Walden University
ScholarWorks

School of Psychology Publications | College of Social and Behavioral Sciences

2014

Maternal Obesity and the Development of Child Obesity

Leann Stadtlander

Follow this and additional works at: https://scholarworks.waldenu.edu/sp_pubs

Recommended Citation

Stadtlander, Leann, "Maternal Obesity and the Development of Child Obesity" (2014). *School of Psychology Publications*. 124.
https://scholarworks.waldenu.edu/sp_pubs/124

This Article is brought to you for free and open access by the College of Social and Behavioral Sciences at ScholarWorks. It has been accepted for inclusion in School of Psychology Publications by an authorized administrator of ScholarWorks. For more information, please contact ScholarWorks@waldenu.edu.

Maternal Obesity and the Development of Child Obesity

by Lee Stadtlander, PhD

Abstract: Research suggests that obese pregnant women are at greater risk for diabetes, pre-eclampsia, and maternal death. Obesity has been shown to increase the risk of fetal death, preterm birth, and congenital abnormalities such as neural tube and cardiovascular defects. These risks are increased with maternal diabetes, thus requiring careful monitoring throughout pregnancy. The relationship between maternal obesity and long-term effects on the child remains conflicted. Some studies have reported that children of obese mothers are more likely to be obese, while others have found only a small relationship. It does appear that extreme maternal obesity (BMI > 40) and metabolic disorders such as diabetes result in long-term effects in the child. Childbirth professionals have the opportunity to intervene through pre-pregnancy planning and careful monitoring throughout the pregnancy.

Keywords: pregnancy, maternal obesity, child obesity

Marie is pregnant with her first child: At a weight of 200 pounds and a height of 5'6", her body mass index (BMI) is 32, which is considered obese according to the Centers for Disease Control (2011). How could her obesity affect her health during pregnancy? How could her weight affect her baby, and are there long-term implications for her child? This article will explore these issues.

Obesity in adults is defined as having a BMI greater than or equal to 30; BMI is calculated from a person's weight and height. As an example, a 5'6" woman weighing 185 lbs. would have a BMI of 30 and be considered obese. The most recent national data on obesity prevalence among United States adults, adolescents, and children show that more than one-third of adults and almost 17% of children and adolescents were obese in 2009–2010 (Ogden, Carroll, Kit, & Flegal, 2012).

Maternal Obesity and the Effect on the Mother

For obese women, the Mayo Clinic (2011) suggests a weight gain of 11-25 pounds during pregnancy. During pregnancy, fat deposits, blood volume expansion, and increases in breast and uterine tissue contribute to 55% of the weight gained, and an additional 15-20% is due to the placenta and amniotic fluid, with the remaining 25-30% due to the weight of the fetus (Fraser & Lawlor, 2012; Institute of Medicine and National Research Council, 2009). It is normal for women to gain between 4.4-13.2 lbs. (2-6 kg) of fat during pregnancy (Nelson, Matthews, & Poston, 2010); this weight is important for providing the fetus with essential nutrients for normal growth and development.

There is evidence that greater gestational weight gain is associated with a higher risk of gestational diabetes (Hedderson, Gunderson, & Ferrara, 2010) and pre-eclampsia (Steegers, von Dadelszen, Duvekot, & Pijnenborg, 2010). Diabetes in the mother also increases the risk of diabetes in the child, while pre-eclampsia is consistently associated with higher blood pressure in the child (Ferreira, Peeters, & Stehouwer, 2009). It has been estimated that up to half of the cases of gestational diabetes can be attributed to pre-pregnancy obesity (Kim et al., 2010). Torloni et al. (2009) provided the metric that for every unit increase in BMI pre-pregnancy, the risk of gestational diabetes increases by 0.92 percent.

