Position of the Academy of Nutrition and Dietetics: Obesity, Reproduction, and Pregnancy Outcomes

ABSTRACT

It is the position of the Academy of Nutrition and Dietetics that all women of reproductive age receive education about maternal and fetal risks associated with prepregnancy obesity, excessive gestational weight gain, and significant postpartum weight retention, including potential benefits of lifestyle changes. Behavioral counseling to improve dietary intake and physical activity should be provided to overweight and obese women, beginning in the preconception period and continuing throughout pregnancy, for at least 12 to 18 months postpartum. Weight loss before pregnancy may improve fertility and reduce the risk of poor maternal–fetal outcomes, such as preterm birth, gestational diabetes, gestational hypertension, pre-eclampsia, assisted delivery, and select congenital anomalies. Lifestyle interventions that moderate gestational weight gain may reduce the risk of poor pregnancy outcomes, such as gestational diabetes, gestational hypertension, large for gestational age, and macrosomia, as well as lower the risk for significant postpartum retention. Postpartum interventions that promote healthy diet and physical activity behaviors may reduce postpartum weight retention and decrease obesity-related risks in subsequent pregnancies. Analysis of the evidence suggests that there is good evidence to support the role of diet, physical activity, and behavior changes in promoting optimal weight gain during pregnancy; however, there is currently a relative lack of evidence in other areas related to reproductive outcomes.

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POSITION STATEMENT

It is the position of the Academy of Nutrition and Dietetics that all women of reproductive age receive education about maternal and fetal risks associated with prepregnancy obesity, excessive gestational weight gain, and significant postpartum weight retention, including potential benefits of lifestyle changes. Behavioral counseling to improve dietary intake and physical activity (PA) should be provided to overweight and obese women, beginning in the preconception period and continuing throughout pregnancy, for at least 12 to 18 months postpartum.

The prevalence of overweight and obesity has increased significantly over the past several decades. Increased rates of overweight and obesity among women of reproductive age* pose a challenge for health care providers who provide preconception, prenatal, and postpartum services. Both maternal and fetal outcomes are influenced by a woman's nutrition and weight status before and during pregnancy. A thorough understanding of the risks associated with overweight and obesity, as well as potential interventions to mitigate these risks, is of paramount importance for health care professionals.

*Reproductive age is defined as between the ages of 15 and 49 years by the Population Reference Bureau. Glossary of Demographic Terms (http://www.prb.org/Publications/Lesson-Plans.aspx).

EPIDEMIOLOGY OF OBESITY

TWO-THIRDS OF US WOMEN 20 years of age and older have a body mass index (BMI; calculated as kg/m^2) of ≥25, signifying they are overweight or obese, with 36% of women classified as obese (BMI ≥30) (see Table 1). Overweight and obesity is also common among adolescent females, with 33.8% of 12- to 19-year-old females (those entering or already of reproductive age) having a BMI ≥85th percentile.[1] National data from the Pregnancy Risk Assessment and Monitoring System (PRAMS) and the Behavioral Risk Factor Surveillance Survey (BRFSS) suggest that 25% of women who give birth are overweight before pregnancy, and 22% are obese.[2]

This Academy position paper includes the authors' independent review of the literature in addition to systematic review conducted using the Academy's Evidence Analysis Process and information from the Academy Evidence Analysis Library (EAL). Topics from the EAL are clearly delineated. The use of an evidence-based approach provides important added benefits to earlier review methods. The major advantage of the approach is the more rigorous standardization of review criteria, which minimizes the likelihood of reviewer bias and increases the ease with which disparate articles may be compared. For a detailed description of the methods used in the evidence analysis process, access the Academy's Evidence Analysis Process at http:www.andevidencelibrary.com/eaprocess.

Conclusion Statements are assigned a grade by an expert work group based on the systematic analysis and evaluation of the supporting research evidence. Grade I=Good; Grade II=Fair; Grade III=Limited; Grade IV=Expert Opinion Only; and Grade V=Not Assignable (because there is no evidence to support or refute the conclusion).

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Data from the PRAMS and the BRFSS suggest that obesity rates among women who give birth vary by race/ethnicity and age (Table 1). It is important to note that obesity is most common among non-Hispanic black women and least common among non-Hispanic Asian women, and increases with age among all groups. The prevalence rates of obesity before pregnancy among women who recently gave birth were 31% among non-Hispanic black women, 24% among Hispanic women, 21% of non-Hispanic white women, and 13% of women from other racial/ethnic categories according to PRAMS and BRFSS.[2] Twenty-one percent of women 18 to 24 years old who gave birth were obese before pregnancy, compared with 23% of women 25 to 34 and 24% of women 35 to 44 years of age.[2]

Obesity carries many risks for women including increased lifetime risks for type 2 diabetes mellitus, cardiovascular disease, orthopedic disorders, depression, and certain types of cancer, particularly reproductive cancers. Obesity in women does not just affect their own health, but has a direct bearing on the health of their offspring, and possibly on future generations.[3] The fetal origins of disease (Barker) hypothesis suggests that the weight and nutritional status of a woman before and during pregnancy can affect the long-term health of their children through programming of the adrenal-pituitary-hypothalamic axis during gestation.[4] These effects can include increased risks for obesity, hypertension, cardiovascular disease, diabetes, and depression.[4] The effects of this fetal programming affect the health of the individual, as well as their reproductive function and outcomes, thus altering the health of future generations.

Table 1. The prevalence of varying degrees of obesity among US women, 20 years and older, for different ages and racial and ethnic backgrounds, National Health and Nutrition Examination Survey 2010-2011[a]

Characteristics	≥20 Years (total)	20 to 39 Years	40 to 59 Years
BMI[b] ≥25			
All races/ethnicities	65.8	58.5	71.7
Non-Hispanic white	63.2	55.0	69.1
Non-Hispanic black	82.0	80.0	85.2
Non-Hispanic Asian	34.7	26.2	39.4
Hispanic	77.2	69.5	84.0
BMI ≥30 (grade 1)			
All races/ethnicities	36.1	31.8	39.5
Non-Hispanic white	32.8	27.8	36.3
Non-Hispanic black	56.6	55.8	58.6
Non-Hispanic Asian	11.4	10.9	11.8
Hispanic	44.4	35.8	51.9
BMI ≥35 (grade 2)			
All races/ethnicities	17.0	15.4	19.1
Non-Hispanic white	15.3	13.7	16.9
Non-Hispanic black	29.2	30.6	30.4
Non-Hispanic Asian	3.0	1.1	4.6
Hispanic	20.2	15.1	25.5
BMI ≥40 (grade 3)			
All races/ethnicities	8.3	7.7	9.8
Non-Hispanic white	7.4	6.8	8.8
Non-Hispanic black	16.4	17.5	17.9
Non-Hispanic Asian	1.4	1.1	1.9
Hispanic	7.6	5.8	9.1

[a]Adapted from Ogden and colleagues.[1]
[b]BMI=body mass index; calculated as kg/m^2.

EFFECT OF OBESITY ON FERTILITY AND CONCEPTION

Adipose tissue plays an important role in the metabolism of sex hormones through the production, storage, and/or release of hormones and related enzymes, such as aromatase, adiponectin, leptin, and other cytokines. The production of estrogen and circulating levels of sex hormone–binding globulin are correlated with the presence and distribution of body fat. Obesity has been associated with reduced fertility in females, particularly among those with central adiposity.[5,6] Compared to women with a BMI ≤25, women who were overweight had a fecundity ratio (FR; probability of conception during a specific cycle) of 0.72, while obese women had an FR of 0.60, and very obese women had the lowest FR (0.48) after controlling for waist circumference.[5] Women who had experienced a weight gain of ≥15 kg after age 17 years had significantly lower fecundity (FR=0.72) than those whose weight had remained more steady in early adulthood.[5] Obesity is a significant cause of anovulatory infertility; it has been estimated that among obese women, the infertility rate may increase by 4% per BMI unit.[6] Racial disparities in fertility and fecundity rates have been noted; populations with high preconception obesity rates are also more likely to report experiencing infertility.[7]

Polycystic ovary syndrome, an endocrine disorder that is characterized by enlarged ovaries with multiple cysts and irregular menses, is a leading cause of infertility, affecting up to 18% of women of reproductive age.[8] Central adiposity increases the risk of polycystic ovary syndrome and contributes to anovulation through insulin resistance, hyperinsulinemia, and hyperandrogenemia. Obesity may affect fertility in ways other than anovulation, as reduced fecundity is also observed among ovulating, obese women.[8-10]

The odds of achieving a pregnancy as a result of assisted reproductive technology (ART) fall as BMI increases among women.[11,12] Pregnancy rates after ART appear lower among both overweight (relative risk [RR]=0.91; 95% CI 0.86 to 0.96) and obese women (RR=0.87; 95% CI 0.80 to 0.95).[11,12] Live birth rates were reduced by 9% among overweight women and 20% among obese women, and miscarriage rates were 24% higher among overweight women and 36% higher among obese women after ART.[12]

Conversely, the risk of unintended pregnancy may be higher among obese ovulating women, as they are less likely to use reliable contraception.[13] Contraception might be avoided by women due to fears of weight gain; however, a recent Cochrane review found no association between contraceptive use and weight gain beyond that of normal aging.[14] While there are limited data to determine whether contraceptive effectiveness is altered by obesity, a 24% reduction in the effectiveness of low-dose oral contraceptives has been reported[15,16] as has a significant reduction in the effectiveness of some types of emergency contraception.[17]

Obesity may also be associated with reduced fertility among males. Relationships between BMI and waist circumference, as well as sperm count, concentration, motility, and morphology have been noted.[18,19] Reductions in androgens and sex hormone–binding globulin, as well as increases in estrogen levels, may contribute to subfertility among males.[18,19]

EFFECT OF OBESITY ON MATERNAL–FETAL OUTCOMES

Pre-pregnancy obesity status has been shown to increase the risk for many poor maternal and fetal outcomes. Positive, linear relationships have been found between both increasing total body weight and BMI values and increasing risk for maternal complications, such as gestational hypertension, pre-eclampsia, gestational diabetes, and cesarean-section delivery.[20-27] Positive, linear relationships have also been found for increased risk of poor fetal outcomes, including preterm birth, macrosomia, shoulder dystocia, select birth defects, and stillbirth. The degree of increased risk for poor maternal and fetal outcomes will be discussed in greater detail.

