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Commentary

Progression of Type 2 Diabetes Mellitus: Early Life Factors

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ABSTRACT

Type 2 diabetes mellitus is multifactorial disorder with a complicated Aetiology that includes genetic, epigenetic, and environmental aspects. In recent years, evidence from both human and animal models revealed early life variables to adult diabetes risk programming. Organ development is critical during the foetal and neonatal phases. Many maternal factors during pregnancy, including malnutrition, health (hyperglycemia and obesity), lifestyle (smoking, drinking, and a junk food diet), hormone administration, and even stress, may contribute to the risk of diabetes in infants later in life. Catch-up growth, lactation, glucocorticoids, and stress have all been implicated in the development of insulin resistance or T2DM in neonates.

Keywords: Glucocorticoids; Malnutrition; Insulin resistance

DESCRIPTION

T2DM is a metabolic condition caused by a combination of hereditary and environmental factors. The "barker hypothesis" is based on epidemiological and experimental findings that suggest harmful early life conditions may predispose to a high incidence of cardiovascular disease and metabolic illnesses later in life. From the embryonic stage to childhood, organs are developing and functionally maturing. Disruption of homeostasis during these critical years may raise the risk of insulin resistance and probably T2DM later in life.

Prenatal factors

Nutrition and diet has been proposed that the fetus's diet, both in terms of quality and quantity, may have long-term consequences. Despite changed gene expression, the altered chromosome structure during this process may be the cause of cell malfunction and increased susceptibility to disorders. Low protein diet during pregnancy may enhance oxidative stress, decreased HNF4a expression, defected mitochondriogenesis and mitochondria dysfunction, fibrosis and increased cell differentiation instead of proliferation was found in adult animal offspring cells, potentially contributing to ß-cell dysfunction and increasing the incidence of T2DM.

Over the last two decades, maternal obesity has shown rise dramatically. Human and animal findings implied that maternal obesity during pregnancy predisposes the offspring developing hyperinsulinemia, insulin resistance and T2DM. Long term exposure to testosterone during foetal development may disrupt insulin sensitivity pathways in the liver and muscle, increase hepatic gluconeogenesis, and impair the offspring's pancreas islet response to glucose.

Postnatal factors

Lactation, which includes early weaning as well as overfeeding by consuming extra milk, can lead to insulin resistance later in life. Reduced insulin sensitivity in the offspring may be caused by maternal stress, obesity, hyperglycemia, and even smoking during lactation, suggesting that breast milk can act as a "agent," transmitting altered hormone levels, insulin, or fatty acid contents from maternal circulation to the neonate.

Health disparities, particularly the risk of type 2 diabetes, are influenced by socioeconomic status. Low childhood socioeconomic status was found to be a strong independent predictor of adulthood type 2 diabetes, with the risk being increased when childhood socioeconomic status was coupled with adult obesity. Poor nutrition, harmful behaviours, and limited access to financial goods and socioeconomic

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CONCLUSION

There is a greater realization that prenatal, neonatal, and childhood factors might increase the risk of type 2 diabetes. Unfavorable conditions (such as poverty and starvation) or obesity cause long-term unfavourable consequences for children by enhancing adult sensitivity to T2DM. As a result, modifying prenatal or postnatal factors may be able to prevent or delay the onset of T2DM.