continued on next page

continued from previous page

Maternal obesity has been implicated in leading to maternal death. In a study conducted in the United Kingdom by the Centre for Maternal and Child Enquiries (2011), 27% of the 261 pregnancy-related deaths reported between 2006 and 2008 were in obese women. Similarly, the California Department of Public Health (2011) reported that 30% of the 386 women who died in pregnancy between 2002 and 2003 were obese. These findings reflect that the leading causes of mortality in pregnant women, thrombo-embolism, pre-eclampsia, and cardiovascular diseases, have a higher prevalence in the obese compared to the non-obese (Oteng-Ntim & Doyle, 2012). In the Centre for Maternal and Child Enquiries study, 75% of the mothers dying from thrombo-embolism were overweight or obese, as were 61% of mothers dying from heart disease.

April Mathews

obese pregnant women are at greater risk for diabetes, pre-eclampsia, and maternal death

Summary and Implications for Child Birth Professionals

The research suggests that obese pregnant women are at greater risk for diabetes, pre-eclampsia, and maternal death. Such evidence suggests that obesity should be considered as a high risk in pregnancy, requiring frequent monitoring of weight gain, blood pressure, and blood glucose levels.

Effects on the Fetus and Newborn with Maternal Obesity

Elevated maternal BMI has been shown to increase the risk of miscarriage, fetal death, preterm birth, congenital defects, and other neonatal complications, which are elevated further with maternal diabetes (Ojha, Budge, & Symonds, 2012).

Fetal Death

Nohr et al. (2005) explored the relationship between fetal death and gestational age by maternal BMI. Nohr et al. reported the risk associated with maternal obesity and fetal death increased with increasing gestational age, being greatest at term (over 37 weeks). The current data suggests that obesity increases the risk of fetal death from the second trimester onward, with a greater chance of stillbirth at or beyond term. The underlying mechanism between obesity and fetal death appears to be related to metabolic risk factors in the mother, such as reduced insulin sensitivity, lipid disorders, and increased levels of inflammatory indicators (Bell, Tennant, & Rankin, 2012). These are thought to directly contribute to the development of gestational hypertension, pre-eclampsia, and other disturbances of prenatal function, and to impair glucose tolerance and gestational diabetes (Sattar, Ramsey, Crawford, Cheyne, & Greer, 2003; Wolf et al., 2001).

Obesity leads to other issues. It is difficult to assess fetal size both clinically and by ultrasound in women with high BMI (Phatak & Ramsay, 2010). Therefore, some of the risk of stillbirth in obese pregnant women may be due to lower rates of recognition of poor fetal growth and fetal compromise. More speculative explanations of the mechanisms lead-

continued on next page

continued from previous page

ing to fetal death with obesity include the presence of sleep apnea and resulting decrease in oxygen to the fetus (Franklin et al., 2000), the reduced ability to detect fetal movement (Fretts, 2005), and lower rates of diagnosis of congenital abnormality (Phatak & Ramsay, 2010).

Childbirth educators have the opportunity to affect this cycle

Congenital Abnormalities

Congenital abnormality is a term used to describe a range of disorders in fetal development that result in structural or other abnormalities present at birth. Congenital abnormalities are one of the major causes of stillbirths and infant deaths, accounting for 20% of infant deaths in the United States (Heron et al., 2009). Evidence suggests that there may be a link between maternal obesity, neural tube defects (e.g., spinal bifida), and cardiovascular anomalies (Rasmussen, Chu, Kim, Schmid, & Lau, 2008; Stothard, Tennant, Bell, & Rankin, 2009).

One likely mechanism for such abnormalities is maternal diabetes and early pregnancy hyperglycemia (elevated blood glucose). Pre-existing diabetes is a well-established risk factor for congenital abnormalities, particularly cardiovascular and neural tube defects (Balsells, Garcia-Patterson, Gich, & Corcoy, 2009). Zabihi and Loeken (2010) speculate that hyperglycemia results in oxidative stress, which disrupts critical genes controlling embryonic development.

