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Commentary

Type 2 Diabetes and Its Correlation with Pancreatic Insulin

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DESCRIPTION

Diabetes mellitus is a broad term that encompasses a wide range of problems with various aetiologies that are all linked by one feature: A pathological elevation of blood glucose. Sustained hyperglycemia causes tissue damage in vulnerable organs, leading to secondary complications such as retinopathy, nephropathy, peripheral neuropathy, cardiovascular disease, and stroke. Diabetes currently affects 387 million people worldwide. The disease's dramatic rise in recent years not only causes individual misery, but also places a massive and growing burden on healthcare systems and the global economy. Indeed, diabetes and its complications consume up to 10% of many countries healthcare budgets.

Type 2 diabetes (T2D) is the most common type of diabetes, accounting for roughly 90% of cases. It has a significant genetic component, which is exacerbated by factors such as age, obesity, diet, physical activity, and pregnancy. T2D is characterized by insufficient insulin secretion from pancreatic islet cells, as well as impaired insulin action in target tissues such as muscle, liver, and fat (a condition termed insulin resistance). When insulin secretion is insufficient to compensate for insulin resistance, hyperglycemia occurs.

Type 1 diabetes (T1D) is much less common than T2D, accounting for only 10% of cases. It is caused by an autoimmune attack on the cells, which results in insulin deficiency, though a small number of functioning cells may survive. T1D usually manifests itself in childhood or early adulthood.

Furthermore, there are rare inherited monogenic forms of diabetes that typically manifest in childhood and account for only 1 to 2% of all diabetes cases. Unlike T2D, which is thought to be caused by multiple genes, monogenic diabetes is thought to be caused by mutations in a single gene. Many of these genes encode transcriptional regulators, metabolic enzymes, and ion channels that control cell stimulus-secretion coupling or may affect pancreas development. Interestingly, common genetic variants in many of the genes known to cause monogenic diabetes increase T2D risk; thus, their investigation may aid in understanding the aetiology of T2D.

Due to a lack of cells, T1D must be treated with insulin injections. T2D therapy begins with dietary and lifestyle changes, followed by oral hypoglycemic agents that may increase insulin secretion (for example, sulfonylureas) or reduce insulin resistance or hepatic glucose output (for example, metformin). If these fail to control hyperglycemia, insulin is administered. Depending on the gene involved, monogenic diabetes is treated in a variety of ways.

β-cells avoid excessive insulin secretion

To avoid excessive insulin secretion and hypoglycemia, cells have evolved important metabolic features, particularly during exercise. For starters, insulin secretion is extremely sensitive to changes in blood glucose levels. This is accomplished by altering intracellular ATP levels, cell electrical activity, and insulin vesicle release to link glucose metabolism and insulin secretion. When blood glucose levels rise, the majority of the glucose taken up by the cell is metabolised via oxidative phosphorylation, raising intracellular ATP. This causes KATP channels to close, resulting in cell electrical activity and calcium influx (*via* voltage-gated calcium channels), which stimulates insulin release. When blood glucose levels fall, insulin secretion is rapidly turned off due to a decrease in intracellular ATP in cells, resulting in the opening of KATP channels, membrane hyperpolarization, reduced calcium entry, and thus inhibition of insulin secretion.

Second, while many metabolic genes are expressed in other tissues, they are not expressed in pancreatic cells. Lactate Dehydrogenase (LDHA) and the monocarboxylate transporter 1 (MCT1/SLC16A1), which are involved in the metabolism of lactate and pyruvate, are examples of 'disallowed' genes. During exercise, this prevents insulin secretion in response to circulating lactate and pyruvate. Mutations in the SLC16A1 gene cause aberrant expression in cells, which causes exercise-induced hypoglycemia by allowing pyruvate-induced insulin secretion. Exercise-induced hypoglycemia could have been lethal in early humans because it hampered escape from a predator; the absence of MCT1 ensures that insulin secretion remains turned off during exercise. Adrenaline also inhibits insulin secretion, ensuring that blood glucose levels do not drop during exercise or the "fight-or-flight" response.

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