from the association

Position of the American Dietetic Association and American Society for Nutrition: Obesity, Reproduction, and Pregnancy Outcomes

ABSTRACT

Given the detrimental influence of maternal overweight and obesity on reproductive and pregnancy outcomes for the mother and child, it is the position of the American Dietetic Association and the American Society for Nutrition that all overweight and obese women of reproductive age should receive counseling on the roles of diet and physical activity in reproductive health prior to pregnancy, during pregnancy, and in the interconceptional period, in order to ameliorate these adverse outcomes. The effect of maternal nutritional status prior to pregnancy on reproduction and pregnancy outcomes is of great public health importance. Obesity in the United States and worldwide has grown to epidemic proportions, with an estimated 33% of US women classified as obese. This position paper has two objectives: (a) to help nutrition professionals become aware of the risks and possible complications of overweight and obesity for fertility, the course of pregnancy, birth outcomes, and short- and long-term maternal and child health outcomes; and (b) related to the commitment to research by the American Dietetic Association and the American Society for Nutrition, to identify the gaps in research to improve our knowledge of the risks and complications associated with being overweight and obese before and during pregnancy. Only with an increased knowledge of these risks and complications can health care professionals develop effective strategies that can be implemented before and during pregnancy

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POSITION STATEMENT

Given the detrimental influence of maternal overweight and obesity on reproductive and pregnancy outcomes for the mother and child, it is the position of the American Dietetic Association and the American Society for Nutrition that all overweight and obese women of reproductive age should receive counseling prior to pregnancy, during pregnancy, and in the interconceptional period on the roles of diet and physical activity in reproductive health, in order to ameliorate these adverse outcomes.

besity in pregnancy carries with it not just increased risks for the pregnant woman during gestation, but also risks for the future health of the child, or, in public health terms, the health of the next generation. The long-term goal of health care professionals must be to reduce the proportion of women who are obese during the reproductive period and increase public awareness about the importance of a healthful lifestyle (healthful diet, moderate to vigorous levels of physical activity, and emotional well-being) before and during pregnancy. In accordance with a recent recommendation by the American College of Obstetrics and Gynecology (ACOG) (1), the American Dietetic Association (ADA) and the American Society for Nutrition recommend that preconceptional and interconceptional counseling about possible complications associated with obesity and how to prevent those problems be available to all women of reproductive age.

Members of ADA work in various settings that provide care to women of

reproductive age. Thus, the first objective of this position paper is for registered dietitians: dietetic technicians, registered; and other health care professionals to become aware of the risks and possible complications of overweight and/or obesity for fertility, the course of pregnancy, birth outcomes, and short- and long-term maternal and child health outcomes. The second objective, related to the commitment to research by the ADA and the American Society for Nutrition, is to identify the research gaps that need to be filled to improve our understanding of the risks and complications associated with being overweight or obese before and during pregnancy. Only with an increased understanding of these risks and complications can health care professionals develop effective strategies that can be implemented prior to and during pregnancy, as well as during the interconceptional period, to ameliorate adverse outcomes.

CONTEXT FOR THIS POSITION STATEMENT

The effect of maternal nutritional status prior to pregnancy on reproduction and pregnancy outcomes is of great public health importance and has been extensively studied over time. A woman's prepregnancy weight has been used as a marker of nutritional status. Being underweight, defined as a body mass index (BMI; calculated as weight kg/m^2) less than 18.5, may reflect chronic nutritional deficiency, whereas a high BMI (>25) reflects an imbalance between energy intake and expenditure, and thus varying degrees of adiposity. The National Institutes of Health and the International Obesity Task Force have defined overweight (or preobese) as a BMI of 25 to 29.9, and obese as a BMI of 30 or more (2). In most clinical

settings and epidemiological studies, BMI is used to estimate adiposity because of its strong correlation with fat mass as measured by hydrodensitometry and dual-energy x-ray absorptiometry. It has limitations, however, in that it does not account for differences that may exist in fat mass by sex, age, and race/ethnicity (3).

Obesity in the United States and worldwide has grown to epidemic proportions. The latest data from the National Health and Nutrition Examination Survey (1999-2004) indicate that among nonpregnant women age 20 to 39 years, approximately 25% are overweight and 28% are obese, of whom 6% are considered extremely obese (BMI \geq 40) (4). Among adolescent girls, 12 to 19 years old, approximately 30% are at or above the 85th percentile of BMI-for-age and 16.4% are considered obese (4). On average, obesity among women of all ages seems to have peaked at 33%, with no appreciable increase between 1999-2000 and 2003-2004 (4). Overweight and obesity are associated with many comorbidities that affect a woman's health, including reduced fertility (5).

In any given year, approximately 4 million women in the United States become pregnant. Among women who become pregnant, the shift toward higher prepregnancy weight in recent years is evident (6). Obesity during pregnancy has been associated with gestational diabetes, gestational hypertension, pre-eclampsia, birth defects, Cesarean delivery, fetal macrosomia, perinatal deaths, postpartum anemia, and childhood obesity (7,8). Not only are more women beginning pregnancy with high BMIs, but more are also gaining in excess of the 1990 Institute of Medicine (IOM) recommendations for gestational weight gain (8,9).* This excessive weight gain compounds the pregnancy complications mentioned previously. It is particularly problematic for overweight and obese women for whom the optimal range of weight gain is

*The 1990 Institute of Medicine report uses the following body mass index cut points to define weight status groups which are different from the National Heart, Lung, and Blood Institute; <19.8 underweight, >19.8 to 26 normal weight, >26 to 29 overweight, and >29 obese. uncertain because there were limited data in 1990 when the guidelines were created and, as such, the recommended range of weight gain (15 to 25 lb for overweight and at least 15 lb for obese) is exceeded by most overweight and obese women. It is also uncertain if overweight and obese adolescents should gain in the upper range of the recommendation (10). Furthermore, overweight and obese women are more likely to maintain excess weight after delivery (8). In affluent countries, many women retain some weight with each successive pregnancy, gaining more weight than their nonpregnant counterparts (8). Those who gain more weight during pregnancy are more likely to retain more weight and continue on a higher weight trajectory throughout their lifetime compared with women who gain less weight (8). Weight gain during pregnancy has also been shown to have implications for the child's future risk of being overweight (8).

THE EFFECT OF OVERWEIGHT AND OBESITY ON FERTILITY AND CONCEPTION

Obesity is a state of excess adipose tissue, which is critical in controlling the regulation of sex hormone availability due to its ability to store lipid steroids such as androgens. Estrogen production and the concentration of sex hormone-binding globulin in the blood are correlated with various measures of body fat. There also seems to be a strong association between obesity and insulin resistance, which is thought to reduce fertility (5).

Body weight and composition are believed to play an important role in pubertal maturation, with leptin also being important in this biological process (11). Excessive weight gain at younger ages is associated with earlier menarche (12,13) and both high absolute weight and change in weight are associated with menstrual problems. Obesity in adolescence and young adulthood, as opposed to during infancy, is more strongly associated with amenorrhea, oligomenorrhea, and long menstrual cycles (14,15).

