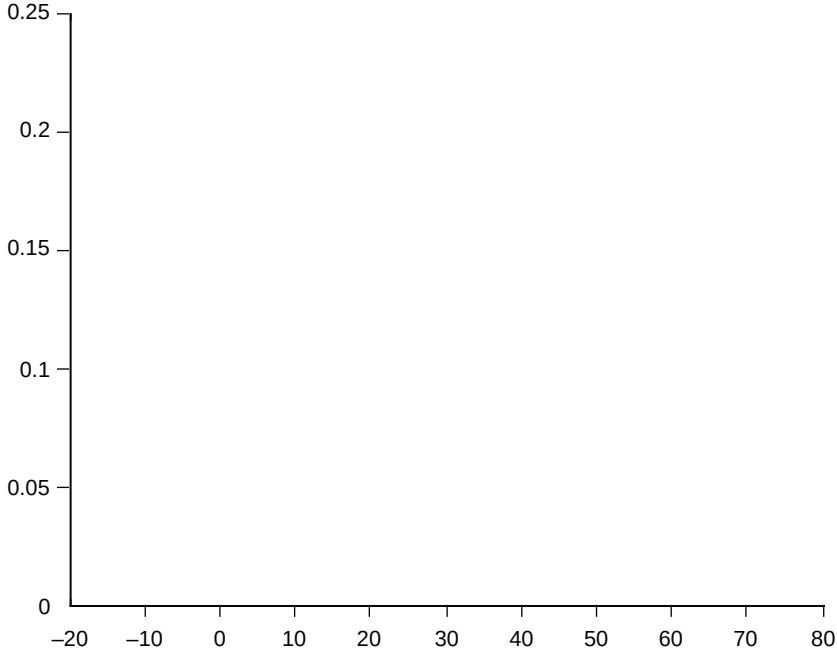


FIGURE G-41 Weight gain (lbs) and probability of cesarean delivery.

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 rela-

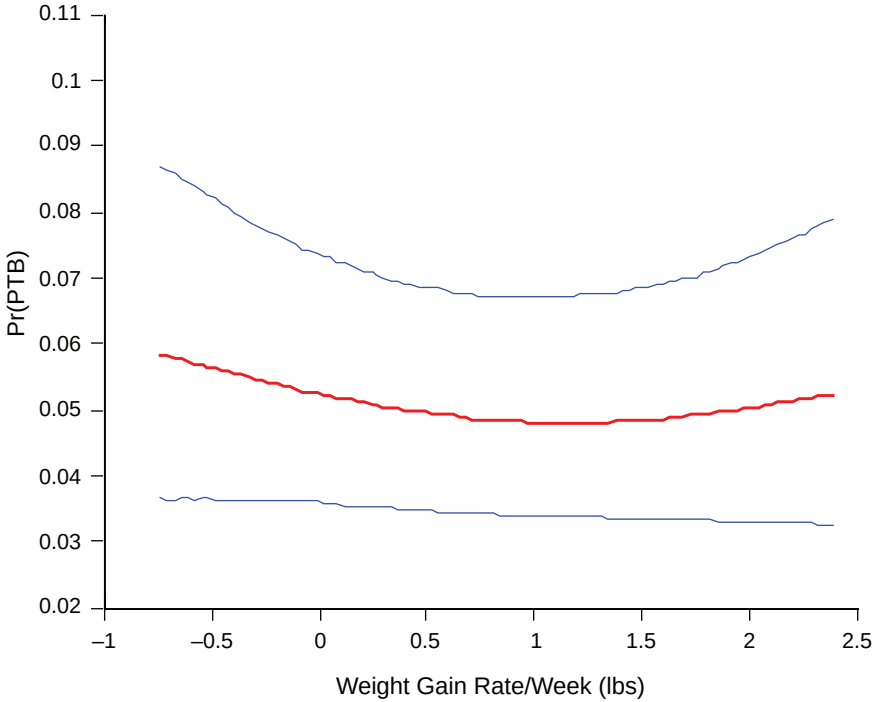


FIGURE G-42 Relationship of weight gain to preterm birth probability.

