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ISSN: 2572-5629
Diabetes Case Reports

Perspective

External Variables and Prenatal Gestational Diabetes in Mice during Pregnancy

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DESCRIPTION

The exact causes of GDM are not fully understood, but several factors have been implicated, such as genetic predisposition, hormonal changes, placental factors, and environmental influences. Gestational Diabetes Mellitus (GDM) is a form of diabetes that occurs during pregnancy and affects 7%-18% of all pregnancies. GDM can have adverse effects on both the mother and the fetus, such as increased risk of preeclampsia, macrosomia, birth complications, and future development of type 2 diabetes. One of the emerging factors that have gained attention in recent years is the role of the gut microbiota in GDM. The gut microbiota is the collection of microorganisms that inhabit the gastrointestinal tract and influence various aspects of human health and disease. The gut microbiota can modulate glucose metabolism, inflammation, immunity, and hormone secretion through various mechanisms, such as producing Short-Chain Fatty Acids (SCFAs), bile acids, and metabolites; affecting intestinal permeability and barrier function; and interacting with host receptors and signaling pathways.

Several studies have shown that women with GDM have altered gut microbiota composition and diversity compared to healthy pregnant women. For example, women with GDM tend to have lower abundance of beneficial bacteria, such as Bifidobacterium and Lactobacillus, and higher abundance of potentially harmful bacteria, such as *Escherichia coli*, *Bacteroides fragilis*, and *Prevotella copri*. These changes in the gut microbiota may contribute to the development of GDM by increasing intestinal inflammation, impairing insulin sensitivity, and inducing dyslipidemia. Moreover, some studies have suggested that the gut microbiota of women with GDM may also affect the offspring's health and predispose them to metabolic disorders later in life. For instance, it has been reported that the offspring of women with GDM have lower diversity and richness of gut microbiota than the offspring of healthy pregnant women. Furthermore, some studies have shown that transplanting the fecal microbiota from women with GDM to germ-free mice can induce hyperglycemia and insulin resistance in the recipient mice.

Therefore, understanding the role of the gut microbiota in GDM may provide new insights into the pathogenesis and prevention of this condition. One of the challenges in studying the gut microbiota in human subjects is the difficulty in controlling for confounding factors, such as diet, medication, lifestyle, and genetic background. Animal models can overcome some of these limitations and allow for more controlled experiments. However, there is no widely accepted animal model of GDM that can mimic the human condition. Recently, a novel mouse model of GDM has been developed by feeding pregnant C57BL/KsJ db/+ mice with a High Fat High Sugar (HFHS) diet for one week. This model can induce glucose intolerance and beta cell dysfunction in pregnant mice by mid-gestation without affecting body weight or adiposity. Moreover, this model can also cause placental and fetal abnormalities, such as increased glucose uptake and fetal weight. The advantage of this model is that it can simulate the acute onset of GDM in humans without requiring genetic manipulation or long-term dietary intervention.

He investigated the effect of astaxanthin, a natural carotenoid with antioxidant and anti-inflammatory properties, on GDM symptoms and reproductive outcomes. Astaxanthin has been shown to have beneficial effects on obesity and diabetes by improving insulin sensitivity, glucose uptake, lipid metabolism, and oxidative stress. The authors found that astaxanthin treatment significantly alleviated glucose intolerance and beta cell insufficiency in pregnant HFHS-fed mice. Astaxanthin also inhibited oxidative stress in the liver and enhanced the activity of antioxidant enzymes. Moreover, astaxanthin improved reproductive outcomes by reducing placental and fetal glucose uptake and fetal weight. The authors suggested that astaxanthin exerted its effects by restoring the nuclear factor erythroid 2-related factor 2 (Nrf2)/Heme Oxygenase-1 (HO-1) antioxidant pathway in the liver of GDM mice.

CONCLUSION

This study demonstrated that astaxanthin could be a potential therapeutic agent for GDM by modulating oxidative stress and

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Received: 03-Jul-2023, Manuscript No. DCRS-23-22685; **Editor assigned:** 06-Jul-2023, PreQC No. DCRS-23-22685 (PQ); **Reviewed:** 19-Jul-2023, QC No DCRS-23-22685; **Revised:** 26-Jul-2023, Manuscript No. DCRS-23-22685 (R); **Published:** 02-Aug-2023, DOI: 10.35841/2572-5629-23.8.171.

Citation: Guzman M (2023) External Variables and Prenatal Gestational Diabetes in Mice during Pregnancy. Diabetes Case Rep. 8:171.

glucose metabolism. However, one limitation of this study was that it did not examine the impact of astaxanthin on the gut microbiota of GDM mice. Since previous studies have indicated that astaxanthin can alter the gut microbiota composition and function in obese and diabetic mice, it would be interesting to explore whether astaxanthin could also affect the gut microbiota of GDM mice and whether this could mediate its effects on GDM symptoms and reproductive outcomes. Future studies should also investigate the long-term effects of astaxanthin on the offspring's health and metabolic status. The mouse model of GDM provides a useful tool for studying the pathophysiology and treatment of GDM. Astaxanthin is a potential natural compound that can ameliorate GDM by reducing oxidative stress and improving glucose metabolism. However, more research is needed to elucidate the role of the gut microbiota in GDM and the potential interactions between astaxanthin and the gut microbiota in this condition.