There is some evidence of increased time to conception for obese compared with normal-weight women (16, 17), particularly among women who smoke cigarettes (18). Obesity is also a strong risk factor for polycystic ovarian syndrome, which results in menstrual irregularities and chronic anovulation. The central distribution of fat, as measured by waist-to-hip ratio, is also related to reproductive functioning, with higher rates of infertility associated with higher waistto-hip ratios (19,20).

It is estimated that 25% of ovulatory infertility in the United States may be attributable to overweight and obesity among women of reproductive age (16). The adverse effect of obesity on conception is manifested even among women who seek assisted reproductive technology, with obese women manifesting lower implantation and pregnancy rates, as well as higher miscarriage rates and increased pregnancy complications (14).

THE EFFECT OF OVERWEIGHT AND OBESITY ON PREGNANCY OUTCOMES

Maternal Complications during Pregnancy

During pregnancy, numerous metabolic adjustments occur to increase the availability of energy, nutrients, and oxygen to the developing fetus. In nonobese women, these metabolic adjustments pose no increased risk for complications. However, in obese women, who already have aberrations in glucose and lipid metabolism, the further adjustments induced by hormonal changes in pregnancy create a metabolic milieu that enhances the risk for metabolic disorders. such as gestational diabetes mellitus (GDM) and pre-eclampsia. The greater the degree of maternal obesity, the higher the risk of developing these metabolic disorders (21). For example, the risk of GDM is increased twofold in overweight compared with normal-weight women, and it is increased eightfold in the severely obese (BMI>40) (22). Pre-eclampsia is approximately twice as prevalent in overweight women (BMI 25 to 30) and approximately three times as high in obese women (BMI \geq 30) (21,23). Pre-eclampsia is more common in obese women with GDM than in women without GDM. The coexistence of these two metabolic disorders suggests a similarity in the underlying biological mechanisms. Tight glucose control in women with GDM seems to reduce the risk for preeclampsia (23). Surveys show that GDM tends to occur more frequently in Asian, Hispanic, and Native American women than in African-American and white women (24-26).

Because maternal obesity is a risk factor for GDM, obese women are usually screened for glucose intolerance early in prenatal care. Initially, a 50-g oral glucose challenge (ie, the glucose challenge test) is given and plasma glucose values are measured 1 hour later. If the values exceed a cutoff, usually between 130 and 140 mg/dL (7.2 and 7.8 mmol/L), an oral glucose tolerance test is done. A diagnosis of GDM is made if two or more plasma glucose values exceed established cutoffs (27). If one postload plasma glucose level exceeds the standard, glucose tolerance may be considered to be impaired, but usually no counseling or treatment is given. Evidence is accumulating, however, that one abnormal glucose value is associated with complications similar to those seen with GDM (28).

Normally, the insulin sensitivity of peripheral tissue decreases approximately 50% to 60% in late pregnancy in lean women; the decrease is greater in obese women (23). Thus, hyperinsulinemia is common in all pregnant women, with higher levels seen in overweight and obese women. If pancreatic insulin secretion is adequate, women will remain glucose tolerant throughout gestation and GDM does not develop. It is thought that the shift toward reduced insulin sensitivity (or increased insulin resistance) during late pregnancy occurs to limit maternal glucose utilization and conserve it for diffusion across the placenta to the fetus. Maternal hyperinsulinemia also enhances the rate of maternal adipocyte fat oxidation, which releases more fatty acids into circulation for use as a fuel source by the mother (29). The free fatty acids are converted to triglycerides in the liver and returned to circulation as very-low-density lipoproteins, resulting in high verylow-density lipoprotein concentrations in late pregnancy (30). This dyslipidemia usually disappears after delivery.

It is not unusual for a mild inflammatory state to occur in obese pregnant women with glucose intolerance (31) because proinflammatory cytokines (ie, interleukin-6 and tumor necrosis factor- α) are produced by the placenta as well as adipose tissue (32). Research suggests that these proinflammatory cytokines may contribute to the decrease in insulin sensitivity seen in obese women with GDM (31).

The risk of GDM in obese women may be reduced by increasing noninsulin-mediated glucose use by peripheral tissues, primarily skeletal muscle (23) by increasing physical activity. An increased use of large skeletal muscles during physical activity may be beneficial. Also, a high-fiber and high—complex carbohydrate diet or a low—glycemic index diet may reduce the insulin need after a meal, and theoretically decrease beta cell failure, but efficacy data are limited.

Gestational hypertension and preeclampsia are also more common in overweight and obese pregnant women. Gestational hypertension, defined as a systolic blood pressure of at least 140 mm Hg or a diastolic blood pressure of at least 90 mm Hg, affects approximately 6% to 17% of nulliparous women and 2% to 4% of multiparous women (33). Approximately 50% of women with gestational hypertension diagnosed before 30 weeks' gestation develop pre-eclampsia, a syndrome involving gestational hypertension plus proteinuria (34). The cause of pre-eclampsia is unknown, but it is currently thought to be related to an inadequate placental blood supply, possibly due to maternal hypertension, which causes placental oxidative stress and the release into maternal circulation of placental factors that trigger an inflammatory response. Because subclinical inflammation is more common in obese individuals, obese women may enter pregnancy with pre-existing inflammation that enhances their risk for pre-eclampsia.

Maternal Complications in the Peripartum Period

Cesarean deliveries and associated morbidities are more common among obese women. For example, in one large multicenter trial of overweight and obese women, the Cesarean delivery rate was 30% for nulliparous women with a BMI less than 30, 34% for those with a BMI of 30 to 34.9, and 48% for women with a BMI of 35 to 39.9 (35). The effect of weight loss between two deliveries on the risk of a subsequent Cesarean delivery has not been carefully assessed (23). After Cesarean delivery, overweight or obese women have more postoperative complications, such as wound infection/breakdown, excessive blood loss, deep venous thrombophlebitis, and postpartum endometritis than do normal-weight women. The length of labor also is longer in overweight and obese women (36).

Although high BMIs are generally associated with higher hemoglobin levels during pregnancy, they are associated with an increased risk of postpartum anemia (37). It is thought that these inconsistent findings are due to the higher prevalence of postpartum hemorrhage and abdominal deliveries among obese women. Macrosomia may also cause significant postpartum blood loss by causing perineal rupture and hemorrhage and lengthening the period of vaginal discharge of blood after delivery.