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 G-43. 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

TABLE G-36 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)

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 G-44 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. (20 49 /Span <</MCI /Span <</04MCI /Spaasu-3( )TN3u-3((s)-3)-3(u3(l)-3(i)- /Span <<

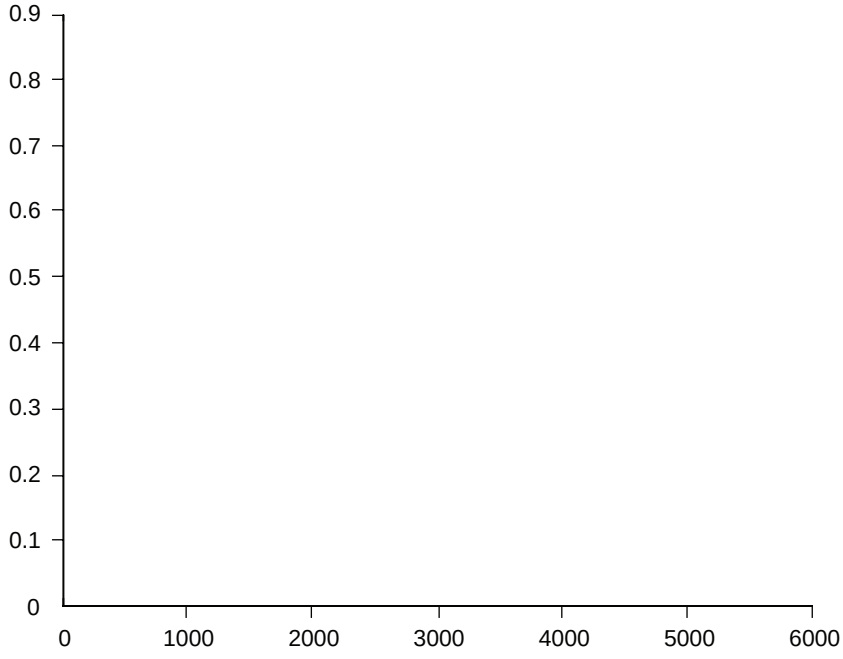


FIGURE G-44 Birth weight density, predicted birth weight distribution by hypothetical weight gain.

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 G-45 and G-46, 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.

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 G-37.

Large-for-Gestational Age

Zhang and Bowes (1995) cutoff points 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

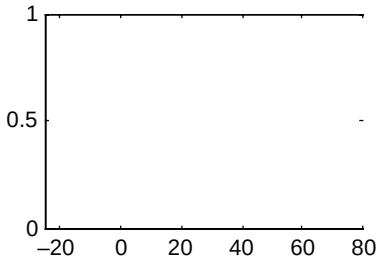


FIGURE G-46 Risk of SGA birth in black women by weight gain (lbs) and pregravid BMI.

8 percent (7 percent, 9 percent) of births are LGA. Predicted probabilities of LGA by pregravid BMI category are in Table G-38.

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 G-40, the interval estimates about these probabilities are quite wide. Analysis of these outcomes is not included due to

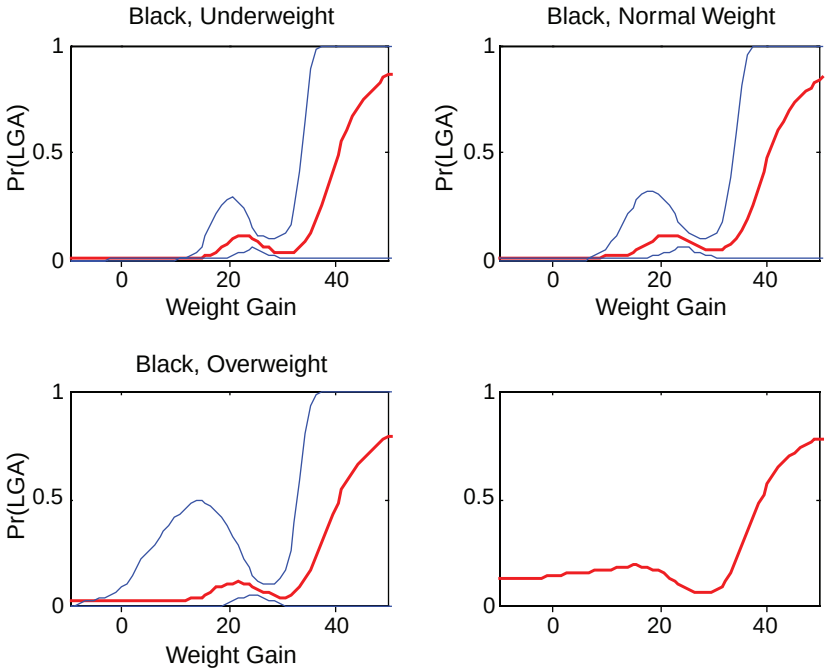
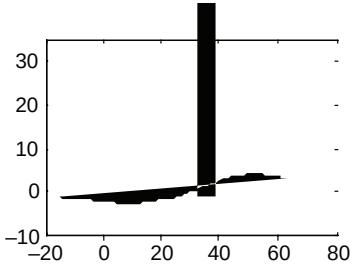