Maternal Outcomes

Obesity and Hypertensive Disorders of Pregnancy. Hypertension is one of the most common chronic health conditions among women and is one of the top three causes of maternal mortality.[21] Nationally, the prevalence of hypertension is approximately 10% among women of reproductive age (18 to 44 years old).[2] Hypertension rates increase among women as they age, with 15% of women 35 to 44 years old reporting a diagnosis of hypertension. Among women of reproductive age, hypertension also varies by race and ethnicity, with a prevalence of 19% among non-Hispanic black women, 9% among non-Hispanic white women, and 8% among other racial/ethnic categories.[2] Hypertension before pregnancy is associated with a higher risk of serious maternal–fetal complications, including pre-eclampsia, placental abruption, gestational diabetes, preterm delivery, small for gestational age (SGA) delivery, and fetal mortality.[21,28]

Obesity may increase the risk of hypertensive disorders among both pregnant and nonpregnant women. Rates of maternal hypertension have increased almost twofold in the past 2 decades, an increase that has been attributed to the concurrent rise in obesity rates among women.[29] In fact, increases in rates of gestational hypertension follow the same racial/ethnic and age variations as rates of preconception obesity. The possibility of developing pre-eclampsia among obese women of reproductive age is two times more likely than normal-weight women of reproductive age.[30] The prevalence of pregestational hypertension was found to be significantly higher among obese compared to normal weight women (6.3% vs 0.4%) in a cohort of pregnant women.[22]

Studies of pregnant women suggest that the odds of developing gestational hypertension is more than six times higher among those who enter pregnancy obese compared to women who enter pregnancy at an ideal weight (odds ratio=6.31; 95% CI 4.30 to 9.26).[22-24] Recent research also suggests that obesity before pregnancy can increase a woman's risk of developing pre-eclampsia three- to eightfold.[23,31] Pre-eclampsia has been estimated to occur in 10% of pregnancies among women with class I (BMI ≥30), 12.8% of women with class II (BMI ≥35), and 16.3% of women with class III (BMI ≥40) obesity, compared to 3.4% among women with a normal BMI before pregnancy.[20]

Hypertension during pregnancy has been associated with increased insulin resistance during pregnancy, even in the absence of diabetes or prediabetes.[32] Gestational hypertension may also increase long-term health risks of women. Both gestational hypertension and pre-eclampsia have been shown to double the risk for development of type 2 diabetes mellitus within 17 years postpartum, even when gestational diabetes mellitus (GDM) was not diagnosed during pregnancy.[32] The occurrence of both GDM and hypertension or pre-eclampsia can dramatically increase a woman's chances of developing type 2 diabetes (as much as 13 times the risk) within 2 decades postpartum.[32] Women who experience pre-eclampsia without GDM during pregnancy have been found to be at three times the risk for developing type 2 diabetes within a year of delivery, suggesting that the insulin resistance associated with gestational hypertension and pre-eclampsia may persist after birth.[33] Women who experienced gestational hypertension without GDM have been found to be at three times the risk for being prescribed medications to treat diabetes within 4 years of delivery, with 4.2 times the risk of using both insulin and oral diabetes medications compared to women without GDM or pre-eclampsia.[33]

Obesity and Gestational Diabetes. Diabetes is a concern during pregnancy because it can increase the risk of congenital anomalies, large for gestational age (LGA) delivery, assisted delivery, and preterm delivery.[2] Data from US women who recently gave birth suggest that about 2% of women report having diabetes before pregnancy.[2] As with other prepregnancy chronic conditions, the risk of preconceptional diabetes varies by age, with 3% of 35- to 44-year-old new mothers reporting diabetes before pregnancy, compared to ≤2% among younger women.[2] Hispanic and

non-Hispanic white women were least likely to report prepregnancy diabetes (prevalence of 1.8% and 2%, respectively) with 2% of non-Hispanic black women and 3% of other women (including Asian/Pacific Islander and American Indian women) reporting this diagnosis. Obesity before pregnancy is a major risk factor for entering pregnancy with pre-existing type 2 diabetes. Data suggest that women who enter pregnancy obese are up to six times more likely to develop GDM than are women who enter pregnancy with BMI <25.[22-24,30] The risk of developing GDM increases with the degree of obesity, such that women with class I obesity are at almost three times the risk for developing GDM, those with class II obesity have approximately four times the risk, and those with class III obesity are at approximately six times the risk compared to women who enter pregnancy at a normal weight.[20,24] Schummers and colleagues[20] found rates of GDM of 9.7% among women with class I, 13.7% among women with class II, and 16.6% among women with class III obesity, compared to 6.1% among women with a normal prepregnancy BMI.

While gestational diabetes creates immediate concerns during pregnancy, there are also long-term concerns for women postpartum. Population-based cohort data suggest that women who experience GDM are at almost 13 times the risk for developing subsequent diabetes than women without GDM.[32] Experiencing both GDM and either pre-eclampsia or gestational hypertension further increase a woman's risk of developing subsequent diabetes, with 16 times the risk for diabetes after dual diagnoses of pre-eclampsia and GDM and more than 18 times the risk after dual diagnoses of gestational hypertension and GDM.[32] Women with GDM during pregnancy were found to be 41 times more likely to require medication to treat diabetes within 4 years of giving birth, with a 22-fold increased risk of using oral medications and a 118-fold increased risk for using insulin; the relative risk of using both insulin and oral medications within 4 years of delivery was 184 times that of women without a diagnosis of GDM.[32,33] Women who experienced both GDM and pre-eclampsia were at an even more elevated risk for requiring medications to treat diabetes within 4 years of delivery, with a 79-fold risk for using any type of medication, 52-fold risk for using oral medications alone, a 145-fold risk for using insulin alone, and a 430-fold risk for using both insulin and oral medications compared to women without GDM or pre-eclampsia.[33]

Table 2. Institute of Medicine guidelines for weight gain during pregnancy, 2009[a]

Prepregnancy BMI[b]	BMI	Total weight gain, lb, range	Weight gain, lb/wk, 2nd and 3rd trimester, mean (range)
Underweight	<18.5	28-40	1 (1-1.3)
Normal weight	18.5-24.9	25-35	1 (0.8-1)
Overweight	25.0-29.9	15-25	0.6 (0.5-0.7)
Obese (all classes)	≥30.0	11-20	0.5 (0.4-0.6)

[a]Recommendations are for singleton pregnancies; consult the Institute of Medicine[35] for information for higher-order births.
[b]BMI=body mass index; calculated as kg/m^2.

Obesity and Delivery. Women who are obese before pregnancy are more likely to experience difficulties during labor and delivery. Obese women are almost two times more likely to experience induction of labor compared to women with a BMI <25.[24,31] The likelihood of induction secondary to a chronic condition during pregnancy is even higher, with the risk of induction quadrupled among obese women with hypertension and the risk of induction increased more than 11-fold among obese women with GDM. Cesarean-section deliveries occur twice as frequently among obese women.[20,22,24,31] Women with a prepregnancy BMI ≥30 are more than three times more likely to experience a cesarean section secondary to pre-eclampsia, and nearly twice as likely to have a cesarean-section delivery as a result of fetal distress or failure to progress in labor compared to normal-weight women. Cesarean section delivery has been estimated to occur in 26.5% of deliveries among normal-weight women, 38.2% of deliveries to women with class I obesity, 43.1% of deliveries to women with class II obesity, and 49.7% of women with class III obesity.[20]

Postpartum hemorrhage is one of the leading causes of maternal mortality. Maternal obesity is thought to be one of the main drivers of the global increase in postpartum hemorrhage that has occurred in the past few decades.[34] Nulliparous obese women have been found to be at double the risk for postpartum hemorrhage, irrespective of the mode of delivery (vaginal vs cesarean section). Women with pre-eclampsia are also at high risk for postpartum hemorrhage, with obesity being a risk factor for both conditions.[34]

Obesity, Gestational Weight Gain, and Postpartum Weight Retention. Maternal obesity can be a risk factor as well as a consequence of excessive gestational weight gain and/or postpartum weight retention. The 2009 Institute of Medicine (IOM) guidelines for gestational weight gain provide ranges for weight gain based on prepregnancy BMI status (Table 2).[35] These guidelines recommend weight-gain levels that promote fetal growth but also minimize postpartum weight retention. National cohort studies suggest that among obese women, up to 66% of nulliparous women and 56% of multiparous women experience gestational weight gains that exceed the IOM guidelines.[36-38]

Pregnancy has been associated with increases in visceral fat stores and weight circumference, with abdominal fat mass increasing during pregnancy and nonsubcutaneous abdominal fat mass rising from 6 to 12 months postpartum.[39-41] These gains may be more pronounced among women who gain above the IOM's recommended weight ranges.