Birth Outcomes

Infants born to obese mothers have a higher prevalence of congenital anomalies than do offspring of normal-weight women, suggesting that maternal adiposity alters development in the sensitive embryonic period (30). In a study of 10,240 US women enrolled in the National Birth Defects Prevention Study, 1997-2002, the odds ratio of structural birth defects ranged from 1.3 to 2.1 among obese compared with nonobese mothers (7) Neural tube defects (NTDs) are approximately twice as high among children of obese women, with spina bifida being more common than anencephaly (7). Other birth defects more frequent in offspring of obese women include oral clefts, heart anomalies, hydrocephaly, and abdominal wall abnormalities. The underlying metabolic basis for increased anomalies in obese women is not known. It is thought that poor glycemic control may play a role. Consuming a diet high in sucrose and other high-glycemic foods increased the risk of NTDs by twofold in women among all weight groups, but the risk was fourfold among obese women (BMI >29) (38). Although low folic acid status has been associated with NTDs, the relationship between maternal obesity and NTDs persists after controlling for self-reported folic acid intakes, suggesting that other factors such as poor glycemic control contribute to congenital anomalies in obese women (30).

Recently, maternal obesity has emerged as a risk factor for intrauterine fetal death and stillbirth (death of fetus before onset of labor) (13). In a Canadian study of more than 84,000 women, a maternal pregravid body weight more than 68 kg increased the risk of fetal death by 2.9-fold after adjusting for age, diabetes, and hypertensive disorders (39). Also, a systematic review of articles on risk factors for antepartum stillbirth reported a threefold increased risk for stillbirth among obese women after adjusting for age, parity, maternal diabetes, and hypertension as well as a number of social factors (40). It is thought that the relatively recent increase in antepartum stillbirth in developed countries may be linked to maternal age or obesity because both of those factors influence metabolic adjustments to pregnancy.

The incidence of preterm birth tends to decrease, rather than increase, with increasing pregravid BMI once medical inductions are accounted for in the analysis (23). An independent effect of maternal obesity on preterm labor or premature rupture of membranes has not been identified after controlling for underlying medical or obstetric issues. Some have even suggested that maternal obesity protects against spontaneous preterm labor (41). The risk of having a small-for-gestational age full-term baby (ie, newborn with weight <10th percentile for gestational age) tends to decrease with increasing maternal BMI, whereas the risk of having a large-for-gestational age (LGA) baby (>90th percentile weight-for-gestational age) increases by approximately 60% compared with normal-weight women (42) Although pregestational diabetes has a greater effect on the frequency of LGA than maternal obesity does (42), more of the LGA babies are born to obese women than women with diabetes because maternal obesity is more prevalent than diabetes (43). Several countries have reported an increase in mean birth weight over the past decade and an increase in the proportion of large babies (44). One explanation for this increase could be the increase in maternal BMI over the same time period. No consistent effect of maternal BMI on infant birth length has been reported.

EFFECTS OF MATERNAL OVERWEIGHT/ OBESITY ON SHORT- AND LONG-TERM CHILD HEALTH STATUS

Using a lifecourse approach, it has been shown that maternal pregravid weight has an early and persistent effect on childhood overweight status as well as a dynamic effect on the process of overweight development (33,45). Most studies that have examined maternal pregravid BMI and childhood weight status have found a positive association with adjusted odds ratios ranging from two to four (46-49).

There is some evidence of an association between maternal overweight or obesity and decreased rates of breastfeeding. Breastfeeding has been well-demonstrated to have many protective effects against childhood morbidities, including the development of obesity later in life (45,50-53). Specifically, a high BMI before conception has been shown to be inversely related to the successful initiation of breastfeeding, the duration of lactation, and the amount of milk produced (54). This is especially problematic given the finding from the 1996 National Longitudinal Survey of Youth (47), which showed that children whose mothers were obese prior to pregnancy and who were never breastfed had a six times greater risk of being overweight compared with children whose mothers were normal weight and who breastfed for at least 4 months. The mechanisms by which overweight and obesity adversely affect lactation performance include mechanical difficulties associated with latching on and proper positioning of the infant; the high Cesarean section rates among this subpopulation, which delays the onset of first suckling; and a lower prolactin response to suckling at 48 hours and more after delivery, which may compromise milk production and, over time, lead to early cessation of lactation (55).

There is some evidence linking maternal overweight and obesity, independent of GDM, to the development of the metabolic syndrome (obesity, hypertension, dyslipidemia, and glucose intolerance) in the offspring. In a cohort of 84 children who were LGA and 95 who were appropriate-for-gestational age at birth, Boney and colleagues (56) showed that the risk of having two or more components of the metabolic syndrome at age 11 was 1.81 (95% confidence interval=1.03, 3.19), if the mother was obese prior to pregnancy.

Overall, these findings on the effect of maternal overweight and obesity on long-term child health status have grave implications for perpetuating the cycle of obesity and its correlates in subsequent generations.

INTERVENTIONS TO REDUCE OBESITY-RELATED PROBLEMS PRIOR TO AND DURING PREGNANCY

Weight loss seems to improve menstrual functioning, ovulation, and infertility in obese women (5,17). Even among women with polycystic ovarian syndrome, as little as a 5% reduction in weight has been associated with improved fertility (5). However, maintaining this weight loss can pose problems for women of childbearing age. Women who have reported restrained eating, dieting, and or weight cycling prior to pregnancy have been shown to gain more weight during pregnancy than those who do not report these behaviors (57).

Among morbidly obese women, bariatric surgery is becoming more common and its effect on reproductive outcomes is emerging. After surgery, women are instructed to consume 1,000 to 1,200 kcal/day and to take a daily multivitamin and some additional form of a vitamin B-12 supplement. Those who are interested in becoming pregnant are told to wait until their weight loss stabilizes, after approximately 12 to 18 months, and to seek care from an obstetrician specializing in high-risk pregnancy. Case series studies report improved pregnancy and fertility rates with weight losses in the range of 10.6 to 44 kg (58). Complications of bariatric surgery that are particularly worrisome during pregnancy include vomiting, malabsorption of several nutrients, and inadequate pregnancy weight gain, which may have adverse effects on the fetus. Two studies using laparoscopic adjustable gastric banding found mean weight gains of approximately 10 kg, mean birth weights of 3 to 3.5 kg, and prevalence of pregnancyinduced hypertension in the range of 7% to 10%, and prevalence of gestational diabetes from 6% to 16% (59,60). In the one study that had a comparison group (59), women after the banding had lower gestational weight gains, lower incidence of gestational diabetes and hypertension, but no difference in mean birth weight compared with a matched obese cohort. In another study that used biliopancreatic diversion with duodenal switch, among 109 women who became pregnant postoperatively, 90 reported weight gains of 9.1 ± 5.9 kg, eight reported weight loss during pregnancy, and 11 reported no weight gain (61). The study reported lower incidence of macrosomia but higher small-for-gestational age when comparing birth outcomes for all women before and after surgery. Lastly, one study used laparascopic Roux-en-Y gastric bypass and examined birth outcomes of 21 women who became pregnant within 1 year of surgery compared with 13 women who delayed pregnancy until after 1 year. They found no differences in mode of delivery, pregnancy complications, or birth weights; however, mean gestational weight gain was significantly lower in the group that became pregnant within 1 year (4 vs 34 lb) (62). Clearly, more studies are warranted to determine if perinatal outcomes are affected among women who become pregnant after using this approach for long-term weight loss. Furthermore, long-range outcomes of these infants also warrant careful study.

