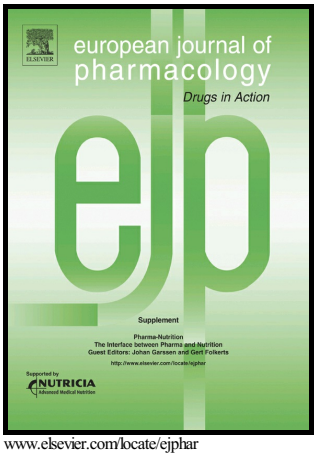

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Molecular mechanism of diabetic neuropathy and its pharmacotherapeutic targets

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Abstract

Diabetic neuropathy is regarded as one of the most debilitating outcomes of diabetes mellitus and may cause pain, decreased motility, and even amputation. Diabetic neuropathy includes multiple forms, ranging from discomfort to death. Prognosis of diabetic neuropathy is an uphill task as it remains silent for several years after the onset of diabetes. Hyperglycemia, apart from inducing oxidative stress in neurons, also leads to activation of multiple biochemical pathways which constitute the major source of damage and are potential therapeutic targets in diabetic neuropathy. A vast array of molecular pathways, including polyol pathway, hexosamine pathway, PKCs signaling, oxidative stress, AGEs pathway, PARP pathway, MAPK pathway, NF-κB signaling, hedgehog pathways, TNF-α signaling, cyclooxygenase pathway, interleukins, lipoxygenase pathway, nerve growth factor, Wnt pathway, autophagy, and GSK3 signaling may be accounted for the pathogenesis and progression of diabetic neuropathy. Although