Metformin; A Review to its Anti-Neuroinflammatory Properties

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Abstract

Neuroinflammatory disorders include a wide spectrum of disorders in which immune system injures components of the nervous system. Despite the advances in therapy, lack of efficient curative therapies for these disorders not only affects the quality of life but also places huge burden on the society. These limitations have necessitated therapeutic interventions for developing more efficient regimen. Recently, emerging new therapeutic approaches like targeting underlying pathways offered some respite and drawn much attention. Accordingly, adenosine monophosphate-activated protein kinase (AMPK), signal transducer and activator of transcription3 (STAT3), P65/NF-κB, mTOR, and PI3K/Akt pathways have been reported to be affected in neuroinflammation, and, thus could be considered as novel molecular targets for therapy. Among the available drugs, metformin has shown a great potential in targeting aforementioned pathways. Studies declared that metformin could suppress neuroinflammation in neurological deficits such as Alzheimer disease, brain injury, and neuropathic pain. Besides, preclinical and clinical studies indicated that metformin not only improves inflammation through affecting metabolic parameters, but also exerts direct anti-inflammatory effects. In this view, metformin could be defined as an available, cost-effective drug with great potency to target multiple signaling pathways in neuroinflammation. However, requiring metformin in clinical stage demands enough knowledge about the mechanisms and pathways involved in its anti-neuroinflammatory activities and comprehensive review of its in-vivo utility. This review discusses the published evidence of anti-neuroinflammatory properties of metformin and its clinical implication in neuroinflammatory disorders with the objective of paving the way for further clinical application and better management of diseases.

Keywords: Neuroinflammation, Metformin, Therapeutic Management, Signaling Pathways

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