Other weight-loss practices for women of reproductive age have been covered in previous ADA positions— "Weight Management" and "Nutrition and Women's Health" (63,64). The ACOG recommends that obese women undertake a weight-loss program before pregnancy (65). That would be ideal, but it is unlikely that many women who have been overweight or obese will reach normal weight prior to conception. Thus, some intervention during pregnancy merits consideration.

The ACOG (1) recommends determining pre- or early pregnancy BMI at the first prenatal visit and then recommending a pregnancy weight gain within the IOM guidelines for each BMI category (9). There is some epidemiological evidence suggesting that women are more likely to gain within the IOM guidelines if their health care provider makes this recommendation (66). Four groups have studied behavioral interventions to prevent excessive weight gain in pregnancy (67-69). The studies were not done exclusively in overweight or obese women. One of the four studies. a community-based intervention (69), failed to find an effect of the intervention on gestational weight gain. Two of the three interventions were based in the United States (67.70). In Pollev and colleagues' study (67), normalweight women in the intervention group had lower percentages that exceed the IOM recommendation; however, the intervention did not work for overweight/obese women. In the Olson and colleagues study (70), a significant effect was seen only among the low-income women; adjusted odds ratio=0.41 (95% confidence interval=0.20, 0.81) for excessive gestational weight gain associated with the intervention. The fourth study, done in Finland, found that women increased their intakes of fruit, vegetables, and high-fiber bread, but the prevalence of excessive weight gain was not reduced (68). Clearly, more work is warranted in this area.

Only two groups have initiated intervention programs to limit weight gain, not just prevent excessive gain, in obese pregnant women (71,72). Artal and colleagues (71) studied a dietary intervention, with or without exercise, to restrict weight gain in obese women with GDM. No control group was included in the study. Weight gain averaged 0.3 kg/week during the last trimester in the diet group and 0.1 kg/week in the diet with exercise group. The 1990 IOM recommended that overweight women (BMI 26 to 29) should gain approximately 0.3 kg/week (9). Thus, the investigators achieved that goal with the diet intervention and reduced the rate of gain further when exercise was added. When the women were divided into two groups-those who gained and those who lost weight or had no weight change-no differences in the measured pregnancy and fetal outcomes were observed. Claesson and colleagues (72) initiated an intervention with weekly counseling and aqua aerobic classes in 155 obese women (BMI \geq 30) and 193 control subjects. The intervention women gained less weight than the control

subjects during pregnancy (8.7 vs 11.3 kg) and weighed less at the postpartum check-up. Birth weight did not differ between the two groups in this small study. These two studies suggest that intervention programs initiated during pregnancy can control weight gain in obese women, but further studies are needed in larger samples of women, particularly to evaluate possible adverse outcomes.

Interventions to Prevent Metabolic Disorders in Obese Women

As mentioned earlier, obese women have an increased risk for gestational diabetes, gestational hypertension, and pre-eclampsia. Interventions involving dietary components or nutrient supplements have been used to prevent these disorders in pregnancy, although the studies have not been done exclusively in obese women. Because these problems are common in obese women, a brief review follows.

The role of dietary carbohydrate intakes in reducing the risk of GDM or glucose intolerance has been investigated by several groups. In an observational study, Saldana and colleagues (73) found that higher carbohydrate intakes, presumably from refined carbohydrate sources, increased the risk of glucose intolerance in nondiabetic women. Intervention studies also show that reducing refined carbohydrates decreases glucose intolerance. Fraser and colleagues (74) reported that postprandial insulin responses were attenuated in nonobese pregnant women consuming more than 50 g of fiber per day, compared with control subjects' consumption of approximately 12 g of fiber per day. The authors concluded that the usual increase in plasma insulin levels after meals among pregnant women in the Western hemisphere are an unphysiological response to dietary fiber depletion. In a small study of 12 lean women, Clapp (75) compared the effect on glycemic control of either a low- or high-glycemic index diet consumed from before conception to term. The amount of carbohydrate was similar in both groups, but at late pregnancy the glycemic response to a meal in the low-glycemic diet group was similar to prepregnancy values, whereas it was nearly doubled in the high-glycemic diet group. Bronstein and colleagues (76) compared the serum glucose and insulin response to low- and high-glycemic test meals and found that the response of both was lower with low glycemic meals in *both* lean and obese women. Epidemiologic data from the Nurses' Health Study also showed that a low-glycemic, highcereal fiber diet reduced the risk for GDM by approximately one half (77).

In addition to modifications in the type of carbohydrate, adjustments in the type of fat may also reduce glucose intolerance and GDM. In a study of 171 pregnant Chinese women, increased body weight, decreased polyunsaturated fat intake, and a low ratio of dietary polyunsaturated to saturated fat independently predicted glucose intolerance (78). Bo and colleagues (79) also found that glucose intolerance in pregnant women without conventional risk factors (eg, family history, age, and BMI) was increased with high intakes of saturated fat and reduced with high intakes of polyunsaturated fat. Further studies are needed in obese women, but these data suggest that reducing the glycemic load and increasing cereal fiber and polyunsaturated fatty acid intakes may reduce GDM or glucose intolerance during pregnancy.

Nutritional interventions to reduce the risk of gestational hypertension or pre-eclampsia have primarily involved nutrient supplements. Several calcium supplementation trials have been done, and the results were reviewed in a recent meta-analysis (80). It was thought that supplemental calcium might prevent pre-eclampsia because low levels of cellular calcium cause vasoconstriction by stimulating release of either parathyroid hormone or renin. Data show that calcium supplementation is effective among women with low calcium intakes; calcium reduced the risk of pre-eclampsia by approximately 50% without any adverse effects. No benefit has been observed in women with adequate calcium intakes.

Antioxidant supplementation has also been tested as a means to prevent pre-eclampsia. However, two randomized controlled trials providing 1,000 mg vitamin C and 400 IU of vitamin E from the first or second trimesters to term were not beneficial (81,82). Initiating the intervention prior to conception or very early in pregnancy when the placenta is developing may be necessary. n-3 fatty acids reduce blood pressure in nonpregnant individuals, but attempts to reduce blood pressure with n-3 fatty acid supplements in pregnancy have yielded contradictory results (83). Salt restriction was recommended for several years to prevent edema and/or pre-eclampsia, but a recent review of its efficacy found no benefit (84). Thus, except for the beneficial effects on the risk of pre-eclampsia, as seen with supplemental calcium in women with low calcium intakes, none of the other nutritional interventions have proven to be efficacious.