Less than half of women will revert to their prepregnancy weight and more than one in four women will retain 10 lb or more by 12 months

postpartum.[25,42] A prospective cohort study of postpartum women found that 75% had a higher weight at 12 months postpartum than their prepregnancy weight, with 47% retaining ≥10 lb and 24% retaining ≥20 lb.[37] A cohort study of >56,000 women found that up to 15% of women retained 5 kg by 6 months postpartum, with up to 13% moving from a normal prepregnancy BMI to an overweight BMI classification.[36] Longitudinal research suggests that obese women vary tremendously in the amount of weight they retain; on average, the data suggest they may retain slightly less than underweight and normal-weight women within the first 18 months postpartum, but have higher annual weight gains after 18 months postpartum, thus having higher weights at 3, 4, 7, and 15 years postpartum.[36,38,43] Weight retention at 6 months postpartum has been associated with a higher risk for subsequent postpartum weight gain and higher weight status and larger waist circumferences (controlled for BMI status) at 7 years postpartum, suggesting this is a group of women at high risk for lifelong weight-control challenges.[38] Breastfeeding duration of 6 months or longer may attenuate the risk of postpartum retention in the short term, but data on long-term attenuation are equivocal.[37,38]

The evidence is mixed with regard to which factor, prepregnancy weight status or gestational weight gain, is most predictive of postpartum weight retention. Prepregnancy weight status has been found to predict postpartum weight status, with every 1 kg increase in prepregnancy weight associated with a 0.91-kg higher weight at 7 years postpartum, in some but not all studies.[38] Obesity before pregnancy may be more predictive of very high postpartum weight retention than gestational weight gain. Prepregnancy weight and BMI, but not excessive gestational weight gain, have been shown to be significant risk factors for retaining ≥20 lb at 1 year postpartum.[37] Additional risk factors for retaining ≥20 lb included African-American race, unemployment status, low family income, low educational attainment, being a smoker, unplanned pregnancy, and being single but in a relationship at the time of birth.[37]

Excessive gestational weight gain has been found to be predictive of postpartum weight retention in most studies; however, studies vary tremendously in the timing and frequency of follow-up measurements. Gaining more weight than recommended during pregnancy has been found to increase the risk of retaining >2 kg of weight at 18 months postpartum, with nulliparous women retaining more weight than parous women, and obese women at the highest risk of retaining >2 kg.[36] A meta-analysis with >65,000 women confirmed that gestational weight gain in excess of the IOM guidelines was associated with a 3-kg increase in postpartum weight retention at 3 years and a 4.7-kg increase at 15 years compared to women who gained within the guidelines.[44] Postpartum weight retention has been found to be independent of energy intake at 6 months postpartum (after controlling for gestational weight gain), suggesting that excessive postpartum energy may not drive short-term postpartum retention.[45]

The issue of prepregnancy BMI and gestational weight gain as predictors of postpartum weight retention may be confounded by mental health status. Having a high prepregnancy BMI has been associated with a higher risk for postpartum depression.[46,47] Risk for postpartum depression 6 to 8 weeks after delivery appears to increase in a linear fashion with increasing class of pregravid obesity, with women with class I obesity at no increased risk, while those with class II obesity at approximately three times the risk, and those with class III obesity at approximately four times the risk compared to women who entered pregnancy with a normal BMI.[4] Almost 19% of women with pregravid class I obesity screened positive for postpartum depression (using the Edinburgh Postpartum Depression screening tool); the percentage screening positive was 32% among women with class II and 40% with class III obesity before pregnancy. In addition, the majority of women with postpartum depression (55% to 62%) have experienced excessive gestational weight gain.[48] Excessive gestational weight gain, in turn, can increase the risk of significant postpartum weight retention, thus creating an ongoing cycle of weight and depression issues. The odds of postpartum weight retention of >5 kg have been estimated at 2.9 (95% CI 1.64 to 5.11) for women gaining excessively compared to women gaining within the IOM gestational weight gain guidelines among women with postpartum depression 12 months after birth.[48]

Fetal Outcomes

Obesity, Preterm Birth, and Infant Mortality. Preterm birth is a leading cause of low birth weight, and both preterm birth and low birth weight are significant risk factors for infant mortality.[49] Women who are obese before pregnancy are at a significantly elevated risk for preterm delivery.[23,50-54] Preterm delivery risk may follow an obesity gradient. A population-based cohort study of >226,000 women found that 8.4% of women with class I obesity, 8.8% with class II obesity, and 10.3% with class III obesity delivered preterm, compared to 7.1% of women who began pregnancy with BMI ≤25.[20]

A meta-analysis of data from nearly 2 million subjects found that the risk for medically indicated preterm delivery was elevated by 60% among women with class I obesity before pregnancy, but was more than doubled among women with class II prepregnancy obesity.[55] In this study, higher BMI did not increase the risk for premature rupture of the membranes or other causes of spontaneous preterm birth, rather it only increased the risk of medically indicated preterm delivery. On the other hand, smaller, individual studies have shown associations between pregravid obesity status and spontaneous preterm delivery secondary to premature rupture of the membranes.[20,51,56] Potential mechanisms to explain the relationship between preterm birth and pregravid obesity include the increased risk for gestational hypertension and diabetes, both of which involve increased levels of insulin resistance and can lead to maternal–fetal distress and medically indicated early delivery, and an increased inflammatory response secondary to obesity, which may predispose women to spontaneous preterm delivery.[57]

Maternal obesity has been found to increase the risk of neonatal and infant death.[30,58] A meta-analysis found a 21% increase in the risk of fetal death per five BMI units above ideal weight, with risk rising more quickly at higher BMI levels.[20,50,58] The risk of stillbirth was

found to be 24% higher per five BMI units, while the risk of perinatal death was not statistically significant.[20,50,58] Associations were also found between maternal obesity and neonatal and infant death.[58]

Obesity and Birth Weight. Birth weights among infants born to mothers who are obese before pregnancy are generally larger for full-term infants than those born to women with lower BMI values and body weights. Schummer and colleagues[20] documented increasing birth weights corresponding to higher classes of obesity; women with a BMI in the normal category gave birth to infants with a mean weight of 3,391 g, compared to birth weights of 3,548 g, 3,572 g, and 3,591 g, for obesity classes I, II, and III, respectively. These data are consistent with other studies that found mean birth weights of infants born to obese mothers were 100 g higher than those born to nonobese women (3,376 vs 3,476 g).[31]

Macrosomia, or a birth weight of >4,000 to 4,500 g,[59] occurs two to three times more frequently among women who are obese entering pregnancy.[20,21,50,51,54] While 1.4% of normal-weight women in one study gave birth to macrosomic infants, 3.8% of women with class I obesity, 4.5% of women with class II obesity, and 6.1% of women with class III obesity delivered macrosomic infants.[20] Studies have documented macrosomia in up to 20% of births to obese women.[20,51] Not surprising, obese women are also at two to three times higher risk for delivering an LGA infant, with delivery rates for LGA infants between 16% and 22% in some studies.[23,51,53] The risk of LGA birth may be exacerbated by excessive gestational weight gain.[53]

Shoulder dystocia, which occurs when the infant's head is delivered but the shoulders become obstructed in the birth canal, results in a slow, difficult labor that may result in fetal neural injuries, fetal hypoxia, and higher risk of maternal hemorrhage. It occurs more frequently during the delivery of macrosomic or LGA infants. Maternal obesity is also a risk factor for shoulder dystocia. While 3.5% of women with a BMI ≤25 before pregnancy have been found to experience shoulder dystocia during labor, 4.1% of those with class I and 4.4% of women with class II and III obesity also experience dystocia.[20]

Low birth weight has been documented in up to 3% of deliveries to obese women.[51] Women with class I and II obesity have been found to be at twice the risk for delivering a low-birth-weight infant, while those with class III obesity were at three times the risk.[54] Low birth weight is often secondary to preterm birth, which may occur in higher rates among obese women due to their increased risk for preeclampsia, hypertension, and other chronic conditions during pregnancy. Some research suggests that obese women may also be at risk for SGA infants as well, however, this finding is not consistent among studies.[53,54] Suggested pathways for this relationship include increased risk of gestational hypertension and diabetes among obese women, which can lead to alterations in placental development and perfusion that lead to altered nutrient transmission to the growing fetus.[60] Rates of SGA births among obese women have been estimated at 5.7% to 7.5%.[51,53]

Obesity and Congenital Anomalies. Infants of mothers who enter pregnancy obese are more likely to suffer from congenital anomalies. Pregravid obesity may double the risk for neural tube defects (particularly spina bifida), increase the risk of heart defects by 30% to 40%, and increase the risk of limb reduction by >30%.[26,27,61] Other birth defects that have been shown in the majority of studies to be increased among offspring of obese women include anorectal atresia, hypospadias, omphalocele, hydrocephaly, and cleft lip and palate.[26,27,61] Congenital anomalies occur in about 4.7% in pregnancies among women of ideal weight, compared with up to 5.5% of women who are obese.[20] Possible mechanisms to explain the association of obesity with increased risk of birth defects include growing evidence on the role of hyperglycemia secondary to poorly controlled diabetes or the presence of insulin resistance.[26,27] Overweight and obese women have been found to have lower serum folate levels compared to women with a BMI ≤25, which may place their fetuses at a higher risk for developing neural tube defects and possibly other congenital anomalies.[62] While some studies have found that obese women may have lower folic acid intakes compared to normal-weight women, the literature is equivocal.[26,27,45,60] An interaction between obesity and folic acid that is independent of folic acid intake may partially explain the lower serum folate status and higher risk of neural tube defects among obese women.[26] It is suspected that the presence of excessive abdominal fat may mediate this interaction. Obese women do appear to be at lower risk for one birth defect—gastroschisis.[26,27] Behavioral counseling should be a critical component of routine care for overweight and obese women of reproductive age to achieve a healthy body weight and assure adequate intake of folic acid through diet and supplements. This type of intervention may reduce the incidence of poor maternal and fetal outcomes and could be delivered in both clinical and community-based settings, beginning with well-woman visits and continuing through prenatal and postpartum visits.

INTERVENTIONS TO MITIGATE EFFECTS OF OBESITY ON INFERTILITY AND MATERNAL–FETAL OUTCOMES

All women of reproductive age can be encouraged to adopt lifestyle changes to promote health and to reach and maintain a healthy body weight. This practice should be incorporated across the continuum of care through inpatient, outpatient, wellness, and community health care settings. Lifestyle-modification interventions utilize a multifactorial approach to managing weight that include diet, physical activity (PA), and behavior change; these approaches have been utilized concurrently and independently from each other. Goal setting, daily or weekly weighing, and aids to monitor adherence to goals (such as pedometers and daily journals or diaries) are also used often in lifestyle interventions to promote weight loss or prevent weight gain. A set of recommendations regarding diet and PA interventions to promote a healthy weight among adults, including nonpregnant women of reproductive age, is included in the "Position of the Academy of Nutrition and Dietetics: Interventions for the Treatment of Overweight and Obesity in Adults."[63] To avoid duplication, this position paper will focus on lifestyle interventions to mitigate the effects of obesity on reproductive health outcomes, thus it will focus

on interventions to improve fertility and fecundity, reduce excessive gestational weight gain, and reduce postpartum weight retention.