Another cause of congenital defects with obesity that has been proposed is a folate deficiency (Mojtabai, 2004). Folate is a water-soluble B vitamin that is naturally present in some foods and is available as a dietary supplement. Folate is the generic term for both naturally occurring food folate and folic acid, the form of the vitamin that is used in dietary supplements and fortified foods (National Institutes of Health, 2012). Folate is known to be a protective factor against neural tube defects and possibly other defects, such as cardiovascular abnormalities (Czeizel, 2009). Women with high BMI have been shown to have lower serum folate levels (Mojtabai, 2004); therefore, their children are at risk for such defects.

Summary and Implications for Child Birth Professionals

Obesity has been shown to increase the risk of fetal death, preterm birth, and congenital abnormalities such as neural tube and cardiovascular defects. These risks are increased with maternal diabetes, thus requiring careful monitoring throughout pregnancy. Folate deficiencies have also been implicated, which suggest that folic acid supplements, starting pre-pregnancy continues to be considered beneficial.

Long-Term Effects on the Child

There is conflicting evidence from studies as to whether high maternal BMI results in obesity in offspring. In one compelling study, Rooney, Mathiason, and Schauberger (2011) followed 777 obese mothers and children from prenatal through adulthood. Rooney et al. found that pre-pregnancy maternal obesity was a strong predictor of childhood, adolescence, and early adulthood obesity: Among mothers who were obese at pre-pregnancy, 52% of their offspring were obese at childhood, 62% at adolescence, and 44% at early adulthood.

A second persuasive study, conducted by Smith et al. (2009), examined siblings of mothers with extreme obesity (BMI > 40) who had undergone bariatric (stomach reduction) surgery. Smith et al. found that siblings born after the surgery had lower rates of obesity than those siblings born before the surgery. Smith et al. reported that children born after the bariatric surgery had improved metabolic profiles as compared to their siblings.

Other studies have found a small relationship between maternal obesity and subsequent obesity in the children (for example, see Whitaker, Jarvis, Beeken, Boniface, & Wardle,

continued on next page

continued from previous page

2010). In contrast, several large studies (Lake, Power, & Cole, 1997; Patel et al., 2011; Subramanian, Ackerson, & Smith, 2010) have found the relationship of maternal BMI with offspring obesity is similar to those of paternal BMI and the siblings' BMI. This suggests that both the mother's and the father's genetics and family lifestyle characteristics also influence such relationships.

Implications for Child Birth Professionals

The relationship between maternal obesity and long-term effects on the child remains conflicted. Some studies have reported that children of obese mothers are more likely to be obese, while others have found only a small relationship. It does appear that extreme maternal obesity and metabolic disturbances such as diabetes result in long-term effects in the child.

Childbirth educators have the opportunity to affect this cycle in a number of ways. Prospective obese mothers should be encouraged to work towards a healthier BMI, control diabetes, and to begin folic acid supplements before pregnancy. Obese women's gestational weight gain should be carefully monitored, along with their blood pressure and blood glucose. Extremely obese women (> 40 BMI) who have potentially high risk pregnancies may be referred to an obstetrician. Providing education and literature on healthy eating habits during pregnancy may be a way to offer motivation for life long change for not just the mother but for the entire family.

References

Balsells, M., Garcia-Patterson, A., Gich, I., & Corcoy, R. (2009). Maternal and fetal outcome in women with type 2 vs. type 1 diabetes mellitus: A systematic review and metaanalysis, *The Journal of Clinical Endocrinology and Metabolism, 94*, 4284-4291.

Bell, R., Tennant, P. W. G., & Rankin, J. (2012). Fetal and infant outcomes in obese pregnant women. In M. W. Gillman & L. Poston (Eds.), *Maternal obesity* (pp. 56-69). Cambridge, United Kingdom: Cambridge University Press.