Because the marked increase in lipid and lipoprotein components in late pregnancy may induce proatherogenic changes in the fetal aorta, the effect of a cholesterol-lowering diet on maternal, cord, and neonatal lipids and on pregnancy outcomes was studied in a group of nonobese (mean BMI=24) Norwegian women (85). The dietary intervention (which promoted consumption of fish, low-fat meats and dairy products, whole grains, and fruits and vegetables from mid-pregnancy to term), reduced maternal total and low-density lipoprotein cholesterol levels but did not alter cord and neonatal levels. The marked reduction in the incidence of preterm deliveries in the intervention group suggests that further studies of the role of diet on preterm deliveries are needed.

Although the data are sparse, there is some evidence that moderate physical activity throughout gestation reduces the risk of GDM and preeclampsia by nearly one half (86). Physical activity activates the enzyme AMPK (adenosine monophosphate-activated protein kinase), which increases glucose transport into muscle, enhances fat oxidation, and reduces insulin resistance (87). Also, physical activity may reduce the risk of pre-eclampsia by reducing circulating levels of inflammatory cytokines (88). Women who exercised throughout pregnancy (eg, performing endurance exercises four or more times per week) gained significantly less fat and had significantly lower increases in tumor necrosis factor- α and leptin during gestation (89). Moderate, consistent physical activity may be an effective way to reduce both subclinical inflammation and insulin resistance, two features of metabolic disorders in obese pregnant women. Once again, no intervention studies using physical activity to prevent metabolic disorders have been done with obese pregnant women.

EMPIRICAL RESEARCH NEEDED TO FILL GAPS

We identified the following research gaps that are critical to advancing our understanding of the influence of being overweight or obese on reproduction and pregnancy outcomes. Increased understanding would help guide appropriate nutrition counseling prior to and during pregnancy, as well as in the postpartum and interconceptional periods.

Mechanisms by which body weight influences reproductive function and performance are not well-understood and need further clarification. Maternal subclinical inflammation and vascular dysfunction are associated with many of the complications occurring during pregnancy in overweight and obese women. In addition, the effect of maternal BMI status, insulin resistance, and inflammation on placental function and birth outcomes needs to be examined in greater detail. Finally, the roles of maternal diet and physical activity before and during gestation on these metabolic disorders need further elucidation before standards of clinical and dietary care can be established.

The Dietary Reference Intakes for total energy indicate that for a healthful birth outcome, pregnant women should, on average, consume an extra 340 kcal/day in the second trimester and 452 kcal/day in the third trimester (90). Whether this energy prescription is suitable for overweight and obese women is unknown. Research is needed in this area to support dietary recommendations that result in appropriate weight gain for overweight and obese pregnant women. There also seems to be a lack of research related to how information is given to pregnant women regarding diet and maternal weight gain in various systems of health care, whether they follow it, and how this information influences their behavior.

Limited evidence suggests that the composition and pattern of gestational weight gain is altered by a high prepregnancy BMI; only one study has shown that obese women tend to gain less fat during the second trimester than do normal-weight women (8). Compared with underweight and normal-weight women, in obese women, the relationship between maternal weight gain and birth weight is attenuated (8). This suggests that the maternal metabolic adjustments required to support fetal growth are less dependent on gestational weight gain in obese women than in nonobese women. More research is needed on the relationship of the amount, composition, and pattern of weight gain for overweight, obese, and severely obese women to maternal and child health outcomes in the short and long term. Those data are needed to revise the IOM gestational weightgain recommendations for pregnant women and to suggest an upper limit for obese women; in the 1990 report, obese women were advised only to gain at least 6 kg (15 lb) (9).

The relationship between maternal obesity and decreased breastfeeding needs further elucidation. Given the protective effect of breastfeeding on obesity development in the child and mother, we need to know why obese women are less likely to breastfeed successfully in order to develop successful intervention strategies.

It is unknown whether a high maternal prepregnancy BMI contributes to an intrauterine developmental programming process that increases the risk of childhood overweight and longterm disease, or whether the obesegenic environment of the home and family is the primary determinant of childhood obesity and subsequent disease. It is also uncertain if infants born to obese women are actually fatter or if the higher birth weight is because of greater lean body mass. We need better epidemiological studies that can tease out these effects.

A more systematic approach and longer-term follow-up studies are needed of women who become pregnant after bariatric surgery to evaluate the effects of surgery on perinatal and long-term child health outcomes.

More intervention studies are needed to determine if GDM, preeclampsia, and pregnancy-induced hypertension can be prevented with interventions related to diet and/or physical activity during pregnancy, and if there is a critical time when these interventions should be implemented. This will help in establishing the importance of preconceptional vs prenatal counseling for behaviors that can ameliorate these conditions.

ROLES AND RESPONSIBILITIES OF FOOD AND NUTRITION PROFESSIONALS WORKING WITH OVERWEIGHT/OBESE WOMEN PRIOR TO AND DURING PREGNANCY AND IN POSTPARTUM AND INTERCONCEPTIONAL PERIODS

As experts in food and nutrition throughout the lifespan, registered dietitians and other food and nutrition professionals are in a unique position to provide counseling to overweight and obese women to ameliorate the adverse effect of a high prepregnancy weight on reproductive, pregnancy, and long-term maternal and child health outcomes. We acknowledge that a comprehensive guide for evidencebased counseling of obese women needs to written. Based on the present knowledge it is important to provide counseling to obese women during the preconceptional, prenatal, postpartum, and interconceptional periods regarding the following topic areas:

- During the preconceptional period, overweight and obese women should receive counseling on strategies to obtain and remain at a healthful weight according to guidance provided in the ADA position "Nutrition and Women's Health" (64). This includes information about successful weight-loss practices, healthful eating both in the types of food and quantities, folic acid supplement use, as well as information about being physically active. In addition, they should be counseled about potential pregnancy and fetal complications if they are to become pregnant. Documenting a measured weight and height in their medical records would also be beneficial for later use related to the gestational weight-gain goals.
- **During pregnancy** all overweight and obese women should be informed about current IOM gestational weight gain target goals[†], be advised to not lose weight during pregnancy, and counseled about eating healthful foods during pregnancy as described in the ADA po-

†*At the time of this report the revised Institute of Medicine guidelines were not published.* sition "Nutrition and Lifestyle for a Healthy Pregnancy Outcome" (91) and in MyPyramid for Pregnancy (www.mypyramid.gov/ mypyramidmoms) (92). They should be encouraged to be physically active and be made aware of the ACOG guidelines for exercising during pregnancy (www.acog.org/ publications/patient education/ bp045.cfm) (93). Per the ACOG Committee Opinion report (1), food and nutrition professionals should encourage and support the screening of obese women for gestational diabetes upon presentation at the first clinic visit and repeated screening later in pregnancy if results are initially negative. Early in pregnancy, overweight and obese women should be encouraged to breastfeed and be made aware of the benefits for both her and her child's health.