Improved Fertility and Fecundity

Lifestyle interventions have been found to be successful in treating women with reproductive dysfunctions.[64] A 24-week intervention, which included a cohort of 40 obese anovulatory infertile women with a medical diagnosis of polycystic ovary syndrome, was conducted to determine the efficacy of the intervention program on clinical, hormonal, and metabolic outcomes.[65] Individuals were selected to different protocols according to their preference; the programs included the structured exercise training group (n=20), in which individuals exercised for 30 minutes 3 time per week, and the hypocaloric, hyperproteic diet group (n=20), which included a caloric reduction of 800 kcal/day and a diet composition of 35% protein, 45% carbohydrate, and 20% fat with a multivitamin supplement. Although the study found significant improvements in menses cycles for both intervention groups, the menses frequency rate was significantly higher in the structured exercise training group when compared to the diet group.[65] In addition, the structured exercise training group had significantly better ovulation rates when compared to the diet group. The study concluded that there were benefits from participating in either intervention group that resulted in significant improvements in menstrual cyclicity and infertility in both groups, with additional benefits noted when PA was included.

A retrospective cohort study examined infertile overweight and obese individuals (n=52; BMI ≥25) to determine whether meaningful weight loss (≥10% of body weight) could improve fecundity and live birth rates.[66] The intervention method included lifestyle modifications to decrease caloric intake and increase PA up to 30 minutes per day, five times a week. Individuals who were able to accomplish meaningful weight loss had significantly higher conception and live birth rates. The study concluded that weight loss in overweight individuals is an important contributor to improving infertility rates and that weight loss of ≥10% of body weight can improve the likelihood of a live birth.

One of the earlier studies of lifestyle intervention to promote weight loss and increase fertility found that 6 months of weekly sessions that were 2 or 3 hours long were effective in promoting lifestyle changes and weight loss, which in turn improved fertility among obese women (20 dropouts as a comparison group, 67 subjects who completed the intervention) who had been unable to conceive for at least 2 years.[67,68] The lifestyle intervention consisted of a 60-minute group workout session each week, with women agreeing to participate in at least two to three additional workout sessions on their own each week, along with skills-based nutrition education sessions. These sessions varied in content including food purchasing and preparation activities, education on portion control and nutrient contents of food, and general nutrition advice. Women who completed the intervention reduced their BMI by 3.7 units, and lost a mean of 10.2 kg.[67] Almost all women who completed the intervention (90%) experienced spontaneous ovulation, with 78% conceiving and 67% experiencing a live birth. The total cost (in Australian dollars [A$]) of the program was A$8,828 compared to the cost of one in vitro fertilization cycle for one woman costing A$5,190. The total cost per live birth to women before treatment was A$275,000 compared to A$4,600 per live birth after completing the intervention.

Sim and colleagues randomized 49 obese women younger than 38 years of age (n=27 intervention, n=22 control) to either a control condition consisting of the provision of printed material regarding recommendations for weight loss or a 12-week intervention.[69] The intervention included 6 weeks of a very-low-calorie preformulated dietary supplement (providing 2,550 KJ, 65 g protein, 12 g fat, 54 g carbohydrate), followed by 6 weeks of an individualized diet plan (formulated by a registered dietitian nutritionist [RDN]) combined with a weekly group education program that included dietary monitoring and 10,000 steps of daily walking that was monitored through pedometer use. Eighty percent of participants completed the intervention component. The intervention group lost 5 kg more weight on average (mean loss of 6.6 kg among intervention subjects) and reduced their BMI by 1.8 units more (total of 2.4 BMI unit reduction) when compared to control subjects.[69] Waist circumference dropped by 8.7 cm (8 cm more than among control subjects) indicated loss of abdominal body fat. Pregnancy rates were 14% among control subjects and 48% among intervention subjects; three women in the intervention group conceived naturally and none of the control subjects conceived without assistance.

Dropout rates for infertility intervention programs range from 0% to 31% with a mean of 24% of participants not completing the majority of an intervention.[70] Dropouts rate were slightly higher for interventions with a structured exercise component vs those with diet only or diet and unstructured activity. Longer participation in studies was associated with higher weight loss and greater improvements in fertility.[70]

Available data suggest that lifestyle changes that include both dietary modification and increased PA may be effective in improving fertility, however, the following limitations should be considered: many studies included a relatively small sample of participants, which limits the generalizability of the results; women from racial and ethnic minority groups are vastly underrepresented or often missing from these studies; the use of less-rigorous study methodology, such as using dropouts as controls, hampers the validity of the findings; and the intensity of these interventions results in high dropouts rates, which can limit the usefulness of study interventions in clinical practice. Further investigation into diet- and PA-focused interventions that meet the specific needs of all women, including those from racial/ethnic minority groups, as well as programs that minimize barriers to participation (such as use of Internet, text message, and e-mail or postal mail delivery) are needed. Such programs should be designed with application to clinical and community practice as a key consideration.

Prevention of Excessive Gestational Weight Gain and Related Maternal–Fetal Complications

Dietary and PA interventions have been found to be effective at moderating

maternal weight gain in pregnancy as well as risks associated with obesity and excessive gestational weight gain in pregnancy.[71] Dietary intervention approaches found to be effective consisted of education about a balanced diet (usually provided by an RDN) and the use of daily food diaries. Effective PA-intervention approaches included light-intensity resistance training, walking for 30 minutes or for a set number of steps, and other light-intensity activities. The following supporting strategies are commonly utilized in effective studies: goal setting, regular weight monitoring, use of weight-gain graphs to visualize weight gain, verbal feedback on success toward goals, and self-monitoring of diet and PA through the use of food and/or PA records, pedometers, and food scales.[72] Increases in frequency of contact when failing to meet goals, either through phone contact, postcards, or in person, have been recommended to increase success. It has been suggested that obese women may respond to individualized goal setting more than approaches that include less-personal engagement.[72]

A randomized controlled trial of 425 pregnant women with a BMI ≥30 who were randomly assigned to one of two intervention groups or a control group assessed the primary outcome measure of gestational weight gain.[73] Secondary outcome measures of pregnancy complications and delivery and neonatal outcomes were also examined in this study.[73] Both intervention groups (PA plus diet group and PA group) were counseled by an RDN to increase PA by walking at least 11,000 steps/day. The intervention group PA+diet was counseled by an RDN every 2 weeks about a hypocaloric low-fat diet with 1,200 to 1,675 kcal (based on trimester), based on a Mediterranean-style dietary pattern. The study concluded that gestational weight gain was significantly lower in both intervention groups compared to the control group.[73] Fifty-five percent of women in the PA+diet intervention group, 49% of women in the PA intervention group, and 37% of women in the control group met the IOM's gestational weight gain recommendations of 5 to 9 kg of total weight gain. A multivariate analysis concluded that gestational weight gain was reduced by an additional 1.38 kg among those who used a pedometer as a PA intervention, when compared to the control groups. Women in the PA+diet group had a lower rate of emergency cesarean deliveries compared to the other groups.[73]

Obese Danish women who participated in weekly educational sessions and water aerobics classes were found to experience lower gestational weight gains and postpartum BMI values compared to control subjects.[74] The Lifestyle in Pregnancy Study found that the provision of dietary counseling, free gym membership, and personal training resulted in a 1.6-kg lower gestational weight gain among participants compared to nonparticipants.[75] In this study, 35% of intervention subjects vs 47% of control subjects exceeded the IOM gestational weight gain recommendations.

One hundred women were randomized to a counseling program or control group in an attempt to reduce the number of women who exceeded the IOM recommendations for pregnancy weight gain (n=57 intervention group, n=43 control group).[75] Lifestyle counseling was provided to assist women in consuming a balanced diet (recommended caloric distribution included 40% carbohydrate, 30% protein, 30% fat) and to participate in PA three to five times per week was provided. More than half (61%) of the women in the intervention group gained weight within the IOM guidelines compared to 49% in the control care group. Intervention group participants gained significantly less weight than control group participants (mean of 28.7 lb compared with 35.6 lb).[75]

Guelinckx and colleagues[76] conducted a randomized controlled trial to compare whether the provision of an active or passive intervention was more effective in improving dietary habits, increasing PA, and moderating gestational weight gain in obese pregnant women compared to no intervention. Pregnant women (n=122) were randomized into three groups: control (n=43), passive (n=37), and active (n=42) interventions. Women in the control group received standard care, while women in the passive group received only a brochure on diet and PA during pregnancy. Women randomized to the active group received three 1-hour group sessions with an RDN at 15, 20, and 32 weeks gestation. A balanced diet (caloric distribution of 9% to 11% protein, 30% to 35% fat, and 50% to 55% carbohydrate) was discussed as was the importance of increased PA. Seven-day food records were collected during each trimester. A significantly lower energy intake was noted in the passive and active groups than in the control group, but PA levels did not differ by group.[76] Women in the active group gained slightly, but not significantly, less weight compared to women in the passive and control groups (9.8 kg, 10.9 kg, and 10.6 kg, respectively).

The impact of routine weighing during pregnancy on gestational weight gain was assessed in a randomized controlled trial by Jeffries and colleagues.[77] Pregnant women (n=236) at ≤14 weeks gestation were randomized into the intervention group (n=125 intervention, 20% obese) and control groups (n=111, 19% obese). Intervention-group participants received a personalized weight-management card, advice on optimal gestational weight gain, and instruction to record their weight every 4 weeks. The control group received standard care. No significant difference was found in gestational weight gain of obese women between the intervention and control groups; however, a difference was noted among overweight women.[77] The proportion of overweight women who gained more than the IOM recommendations was 35% in the intervention group compared to 56% in the control group. For obese individuals, 36% in the intervention group exceeded the IOM recommendations compared to 24% in the control group.[77] These findings suggest that regular weight measurements during pregnancy may be effective in controlling weight gain of women who are overweight, but may not be as effective among women who are obese before pregnancy. More research is warranted.

Quinlivan and colleagues[78] evaluated whether a four-step multidisciplinary protocol for overweight and obese women would reduce GDM, with gestational weight gain as a secondary outcome. Pregnant, overweight, or obese women (n=124) were randomized into intervention (n=63) and control (n=61) groups. A 5-minute dietary consultation before each prenatal visit, weight self-monitoring, and psychology evaluation and treatment were

included in the intervention protocol. An increased consumption of water, fresh fruit and vegetables, and home-cooked meals, and a reduction in consumption of carbonated beverages, juices, convenience foods, and fast foods were noted among intervention subjects.[78] On average, participants in the intervention group gained 7 kg, which was significantly less than that gained by the control group (13.8 kg).