California Department of Public Health. (2011). The California pregnancy associated mortality review: Report from 2002 and 2003 maternal death reviews. Retrieved from http://www.cdph.ca.gov/data/statistics/Pages/CaliforniaPregnancy-AssociatedMortalityReview.aspx

Centers for Disease Control. (2011). Healthy Weight - It's not a diet, it's a lifestyle! Retrieved March 14, 2014, from http://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/index.html

Centre for Maternal and Child Enquiries. (2011). Saving mothers' lives: Reviewing maternal deaths to make motherhood safer: 2006-2008. *BJOG: An International Journal of Obstetrics and Gynaecology, 118*(Suppl. 1), 1-203. doi:10.1111/j.1471-0528.2010.02847.x

Czeizel, A. (2009). Periconceptual folic acid and multivitamin supplementation for the prevention of neural tube defects and other congenital abnormalities. *Birth Defects Research. Part A, Clinical and Molecular Teratology, 85*, 260-268.

Ferreira, I., Peeters, L. L. & Stehouwer, C. D. (2009). Preeclampsia and increased blood pressure in the offspring: Meta-analysis and critical review of the evidence. *Journal of Hypertension, 27*(10), 1955-1959.

Franklin, K., Holmgen, P., Jönsson, F., Poromaa, N., Stenlund, H., & Svanborg, E. (2000). Snoring, pregnancy-induced hypertension, and growth retardation of the fetus. *Chest, 117*, 137-141.

Fraser, A., & Lawlor, D. A. (2012). Long-term consequences of maternal obesity and gestational weight gain for offspring obesity and cardiovascular risk: Intrauterine or shared familial mechanisms? In M. W. Gillman & L. Poston (Eds.), *Maternal obesity* (pp. 87-99). Cambridge, United Kingdom: Cambridge University Press.

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

Hedderson M. M., Gunderson E. P., & Ferrara, A. (2010). Gestational weight gain and risk of gestational diabetes. *Obstetrics & Gynecology, 115*(3), 597-604.

Heron, M., Hoyert, D., Murphy, S., Xu, J., Kochanek, K.D., & Tejada-Vera, B. (2009). Deaths: Final data for 2006. *National Vital Statistics Reports, 57*(14), 1-134.

Institute of Medicine and National Research Council. (2009). *Weight gain during pregnancy: Reexamining the guidelines.* Washington, DC: National Academies Press.

Kim, S. Y., England, L., Wilson, H. G., Bish, C., Satten, G. A., & Dietz, P. (2010). Percentage of gestational diabetes mellitus attributable to overweight and obesity. *American Journal of Public Health, 100*(6), 1047-1052.

Lake, J. K., Power, C. & Cole, T. J. (1997). Child to adult body mass index in the 1958 British birth cohort: Associations with parental obesity. *Archives of Diseases in Childhood, 77*(5), 376-381.

Mayo Clinic. (2011). Pregnancy weight gain: What's healthy? Retrieved from http://www.mayoclinic.com/health/pregnancy-weight-gain/PR00111

Mojtabai, R. (2004). Body mass index and serum folate in childbearing age women. *European Journal of Epidemiology, 19*(11), 1029-1036.

Nelson, S. M., Matthews, P., & Poston, L. (2010). Maternal metabolism and obesity: Modifiable determinants of pregnancy outcome. *Human Reproduction Update, 16*(3), 255-275.

National Institutes of Health. (2012). Dietary supplement fact sheet: Folate. Retrieved from http://ods.od.nih.gov/factsheets/Folate-HealthProfessional/

Nohr, E. A., Nech, B. H., Davies, M. J., Frydenberg, M., Henriksen, T. B., & Olsen, J. (2005). Prepregnancy obesity and fetal death: A study within the Danish National Birth Cohort. *Obstetrics & Gynecology, 106*, 250-259.

Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2012). *Prevalence of obesity in the United States, 2009-2010* [NCHS Data Brief No. 82]. Retrieved from http://www.cdc.gov/nchs/data/databriefs/db82.pdf

Ojha, S., Budge, H., & Symonds, M.E. (2012). Adipose tissue development and its potential contribution to later obesity. In M. W. Gillman & L. Poston (Eds.), *Maternal obesity* (pp.124-134). Cambridge, United Kingdom: Cambridge University Press.

continued on next page

continued from previous page

Oteng-Ntim, E., & Doyle,P. (2012). Maternal outcomes in obese pregnancies. In M. W. Gillman & L. Poston (Eds.), *Maternal obesity* (pp. 35-44). Cambridge, United Kingdom: Cambridge University Press.

Patel, R., Martin, R. M., Kramer, M. S., Oken, E., Bogdanovich, N., Matush, L.,... Lawlor, D. A. (2011). Familial associations of adiposity: Findings from a cross- sectional study of 12,181 parental-offspring trios from Belarus. *PLOS One 6*(l), e14607.

Phatak, M., & Ramsay, J. (2010). Impact of maternal obesity on procedure of mid-trimester anomaly exam. *Journal of Obstetrics & Gynecology, 30*, 447-450.

Rasmussen, S. A., Chu, S. Y., Kim, S. Y., Schmid, C. H., & Lau, J. (2008). Maternal obesity and risk of neural tube defects: A metaanalysis. *American Journal of Obstetrics & Gynecology, 198*, 611-619.

Rooney, B. L., Mathiason, M . A., & Schauberger, C. W. (2011). Predictors of boesity in childhood, adolescence, and adulthood in a birth cohort. *Maternal and Child Health Journal, 15*, 1166–1175

Sattar, N., Ramsey, J., Crawford, L., Cheyne, H., & Greer, I. (2003). Classic and novel risk factor parameters in women with a history of preeclampsia. *Hypertension, 42*, 39-42.

Smith, J., Cianflone, K., Biron, S., Hould, F. S., Lebel, S., Marceau, S.,... Marceau, P. (2009). Effects of maternal surgical weight loss in mothers on intergenerational transmission of obesity. *The Journal of Clinical Endocrinology and Metabolism, 94*(11), 4275-4283.

Steegers, E. A., von Dadelszen, P., Duvekot, J. J., & Pijnenborg, R. (2010). Pre-eclampsia. Lancet, 376(9741), 631-644.

Stothard, K. J., Tennant, P. W. G., Bell, R., & Rankin, J. (2009). Maternal overweight and obesity and the risk of congenital anomalies. *Journal of the American Medical Association, 301*, 636-650.

Subramanian, S. V., Ackerson, L. K. & Smith, G. D. (2010). Parental BMI and childhood under nutrition in India: An assessment of intrauterine influence. *Pediatrics, 126*(3), e663-e671.

Torloni, M. R., Betnin, A. P., Horta, B. L., Nakamura, M. U., Atallah, A. N., Moron, A. F., & Valente, O. (2009). Pre-pregnancy BMI and the risk of gestational diabetes: A systematic review of the literature with meta-analysis. *Obesity Review, 10*(2),194-203.

Whitaker, K. L., Jarvis, M. J., Beeken, R. J., Boniface, D., & Wardle, J. (2010). Comparing maternal and paternal intergenerational transmission of obesity risk in a large population-based sample. *The American Journal of Clinical Nutrition, 91*(6), 1560-1567.

Wolf, M., Kettyle, E., Sandler, L., Ecker, J. L., Roberts, J., & Thadhani R. (2001). Obesity and preeclampsia: The potential role of inflammation. *Obstetrics & Gynecology, 98*, 757-762.

Zabihi, S., & Loeken, M. R. (2010). Understanding diabetic teratogenesis: Where are we now and where are we going? *Birth Defects Research. Part A, Clinical and Molecular Teratology, 88*, 779-790.

Lee Stadtlander is a researcher, professor, and the coordinator of the Health Psychology program at Walden University. As a clinical health psychologist, she brings together pregnancy and health care issues.

We want to hear about prenatal education practices for the families of armed forces personnel.

Contact editor@icea.org for guidance on writing an article.