FIGURE G-48 Probability of LGA birth by BMI and weight gain (lbs) in blacks.

smoking during postpartum (none, 1-10 cigarettes per day, > 10 cigarettes



PART III: ANALYSES FROM DR. STEIN AND DR. SAVITZ

THE EFFECT OF MATERNAL RACE/ETHNICITY AND BMI ON THE ASSOCIATION BETWEEN GESTATIONAL WEIGHT GAIN AND BIRTH OUTCOME

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Mount Sinai School of Medicine

To examine the independent and joint effects of maternal race/ethnicity and body mass index (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 (see Tables G-39 and G-40).

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 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 G-41, 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.

TABLE G-39 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[a]	
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)
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[b]	
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)

continued

Characteristic	N (percent)
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)

[a]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.

[b]Excludes 3,502 repeat cesarean and 1,008 vaginal birth after cesarean deliveries.

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 premature rupture of membranes (PROM) (658.1x; 658.2x) were categorized as spontaneous. We then added 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 spontane-

TABLE G-40A Bivariate Association between BMI and Characteristics of Singleton Births, New York City, 1995-2003, *n* = 34,307

TABLE G-40B 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[a]		
	Lower Tertile N = 11,250	Middle Tertile N = 11,416	Upper Tertile N = 11,641
Maternal race/ethnicity	N (percent)	N (percent)	N (percent)
Non-Hispanic white	4,922 (30.2)		

TABLE G-40E Bivariate Association between Gestational Weight Gain and Race/Ethnicity Among Singleton Births, New York City, 1995-2003, *n* = 913,290

	Gestational Weight Gain			
Maternal race/ethnicity	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)
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)

ous. 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) (3(r)-3(o)-3(u)-3(s)-3())-3( )-28 MCID 53930 >>B- /Span <</

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, North Carolina) and Stata Version 10 (Stata Corp, College Station, Texas). 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
-12(c)-3(o10 72 558.28 Tm[S2 Td[(o)-30 Td(-)TjEM Tm[S2 Td[(o)-30 Td(-)TjEM Tm[

TABLE G-42 Odds Ratios and 95% Confidence Intervals for the Association of Rate of Gestational Weight Gain with Preterm Birth <

TABLE G-43 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[a]		
	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.an <</MC.3.9, 1.3)

TABLE G-44 Odds Ratios and 95% Confidence Intervals for the

TABLE G-45 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)				

TABLE G-46 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*				

TABLE G-47A Adjusted[a] 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*n*

TABLE G-47B Adjusted[a] 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[b]		
	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)

[a]Adjusted for maternal age, parity, education, and smoking.

[b]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 G-47C Adjusted[a] 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[b]		
	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)

[a]Adjusted for maternal age, parity, education, and smoking.

[b]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 G-47D Adjusted[a] 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[b]

[a]Adjuse)

TABLE G-47E Adjusted[a] 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[b]	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)

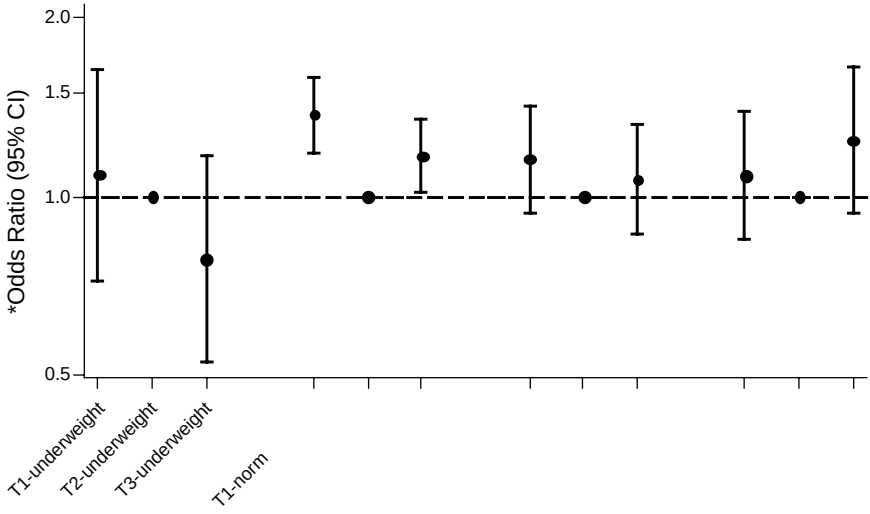

FIGURE G-52 Gestational weight gain and preterm birth, < 37 weeks by body mass index (BMI).

