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Insights of Mitochondrial Uncoupling Proteins and Age-related Oxidative Stress in Diabetes and its Metabolism

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Introduction

Mitochondria are the cell organelles responsible for energy production. Mitochondria are required for aerobic ATP synthesis via oxidative phosphorylation, heme, cholesterol, and phospholipid synthesis, as well as apoptosis and cell signalling. They are distinct from other cell organelles because they have their own genome. Mitochondrial DNA (mtDNA) has the ability to self-replicate and transcribe. Because mtDNA is small and circular, it only encodes proteins required for normal oxidative phosphorylation, such as some subunits of the mitochondrial respiratory chain [1-3] and some tRNA and rRNAs for mitochondrial translational machinery assembly.

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Description

Increased expression of these pathways' gene products causes additional cellular damage. Damage to mtDNA can impair viability and other cellular functions, and maintaining its integrity as we age is critical for survival. As a result, mitochondrial dysfunction has been linked to a variety of aging-related diseases, including type 2 diabetes (T2D), neurodegenerative diseases, cancer, and cardiovascular disease. T2D is characterised by insufficient insulin production, excessive glucagon secretion by pancreatic beta cells, and insulin resistance [4,5], which results in impaired energy metabolism in the pancreas, liver, skeletal muscle, and other organs.

Data for 2021 show that the global prevalence of T2D in people aged 20 to 79 is 10.5%. The prevalence is lowest among young adults aged 20-24 years (2.2%) and gradually rises to 24% among elderly people aged 75-79 years. The projections for 2045 are similar, with the exception that the percentages in each age group will be slightly higher. Most importantly, as the world population ages, the proportion of people with T2D over the age of 60 will increase, as will the incidence of cardiovascular complications and metabolic syndrome. The increased prevalence of comorbidities and the concurrent use of multiple medications, which may result in drug interactions in older diabetic patients, make T2D management particularly complex and challenging. As a result, new approaches to T2D control are required, including individualized treatment strategies.

Conclusion

Although the primary cause of T2D has not been identified, mitochondrial dysfunction in the organs responsible for insulin secretion (pancreatic beta cells), the target organs of insulin action (skeletal and cardiac muscle cells, as well as liver cells), and the organs associated with the major complications of T2D (kidneys, retina, nerves, and vascular cells) may play an important role in the disease's pathophysiology. Because ATP is required for insulin production and release, altered mitochondrial bioenergetics associated with impaired glucose and fatty acid metabolism have been linked to insulin and glucagon secretion defects in T2D.

The inner mitochondrial membrane of various tissues contains a group of five homologous proteins known as UCPs. They are involved in a variety of tasks and cellular functions, ranging from thermoregulation to insulin secretion modulation and neuroprotection. The mitochondria of skeletal muscle have the most diverse spectrum of UCPs, expressing all five UCPs. As a result, skeletal muscle is one of the most well-studied tissues in terms of advancing our understanding of UCP function and associated pathologies.

Conflict of Interest

None.

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