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Influences of Maternal Prepregnancy Obesity and Gestational Diabetes Mellitus on the Infant Gut Microbiome in Full-Term Infants

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ABSTRACT

This review examines the current evidence of how prepregnancy obesity (PPO) and gestational diabetes mellitus (GDM) influence the newborn gut microbiome. Scientific gaps in the literature are described to guide future research in this area. The prevalence of PPO and GDM increased to 64% in the United States over the past decade. Prepregnancy obesity and GDM influence newborn gut microbiome and contribute to adverse short- and long-term outcomes in full-term infants. This review aims to discuss current research findings related to the associations between PPO and GDM, separately, and together, on infant gut microbiome outcomes, provide an overview of short-term and long-term outcomes, describe clinical relevance, and identify avenues for future scientific inquiry. This review found that PPO and GDM influence infant gut microbiomes. Infants born to women with PPO and GDM were found to have lower levels of diversity in gut microbiota than infants born to normal prepregnancy weight women and those born to women without GDM.

Key Words: dysbiosis, gestational diabetes, infants, microbiome, microbiota, prepregnancy obesity

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The human gut microbiota is a dynamic ecosystem that has a reciprocal relationship with its host.[1–4] The gut microbiota has many functions: nutrient extraction from food, protection from harmful pathogens, and contribution to immune function.[5] Disturbances in the composition of the microbial makeup, also known as dysbiosis, and reduction of diversity of the gut microbiome support the development of disease.[1,2,4–6] To determine the diversity of the gut microbiome, statistical analysis of sequencing data involving metagenomics DNA analysis coding for the 16S region of the bacterial gene allows scientists to analyze compositional makeup.[7] In addition, statistical analysis of sequencing data describes diversity of species within the same individual (alpha diversity) and interindividual species diversity (beta diversity).[7] The 2 largest bacterial phyla found in the human gut are Firmicutes and Bacteroidetes. Firmicutes and Bacteroidetes that coevolve with host-microbiome complement the coding of our genome, contributing to dysbiosis.[8] Altered levels of Firmicutes and Bacteroidetes are associated with obesity.[5,8,9] Prepregnancy obesity (PPO) is associated with gut dysbiosis characterized by elevated levels of Firmicutes in infants born to normal-weight mothers and elevated levels of Bacteroidetes in infants born to mothers who are obese. These data are important because the maternal transfer of the gut microbiome is an early-life exposure that contributes to obesity in offspring later in life.[8,10,11]

Obesity rates are rising to significant rates in all populations, including women of childbearing ages. According to the most recent data, in the United States, 27% of women giving birth in 2014 had PPO (body mass index: $\geq$30 kg/m^2),[12] which is strongly associated with an increased risk for cesarean delivery,

vaginal delivery complications, and birth injury leading to unfavorable infant outcomes.[12–14] Data suggest that infants born to a mother with PPO have increased childhood obesity and arterial stiffness, a reliable antecedent for cardiometabolic disease.[15–17] Although associations have been identified, the exact mechanisms involved in PPO-induced infant gut dysbiosis remain uncertain. Recent evidence supports that these effects may be mediated through pregnancy and infancy.[8,15,18]

A significant risk factor for the development of gestational diabetes mellitus (GDM)[19] is obesity before[15] and throughout pregnancy.[12,20,21] Parallel with the prevalence of increasing PPO rates, GDM is consequently increasing, affecting an estimated 9.2% of pregnancies in the United States.[21,22] Multiple neonatal complications are associated with maternal GDM, including glucose instability, hyperbilirubinemia, macrosomia, and subsequent birth-related injuries, prematurity, and respiratory insufficiency.[23] Long term, these infants face an increased risk for childhood obesity and type 2 diabetes.[6,16,24,25] However, GDM is linked to maternal altered glucose tolerance and hypertension, and infant macrosomia and childhood obesity independent of PPO.[24] The risks for developing metabolic syndrome and type 2 diabetes are 10 times more likely when PPO and GDM coexist.[26] The maternal gut microbiome and transfer to their offspring may be linked to these risks.[27] Transfer of maternal gut microbiome to offspring born to women with PPO and GDM together may significantly increase risks for adverse offspring body composition in infancy, childhood, and adolescence.[25,27]

Various factors influence the infant gut microbiome composition, such as perinatal environmental exposures, early life stress, and maternal factors.[15] Placental transfer of maternal metabolites from mothers with GDM (lactate, triglycerides, β-hydroxybutyrate, nonesterified fatty acids, and glycerol) influences the fetal metabolome and fetal metabolism, resulting in absence of phylum-level gut microbiome in offspring born to GDM mothers.[28,29] The influences of PPO and GDM on the gut microbiome of newborns have critical implications for early identification of disease development later in life. Specifically, knowledge of influences on the infant microbiome may help in explaining the increase in obesity and cardiometabolic disease rates in infancy, childhood, and adolescence.