Another randomized controlled trial compared nutritional and behavioral intervention with conventional prenatal management in 232 obese pregnant women (n=116 control group, n=116 intervention group).[79] Intervention-group participants were placed on a balanced diet of 18 kcal to 24 kcal per kg with 40% of energy from carbohydrate, 30% from protein, and 30% from fat. Food records were kept and reviewed at prenatal visits. Significant differences between the intervention and control groups for pregnancy weight gain and 6-week postpartum weight were noted. Nine percent of women in the control group experienced gestational hypertension compared to 3% of women in the intervention group.

A study of 50 obese Danish women randomized into intervention and control groups (n=23 intervention, n=27 control) found that 10 1-hour consultation sessions with an RDN during the pregnancy were effective at moderating gestational weight gain.[80] A balanced diet with 30% of energy from fat, 15% to 20% from protein, and 50% to 55% from carbohydrate was prescribed with an energy intake restriction (based on individual estimated energy requirements plus sufficient for fetal growth) designed to restrict gestational weight gain to 6 to 7 kg. Women in the intervention group had a mean gestational weight gain of 6.6 kg compared to 13.3 kg in the control group.[80] Mean weekly weight gain from time of enrollment to 36 weeks gestation was significantly reduced (0.18 kg per week in the intervention group compared to 0.26 per week in the control group). The intervention group reported a significantly lower weight compared to the control group at 4 weeks postpartum.

A meta-analysis of lifestyle interventions found a 0.97-kg reduction in gestational weight gain among intervention subjects, with the largest reduction seen in the intervention studies that included a dietary counseling component; these studies had a 3.36-kg mean reduction in gestational weight gain.[71] In particular, interventions that included at least 6 weeks of educational classes or counseling that focused on behavioral change strategies to improve dietary intake and access to structured PA appeared to be effective in moderating gestational weight gain.[71] Women who participated in lifestyle-intervention studies were 26% less likely to develop pre-eclampsia.[71] Dietary interventions had the most significant effect with a 33% reduction in pre-eclampsia risk and 70% reduction in risk for gestational hypertension.[71] Preterm birth was reduced by 32% in dietary interventions, while a trend toward the reduction in risk of 48% for gestational diabetes was seen. Infants born to women who participated in interventions were 0.07 kg lighter than their peers, indicating that the reduction in weight gain did not inhibit normal fetal growth.[71] A 27% reduction in the risk for LGA and 69% reduction in shoulder dystocia were observed, with no effects on risk for low birth weight or SGA.[71]

Metformin has been utilized during pregnancy among women with polycystic ovary syndrome to moderate weight gain and prevent fetal loss.[81,82] Kumar and Khan[81] found reduced rates of pregnancy loss and lower rates of SGA and LGA deliveries among obese women with polycystic ovary syndrome who took metformin during pregnancy. This study also suggested that the risk of gestational hypertension and GDM were both dramatically reduced (by up to 90%) among women who used metformin. The use of metformin during pregnancy has also been shown to moderate weight gain during pregnancy; however, women who stopped taking metformin at delivery were heavier at 1 year postpartum (+1 BMI unit vs +0.2 units in placebo subjects), as were infants born to mothers who took metformin.[81] Additional research into both the short- and long-term effects of metformin use during pregnancy as a means of reducing maternal–fetal complications is warranted.

The prevention of excessive gestational weight gain can result in significant improvements in maternal–fetal health outcomes, reducing the risks for gestational hypertension, pre-eclampsia, cesarean-section delivery, preterm birth, and gestational diabetes. Intervention type and intensity seem to affect the efficacy of programs; effective programs tended to last 6 weeks or longer, focus on improving both dietary intake and PA levels, and actively engage women through routine monitoring of weight gain and/or food intake and PA levels. Few data are available to guide the development of programs for specific subpopulations of women, including racial/ethnic minority groups, various socioeconomic status groups, or differing age groups. Additional research is needed to answer these questions and to determine whether research interventions can be successfully implemented and sustained within clinical and community health care practice settings.

Reduction of Postpartum Weight Retention

Postpartum weight retention may contribute to a woman's lifelong development of obesity. Data from studies of predominantly low-income women suggest that the mean weight retention between pregnancies is approximately 5 kg, with 20% of women retaining ≥5 kg after a pregnancy.[83,84] The effects of postpartum weight retention may be cumulative over a woman's lifetime, with higher parity associated with a higher BMI among women in their 40s and 50s.[85]

Breastfeeding for 3 months or longer has been associated with lower postpartum weight retention, with intensity and frequency negatively associated with postpartum weight retention at 8 and 15 years post pregnancy.[83,86,87] However, obese women have been shown to have lower intention to breastfeed before pregnancy, as well as more difficulty with initiating and continuing lactation after delivery.[88,89] There is a need for the development of interventions to increase breastfeeding initiation rates and duration among obese women, which may have the added effect of moderating postpartum weight retention. Additional research into effective intervention methodologies and platforms of delivery are needed.

PA has been successful in long-term weight management among

adults.[90,91] PA interventions in postpartum period have been found to reduce postpartum weight by 2.57 kg on average.[91] Augmenting PA with heart rate monitors or pedometers to provide feedback on intensity and/or duration of activity may produce a larger mean weight loss (4.09 kg).[91]

A recent Cochrane Review examined varying intervention methods and whether or not they were successful in weight reduction among women after childbirth.[92] Included in this review were two studies that compared PA vs usual care among women who were exclusively breastfeeding. Although they did not see a significant change in body weight, they did see a significant change in fat-free mass in the PA intervention groups compared to the control groups.[92] The addition of diet to PA intervention resulted in the greatest weight loss (4.34 kg).[92] Diet-only intervention resulted in a 1.70-kg weight loss compared to usual care, while diet plus exercise results in a 1.93-kg weight loss.[92] Diet-only interventions resulted in a loss of both fat and fat-free mass, while the addition of exercise helped to preserve fat-free mass. The evidence suggests that exercise interventions are best implemented in conjunction with dietary interventions and should be augmented with technology to help women track intensity and duration of activity.

Similar to interventions to prevent excessive gestational weight gain, interventions to reduce postpartum weight loss can benefit from the addition of self-monitoring strategies.[93] Goal setting was found to be related to lower weight retention at 6 months postpartum.[72] The postpartum period can be a difficult time for women to participate in ongoing intervention studies, given the demands of motherhood. A review of postpartum weight-intervention studies found that dropout rates ranged from 20% to >50% of enrolled participants.[93] Weekly sessions were found to be difficult for some women to participate in, and the review concluded that home-based interventions that allowed women to participate at times that were most convenient for them might be a more appropriate option for postpartum women.

Postpartum women commonly use technology on a daily basis. Nearly all (84%) of the postpartum women in a cross-sectional study had Internet access at home, and those that did not could access it at a friend or relative's home.[94] In fact, 78% of postpartum women reported daily use of the Internet, 75% viewed e-mail daily, 97% used their cell phones daily, and 67% texted daily.[94] Web-based programs have been investigated as an option among postpartum, lactating women and were found to be effective in promoting weight loss.[95] The use of text messaging has been found to be effective among obese, nonpregnant women and may be an appealing option for postpartum women for weight-control intervention.[96,97] Mail-based interventions programs can also be an option for women in rural areas or those without high-speed Internet connection. Thirty-nine percent of postpartum women in a Texas-based study were interested in participating in a mail-based weight-loss program.[94] Mail-based education has been found to be an effective means of reaching pregnant and postpartum women, resulting in a 2.27-kg lower weight retention among participants compared to nonparticipants.[98]

Bariatric Surgery and Pregnancy Outcomes

It is estimated that >50,000 women of reproductive age experience bariatric surgery each year in the United States.[99,100] Women who undergo bariatric surgery are encouraged to avoid pregnancy for at least 12 to 24 months after surgery to avoid complications that could result from potential nutrient deficiencies secondary to reduced food intake and malabsorption.[101] Women who become pregnant after bariatric surgery may be at lower risk for GDM, gestational hypertension, and pre-eclampsia, but may be at elevated risk for preterm delivery and SGA delivery.[102,103] A study of 596 postsurgical births with 2,356 control births found significantly lower rates of GDM among postsurgical women (1.9%) compared to BMI-matched control women (6.8%), a 75% reduction in risk for development of GDM[102]; these findings are consistent with several other studies showing reduced risk of GDM in pregnancies after bariatric surgery compared to obese women.[104,105] The risk for developing hypertensive disorders of pregnancy may also be reduced by up to 75% after bariatric surgery.[106,107]

Prematurity rates have been reported to be higher among postsurgical women when compared to matched controls (9.6% compared to 6.1%, respectively) with risk found among both spontaneous (5.2% vs 2.6%) and medically indicated (4.5% vs 2.5%) preterm births.[108] Birth weight may also be affected by prepregnancy bariatric surgery; a review of 17 studies found lower birth weights among infants born to postsurgical mothers.[109] Increased rates of SGA delivery has been found among women who have undergone surgery compared to matched controls (5.2% compared to 3.0% and 17.4% compared to 5%).[108,110] At least one study has suggested that the risk for SGA is much higher among women who underwent gastric bypass surgery compared to those who underwent restrictive procedures (such as adjustable gastric banding).[109]

It appears that bariatric surgery may ameliorate some of the increased risks associated with pregravid obesity, such as hypertensive disorders and GDM, but may also increase the risk for other poor outcomes, such as preterm delivery and SGA. Women who have undergone bariatric surgery may comprise a high-risk population despite their weight loss and merit special attention before, during, and after pregnancy.

FUTURE RESEARCH AND PRACTICE NEEDS

A workgroup made up of members from the Academy of Nutrition and Dietetics (Academy) who are experts in the area of maternal and infant nutrition utilized the Academy's Evidence Analysis Process to develop five practice-based questions that informed a subsequent systematic review of the literature on structured nutrition programs and interventions that could improve maternal and fetal outcomes. The Figure illustrates these questions along with the evidence compiled and available through the Academy's Evidence Analysis Library.

