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Editorial

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Molecular and Cellular Mechanisms Underlying the Development and Progression of Diabetic Neuropathy

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Introduction

Diabetic neuropathy is one of the most common and debilitating chronic complications of diabetes mellitus, affecting sensory, motor, and autonomic nerves. It significantly impairs quality of life and increases the risk of foot ulcers, infections, and lower-limb amputations. Persistent hyperglycemia is the primary driving factor in the development of diabetic neuropathy, triggering a cascade of metabolic and vascular disturbances that damage peripheral nerves. Understanding the underlying mechanisms of diabetic neuropathy is essential for developing effective strategies for prevention, early diagnosis, and treatment [1,2].

Discussion

Multiple interconnected biochemical and cellular pathways contribute to nerve injury in diabetic neuropathy. One of the key mechanisms is chronic hyperglycemia-induced metabolic stress. Excess intracellular glucose is shunted into alternative metabolic pathways, including the polyol pathway, where glucose is converted to sorbitol by aldose reductase. Accumulation of sorbitol increases osmotic stress and reduces levels of important antioxidants such as nicotinamide adenine dinucleotide phosphate, leading to increased oxidative stress and nerve damage [3,4].

Oxidative stress plays a central role in diabetic neuropathy. Elevated glucose levels enhance the production of reactive oxygen species in mitochondria, resulting in mitochondrial dysfunction and impaired energy production. Oxidative damage affects neuronal membranes, proteins, and DNA, ultimately leading to axonal degeneration. In parallel, advanced glycation end products formed through non-enzymatic glycation of proteins accumulate in neural and vascular tissues. Interaction of these products with their receptors activates inflammatory signaling pathways and further increases oxidative stress [5].

Microvascular dysfunction is another critical contributor to diabetic neuropathy. Hyperglycemia damages endothelial cells, reducing nitric oxide availability and impairing blood flow to peripheral nerves. This ischemia leads to reduced oxygen and nutrient delivery, exacerbating nerve injury. Additionally, chronic inflammation contributes to neuropathic progression, with elevated levels of pro-inflammatory cytokines promoting neuronal apoptosis and Schwann cell dysfunction.

Impaired neurotrophic support also plays a role in diabetic neuropathy. Reduced levels of nerve growth factor and other neurotrophins compromise nerve regeneration and repair. Insulin deficiency or resistance further disrupts neuronal metabolism, as insulin has direct neuroprotective and trophic effects on peripheral nerves.

Conclusion

Diabetic neuropathy results from a complex interplay of metabolic, oxidative, vascular, and inflammatory mechanisms driven primarily by chronic hyperglycemia. These interconnected pathways lead to progressive nerve damage and functional impairment. Effective management of diabetic neuropathy requires early glycemic control and therapeutic strategies targeting oxidative stress, inflammation, and microvascular dysfunction. Continued research into these mechanisms is essential for the development of disease-modifying treatments that can prevent or slow the progression of diabetic neuropathy.

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