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Experimental Redox Homeostasis in Type 2 Diabetes Mellitus: Molecular Genetics

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INTRODUCTION

One in 10 people worldwide are affected by diabetes mellitus, one of the most prevalent metabolic diseases. More than 537 million people worldwide have diabetes, according to the most recent edition of the IDF Diabetes Atlas. By 2030, 6.43 billion people worldwide, or 11.3% of the population, will have diabetes. By 2045, if current trends hold, this figure will soar to a startling 783 million (12.2%). Type 2 diabetes mellitus, also known as non-insulin dependent diabetes mellitus, affects the vast majority of diabetics worldwide. This chronic illness is serious and results from inadequate insulin production or ineffective insulin use. T2D is a heterogeneous disease determined by genetic, epigenetic, and environmental risk factors that interact strongly, according to epidemiological studies carried out over the past few decades. Numerous disease-associated gene polymorphisms have been discovered as a result of the extensive genetic research that has been done to understand the molecular mechanisms underlying T2D, including beta cell dysfunction, insulin resistance, an imbalance in redox homeostasis, and impairment of incretin signalling. However, many aspects of the molecular mechanisms underlying disease pathogenesis are still not well understood. One of the main pathological disorders underlying the onset and progression of type 2 diabetes is oxidative stress, which is defined as the imbalance between the production of free radicals and their neutralization by antioxidant enzymes. This imbalance is caused by excess ROS or oxidants over the capacity of the cell to realize an effective antioxidant response.

DESCRIPTION

It is generally accepted that pancreatic beta cell failure, which manifests as a gradient reduction in beta cell mass and insulin production in response to glucose, is the cause of chronic hyperglycemia, the main diagnostic indicator of T2D. Beta cells start oxidizing fatty acids instead of glucose when their metabolic flexibility is reduced, which leads to the production of dangerous byproducts (peroxides) and a reduction in insulin secretion. It is noteworthy that the endocrine portion of the pancreas is damaged by at least three biological processes: Beta cell apoptosis, dedifferentiation, and conversion into alpha cells that produce glucagon. Numerous studies have examined the disorders that follow a loss of insulin-producing capacity in T2D and demonstrated a decrease in the expression of crucial transcription factors controlling the function of beta cells. Experimental studies on transgenic mice have shown that the islets of Langerhans contain bihormonal cells that produce both glucagon and insulin after the conversion of beta cells into alpha cells. Given that alpha cells are more resilient to the metabolic stress brought on by excessive nutrition, a change in the phenotype of pancreatic beta cells may act as a mechanism for cell mass conservation.

CONCLUSION

Numerous studies have shown that people with type 2 diabetes have impaired redox homeostasis and oxidative stress, which are caused by an excess of free radicals and, on the other hand, a deficiency in endogenous antioxidants, primarily glutathione. Increased reactive oxygen species and oxidative stress levels have been linked to pathological conditions like beta cell dysfunction and insulin resistance in type 2 diabetes. Compared to healthy people, people with T2D have lower levels of reduced glutathione and a slower rate of its biosynthesis. The lower levels of glutathione are correlated with higher blood glucose levels and the development of the disease.

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