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An Editorial on Genetics of Diabetes Mellitus Patients

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Editorial

Diabetes mellitus (DM) is a metabolic disorder characterized by biochemical hyperglycemia. It is currently divided into four major categories: type-1 diabetes (T1DM), type 2 diabetes (T2DM), gestational diabetes (GDM), and other diabetes. The etiology is still variable within each category. Most DM pathogenesis is multifactorial, involving numerous genetic and environmental factors. T1DM, T2DM, and GDM are all polygenic diseases. There are at least six maturity-onset diabetes of the young (MODY) and many other genetic syndromes associated with DM that are monogenic in origin in the category of other DM. Long-term exposure to the hyperglycemia that characterises diabetes patients causes serious, often disabling or fatal complications. Emerging evidence suggests that genes play a significant role in a person's risk of developing complications.

This evidence comes from assessments of familial aggregation, racial and ethnic differences in incidence, and statistical analysis of family data. To date, evidence suggests that complication genes are distinct from diabetes genes. To identify genes that contribute to complications, molecular geneticists have used a variety of approaches, ranging from relatively simple analyses of specific candidate genes in small case-control comparisons to systematic evaluations of the human genome [1-3] using genome scans and linkage analysis in large collections of families. The findings indicate that genetic contributions to diabetes complications are diverse and complex, posing a significant challenge to researchers. Diabetes patients and their families are frequently predisposed to complications such as cardiovascular disease or nephropathy. Aside from their importance in the study of diabetes complications, such families may also be useful in understanding cardiovascular disease and nephropathy in the non diabetic population. The main risk factors for the development of micro- and macrovascular complications of diabetes are chronic hyperglycemia and diabetes duration.

Although it is thought that hyperglycemia causes damage to specific cell subtypes, such as meningeal cells in the renal glomerulus, capillary endothelial cells in the retina, and neurons and Schwann cells in peripheral nerves, the exact mechanisms underlying these damaging defects are unknown. The clustering of micro- and macro vascular complications in diabetes families suggests a strong genetic susceptibility. However, only a few genetic variants have been linked to either nephropathy [4,5] (ACE, ELMO1, FRMD3, and AKR1B1) or retinopathy (VEGF, AKR1B1, and EPO), and only a few studies have been conducted for genetic susceptibility to cardiovascular disease (ADIPOQ, GLUL) in diabetic patients. More data from larger studies and better phenotypically characterized cohorts are clearly required to facilitate genetic discoveries and uncover novel insights into the pathogenesis of diabetic complications.

Diabetes mellitus is a clinically diverse disorder characterized by hyperglycemia caused by an absolute or relative insulin deficiency. Its development is influenced by both genetic and non-genetic factors, making it a multifactorial disorder. Furthermore, in some cases, it may be a polygenic disorder caused by the combined effects of several genes with or without environmental factors. In the HLA-D region of chromosome 6 and near the insulin gene on chromosome 11, serological and/or DNA markers for genes that confer susceptibility to the insulin-dependent form of the disorder (IDDM; type 1) have been identified. Patients with non-insulin-dependent diabetes mellitus (NIDDM; type 2) are a more heterogeneous group than those with IDDM, and it is likely that similar clinical phenotypes are caused by different genetic defects in these patients.

Susceptibility to NIDDM can be conferred by the synthesis of an abnormal insulin/proinsulin molecule or an abnormal insulin receptor. The insulin and insulin receptor genes are located on chromosomes 11 and 19, respectively. Furthermore, studies of restriction fragment length polymorphism and disease associations suggest that two other genes on chromosome 11 may contribute to the development of NIDDM, one near the insulin gene on the short arm and the other near the Apo lipoprotein A-I gene on the long arm. In the absence of other genetic or non-genetic contributing factors, none of the susceptibility genes identified to date cause diabetes, indicating a multifactorial or polygenic origin for this disorder.

Conflict of Interest

The author declares that there is no conflict of interest associated with this paper.

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