

Hesperidin

There is increasing evidence showing that [mild traumatic brain injury](#) (mTBI) is associated with increased [depression](#)-related disorders in humans.

Studies suggested that dietary intake or supplementation of natural [flavonoids](#) like hesperidin can be used for therapy of patients with brain injury and depression. However, the exact mechanisms by which hesperidin indicates its neuroprotective effects are not fully understood. The purpose of this study was to explore the influence of hesperidin on depression-related symptoms in a mouse model of mTBI, and that what mechanisms are primarily involved in the antidepressant effects of this bioflavonoid.

Ten days after mTBI-induction, mice received oral hesperidin treatment (50 mg/kg/14 days), then animals were subjected to different depression tests including sucrose preference test, forced swim test, novelty-suppressed feeding test, and tail suspension test. We also measured levels of tumor necrosis factor (TNF)- α , interleukin-(IL)-1 β , malondialdehyde (MDA), and brain-derived-neurotrophic-factor ([BDNF](#)) in the hippocampus.

Our results show that mTBI induction induced depressive-like behaviors in mice by increasing inflammatory cytokines (IL-1 β and TNF- α) and oxidative stress marker (MDA), and reducing BDNF levels in the hippocampus. Interestingly, hesperidin treatment was effective to significantly reduce depression-related symptoms in mTBI-induced mice. In addition, hesperidin decreased the levels of IL-1 β , TNF- α and MDA, and increased BDNF levels in the hippocampus. The major strength of our study is that four behavioral tests gave similar results.

This study suggests that antidepressant-like effect of hesperidin may be mediated, at least in part, by decreased neuroinflammation and oxidative damage, and enhanced BDNF production in the hippocampus ¹⁾.

¹⁾

Kosari-Nasab M, Shokouhi G, Ghorbanihaghjo A, Abbasi MM, Salari AA. Hesperidin attenuates depression-related symptoms in mice with mild traumatic brain injury. Life Sci. 2018 Oct 20. pii: S0024-3205(18)30665-9. doi: 10.1016/j.lfs.2018.10.040. [Epub ahead of print] PubMed PMID: 30352242.

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