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Genetic Variants of Diabetes

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Introduction

Diabetes mellitus is a complicated and heterogeneous metabolic illness that is frequently preceded or followed by consequences such as cardiovascular disease, hypertension, inflammation, chronic kidney disease, diabetic retinopathy, and nephropathy. However, the prevalence of these co-morbidities varies greatly between individuals and populations. As a result, it's not out of the question that specific genetic variants have a role in these diseases. We found four single nucleotide polymorphisms in AGTR1, IL6, NOS3, and TNFA genes (rs5186, rs1800795, rs1799983, and rs1800629, respectively) to be linked with each of these diseases. In different European and admixed American populations, the variant alleles are notably common. The fact that these polymorphisms are distributed differently in different ethnic groups suggests that some medications may be more successful in specific populations rather than all. Before selecting a medication for these disorders, population-specific genetic architectures should be examined. Insulin is a master regulator of cellular metabolism, and its diminished supply and/or influence on metabolic pathways has pleiotropic effects. Long-term micro- and macrovascular problems are the primary cause of morbidity and mortality among diabetics.

Description

Microangiopathy is a common symptom of diabetes that manifests itself most notably in the eyes (diabetic retinopathy) and kidneys (diabetic nephropathy). Microvascular damage, such as basement membrane thickening in small blood vessels, may be visible even in the early stages of diabetes. The risk of vascular problems in diabetics increases as comorbidities accumulate. High blood pressure (140/90 mmHg) is strongly linked to diabetes problems and is a risk factor for atherosclerotic cardiovascular disease (ASCVD) and microvascular consequences. Antihypertensive medicines minimize microvascular consequences and ASCVD occurrences in persons with diabetes. Hyperglycemia and hypertension are two major risk factors for chronic kidney disease (CKD), which affects about 40 percent of diabetic individuals. Diabetic Nephropathy (DN) and hypertensive nephropathy (HTN) are the primary causes of end-stage renal disease (ESRD) in both developed and developing countries. Albuminuria is a common symptom of diabetic kidney disease, and it is often associated with high blood pressure as the condition progresses [1,2].

Hypertension not only accelerates the course of CKD, but it also damages the kidneys by increasing arterial stiffness, endothelial dysfunction, the renin–angiotensin–aldosterone system (RAAS), and salt retention. Diabetes pathogenesis and micro- and macrovascular consequences are also linked to RAAS. Additionally, in people with chronic renal illness, a rise in urea in the blood produces a deficiency in pancreatic cell insulin secretion. Such interactions between diabetes and its consequences point to similar contributors. Single nucleotide polymorphisms (SNPs) can influence gene expression, messenger RNA (mRNA) stability, and/or translational efficiency, all of which can contribute to illness onset and progression. Diabetes risk alleles have been found in genes that control pancreatic beta cell growth and function, as well as insulin gene expression, secretion, and action. Hypertension, inflammation, chronic kidney disease, cardiovascular disease, diabetic retinopathy, and nephropathies have all been linked to SNPs.

The prevalence of diabetes-related complications differs by ethnic group. Variations in variant allele frequencies among ethnic groups may lead to a wide range of illness susceptibilities. The necessity of early diabetes screening and risk assessment in reducing these consequences is critical. T1DM is a kind of diabetes that affects 5–10% of diabetic persons. T1DM is caused by cellular-mediated autoimmune destruction of pancreatic beta-cells. It mostly affects children, and those who are affected are insulin-dependent for the rest of their lives and are prone to ketosis. T2DM affects 90–95 percent of diabetic people. For long periods of time, patients with type 2 diabetes may be asymptomatic. These patients may develop vascular problems such as nephropathy, neuropathy, retinopathy, and cardiovascular disease. In T2DM, the genetic component appears to have a greater impact than in T1DM. Another kind of diabetes that develops during pregnancy is gestational diabetes mellitus (GDM), which affects 1 to 14 percent of all pregnancies [3-5].

Conclusion

Diabetes mellitus is a complex disease whose presence and incidence are influenced by both environmental and hereditary factors. Genome-wide association studies have highlighted the genetic heterogeneity of diabetes and the fact that ethnicity might lead to various diabetes-susceptible genes. PPRAG (peroxisome proliferator-activates receptor gamma), IRS1 and 2 (insulin receptor substrate), KCNJ11 (potassium inwardly rectifying channel), and HNFA are among the genes linked to T2DM. Many genes, including Calpain 10 and TCF7L2 (transcription factor 7-like 2), have been linked to T2DM in genome-wide association studies (GWAS). HHEX (hematopoietic expressed homeobox), SLC30A8 (solute carrier family 30 (zinc transporter), member 8), CDK2A/B (cyclin-dependent kinase inhibitor 2A/B), and IG2BP2 (insulin-like growth factor 2) are some of the additional genes linked to T2DM. Some of these genes are found in beta cells or are involved in the secretion of insulin. Another key gene linked to T2DM and other related issues is MTHFR. Africans, Asians, and Caucasians had no connection with the C677T variant of the MTHFR gene. TCF7L2 is one of the most powerful genes linked to T2DM among candidate genes.

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