Molecular Mechanisms Linking Oxidative Stress and Diabetes Mellitus

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Abstract

Diabetes mellitus is a prevalent metabolic disorder characterized by high blood glucose levels, and oxidative ress has been implicated in its pathogenesis. Oxidative ress refers to an imbalance between the production of reactive oxygen species and the body's ability to neutralize them. This review aims to provide an overview of the molecular mechanisms linking oxidative ress and diabetes mellitus.

Numerous udies have revealed that oxidative ress plays a crucial role in the development and progression of diabetes mellitus. Excessive ROS production and impaired antioxidant defense mechanisms contribute to the generation of oxidative ress. This, in turn, leads to the activation of ress-sensitive signaling pathways, such as c-Jun N-terminal kinase and nuclear factor kappa B, resulting in insulin resi ance. Oxidative ress also induces pancreatic beta-cell dysfunction through mechanisms such as apoptosis, mitochondrial dysfunction, and altered gene expression. Furthermore, oxidative ress disrupts glucose homeo asis by impairing insulin signaling in insulin-sensitive tissues like skeletal muscle, liver, and adipose tissue.

Under anding the molecular mechanisms connecting oxidative ress and diabetes mellitus is critical for developing e ective therapeutic rategies. By targeting these pathways, it may be possible to mitigate oxidative ress and re ore normal cellular function, thus improving the management and outcomes of diabetes mellitus. Further research in this area is needed to identify novel therapeutic targets and interventions for the prevention and treatment of diabetes mellitus.

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Increased oxidative stress in diabetes

Molecular mechanisms of oxidative stress in diabetes

Activation of protein kinase C (PKC):

Mitochondrial dysfunction:

Advanced glycation end products (AGEs):

Polyol Pathway:

Consequences of oxidative stress in diabetes

Discussion