While there is a growing body of evidence to guide the development of interventions to moderate gestational weight gain and reduce postpartum weight retention, there is limited

Question	Evidence Grade[a] and Conclusion Statement
Preconception Period	
In overweight and obese women, what is the impact of intentional weight loss during the preconceptional period on conception?	Grade V: No evidence was identified to evaluate the impact of intentional weight loss during the preconceptional period on conception.
Gestational Period	
In overweight and obese women who become pregnant, what is the impact of a structured weight-management program in gestational weight gain?	Grade I: Among overweight and obese women, gestational weight gain appears to be controlled by a lifestyle-intervention approach to weight management. Limited evidence supports any combination of behavioral counseling, weight monitoring, diet, and physical activity as weight-management methods to control excessive gestational weight gain. Physical activity interventions independent of other weight-management strategies did not appear to influence gestational weight gain.
In post–weight-loss surgery females who become pregnant, what is the impact of nutrition intervention on gestational weight gain?	Grade V: No evidence was identified to evaluate the impact of nutrition intervention on gestational weight gain in post–weight-loss surgery females who become pregnant.
Postpartum Period	
Among women who are overweight or obese before pregnancy, what is the impact of exclusive breastfeeding on postpartum weight?	Grade V: No evidence was identified to evaluate the impact of exclusive breastfeeding on postpartum weight.
Among women who are overweight or obese before pregnancy, what is the effect of a structured breastfeeding program on the duration of breastfeeding?	Grade III: Limited evidence suggests that a structured breastfeeding program may extend the duration of exclusive and partial breastfeeding in women who were obese before pregnancy.
[a]Grade I=Good/Strong; Grade II=Fair; Grade III=Limited/Weak; Grade IV=Expert Opinion Only; and Grade V=Grade Not Assignable.	

Figure. Evidence Analysis Library questions related to the role of obesity on reproductive outcomes.

evidence on the effects of intentional weight loss on fertility and conception. There is also a relative lack of information available about the impact of weight-loss surgery and gestational weight-gain rates among obese women, and on the effects of breastfeeding interventions on postpartum weight status. Finally, there is a dearth of evidence relating to cost effectiveness or cost savings for lifestyle interventions to prevent excessive gestational weight gain and/or postpartum weight retention. In addition, cost savings estimates for helping women achieve a healthy weight before pregnancy are also lacking.

A glaring gap in the literature exists with regard to how to tailor preconception, prenatal, and postpartum weight-control interventions to meet the needs of women from varying racial/ethnic groups, socioeconomic backgrounds, and of varying health literacy abilities. It is unclear whether the types of interventions utilized in studies consisting of mainly white, middle- to upper-middle-class women will be as successful among women from other populations. In addition, the economic feasibility of providing the research intervention protocols that have been found to be effective in general health care settings, without grant funding, is unexplored. Finally, data that compare various platforms for providing intervention programs to women (web-based vs text messaging vs in person vs small group vs postal mail) are needed to help assess both the comparative effectiveness and the economic feasibility of integrating nutrition and other lifestyle health-promotion services for women into routine wellness exams, as well as prenatal and postpartum care visits to help them achieve and maintain a healthy weight before, during, and after pregnancy.

ROLES AND RESPONSIBILITIES OF RDNs AND NUTRITION AND DIETETICS TECHNICIAN, REGISTERED

All women, particularly overweight and obese women, should have access to nutrition education and counseling regarding the potential maternal and fetal complications that can accompany obesity before and during pregnancy. RDNs and nutrition and dietetic technicians, registered (NDTRs) are uniquely qualified to provide lifestyle-focused nutrition education and counseling, including medical nutrition

therapy, to achieve this goal. Based on current evidence, recommendations for roles and responsibilities of RDNs and NDTRs include:

- During the preconception period, all women should be screened during routine care to determine their weight status. Overweight and obese women should be offered counseling and interventions that assist them in reaching and maintaining a healthy body weight and BMI. The Academy's position paper on weight management[63] provides guidance on effective interventions to assist nonpregnant women in reaching and maintaining a healthy body weight. Obese women who experience infertility can benefit from intensive weight-loss counseling, with medical nutrition therapy provided by an RDN, as appropriate, to treat comorbid medical conditions before and during infertility treatment. All women can be encouraged to consume a varied diet that includes foods fortified with folic acid, in addition to a supplement that contains 400 μg folic acid; this is particularly important among obese women, who are already at an elevated risk for bearing a child with neural tube defects and who can benefit from a supplement containing 800 μg folic acid.[111] Obese and overweight women with a family history of birth defects should be encouraged to consume up to 4-mg folic acid supplements before and between pregnancies.[112]
- During pregnancy, all women should be provided with education about appropriate weight-gain goals based on their prepregnancy BMI, as well as potential risks of excessive gestational weight gain. Overweight and obese women should have access to routine nutrition education and lifestyle counseling to assist them with achieving appropriate weight gain. The use of tools to monitor lifestyle changes, such as food diaries and pedometers, can be encouraged along with goal setting. Gestational weight-gain grids can assist with monitoring weight-gain appropriateness and can be an effective tool for nutrition education. Technology can be used as appropriate to assist women in monitoring lifestyle changes. MyPlate for Moms is an online resource that provides the ability to track dietary intake and PA patterns across pregnancy, with general nutrition advice based on trimester of pregnancy provided. RDNs can provide medical nutrition therapy to overweight and obese women who develop chronic conditions during pregnancy, such as gestational hypertension and GDM.
- During the postpartum and interconception period, women should have access to nutrition education and lifestyle counseling to help them reduce postpartum weight retention. Behavioral counseling that is focused on improving dietary intake and PA levels and tailored to the needs of postpartum women should be provided for the first 12 to 18 months postpartum. Regular attendance at group sessions may be difficult for new mothers; thus, alternative methods of education, such as text messages and online programs, can be explored. All women can be encouraged to initiate and maintain breastfeeding in the postpartum period. Obese women may experience more difficulties in establishing lactation, so assistance from RDNs and NDTRs who are trained in lactation support can be made available after hospital discharge. Women who participate in the Special Supplemental Nutrition Program for Women, Infants, and Children can be encouraged to utilize the breastfeeding peer support programs that are an integral part of that program.

References

1. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011-2012. *JAMA*. 2014;311(8):806-814.
2. Robbins CL, Zapata LB, Farr SL, et al. Core state preconception health indicators—Pregnancy risk assessment monitoring system and behavioral risk factor surveillance system, 2009. *MMWR Surveill Summ*. 2014;63(3):1-62.
3. Kulie T, Slattengren A, Redmer J, Counts H, Eglash A, Schrager S. Obesity and women's health: An evidence-based review. *J Am Board Fam Med*. 2011;24(1):75-85.
4. de Boo HA, Harding JE. The developmental origins of adult disease (Barker) hypothesis. *Aust N Z J Obstet Gynaecol*. 2006;46(1):4-14.
5. Robker RL, Akison LK, Bennett BD, et al. Obese women exhibit differences in ovarian metabolites, hormones, and gene expression compared with moderate-weight women. *J Clin Endocrinol Metab*. 2009;94(5):1533-1540.
6. Wise LA, Rothman KJ, Mikkelsen EM, Sorensen HT, Riis A, Hatch EE. An Internet-based prospective study of body size and time-to-pregnancy. *Hum Reprod*. 2010;25(1):253-264.
7. Chandra A, Copen CE, Stephen EH. Infertility and impaired fecundity in the United States, 1982-2010: Data from the national survey of family growth. *Natl Health Stat Report* 2013;(67):1-18. 1 p following 19.
8. van der Steeg JW, Steures P, Eijkemans MJ, et al. Obesity affects spontaneous pregnancy chances in subfertile, ovulatory women. *Hum Reprod*. 2008;23(2):324-328.
9. Motta AB. The role of obesity in the development of polycystic ovary syndrome. *Curr Pharm Des*. 2012;18(17):2482-2491.
10. Gesink Law DC, Maclehose RF, Longnecker MP. Obesity and time to pregnancy. *Hum Reprod*. 2007;22(2):414-420.
11. Luke B, Brown MB, Missmer SA, et al. The effect of increasing obesity on the response to and outcome of assisted reproductive technology: A national study. *Fertil Steril*. 2011;96(4):820-825.
12. Rittenberg V, Seshadri S, Sunkara SK, Sobaleva S, Oteng-Ntim E, El-Toukhy T. Effect of body mass index on IVF treatment outcome: An updated systematic review and meta-analysis. *Reprod Biomed Online*. 2011;23(4):421-439.
13. Bajos N, Wellings K, Laborde C, Moreau C; ; CSF Group. Sexuality and obesity, a gender perspective: Results from French national random probability survey of sexual behaviours. *BMJ*. 2010;340:c2573.
14. Gallo MF, Lopez LM, Grimes DA, Carayon F, Schulz KF, Helmerhorst FM. Combination contraceptives: Effects on weight. *Cochrane Database Syst Rev*. 2014;1:CD003987.
15. Lopez LM, Grimes DA, Chen M, et al. Hormonal contraceptives for contraception in overweight or obese women. *Cochrane Database Syst Rev*. 2013;4:CD008452.
16. Burkman RT, Fisher AC, Wan GJ, Barnowski CE, LaGuardia KD. Association between efficacy and body weight or body mass index for two low-dose oral contraceptives. *Contraception*. 2009;79(6):424-427.