- During postpartum and inter**conceptional periods**, special emphasis should be placed on the support of breastfeeding initiation and duration. Frequent early contact after hospital discharge would be advantageous for this purpose. During lactation, the overweight and obese woman should be counseled on a healthful diet that is nutrient-dense and also advised to not increase energy intake to compensate for milk production because that will help minimize postpartum weight retention (94). At the 6-week postpartum visit, women should be encouraged to consider a weight-loss program that includes a physical activity component (95) and be screened for postpartum depression and type 2 diabetes if gestational diabetes was confirmed during the pregnancy (1). They may need encouragement in taking time out for themselves to relieve the stress of motherhood, and guidance on the developmentally appropriate introduction of solid foods for the infant. These guidelines should address five key areas:
 - developmental signs of when a baby is ready for solids;
 - what foods are appropriate to feed and why;
 - developing healthful eating habits to last a lifetime;
 - $\circ~$ food safety concerns specific to infants; and

 developing a child's motor skills and physical activity patterns (96).

Other visits during the postpartum and interconceptional period would be advantageous because this would allow monitoring and supporting weight-loss practices and encouraging adherence to the 2005 Dietary Guidelines for Americans (97).

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References

- American College of Obstetricians and Gynecologists. Obesity in pregnancy. Obstet Gynecol. 2005;315:671-675.
- World Health Organization. Physical status: The use and interpretation of anthropometry. WHO Technical Report Series 854. Geneva, Switzerland: World Health Organization; 1995:1-452.
- Gallagher D, Visser M, Sepulveda D, Pierson RN, Harris T, Heymsfield SB. How useful is body mass index for comparison of body fatness across age, sex, and ethnic groups? *Am J Epidemiol.* 1996;143:228-239.
- Ogden ČL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999-2004. JAMA. 2006;295:1549-1555.
- Sarwer DB, Allison KC, Gibbons LM, Markowitz JT, Nelson DB. Pregnancy and obesity: A review and agenda for future research. J Womens Health (Larchmt). 2006; 15:720-733.
- Kim SY, Dietz PM, England L, Marrow B, Callaghan WM. Trends in pre-pregnancy obesity in nine states: 1993-2003. Obesity. 2007;15:986-993.
- Waller DK, Shaw GM, Rasmussen SA, Hobbs CA, Canfield MA, Siega-Riz AM, Gallaway MS, Correa A. Prepregnancy obesity as a risk factor for structural birth defects. *Arch Pediatr Adolesc Med.* 2007;161:745-750.
- Viswanathan M, Siega-Riz AM, Moos MK, Deierlein A, Mumford S, Knaack J, Thieda P, Lux LJ, Lohr KN. 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.) Rockville, MD: Agency for Healthcare Research and Quality; 2008. AHRQ Pub No. 08-E-09.
- Institute of Medicine. Nutrition During Pregnancy. Part I. Weight Gain. Part II. Nutrient Supplements. Washington, DC: The National Academies Press; 1990.
- Groth S. Adolescent gestational weight gain: Does it contribute to obesity? MCN Am J Matern Child Nurs. 2006;31:101-105.
- Farooqi IS, Jebb SA, Langmack G, Lawrence E, Cheetham CH, Prentice AM, Hughes IA, McCamish MA, O'Rahilly S. Effects of recombinant leptin therapy in a child with congenital leptin deficiency. N Engl J Med. 1999;341:879-884.

- Moisan J, Meyer F, Gingras S. A nested case-control study of the correlates of early menarche. Am J Epidemiol. 1990;132:953-961.
- Maclure M, Travis LB, Willett W, MacMahon B. A prospective cohort study of nutrient intake and age at menarche. Am J Clin Nutr. 1991;54:649-656.
- Pasquali R, Pelusi C, Genghini S, Cacciari M, Gambineri A. Obesity and reproductive disorders in women. *Hum Reprod Update*. 2003;9:359-372.
- Lake JK, Power C, Cole TJ. Women's reproductive health: The role of body mass index in early and adult life. *Int J Obes Relat Metab Disord*. 1997;21:432-438.
- Rich-Edwards JW, Goldman MB, Willett WC, Hunter DJ, Stampfer MJ, Colditz GA, Manson JE. Adolescent body mass index and infertility caused by ovulatory disorder. *Am J Obstet Gynecol.* 1994;171:171-177.
- 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;13:1502-1505.
- Bolumar F, Ölsen J, Rebagliato M, Saez-Lloret I, Bisanti L. Body mass index and delayed conception: A European Multicenter Study on Infertility and Subfecundity. Am J Epidemiol. 2000;151:1072-1079.
- 19. Zaadstra BM, Seidell JC, Van Noord PA, te Velde ER, Habbema JD, Vrieswijk B, Karbaat J. Fat and female fecundity: Prospective study of effect of body fat distribution on conception rates. Br Med J. 1993;306:484-487.
- Jenkins JM, Brook PF, Sargeant S, Cooke ID. Endocervical mucus pH is inversely related to serum androgen levels and waist to hip ratio. *Fertil Steril.* 1995;63:1005-1008.
- Baeten J, Bukusi E, Lambe M. Pregnancy complications and outcomes among overweight and obese nulliparous women. Am J Public Health. 2001;91:436-440.
- Chu SY, Callaghan WM, Kim SY, Schmid CH, Lau J, England LJ, Dietz PM. Maternal obesity and risk of gestational diabetes mellitus: A meta-analysis. *Diabetes Care*. 2007; 30:2070-2076.
- Catalano PM. Management of obesity in pregnancy. Obstet Gynecol. 2007;109:419-433.
- 24. Beckles G, Thompson-Reid P. Diabetes and Women's Health Across the Life Stages: A Public Health Perspective. Atlanta, GA: Department of Health and Human Services, Center for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Division of Diabetes Translation; 2001.
- 25. Kieffer EC, Carman WJ, Gillespie BW, Nolan GH, Worley SE, Guzman JR. Obesity and gestational diabetes among African-American Women and Latinas in Detroit: Implications for Disparities in Women's Health. J Am Med Womens Assoc. 2001;56: 181-187.
- 26. Ferrara A, Hedderson MM, Quesenberry CP, Selby JV. Prevalence of gestational diabetes mellitus detected by the national diabetes data group or the carpenter and coustan plasma glucose thresholds. *Diabetes Care*. 2002;25:1625-1630.
- American Diabetes Association. Clinical practice recommendations 2007. Diabetes Care. 2007;30(Suppl):S1-103.
- 28. Schafer-Graf UM, Dupak J, Vogel M, Du-

denhausen JW, Kjos SL, Buchanan TA, Vetter K. Hyperinsulinism, neonatal obesity and placental immaturity in infants born to women with one abnormal glucose tolerance test value. J Perinat Med. 1998;26:27-36.

