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An Overview on Genetic Diabetic Disorder

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Introduction

Individual susceptibilities to genetic illnesses are determined by genetic variation in the human population. This is especially true in the case of diabetes. There are two main prerequisites for a gene to give diabetes genetic vulnerability. The gene must first play a role in the pathogenetic pathway. Second, the gene's function must be altered by Deoxyribonucleic Acid (DNA) variations. All living species have DNA as their genetic foundation. A DNA molecule is made up of a lengthy chain of nucleotides, each of which contains one of the four bases: adenine (A), cytosine (c), and guanine (G). DNA sequences in the human genome are arranged in a double-helix shape. In total, three billion DNA base pairs are dispersed throughout 22 pairs of autosomes and two sex chromosomes (X and Y). From longest to shortest, chromosomes are numbered 1 (Chr1) through 22. The human nuclear genome contains around 20,000-25,000 protein-coding genes. In addition to the nuclear genome, the human mitochondrial genome is made up of a tiny circular DNA molecule. The human mitochondrial genome is 16.6 kilo base pairs (kb) long, with varied copy counts in different tissues' cells. The mitochondrial genome encodes 37 genes, according to research.

Description

A single base alteration is the most prevalent type of DNA variation in the human genome. These sequence variations can range in frequency from extremely rare to 50%. (in which case the two alleles have equal frequencies). Single-nucleotide polymorphisms are what they're called (SNPs). SNPs (single nucleotide polymorphisms) are the most researched DNA variations, occurring every 100-300 bases in the human genome. The majority of SNPs in the human genome have no effect on gene activity. By producing an amino acid change in a peptide (i.e., a nonsynonymous SNP) or by controlling gene expression and/or alternative splicing, SNPs can alter the function of a gene. The substituted nucleotide may correspond to either enhanced (gain) or diminished (loss) gene function when compared to the original nucleotide.

Other types of frequent genetic polymorphisms include tandem repeat polymorphisms and insertion/deletion polymorphisms, in addition to SNPs. Through changes in gene function, any sort of polymorphism might alter susceptibility to a genetic disease (or phenotypic trait). Because polymorphisms from many different genes must contribute to the disease, most polymorphism-associated diseases are not inherited in a Mendelian pattern (genetically complex disease). In healthy people, a polymorphism can cause a shift in gene function. A Mendelian hereditary (monogenic) disease caused by a single defective gene, on the other hand, is usually a rare mutation with a significant impact on gene function [1-5].

Typ-2 diabetes is assumed to be caused by a combination of environmental, behavioural, and genetic variables, with heritability estimates ranging from 25 percent to 72 percent based on family and twin studies. Since early 2007, data on the genetics of type 2 diabetes and related variables has exploded thanks to Genome Wide Association Studies (GWAS). These GWAS took place in the context of genotyping arrays populated by common Single Nucleotide Polymorphisms (SNPs), which were used in a variety of cohorts that eventually merged to form big worldwide consortia.

Conclusion

As a result, researchers have begun to compile a list of genetic loci that influence type 2 diabetes and quantitative glycemic features. Over 100 type 2 diabetes-associated loci have been found, in addition to others implicated in determining quantitative glycemic features including insulin resistance. However, no widely common variant has been identified to have a bigger effect than the TCF7L2 rs7903146 SNP, which has a small effect (odds ratio 1.4). GWAS discoveries, on the other hand, have revealed new pathways, pointed to fundamental biology, highlighted the significance of beta cell dysfunction in type 2 diabetes, corroborated past epidemiologic findings, and given potential targets for pharmacotherapy and pharmacogenetic clinical trials.

Conflict of Interest

None.

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