This review provides a discussion of the current body of literature on influences of PPO and GDM, separately, and together, on gut microbiome infant outcomes. In addition, an overview of the identification of areas of future research is discussed. Essential knowledge can be gained from human cohort and population health studies emphasizing how early-life exposure to PPO and GDM influences the infant gut microbiome.

CURRENT EVIDENCE

Current evidence examining the influences of PPO and GDM gut microbiome infant outcomes, such as alterations in diversity, remains limited but is the focus of recent research. Gestational diabetes mellitus is characterized by carbohydrate intolerance, which is healthy for glucose metabolism before pregnancy but leads to diabetes during pregnancy.[21,30] Women diagnosed with GDM are considered high-risk, as complications are common during perinatal and newborn periods and may also adversely affect the neonatal gut microbiota.[2] Human and animal studies exploring causal mechanisms of disease programming suggest that gut microbiota dysbiosis negatively affects metabolic health triggering cardiometabolic disease onset later in life.[6] In alignment with the "developmental origin of health and disease" hypothesis, increasing evidence supports that exposure to prenatal metabolic disorders during fetal growth may contribute to health outcomes in the offspring.[15,19] In the studies reviewed, microbiota determinants from infant stool samples are operationalized through the utilization of high-throughput sequencing and 16S rRNA sequence analysis. It has been hypothesized that scrutiny of gut microbiota may explain the mechanisms of transgenerational obesity through transfer of the maternal microbiota to their offspring.[31]

THE INFANT MICROBIOME

Among full-term infants, gut microbiota consists primarily of anaerobic organisms. The "normal" infant gut microbiota develops by the colonization of facultative anaerobic organisms, and later developing obligate anaerobes, including *Bifidobacterium*, *Bacteroides*, and *Clostridium*.[32] These anaerobes are associated with producing polysaccharides that mediate microbiota colonization, immune modulation, and host-gut cross talk.[33] For example, *Clostridium* in the infant's gut, at high levels, is pathogenic and considered unhealthy.[33] After the age of 3 years, the microbial environment changes rapidly; compositional stability occurs to resemble an adult becoming dominated by Firmicutes and Bacteroidetes.[8,34]

Gut microbiota is associated with the regulation of metabolic and immune-inflammatory axes in the liver, muscle, and brain through host pathways.[10] Dysbiosis, or imbalance of the infant gut microbiome, may be facilitated by early exposure to environmental factors such as bacteria and viruses, which can also alter host microbiota. This dysbiosis of microbiota has long-term effects on host metabolism, leading to metabolic changes, in particular, type 1 diabetes, autoimmune disease, and obesity.[2,33] In humans, it is suggested that early microbial patterns may predict excessive weight gain in

offspring during childhood and later in life.[3,33] In addition, recent literature implicates microbiota-related epigenetic changes during early development, thus affecting phenotypic characteristics such as obesity later in life.[3,28] In other words, the infant's early exposure to maternal microbiomes through a transfer of maternal gut microbiota may alter the composition of the infant's gut microbiome.

PPO AND INFANT GUT MICROBIOME

Recent research identified that overweight and obese pregnant women have higher levels of *Bacteroides*, *Clostridium*, and *Staphylococcus*, and lower levels of *Bifidobacterium* in their feces than the normal-weight women.[8,15,19,35] Lower levels of *Bifidobacterium* are significant because they are major contributors to their host breakdown of glucans and carbohydrates.[36] Diversity of the human gut microbiome refers to the variability of the microbiota. Compositionally, alpha diversity describes the most diverse microbiota, while beta diversity describes factors, such as disease, age, or culture, that correlate with overall compositional differences.[5] Infants born to mothers with PPO microbiomes differ in diversity; however, studies are conflicting. For example, some literature reported that lower levels of diversity (less variability) were found[28,37]; conversely, other studies reported no significant changes in diversity.[31,38] A similar study found that changes in diversity occurred, finding that Firmicutes was significantly enriched in infants born to normal-weight mothers, whereas Bacteroidetes was significantly enriched in children born to obese women. This difference is not surprising, given gut microbiota differs among infants and is strongly affected by other factors such as mode of delivery, antibiotic usage, and breastfeeding.[31] Prepregnancy obesity influences mode of delivery because the risk of cesarean delivery increases with maternal PPO.[39] Therefore, mode of delivery is known as the first environmental exposure and influences the infant gut microbiome.[40] Infants born vaginally have more gut microbiome similarities to their mothers with high abundance of the genera *Bifidobacterium*, *Bacteroides*, *Streptococcus*, and *Clostridium*.[41] Similarly, studies indicate that excess maternal prepregnancy weight is associated with differences in neonatal acquisition of microbiota during vaginal delivery but not cesarean delivery.[38] It has been well established that the gut microbiota is an important factor in the onset and development of metabolic diseases.[24,28] Recent advances in microbiome research suggested that healthy colonization, the construction of a complex microbial community, of gut microbiomes begins before birth and rupture of membranes.[1,15] The infant microbiota increases in diversity and bacterial abundance during the first days of life as exposure to the environment and diet changes.[34] These associations prompt future investigation since the influence of PPO on the newborn microbiome is not entirely known.