17. Mody SK, Han M. Obesity and contraception. *Clin Obstet Gynecol*. 2014;57(3):501-507.
18. Hammoud AO, Gibson M, Peterson CM, Meikle AW, Carrell DT. Impact of male obesity on infertility: A critical review of the current literature. *Fertil Steril*. 2008;90(4):897-904.
19. Eisenberg ML, Kim S, Chen Z, Sundaram R, Schisterman EF, Buck Louis GM. The relationship between male BMI and waist circumference on semen quality: Data from the LIFE study. *Hum Reprod*. 2014;29(2):193-200.
20. Schummers L, Hutcheon JA, Bodnar LM, Lieberman E, Himes KP. Risk of adverse pregnancy outcomes by prepregnancy body mass index: A population-based study to inform prepregnancy weight loss counseling. *Obstet Gynecol*. 2015;125(1):133-143.
21. Lo JO, Mission JF, Caughey AB. Hypertensive disease of pregnancy and maternal mortality. *Curr Opin Obstet Gynecol*. 2013;25(2):124-132.
22. Bautista-Castano I, Henriquez-Sanchez P, Aleman-Perez N, et al. Maternal obesity in early pregnancy and risk of adverse outcomes. *PLoS One*. 2013;8(11):e80410.
23. Gaillard R, Durmus B, Hofman A, Mackenbach JP, Steegers EA, Jaddoe VW. Risk factors and outcomes of maternal obesity and excessive weight gain during pregnancy. *Obesity (Silver Spring)*. 2013;21(5):1046-1055.
24. El-Chaar D, Finkelstein SA, Tu X, et al. The impact of increasing obesity class on obstetrical outcomes. *J Obstet Gynaecol Can*. 2013;35(3):224-233.
25. Overcash RT, Lacoursiere DY. The clinical approach to obesity in pregnancy. *Clin Obstet Gynecol*. 2014;57(3):485-500.
26. Carmichael SL, Rasmussen SA, Shaw GM. Prepregnancy obesity: A complex risk factor for selected birth defects. *Birth Defects Res A Clin Mol Teratol*. 2010;88(10):804-810.
27. Waller DK, Shaw GM, Rasmussen SA, et al. Prepregnancy obesity as a risk factor for structural birth defects. *Arch Pediatr Adolesc Med*. 2007;161(8):745-750.
28. Livingston JC, Maxwell BD, Sibai BM. Chronic hypertension in pregnancy. *Minerva Ginecol*. 2003;55(1):1-13.
29. Martin JA, Hamilton BE, Ventura SJ, et al. Births: Final data for 2009. *Natl Vital Stat Rep*. 2011;60(1):1-70.
30. Black RE, Victora CG, Walker SP, et al. Maternal and child undernutrition and overweight in low-income and middle-income countries. *Lancet*. 2013;382(9890):427-451.
31. Athukorala C, Rumbold AR, Willson KJ, Crowther CA. The risk of adverse pregnancy outcomes in women who are overweight or obese. *BMC Pregnancy Childbirth*. 2010;10. 56-2393-10-56.
32. Feig DS, Shah BR, Lipscombe LL, et al. Preeclampsia as a risk factor for diabetes: A population-based cohort study. *PLoS Med*. 2013;10(4):e1001425.
33. Engeland A, Bjorge T, Daltveit AK, et al. Risk of diabetes after gestational diabetes and preeclampsia. A registry-based study of 230,000 women in Norway. *Eur J Epidemiol*. 2011;26(2):157-163.
34. Fyfe EM, Thompson JM, Anderson NH, Groom KM, McCowan LM. Maternal obesity and postpartum haemorrhage after vaginal and caesarean delivery among nulliparous women at term: A retrospective cohort study. *BMC Pregnancy Childbirth*. 2012;12. 112-2393-12-112.
35. The American College of Obstetricians and Gynecologists Committee on Obstetrics Practice. Weight gain during pregnancy. Committee Opinion 548, January 2013. http://www.acog.org/Resources-And-Publications/Committee-Opinions/Committee-on-Obstetric-Practice/Weight-Gain-During-Pregnancy. Accessed January 29, 2016.
36. Haugen M, Brantsaeter AL, Winkvist A, et al. Associations of pre-pregnancy body mass index and gestational weight gain with pregnancy outcome and postpartum weight retention: A prospective observational cohort study. *BMC Pregnancy Childbirth*. 2014;14. 201-2393-14-201.
37. Endres LK, Straub H, McKinney C, et al. Postpartum weight retention risk factors and relationship to obesity at 1 year. *Obstet Gynecol*. 2015;125(1):144-152.
38. Kirkegaard H, Stovring H, Rasmussen KM, Abrams B, Sorensen TI, Nohr EA. How do pregnancy-related weight changes and breastfeeding relate to maternal weight and BMI-adjusted waist circumference 7 y after delivery? Results from a path analysis. *Am J Clin Nutr*. 2014;99(2):312-319.
39. Gunderson EP, Murtaugh MA, Lewis CE, Quesenberry CP, West DS, Sidney S. Excess gains in weight and waist circumference associated with childbearing: The Coronary Artery Risk Development in Young Adults Study (CARDIA). *Int J Obes Relat Metab Disord*. 2004;28(4):525-535.
40. Gunderson EP, Sternfeld B, Wellons MF, et al. Childbearing may increase visceral adipose tissue independent of overall increase in body fat. *Obesity (Silver Spring)*. 2008;16(5):1078-1084.
41. Fraser A, Tilling K, Macdonald-Wallis C, et al. Associations of gestational weight gain with maternal body mass index, waist circumference, and blood pressure measured 16 y after pregnancy: The Avon Longitudinal Study of Parents and Children (ALSPAC). *Am J Clin Nutr*. 2011;93(6):1285-1292.
42. Olson CM, Strawderman MS, Hinton PS, Pearson TA. Gestational weight gain and postpartum behaviors associated with weight change from early pregnancy to 1 y postpartum. *Int J Obes Relat Metab Disord*. 2003;27(1):117-127.
43. Rong K, Yu K, Han X, et al. Pre-pregnancy BMI, gestational weight gain and postpartum weight retention: A meta-analysis of observational studies. *Public Health Nutr*. 2015;18(12):2172-2182.
44. Nehring I, Schmoll S, Beyerlein A, Hauner H, von Kries R. Gestational weight gain and long-term postpartum weight retention: A meta-analysis. *Am J Clin Nutr*. 2011;94(5):1225-1231.
45. von Ruesten A, Brantsaeter AL, Haugen M, et al. Adherence of pregnant women to nordic dietary guidelines in relation to postpartum weight retention: Results from the Norwegian mother and child cohort study. *BMC Public Health*. 2014;14. 75-2458-14-75.
46. Laraia BA, Siega-Riz AM, Dole N, London E. Pregravid weight is associated with prior dietary restraint and psychosocial factors during pregnancy. *Obesity (Silver Spring)*. 2009;17(3):550-558.
47. LaCoursiere DY, Barrett-Connor E, O'Hara MW, Hutton A, Varner MW. The association between prepregnancy obesity and screening positive for postpartum depression. *BJOG*. 2010;117(8):1011-1018.
48. Herring SJ, Rich-Edwards JW, Oken E, Rifas-Shiman SL, Kleinman KP, Gillman MW. Association of postpartum depression with weight retention 1 year after childbirth. *Obesity (Silver Spring)*. 2008;16(6):1296-1301.
49. US Health Resources and Services Administration. Report of the Secretary's Advisory Committee on Infant Mortality (SACIM): Recommendations for HHS action and framework for a national strategy. 2013. http://www.hrsa.gov/advisorycommittees/mchbadvisory/InfantMortality/Correspondence/recommendationsjan2013.pdf. Accessed October 21, 2015.
50. Leddy MA, Power ML, Schulkin J. The impact of maternal obesity on maternal and fetal health. *Rev Obstet Gynecol*. 2008;1(4):170-178.
51. Li N, Liu E, Guo J, et al. Maternal prepregnancy body mass index and gestational weight gain on pregnancy outcomes. *PLoS One*. 2013;8(12):e82310.
52. Gould JB, Mayo J, Shaw GM, Stevenson DK. March of Dimes Prematurity Research Center at Stanford University School of Medicine. Swedish and American studies show that initiatives to decrease maternal obesity could play a key role in reducing preterm birth. *Acta Paediatr*. 2014;103(6):586-591.
53. Shin D, Song WO. Prepregnancy body mass index is an independent risk factor for gestational hypertension, gestational diabetes, preterm labor, and small- and large-for-gestational-age infants. *J Matern Fetal Neonatal Med*. 2015;28(14):1679-1686.
54. Scott-Pillai R, Spence D, Cardwell CR, Hunter A, Holmes VA. The impact of body mass index on maternal and neonatal outcomes: A retrospective study in a UK obstetric population, 2004-2011. *BJOG*. 2013;120(8):932-939.
55. Torloni MR, Betran AP, Daher S, et al. Maternal BMI and preterm birth: A systematic review of the literature with meta-analysis. *J Matern Fetal Neonatal Med*. 2009;22(11):957-970.
56. Nohr EA, Bech BH, Vaeth M, Rasmussen KM, Henriksen TB, Olsen J. Obesity, gestational weight gain and preterm birth: A study within the Danish National Birth Cohort. *Paediatr Perinat Epidemiol*. 2007;21(1):5-14.

57. Jarvie E, Hauguel-de-Mouzon S, Nelson SM, Sattar N, Catalano PM, Freeman DJ. Lipotoxicity in obese pregnancy and its potential role in adverse pregnancy outcome and obesity in the offspring. *Clin Sci (Lond)*. 2010;119(3):123-129.

58. Aune D, Saugstad OD, Henriksen T, Tonstad S. Maternal body mass index and the risk of fetal death, stillbirth, and infant death: A systematic review and meta-analysis. *JAMA*. 2014;311(15):1536-1546.

59. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 22: Fetal macrosomia. *Obstet Gynecol*. 2000;96(5).

60. Higgins L, Greenwood SL, Wareing M, Sibley CP, Mills TA. Obesity and the placenta: A consideration of nutrient exchange mechanisms in relation to aberrant fetal growth. *Placenta*. 2011;32(1):1-7.

61. Stothard KJ, Tennant PW, Bell R, Rankin J. Maternal overweight and obesity and the risk of congenital anomalies: A systematic review and meta-analysis. *JAMA*. 2009;301(6):636-650.

62. Mojtabai R. Body mass index and serum folate in childbearing age women. *Eur J Epidemiol*. 2004;19(11):1029-1036.

63. Raynor H, Champagne CM. Position of the Academy of Nutrition and Dietetics: Interventions for the treatment of overweight and obesity in adults. *J Acad Nutr Diet*. 2016;116(1):129-147.

64. Moran LJ, Dodd J, Nisenblat V, Norman RJ. Obesity and reproductive dysfunction in women. *Endocrinol Metab Clin North Am*. 2011;40(4):895-906.

65. Palomba S, Giallauria F, Falbo A, et al. Structured exercise training programme versus hypocaloric hyperproteic diet in obese polycystic ovary syndrome patients with anovulatory infertility: A 24-week pilot study. *Hum Reprod*. 2008;23(3):642-650.

66. Kort JD, Winget C, Kim SH, Lathi RB. A retrospective cohort study to evaluate the impact of meaningful weight loss on fertility outcomes in an overweight population with infertility. *Fertil Steril*. 2014;101(5):1400-1403.