FIGURE G-54 Gestational weight gain and cesarean section by body mass index (BMI).

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 valu-3(e)-3(q)-52(h)-3(e)

TABLE G-48B Infant Mortality (Herring)

BMI	GWG (kg)	Prevalence (%)
Underweight (< 18.5)	< 0	2.60
	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 (18.5-24.9)	< 0	1.66
	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 (25-29.9)	< 0	1.30
	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 (≥ 30)	< 0	1.15
	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

NOTES: BMI = body mass index; GWG = gestational weight gain.

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.

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.

Postpartum 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 mass 6 months after birth. Prevalence estimates were provided for four GWG classes (< 10, 10-15, 16-19, ≥ 20 kg), as reported in Table G-49. Third order polynomial functions were fit to these estimates.

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

TABLE G-49 Post-Partum Weight Retention (Nohr)

BMI	GWG (kg)	Prevalence (%)
Underweight (< 18.5)	< 10	7.9
	10-15	13.1
	16-19	27.6
	≥ 20	46.5
Normal (18.5-24.9)	< 10	5.6
	10-15	13.0
	16-19	26.1
	≥ 20	49.7

Overweight 5>>BDC /T1_EMC /Span <</MCID 56249 >>BDC -62.25 -1.25 Td[(()-3(1)-3(8)-3(.)-3(5)-3(-)
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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 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,

TABLE G-50 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	8
0.30-0.45	12-18	r	≥ < 0.15

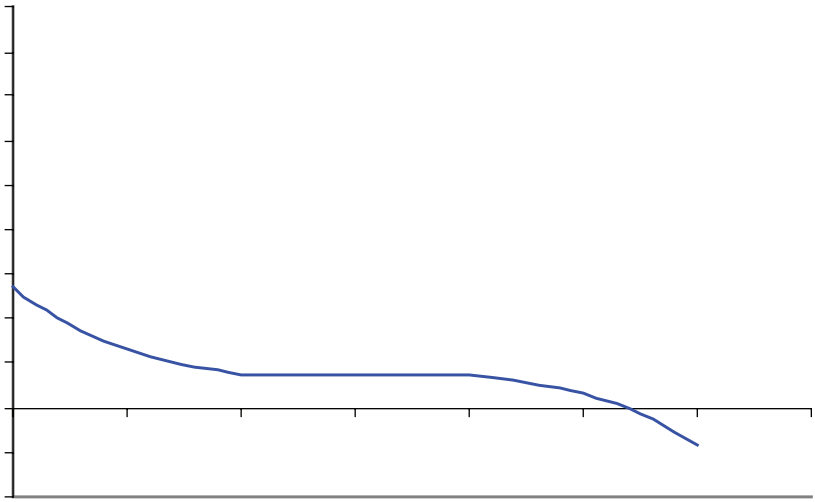

FIGURE G-57 Total expected quality-adjusted life-years (QALYs) lost (Chen et al. [2008] mortality estimates).

ing the Chen et al. (2008) estimates of infant mortality and in Figure G-58 using the Herring estimates.

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 G-58, 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 G-48B. 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 G-57 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

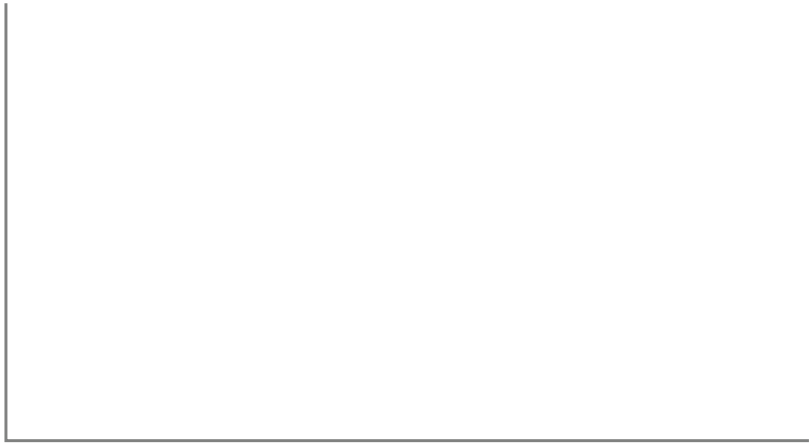

FIGURE G-58 Total expected quality-adjusted life-years (QALYs) lost (Herring infant mortality estimates).

IOM (Institute of Medicine). 1990.

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