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Limonin as serotonin mimetic to protect from Alzheimer's disease

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Limonin (LM), a limonoid glycoside isolated from leaves, fruits, and seeds of citrus plants, belongs to the class of furanolactones [1]. Recently, LM has gained significant interest due to its multiple biological activities such as anti-inflammatory, antitumor, antimicrobial, and antifeedant activity. Lu et al. [2] demonstrated that pretreatment of LPS-induced microglial cells with LM substantially prohibited the formation of ROS and prevented the deposition of NO, PGE2, IL-1β, IL-6, and TNF-α [2]. Furthermore, LM significantly reduced the expressions of iNOS and COX-2 [3]. Their results indicated that LM inhibited inflammatory response through repressing the formation of ROS, accretion and discharge of cytokines, and activation of inflammatory mediators, suggesting its potential as a neuroprotective agent.

LM could act as a serotonin mimic, presenting an intriguing therapeutic avenue for treating Alzheimer's Disease (AD). Serotonin (5-HT) is a neurotransmitter crucial for mood regulation, cognition, and neuroprotection. Dysregulation of serotonin levels has been implicated in the pathophysiology of AD [4]. Given LM's structural similarity to serotonin, it could potentially bind to serotonin receptors, mimicking its effects and restoring normal neurotransmitter function. This interaction might modulate neural signalling pathways associated with neuroprotection and cognitive enhancement. By mimicking serotonin, LM could significantly improve synaptic plasticity, reduce amyloid-beta accumulation, and mitigate neuroinflammatory responses. Additionally, LM's antioxidant properties might reduce oxidative stress, and its activation of pathways such as PI3K/Akt could inhibit apoptosis, protecting neurons from amyloid-beta toxicity. Furthermore, LM might promote amyloid-beta clearance and stabilize tau proteins, potentially mitigating AD pathology. Thus, if LM functions as a neuroprotectant and serotonin mimic, it could hold significant promise for treating AD and other neurodegenerative disorders, warranting further exploration.

Conflict of interest

The authors declared no conflict of interest.

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