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Commentary

Effect of Maternal Obesity on the Fetus

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DESCRIPTION

Obesity in mothers is now a global problem. It has been demonstrated that obesity and a high-fat diet negatively affect foetal programming, predisposing children to poor cardio metabolic and neurodevelopmental outcomes. Large epidemiological studies have linked maternal obesity with poor outcomes for offspring, but the underlying mechanisms are still unknown. In the context of maternal obesity, the mechanistic foundations of foetal malprogramming have been clarified in large part by the use of molecular methods. The evaluation of specific mRNA expression in fetuses and the offspring of obese females using quantitative Reverse Transcription Polymerase Chain Reaction (RT-qPCR) is one of these approaches. Other approaches include characterization of epigenetic modifications, microRNA expression, the gut microbiome, the transcriptome, and the evaluation of specific mRNA expression. Regarding the impact of maternal obesity on foetal and child neurodevelopment and cardio metabolic outcomes, with a focus on molecular methods, animal models and human fluids/cells are used.

There has been a great deal of interest in the early stages of disease since the theory associating low birth weight and poor prenatal growth with risk of cardiovascular disease was first put forth. Interest in the early causes of obesity has increased as obesity rates rise and maternal obesity becomes more prevalent. It is anticipated that a complicated interaction between inherited gene effects and the uterine environment will interact to program pathways that contribute to eventual obesity in the growing embryo. It is evident those pregnant obese women have disrupted maternal metabolism, and that children of obese mothers have greater body fat percentages and are insulin resistant. In addition to discussing numerous potential processes that may cause obesity programming and vulnerability to future metabolic and vascular disease, this review covers the theories underlying the currently accepted concept of obesity programming.

Low circulating levels of Adiponectin (ADN) are found in pregnant women with obesity or gestational diabetes mellitus, and these women typically give birth to large, fatter babies who are more prone to perinatal problems and the later development of metabolic syndrome. Whether there is a cause-and-effect connection between maternal ADN and foetal growth is still uncertain. When put to the test, the following predictions were found to be true: ADN supplementation in obese pregnant dams increases maternal insulin sensitivity, returns normal placental insulin/mechanistic Target of Rapamycin Complex 1 (mTORC1) signaling and nutrient transport, and inhibits foetal overgrowth. Female C57BL/6J mice fed an obesogenic diet prior to ovulation and throughout pregnancy exhibited higher fasting serum levels of leptin, insulin, and C-peptide and lower levels of high-molecular-weight ADN at Embryonic day (E) 18.5. Peroxisome Proliferator-Activated Receptor (PPAR) phosphorylation was decreased, placental transport of glucose and amino acids *in vivo* was increased, and foetal weights were 29% greater in fat dams. Placental insulin and mTORC1 signaling were also activated. During the period between E14.5 and E18.5, maternal ADN infusion in obese dams corrected maternal insulin sensitivity, placental insulin/mTORC1 and PPAR signaling, nutrition transport, and foetal development without changing the maternal fat mass. We show that ADN acts as an endocrine connection between maternal adipose tissue and foetal growth by influencing placental function in a mouse model with significant similarities to obese pregnant women. The negative effects of maternal obesity on placental function and foetal growth were important reversed by maternal ADN administration. The prevention of foetal overgrowth brought on by maternal obesity may be achieved by raising maternal ADN levels.

Future generations will continue to be at risk for metabolic and neurodevelopmental morbidities as maternal obesity and HFD consumption rise globally. It is essential to understand the molecular causes of foetal and child morbidity in order to develop efficient therapeutic approaches that can stop or reverse the malprogramming caused by maternal obesity. Increased maternal obesity has no effect on or effect on the accuracy of clinical or sonographic estimates of foetal weight. Therefore, regardless of a woman's body size, forecasts of foetal weight offer equally precise and reliable instructions for making management decisions.

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