Prepregnancy obesity and birth defects: what’s next?1–3

Sonja A Rasmussen and Deborah A Galuska

Recent data from the National Health and Nutrition Examination Survey for the years 2007–2008 showed that over a third of women ages 20–39 y in the United States are obese [defined as a body mass index [in kg/m²] ≥30] (1). The frequency of obesity among women of childbearing age makes it critical to understand the effects of prepregnancy obesity on maternal and infant outcomes. Prepregnancy obesity has been associated with a wide range of adverse outcomes in the mother and in the fetus and infant. Overall, women who are obese are more likely to experience chronic diseases such as cardiovascular disease, type 2 diabetes, osteoarthritis, and some cancers (2). Additional risks faced by reproductive-aged women who are obese comprise an increased risk of infertility as well as complications during pregnancy, including gestational diabetes, hypertensive disorders, thromboembolic disease, and cesarean delivery. Prepregnancy obesity also has adverse effects on the fetus and infant, with associations observed between maternal obesity and spontaneous abortion, fetal death, macrosomia, shoulder dystocia, and childhood obesity (3, 4).

Among the adverse outcomes associated with maternal prepregnancy obesity is delivering an infant with a birth defect. Previously, the most consistent data showing an association were for neural tube defects (eg, spina bifida and anencephaly) (5, 6). However, data are emerging that obese women are also at increased risk of other birth defects (6). An article in this issue of the Journal by Mills et al (7) provides further support for an association between maternal prepregnancy obesity and congenital heart defects (CHDs). These authors showed a modest but significantly increased odds of CHDs among women who are obese, similar to data from the meta-analysis by Stothard et al (6) that showed an odds ratio of 1.30 (95% CI: 1.12–1.51).

The public health significance of these findings is substantial. Not only is maternal obesity common but CHDs are the most common type of birth defect. Mills et al (7) estimated that the increased odds observed in their study could result in 1500 more infants born with CHDs each year. These defects contribute significantly to infant mortality: in the United States, birth defects are a leading cause of infant mortality, with ∼20% of infant deaths due to birth defects and CHDs accounting for about a third of these deaths (8).

Accumulating evidence for adverse outcomes associated with prepregnancy obesity has led several groups to develop guidelines for pregnant women who are obese. Guidelines from the American College of Obstetricians and Gynecologists in 2005 (3) and the Society of Obstetricians and Gynaecologists of Canada in 2010 (4) both emphasize the need for women to be of healthy weight prepregnancy and provide guidance for care of obese women during pregnancy. However, efforts to address the problem of obesity cannot be limited to the clinical care setting. Other sectors, such as communities and worksites, can support individuals in their attempts to follow recommendations for a healthy diet and physical activity (9, 10). Examples of these environmental and policy supports include increasing the availability and access to affordable, healthy foods; providing access to places for physical activity; and designing streets and neighborhoods to facilitate walking and bicycling.

In addition, research in 2 key areas is needed. First, we need to understand the mechanisms involved with the increased risk of birth defects and other adverse outcomes associated with maternal obesity; identification of the mechanisms responsible for the observed association could lead to the development of strategies to decrease the risk among women who are not at a healthy weight at the start of pregnancy. Several potential mechanisms have been proposed to explain the relation between maternal obesity and neural tube defects (5). For example, studies have shown that women who are obese have lower concentrations of folate, which have been associated with an increased risk of some birth defects (5). If this mechanism is identified as a cause of the increased risk of birth defects among obese women, supplementing women who are obese with higher doses of folic acid might be an effective strategy. Another mechanism frequently proposed is related to glucose control during pregnancy. Prepregnancy diabetes is a well-known risk factor for birth defects (5); however, careful glycemic control during pregnancy can decrease the risk of birth defects among diabetic mothers so this risk approaches that of the general population. The association observed between obesity and birth defects could be related to undiagnosed diabetes; if this is identified as a mechanism responsible for the increased risk of birth defects among obese women, an approach focusing...
on preconceptional testing of all obese women for diabetes could identify affected women early so that strategies to control their glucose could be instituted before pregnancy.

Second, we need research that further identifies and effectively translates evidence-based strategies to prevent obesity and to facilitate weight loss to achieve a healthy weight. Areas of focus might include the development of interventions that address the cultural and environmental barriers to a healthy diet and physical activity in reproductive-aged women at high risk of obesity, such as African American women; the identification of the most effective environmental and policy interventions for obesity prevention; and the characterization of the relative cost-effectiveness of different intervention options to help guide the decisions of policy makers.

Obesity is a problem that deleteriously affects the health and health care costs of the population. Concerted efforts across multiple sectors of society to address obesity will benefit the health of the population including that of reproductive-aged women and their children and should serve as one strategy in the prevention of birth defects.

Neither author had a conflict of interest to declare.

REFERENCES