- Hollingsworth DR. Alternations of maternal metabolism in normal and diabetic pregnancies: Differences in insulin-dependent, noninsulin-dependent, and gestational diabetes. *Am J Obstet Gynecol.* 1983;146:417-429.
- King JC. Maternal obesity, metabolism, and pregnancy outcomes. Ann Rev Nutr. 2006; 26:271-291.
- 31. King JC. Maternal obesity, glucose intolerance, and inflammation in pregnancy. In: Packer L, Sies H, eds. Oxidative Stress and Inflammatory Mechanisms in Obesity, Diabetes, and the Metabolic Syndrome. Boca Raton, FL: Taylor & Francis CRC Press; 2007: 93-106.
- Hauguel-de Mouzon S, Guerre-Millo M. The placenta cytokine network and inflammatory signals. *Placenta*. 2006;27:794-798.
- Sibai BM. Diagnosis and management of gestational hypertension and preeclampsia. Obstet Gynecol. 2003;102:181-192.
- Borzychowski AM, Sargent IL, Redman CW. Inflammation and pre-eclampsia. Semin Fetal Neonatal Med. 2006;11:309-316.
- 35. Weiss JL, Malone FD, Emig D, Ball RH, Nyberg DA, Comstock CH, Saade G, Eddleman K, Carter SM, Craigo SD, Carr SR, D'Alton ME. Obesity, obstetric complications and cesarean delivery rate-a population-based screening study. Am J Obstet Gynecol. 2004;190:1091-1097.
- Vahratian A, Zhang J, Troendle JF, Savitz DA, Siega-Riz AM. Maternal prepregnancy overweight and obesity and the pattern of labor progression in term nulliparous women. *Obstet Gynecol.* 2004;104:943-951.
- Bodnar LM, Siega-Riz AM, Cogswell M. High prepregnancy BMI increases the risk of postpartum anemia. *Obesity Research*. 2004;12:941-948.
- 38. Shaw GM, Quach T, Nelson V, Carmichael SL, Schaffer DM, Selvin S, Yang W. Neural tube defects associated with maternal periconceptional dietary intake of simple sugars and glycemic index. Am J Clin Nutr. 2003;78:972-978.
- Huang DY, Usher RH, Kramer MS, Yang H, Morin L, Fretts RC. Determinants of unexplained antepartum fetal deaths. *Obstet Gy*necol. 2000;95:215-221.
- 40. Smith GC. Predicting antepartum stillbirth. Curr Opin Obstet Gynecol. 2006;18:625-630.
- Ramsay JE, Greer I, Sattar N. Obesity and reproduction. Br Med J. 2006;333:1159-1162.
- Ehrenberg HM, Mercer BM, Catalano PM. The influence of obesity and diabetes on the prevalence of macrosomia. Am J Obstet Gynecol. 2004;191:964-968.
- Catalano P, Ehrenberg H. The short- and long-term implications of maternal obesity on the mother and her offspring. *Br J Obstet Gynecol.* 2006.
- Andreasen KR, Andersen ML, Schantz AL. Obesity and pregnancy. Acta Obstet Gynecol Scand. 2004;83:1022-1029.
- Salsberry PJ, Reagan PB. Dynamics of early childhood overweight. *Pediatrics*. 2005;116: 1329-1338.
- 46. Gillman MW, Rifas-Shiman S, Berkey CS, Field AE, Colditz GA. Maternal gestational diabetes, birth weight, and adolescent obesity. *Pediatrics*. 2003;111:e221-e226.

- 47. Li C, Kaur H, Choi WS, Huang TT, Lee RE, Ahluwalia JS. Additive interactions of maternal prepregnancy BMI and breast-feeding on childhood overweight. *Obes Res.* 2005; 13:362-371.
- Whitaker RC. Predicting preschooler obesity at birth: The role of maternal obesity in early pregnancy. *Pediatrics*. 2004;114:29-36.
- 49. Lawlor DA, Smith GD, O'Callaghan M, Alati R, Mamun AA, Williams GM, Najman JM. Epidemiologic evidence for the fetal overnutrition hypothesis: Findings from the materuniversity study of pregnancy and its outcomes. Am J Epidemiol. 2007;165:418-424.
- Gillman MW, Rifas-Shiman SL, Camargo CA Jr, Berkey CS, Frazier AL, Rockett HR, Field AE, Colditz GA. Risk of overweight among adolescents who were breastfed as infants. JAMA. 2001;285:2461-2467.
- Armstrong J, Reilly JJ. Breastfeeding and lowering the risk of childhood obesity. *Lancet*. 2002;359:2003-2004.
- 52. Bergmann KE, Bergmann RL, Von Kries R, Bohm O, Richter R, Dudenhausen JW, Wahn U. Early determinants of childhood overweight and adiposity in a birth cohort study: Role of breast-feeding. Int J Obes Relat Metab Disord. 2003;27:162-172.
- Harder T, Bergmann R, Kallischnigg G, Plagemann A. Duration of breastfeeding and risk of overweight: A meta-analysis. Am J Epidemiol. 2005;162:397-403.
- Rasmussen KM, Hilson JA, Kjolhede CL. Obesity may impair lactogenesis II. J Nutr. 2001;131:3009S-3011S.
- Rasmussen KM, Kjolhede CL. Prepregnant overweight and obesity diminish the prolactin response to suckling in the first week postpartum. *Pediatrics*. 2004;113:465-471.
- Boney C, Verma A, Tucker R, Vohr B. Metabolic syndrome in childhood: Association with birth weight, maternal obesity, and gestational diabetes mellitus. *Pediatrics*. 2005;115:290-296.
- Mumford S, Siega-Riz AM, Herring A, Evenson K. Dietary restraint and gestational weight gain. J Am Diet Assoc. 2008;108: 1646-1653.
- Merhi ZO. Weight loss by bariatric surgery and subsequent fertility. *Fertil Steril*. 2007; 87:430-432.
- Dixon JB, Dixon ME, O'Brien PE. Birth outcomes in obese women after laparoscopic adjustable gastric banding. *Obstet Gynecol.* 2005;106:965-972.
- 60. Bar-Żohar D, Azem F, Klausner J, Abu-Abeid S. Pregnancy after laparoscopic adjustable gastric banding: Perinatal outcome is favorable also for women with relatively high gestational weight gain. *Surg Endosc.* 2006;20:1580-1583.
- Marceau P, Kaufman D, Biron S, Hould FS, Lebel S, Marceau S, Kral JG. Outcome of pregnancies after biliopancreatic diversion. *Obes Surg.* 2004;14:318-324.
- Dao T, Kuhn J, Ehmer D, Fisher T, McCarty T. Pregnancy outcomes after gastric-bypass surgery. Am J Surg. 2006;192:762-766.
- American Dietetic Association. Position of the American Dietetic Association: Weight management. J Am Diet Assoc. 2009; 109:330-346.
- 64. American Dietetic Association, Dietitians of Canada. Position of the American Dietetic Association and Dietitians of Canada: Nutrition and Women's Health. Can J Diet Pract Res. 2004;65:85-89.
- 65. Committee on Obesity Practice. Obesity in

pregnancy. Obstet Gynecol. 2005;106:671-675.

