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Diabetes Case Reports

Perspective

The Genetics behind Diabetes Mellitus and its Mechanism

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DESCRIPTION

Genetic factors play a significant role in both type 1 and type 2 diabetes risk. The environment and one's manner of life are also factors to consider. Diabetes is a chronic disease that affects millions of people in the United Kingdom alone. BACH2, a type 1 diabetes candidate risk gene, promotes apoptosis in pancreatic beta-cells *via* modulating JNK1 and interacting with the candidate gene PTPN2. Type 1 diabetes is a chronic autoimmune illness defined by the immune system's destruction of pancreatic -cells. More than 50 loci across the human genome have been linked to the risk of type 1 diabetes in linkage and genome-wide heritability. BACH2 (basic leucine zipper transcription factor 2 has recently been connected to a hereditary risk of type 1 diabetes, with the effect attributed to the immune system. In human and rodent cells, BACH2 inhibition worsened cytokine-induced apoptosis *via* the mitochondrial mechanism of cell death, whereas BACH2 overexpression showed protective benefits. By up regulating Mitogen-Activated Protein Kinase Kinase 7 (MKK7) and down regulating PTPN2, BACH2 silencing and exposure to proinflammatory cytokines elevated phosphorylation of the proapoptotic protein JNK1. Both JNK1 and BIM knockdown protected cells against cytokine-induced apoptosis in BACH2-silenced cells, and JNK1 enhanced phosphorylation of the proapoptotic protein BIM.

In multiple genome-wide association studies, BACH2 has been found to be genetically related to the risk of type 1 diabetes. This gene is found on chromosome 6q15, in a 365-kb strong linkage disequilibrium block where BACH2 is the only protein-coding gene. Different BACH2 Single Nucleotide Polymorphisms (SNPs) have been linked to the risk of type 1 diabetes. To date, it was assumed that specific SNPs in BACH2 play a role in type 1diabetes and other autoimmune disorders by influencing T-cell and B-cell development and thereby moderating autoimmune illnesses by balancing tolerance and immunity. The development of severe inflammation in the spleen, lungs, and other tissues limits the viability of BACH2 Knockout (KO) mice. Antinuclear and antidouble-stranded DNA autoantibodies are higher in sera from BACH2 KO mice, which are found in autoimmune disorders such lupus erythematous and rheumatoid arthritis. BACH2 is a transcriptional repressor that binds to the Antioxidant-Responsive Element (ARE) and the Maf Recognition Element (MARE). It's involved in virus-mediated cell death in HeLa cells and mild oxidative stress–induced apoptosis in Raji.

Regulation in blood sugar levels

Blood sugar levels are unusually high in people with type 2 diabetes. The body stops using and producing insulin adequately in this type of diabetes. Insulin is a hormone made in the pancreas that aids in blood sugar regulation. Insulin regulates the amount of glucose (a form of sugar) that is transported from the bloodstream into cells and used as an energy source. When blood sugar levels rise (as they do after a meal), the pancreas releases insulin to transport the excess glucose into cells, lowering blood glucose levels. T2D is the outcome of a complex combination of environmental influences and a substantial inherited component. The effect of genes on T2D has been demonstrated by history in genetic and genomic research in this domain.

CONCLUSION

Only a few T2D risk genes have been discovered *via* candidate gene and linkage-based investigations, but the introduction of genome-wide association studies has resulted in the discovery of a slew of new genes, including several that were previously unknown to play a role in T2D. Although many affected individuals have at least one close family member with the condition, such as a parent or sibling, type 2 diabetes does not have a clear pattern of inheritance. The risk of type 2 diabetes increases as the number of affected family members grows. The higher risk is most likely attributable to shared genetic variables, but it is also connected to share behavioral impacts (such as food and exercise habits) among family members.

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