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Opinion Article

Authentication of Next-generation Sequencing Technologies in Diabetes

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DESCRIPTION

One of the most important public health issues in the world, diabetes seriously compromises both the general public's health and the global economy. Diabetes, along with cardiovascular disease, cancer, and respiratory illness, which together account for more than 80% of all premature Non-Communicable Disease (NCD) mortality, has a 2-3 times greater risk of death from all causes. The International Diabetes Federation (IDF) estimates that there are currently more than 537 million people living with diabetes worldwide, an increase of 16% (74 million) since the IDF's previous estimations for 2019. In 2019, diabetes was the tenth leading cause of death, with an estimated 1.5 million fatalities directly attributable to the condition World Health Organization (WHO). Type 1 and type 2 diabetes are differentiated based on how responsive the body is to insulin. Diabetes (T2D), formerly called as insulin independence, is the most common kind of diabetes (95%) and develops when the body does not respond adequately to insulin. It is now recognised in youngsters despite earlier being thought to only affect adults. T1D is an insulin-dependent condition that is characterised by insufficient insulin synthesis from pancreatic -cells, necessitating daily injection.

The most prevalent trait of T2D patients is obesity or a higher than average body fat percentage, mostly in the abdominal area. In this condition, adipokine dysregulation and increased release of Free Fatty acids (FFA) are two examples of how adipose tissue causes insulin resistance (IR). As a consequence, the main causes of the T2D epidemic obesity, sedentary behaviour, high-calorie diets, and population aging are responsible for the epidemic's increased incidence rates of T2D. The malfunction of physiological and cellular feedback processes results in an imbalance between insulin secretion and action, which raises blood glucose levels. When -cells are dysfunctional, the body's ability to maintain physiological glucose levels is limited by decreased insulin production. Adversely, insulin resistance causes an increase in the liver's production of glucose and a fall in the uptake of glucose by muscle, the liver, and adipose tissue. Even while both processes occur early in the pathophysiology and help the disease progress, -cell dysfunction is typically more severe than IR. When IR and -cell dysfunction coexist, hyperglycemia rises, which aids in the emergence of T2D symptoms. Precision medicine to treat diabetes requires a thorough understanding of pathophysiology, social and psychological variables, environmental factors, and the limitations of existing therapy.

Monogenic diabetes is caused by a number of distinct processes gene abnormalities in the pancreatic/-cell development pathway (*PDX1*, *HNF1B*, *GATA4*, *GATA6*, and *PTF1A*) lead to agenesis or hypoplasia of the pancreas. Similar to this, other genes, including those involved in glucose sensing (GCK), ATP responsiveness (ABCC8, KCNJ11), ER stress (INS, EIF2AK3), and transcriptional regulation, are engaged in decreased -cell mass or proliferations (*INS*, *PTF1A*), or -cell dysfunction (*HNF1A*, *HNF1B*, *HNF4A*, *NEUROD1*, *PDX1*). Since single gene mutations are the primary cause of monogenic diabetes, this class appears to offer the advantage of accurate diagnosis of nonoverlapping etiological subgroups for which treatment options can be implemented. As monogenic diabetes is caused by a number of distinct processes; gene abnormalities in the pancreatic cell development pathway (*PDX1*, *HNF1B*, *GATA4*, *GATA6*, and *PTF1A*) lead to agenesis or hypoplasia of the pancreas. Similar to this, other genes, including those involved in glucose sensing (GCK), ATP responsiveness (ABCC8, KCNJ11), ER stress (*INS*, *EIF2AK3*), and transcriptional regulation, are engaged in decreased -cell mass or proliferations (*INS*, *PTF1A*), or -cell dysfunction (*HNF1A*, *HNF1B*, *HNF4A*, *NEUROD1*, *PDX1*). Since single gene mutations are the primary cause of monogenic diabetes, this class appears to offer the advantage of accurate diagnosis of nonoverlapping etiological subgroups for which treatment options can be implemented. As a consequence, monogenic diabetes is the most appropriate target to treat the disease.

The best target for the disease's treatment is monogenic diabetes. Monogenic forms of diabetes, despite they only make up a small portion of all cases, offer a chance to show how successful pharmacogenomics techniques are. It has been more common in recent years to refer to other hereditary types of diabetes, like

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neonatal or syndrome diabetes, as monogenic diabetes. Due to on-going advances in understanding of the human genome and new technology, researchers have witnessed the comprehensive dissection of the genetic causes of these monogenic forms of diabetes. This has provided a wealth of information regarding the mechanisms responsible for the growth and maintenance of insulin-secreting pancreatic beta-cells. Maturity-Onset Diabetes of the Young (MODY) is the term used to describe non-insulin dependent autosomal dominantly inherited diabetes that manifests early in life (before the age of 25 years). The American College of Medical Genetics has determined that there are 10 subtypes of MODY, although the OMIM list previously thought there were 14. Unfortunately, it has been claimed that 50–80% of people with MODY go undiagnosed or receive minimal therapy because they are mistaken for people with type 1 or type 2 diabetes.

Recent years have seen an increase in the use of Next-Generation Sequencing (NGS) methods in diagnostic procedures since they enable the parallel analysis of many genes. There are numerous genetic reasons, but mutations in the three genes account for up to 95% of instances of MODY (HNF1A, HNF4A, and GCK). The genetic origins of Type 2 Diabetes (T2D) can be studied by a variety of methods using Next-Generation Sequencing (NGS), including mapping rare and common genetic variants, identifying epigenetic disease markers, and analysing the disease's associated microbiome.