- Olson CM. A call for intervention in pregnancy to prevent maternal and child obesity. *Am J Prev Med.* 2007;33:435-436.
- Polley BA, Wing RR, Sims CJ. Randomized controlled trial to prevent excessive weight gain in pregnant women. Int J Obes Relat Metab Disord. 2002;26:1494-1502.
- Kinnunen TI, Pasanen M, Aittasalo M, Fogelholm M, Hilakivi-Clarke L, Weiderpass E, Luoto R. Preventing excessive weight gain during pregnancy—A controlled trial in primary health care. *Eur J Clin Nutr.* 2007; 61:884-891.
- 69. Gray-McDonald K, Robinson E, David K, Renaud L, Rodrigues S. Intervening to reduce weight gain in pregnancy and gestational diabetes mellitus in Cree communities: An evaluation. *Can Med Assoc J.* 2000; 163:1247-1251.
- Olson CM, Strawderman MS, Reed RG. Efficacy of an intervention to prevent excessive gestational weight gain. Am J Obstet Gynecol. 2004;191:530-536.
- Artal R, Catanzaro RB, Gavard JA, Mostello DJ, Friganza JC. A lifestyle intervention of weight-gain restriction: Diet and exercise in obese women with gestational diabetes mellitus. *Appl Physiol Nutr Metab.* 2007;32:596-601.
- Claesson IM, Sydsjo G, Brynhildsen J, Cedergren M, Jeppsson A, Nystrom F, Sydsjo A, Josefsson A. Weight gain restriction for obese pregnant women: A case-control intervention study. Br J Obstet Gynecol. 2008; 115:44-50.
- Saldana TM, Siega-Riz AM, Adair LS. Effect of macronutrient intake on the development of glucose intolerance during pregnancy. *Am J Cin Nutr.* 2004;79:479-486.
- 74. Fraser RB, Ford FA, Milner RD. A controlled trial of a high dietary fibre intake in pregnancy--effects on plasma glucose and insulin levels. *Diabetologia*. 1983;25:238-241.
- Clapp JF 3rd. Effect of dietary carbohydrate on the glucose and insulin response to mixed caloric intake and exercise in both nonpregnant and pregnant women. *Diabetes Care*. 1998;21(Suppl 2):B107-B112.
- Bronstein MN, Mak RP, King JC. The thermic effect of food in normal weight and overweight pregnant women. Br J Nutr. 1995;75: 261-275.
- Zhang C, Liu S, Solomon CG, Hu FB. Dietary fiber intake, dietary glycemic load, and the risk for gestational diabetes mellitus. *Diabetes Care*. 2006;29:2223-2230.
- Wang Y, Storlien LH, Jenkins AB, Tapsell LC, Jin Y, Pan JF, Shao YF, Calvert GD, Moses RG, Shi HL, Zhu XX. Dietary variables and glucose tolerance in pregnancy. *Diabetes Care*. 2000;23:460-464.
- Bo S, Menato G, Lezo A, Signorile A, Bardelli C, De Michieli F, Massobrio M, Pagano G. Dietary fat and gestational hyperglycaemia. *Diabetologia*. 2001;44:972-978.
- Hofmeyr GJ, Atallah AN, Duley L. Calcium supplementation during pregnancy for preventing hypertensive disorders and related problems. *Cochrane Database Syst Rev.* 2006;3:CD001059.
- Rumbold AR, Crowther CA, Haslam RR, Dekker GA, Robinson JS. Vitamins C and E and the risks of preeclampsia and perinatal complications. N Engl J Med. 2006;354: 1796-1806.

- Poston L, Briley AL, Seed PT, Kelly FJ, Shennan AH. Vitamin C and vitamin E in pregnant women at risk for pre-eclampsia (VIP trial): Randomised placebo-controlled trial. *Lancet*. 2006;367:1145-1154.
- Olafsdottir AS, Skuladottir GV, Thorsdottir I, Hauksson A, Thorgeirsdottir H, Steingrimsdottir L. Relationship between high consumption of marine fatty acids in early pregnancy and hypertensive disorders in pregnancy. Br J Obstet Gynecol. 2006;113: 301-309.
- Duley L, Henderson-Smart D, Meher S. Altered dietary salt for preventing pre-eclampsia, and its complications. *Cochrane Database Syst Rev.* 2005:CD005548.
- 85. Khoury J, Henriksen T, Christophersen B, Tonstad S. Effect of a cholesterol-lowering diet on maternal, cord, and neonatal lipids, and pregnancy outcome: A randomized clinical trial. Am J Obstet Gynecol. 2005;193: 1292-1301.
- Dye TD, Knox KL, Artal R, Aubry RH, Wojtowycz MA. Physical activity, obesity, and diabetes in pregnancy. Am J Epidemiol. 1997;146:961-965.
- Greenberg AS, Obin MS. Obesity and the role of adipose tissue in inflammation and metabolism. Am J Clin Nutr. 2006;83 (suppl):461S-465S.
- Dempsey JC, Butler CL, Williams MA. No need for a pregnant pause: physical activity may reduce the occurrence of gestational diabetes mellitus and preeclampsia. *Exerc* Sport Sci Rev. 2005;33:141-149.
- Clapp JF 3rd, Kiess W. Effects of pregnancy and exercise on concentrations of the metabolic markers tumor necrosis factor alpha and leptin. Am J Obstet Gynecol. 2000;182: 300-306.
- Institute of Medicine. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids. Part I. Washington, DC: The National Academies Press; 2002.
- American Dietetic Association. Position of the American Dietetic Association: Nutrition and lifestyle for a healthy pregnancy outcome. J Am Diet Assoc. 2008;108:553-561.
- MyPyramid for Pregnancy. MyPyramid Web site. http://www.mypyramid.gov/ mypyramidmoms. Accessed June 3. 2008.
- 93. American College of Obstetricians and Gynecologists. Women's Health: Exercise and Fitness. American College of Obstetricians and Gynecologists Web site. http://www.acog.org/publications/patient_education/bp045.cfm. Accessed June 3, 2008.
- Institute of Medicine. Nutrition During Lactation. Washington, DC: The National Academies Press; 1991.
- Lovelady CA, Garner KE, Moreno KL, Williams JP. The effect of weight loss in overweight, lactating women on the growth of their infants. *N Engl J Med.* 2000;342:449-453.
- 96. Butte N, Cobb K, Dwyer J, Graney L, Heird WC, Rickard KA. Start Healthy Stay Healthy: Feeding Guidelines: Starting Solids Foods. American Dietetic Association Web site. http://www.eatright.org/ada/files/ infant_book.pdf. Published 2005. Accessed February 4, 2009.
- 97. US Department of Health Human Services, US Department of Agriculture. Dietary Guidelines for Americans, 2005. Health.gov Web site. http://www.health.gov/Dietary Guidelines/dga2005/document/default.htm. Published 2005. Accessed February 4, 2009.

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