67. Clark AM, Thornley B, Tomlinson L, Galletley C, Norman RJ. Weight loss in obese infertile women results in improvement in reproductive outcome for all forms of fertility treatment. *Hum Reprod*. 1998;13(6):1502-1505.

68. Clark AM, Ledger W, Galletly C, et al. Weight loss results in significant improvement in pregnancy and ovulation rates in anovulatory obese women. *Hum Reprod*. 1995;10(10):2705-2712.

69. Sim KA, Dezarnaulds GM, Denyer GS, Skilton MR, Caterson ID. Weight loss improves reproductive outcomes in obese women undergoing fertility treatment: A randomized controlled trial. *Clin Obes*. 2014;4(2):61-68.

70. Mutsaerts MA, Kuchenbecker WK, Mol BW, Land JA, Hoek A. Dropout is a problem in lifestyle intervention programs for overweight and obese infertile women: A systematic review. *Hum Reprod*. 2013;28(4):979-986.

71. Thangaratinam S, Rogozinska E, Jolly K, et al. Effects of interventions in pregnancy on maternal weight and obstetric outcomes: Meta-analysis of randomised evidence. *BMJ*. 2012;344:e2088.

72. Brown MJ, Sinclair M, Liddle D, Hill AJ, Madden E, Stockdale J. A systematic review investigating healthy lifestyle interventions incorporating goal setting strategies for preventing excess gestational weight gain. *PLoS One*. 2012;7(7):e39503.

73. Renault KM, Norgaard K, Nilas L, et al. The treatment of obese pregnant women (TOP) study: A randomized controlled trial of the effect of physical activity intervention assessed by pedometer with or without dietary intervention in obese pregnant women. *Am J Obstet Gynecol*. 2014;210(2):134.e1-134.e9.

74. Claesson IM, Sydsjo G, Brynhildsen J, et al. Weight gain restriction for obese pregnant women: A case-control intervention study. *BJOG*. 2008;115(1):44-50.

75. Asbee SM, Jenkins TR, Butler JR, White J, Elliot M, Rutledge A. Preventing excessive weight gain during pregnancy through dietary and lifestyle counseling: A randomized controlled trial. *Obstet Gynecol*. 2009;113(2 Pt 1):305-312.

76. Guelinckx I, Devlieger R, Mullie P, Vansant G. Effect of lifestyle intervention on dietary habits, physical activity, and gestational weight gain in obese pregnant women: A randomized controlled trial. *Am J Clin Nutr*. 2010;91(2):373-380.

77. Jeffries K, Shub A, Walker SP, Hiscock R, Permezel M. Reducing excessive weight gain in pregnancy: A randomised controlled trial. *Med J Aust*. 2009;191(8):429-433.

78. Quinlivan JA, Lam LT, Fisher J. A randomised trial of a four-step multidisciplinary approach to the antenatal care of obese pregnant women. *Aust N Z J Obstet Gynaecol*. 2011;51(2):141-146.

79. Thornton YS, Smarkola C, Kopacz SM, Ishoof SB. Perinatal outcomes in nutritionally monitored obese pregnant women: A randomized clinical trial. *J Natl Med Assoc*. 2009;101(6):569-577.

80. Wolff S, Legarth J, Vangsgaard K, Toubro S, Astrup A. A randomized trial of the effects of dietary counseling on gestational weight gain and glucose metabolism in obese pregnant women. *Int J Obes (Lond)*. 2008;32(3):495-501.

81. Kumar P, Khan K. Effects of metformin use in pregnant patients with polycystic ovary syndrome. *J Hum Reprod Sci*. 2012;5(2):166-169.

82. Vinter CA, Jensen DM, Ovesen P, Beck-Nielsen H, Jorgensen JS. The LiP (lifestyle in pregnancy) study: A randomized controlled trial of lifestyle intervention in 360 obese pregnant women. *Diabetes Care*. 2011;34(12):2502-2507.

83. Ostbye T, Krause KM, Swamy GK, Lovelady CA. Effect of breastfeeding on weight retention from one pregnancy to the next: Results from the North Carolina WIC program. *Prev Med*. 2010;51(5):368-372.

84. Gunderson EP, Abrams B. Epidemiology of gestational weight gain and body weight changes after pregnancy. *Epidemiol Rev*. 1999;21(2):261-275.

85. Weng HH, Bastian LA, Taylor DH Jr, Moser BK, Ostbye T. Number of children associated with obesity in middle-aged women and men: Results from the health and retirement study. *J Womens Health (Larchmt)*. 2004;13(1):85-91.

86. Amorim AR, Rossner S, Neovius M, Lourenco PM, Linne Y. Does excess pregnancy weight gain constitute a major risk for increasing long-term BMI? *Obesity (Silver Spring)*. 2007;15(5):1278-1286.

87. Rooney BL, Schauberger CW, Mathiason MA. Impact of perinatal weight change on long-term obesity and obesity-related illnesses. *Obstet Gynecol*. 2005;106(6):1349-1356.

88. Rasmussen KM. Association of maternal obesity before conception with poor lactation performance. *Annu Rev Nutr*. 2007;27:103-121.

89. Turcksin R, Bel S, Galjaard S, Devlieger R. Maternal obesity and breastfeeding intention, initiation, intensity and duration: A systematic review. *Matern Child Nutr*. 2014;10(2):166-183.

90. Soini S, Mustajoki P, Eriksson JG. Lifestyle-related factors associated with successful weight loss. *Ann Med*. 2015;47(2):88-93.

91. Nascimento SL, Pudwell J, Surita FG, Adamo KB, Smith GN. The effect of physical exercise strategies on weight loss in postpartum women: A systematic review and meta-analysis. *Int J Obes (Lond)*. 2014;38(5):626-635.

92. Amorim Adegboye AR, Linne YM. Diet or exercise, or both, for weight reduction in women after childbirth. *Cochrane Database Syst Rev*. 2013;7:CD005627.

93. van der Pligt P, Willcox J, Hesketh KD, et al. Systematic review of lifestyle interventions to limit postpartum weight retention: Implications for future opportunities to prevent maternal overweight and obesity following childbirth. *Obes Rev*. 2013;14(10):792-805.

94. Walker LO, Im EO, Vaughan MW. Communication technologies and maternal interest in health-promotion information about postpartum weight and parenting practices. *J Obstet Gynecol Neonatal Nurs*. 2012;41(2):201-215.

95. Colleran HL, Lovelady CA. Use of MyPyramid menu planner for moms in a weight-loss intervention during lactation. *J Acad Nutr Diet*. 2012;112(4):553-558.

96. Bertz F, Brekke HK, Ellegard L, Rasmussen KM, Wennergren M, Winkvist A. Diet and exercise weight-loss trial in lactating overweight and obese women. *Am J Clin Nutr*. 2012;96(4):698-705.

97. Haapala I, Barengo NC, Biggs S, Surakka L, Manninen P. Weight loss by mobile phone: A 1-year effectiveness study. *Public Health Nutr*. 2009;12(12):2382-2391.

98. Olson CM, Strawderman MS, Reed RG. Efficacy of an intervention to prevent excessive gestational weight gain. *Am J Obstet Gynecol*. 2004;191(2):530-536.

99. Maggard MA, Yermilov I, Li Z, et al. Pregnancy and fertility following bariatric surgery: A systematic review. *JAMA*. 2008;300(19):2286-2296.

100. Shekelle PG, Newberry S, Maglione M, et al. Bariatric surgery in women of reproductive age: Special concerns for pregnancy. *Evid Rep Technol Assess (Full Rep)*. 2008;169:1-51.

101. American College of Obstetricians and Gynecologists. ACOG practice bulletin no. 105: Bariatric surgery and pregnancy. *Obstet Gynecol*. 2009;113(6):1405-1413.

102. Johansson K, Cnattingius S, Naslund I, et al. Outcomes of pregnancy after bariatric surgery. *N Engl J Med*. 2015;372(9):814-824.

103. Willis K, Lieberman N, Sheiner E. Pregnancy and neonatal outcome after bariatric surgery. *Best Pract Res Clin Obstet Gynaecol*. 2015;29(1):133-144.

104. Amsalem D, Aricha-Tamir B, Levi I, Shai D, Sheiner E. Obstetric outcomes after restrictive bariatric surgery: What happens after 2 consecutive pregnancies? *Surg Obes Relat Dis*. 2014;10(3):445-449.

105. Shai D, Shoham-Vardi I, Amsalem D, Silverberg D, Levi I, Sheiner E. Pregnancy outcome of patients following bariatric surgery as compared with obese women: A population-based study. *J Matern Fetal Neonatal Med*. 2014;27(3):275-278.

106. Bennett WL, Gilson MM, Jamshidi R, et al. Impact of bariatric surgery on hypertensive disorders in pregnancy: Retrospective analysis of insurance claims data. *BMJ*. 2010;340:c1662.

107. Aricha-Tamir B, Weintraub AY, Levi I, Sheiner E. Downsizing pregnancy complications: A study of paired pregnancy outcomes before and after bariatric surgery. *Surg Obes Relat Dis*. 2012;8(4):434-439.

108. Roos N, Neovius M, Cnattingius S, et al. Perinatal outcomes after bariatric surgery: Nationwide population based matched cohort study. *BMJ*. 2013;347:f6460.

109. Kjaer MM, Lauenborg J, Breum BM, Nilas L. The risk of adverse pregnancy outcome after bariatric surgery: A nationwide register-based matched cohort study. *Am J Obstet Gynecol*. 2013;208(6):464.e1-464.e5.

110. Lesko J, Peaceman A. Pregnancy outcomes in women after bariatric surgery compared with obese and morbidly obese controls. *Obstet Gynecol*. 2012;119(3):547-554.

111. Wolf R, Witkop CT, Miller T, Syed S. Folic acid supplementation for the prevention of neural tube defects: An update of the evidence for the U.S. Preventive Services Task Force. *Ann Intern Med*. 2009;150(9):623-639.

112. Centers for Disease Control and Prevention. Effectiveness in disease and injury prevention use of folic acid for prevention of spina bifida and other neural tube defects—1983-1991. *MMWR Morb Mortal Wkly Rep*. 1991;49(30):513-516.

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