

A Short Note History of Gestational Diabetes Mellitus: Genetic Susceptibility

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GDM (gestational diabetes mellitus) is a kind of diabetes that develops or is discovered during pregnancy. It is one of the most prevalent pregnancy problems, with an increasing frequency year after year. The International Diabetes Federation (IDF) estimated the incidence of GDM in pregnant women aged 20 to 49 in 34 countries and regions around the world in 2013. The overall incidence was 14.2%, with Southeast Asia (20.9%) having the highest rate and North America and the Caribbean having the lowest rate. In China, the incidence rate was estimated to be between 5% and 10%. In terms of the pathophysiology of GDM, the traditional idea was that the foetus required more glucose as a result of increased maternal oestrogen and progesterone secretion, which resulted in an increase in insulin secretion and hyperinsulinemia in the early stages of pregnancy. Placental hormone, whose job was to antagonise insulin, rose with gestational age in the second trimester, leading in a steady decrease in the sensitivity of peripheral tissue to insulin.

Description

GDM incidence may be linked to genetic vulnerability, inflammatory cytokines, adipokines, and other risk factors such as obesity, according to experts. We examined the advancement of research into the aetiology of GDM in this publication. As previously stated, the incidence of GDM varies by ethnicity and geography, which partially reflects the hereditary impacts of GDM. Genetics is thought to play a role in disease development, and evidence of this can be found in genes, ethnicity, and family history. The functional integrity of the K-ATP channel, which is made up of eight subunits, four Kir6.2 subunits encoded by potassium channel inwardly rectifying subfamily J member 11 and four sulfonylurea receptor-1 subunits encoded by ATP-binding cassette transporter subfamily C member 8, is required for pancreatic-cell insulin secretion. It is clear that distinct KCNJ11 gene variations are linked to different forms of DM [1].

KCNJ11 and ABCC8 may have a role in insulin release control. Diabetes risk may be increased if these genes' co-expression is reduced. The T allele was linked to an elevated risk of GDM in one meta-analysis. However, the precise functional link between these gene networks and insulin release regulation remains unknown. To be decided Future study will focus on determining the specific role of the KCNJ11 gene mutation in diabetes and how it interacts with other genes, which could lead to the development of better treatments and diagnostics for this common disease [2]. TCF7L2, a transcription factor and part of the Wnt signalling pathway, has been linked to the development of GDM. The TCF7L2 gene's rs7903146 mutation was the most widely researched in relation to GDM, and it revealed a consistent and substantial connection across different groups. A meta-analysis of nine studies, for example, found that the T allele of rs7903146 was linked to an increased risk of GDM. The found heterogeneity for rs7903146 across studies was due to variations in the research populations in a stratification analysis by race/ethnicity; no substantial heterogeneity was observed in Asians, but there was significant variability among Caucasians [3].

The mRNA-binding protein-2 gene for insulin-like growth factor-2

In Asian and Caucasian populations, the relationship between IGF2BP2 and GDM risk had similar effect sizes. Cho, for instance, discovered a link between rs4402960, a genetic variant of the IGF2BP2 gene, and the incidence of GDM in Korean women. Wang and Lauenborg both found the same results among Chinese and Danish Caucasian women. According to a meta-analysis of this research, revealed the T allele of the rs4402960 gene was linked to a higher risk of GDM. The link between the rs1799884 mutation in the GCK gene and the risk of GDM has been studied extensively, and it has been found to be substantial [4].

Glucokinase, which acts as a glucose sensor in pancreatic b-cells, is involved in the regulation of glucose-stimulated insulin release. Mutations in the glucokinase gene (GCK) have been linked to a variety of glucose control problems, according to genetic studies. Heterozygous inactivating GCK mutations were the most common mutations, and they were the pathogenetic reasons of young-onset diabetes. Despite the fact that patients with GCK-MODY have persistent hyperglycemia after giving delivery, early control proved difficult. Furthermore, a meta-analysis of seven studies found a substantial link between the rs1799884 mutation and the risk of GDM. The frequency of GDM in different ethnic groups varies significantly. According to the IDF Atlas, the global prevalence of hyperglycemia in pregnancy (HIP) for women aged 20 to 49 is 16.2%. The prevalence of the disease varied by region, from 10.4 percent in Africa to 24.2 percent in Southeast Asia. However, these divisions obscured significant variance within ethnic groups [5].

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Conflict of Interest

None

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