GDM AND INFANT MICROBIOME OUTCOMES

Recent research reported that GDM alters the microbiota of newborns, contributing to the current understanding of intergenerational obesity and diabetes prevalence.[2] In addition, one study observed a significant reduction in the diversity of various bacterial types in GDM newborns. These findings indicate that there might be serious dysbiosis in the gut of GDM newborns.[28] Compared with those of healthy newborns, GDM newborns could be potentially more predisposed to develop gastrointestinal diseases and metabolic syndrome at later stages in their lives.[28] These findings are consistent with previous findings suggesting that the gut microbiota in the GDM group was associated with lower alpha-diversity level compared with the healthy groups.[42] This finding is important because a lower alpha-diversity level in the gut microbiome is associated with a higher body mass index.[43] The increases in maternal PPO and GDM are linked to increased body mass index before and during pregnancy.

SHORT-TERM OUTCOMES

As demonstrated in the current evidence, infant gut microbiome dysbiosis with PPO and GDM is a vital component leading to disease later in life. The short-term outcomes of early-life exposure to PPO and GDM show conflicting findings of the influences on the infant's gut microbiota. Data support that maternal microbiota may be transferred to offspring, altering infant gut microbiome. The interactions between the microbiome, epigenetics, and metabolic systems are likely to play a significant role in the origin of obesity and metabolic syndrome, yet the mechanisms continue to be poorly explained.[3] Further research is needed on early-life exposure to understand these mechanisms better.

LONG-TERM OUTCOMES

Prepregnancy obesity and GDM are associated with dysbiosis in the infant gut microbiome. Further work is needed to determine specific mechanisms of compositional changes in newborns and infants over time. Research supports that the future health of infants may be affected as the offspring of GDM mothers is more likely to develop obesity during childhood and later in life.[6,25] Another study described that as the alpha diversity of fecal microbiota decreased in children aged

6 to 16 years, body mass index *z* score (adjusted for age and sex of the child) increased.[10] The increasing number of women with PPO and GDM has significant implications for the mother and the offspring, as the dysbiosis of the infant gut microbiome may contribute to childhood obesity and the development of cardiometabolic disease.

CLINICAL RELEVANCE

A clearer understanding of how PPO and GDM influence infant gut microbiota will help guide the development of screening methods that can identify and monitor the development of neonatal dysbiosis. Detection of early biomarkers signaling dysbiosis would drive early interventions to achieve gut symbiosis. For example, prebiotics and probiotics in the infant's diet have been associated with increased gut microbiota diversity. However, it is not known whether this persists after discontinuation of the prebiotics and probiotics.[44] Offspring of mothers who have a healthy lifestyle (normal weight and regular exercise) before pregnancy have a significantly decreased risk of childhood obesity.[45] Increase patient education efforts to reduce PPO and increase adherence to healthy lifestyle before pregnancy to prevent childhood obesity and later in life cardiometabolic consequences. In addition, nurses must be aware of the newborn risks associated with GDM and the increased risks to infants born to mothers with GDM.

CONCLUSION

The incidence of PPO and GDM is increasing, and both of these maternal factors influence infant gut microbiome outcomes. Maternal transfer of disproportionate pathogenic bacteria creates an environment that supports infant gut dysbiosis and may be an important link to understanding how early-life exposure to maternal factors influences disease onset later in life. The current body of evidence examining the effect of PPO and GDM on the infant's microbiome is growing and yet conflicting. The newborn microbiome develops and is similar in composition to the adult by 3 years of age. Short-term outcomes found that PPO and GDM lower the infant's microbiome alpha diversity, which is directly associated with adulthood obesity. Long-term outcomes are that dysbiosis of infant gut microbiome may lead to obesity and cardiometabolic disease in infancy, childhood, and adolescence.

Although the body of research focused on this problem is growing, existing evidence is often conflicting, indicating that further inquiry is warranted to fully explain how early-life exposure to PPO and GDM influences the infant gut microbiome. Specifically, future research must focus on the interconnection between healthy maternal weight, healthy lifestyle, and maternal gut microbial environments and infant gut colonization. Longitudinal studies following the maternal-infant dyad to study ongoing changes with age and lifestyle influences are needed to help describe links between microbiota transfer and external influences. The findings of future studies should advance current knowledge in terms of infant gut microbiome and weight management interventions, important for decreasing risks for obesity and cardiometabolic disorders. Studies that connect diet, microbiota, and metabolism in mothers with obesity/GDM and their offspring remain a critical unmet need. Finally, efforts to identify meaningful biomarkers that detect neonatal dysbiosis are required to define appropriate diagnostic approaches and design effective early intervention strategies to optimize infancy, childhood, and adult health outcomes.

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