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[Inflammation as a Therapeutic Target for Diabetic Neuropathies.](#)

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Abstract

Diabetic neuropathies (DNs) are one of the most prevalent chronic complications of diabetes and a major cause of disability, high mortality, and poor quality of life. Given the complex anatomy of the peripheral nervous system and types of fiber dysfunction, DN has a wide spectrum of clinical manifestations. The treatment of DN continues to be challenging, likely due to the complex pathogenesis that involves an array of systemic and cellular imbalances in glucose and lipid metabolism. These lead to the activation of various biochemical pathways, including increased oxidative/nitrosative stress, activation of the polyol and protein kinase C pathways, activation of polyADP-ribose polymerase, and activation of genes involved in neuronal damage, cyclooxygenase-2 activation, endothelial dysfunction, altered Na(+)/K(+)-ATPase pump function, impaired C-peptide-related signaling pathways, endoplasmic reticulum stress, and low-grade inflammation. This review summarizes current evidence regarding the role of low-grade inflammation as a potential therapeutic target for DN.

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