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The Mitigation of Quercetin on Lead-Induced Neuroinflammation in a Rat Model: Changes in Neuroinflammatory Markers and Memory

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Abstract : The neuroprotective role of inflammation from detrimental intrinsic and extrinsic factors has been reported. However, the overactivation of astrocytes and microglia due to lead toxicity produce excessive pro-inflammatory cytokines, mediating neurodegenerative diseases. The present study investigated the mitigatory effects of quercetin on neuroinflammation, correlating with memory function in lead-exposed rats. In this study, Wistar rats were administered orally with Quercetin (Q: 60 mg/kg) and Succimer as a standard drug (S: 10 mg/kg) for 21 days after lead exposure (Pb: 125 mg/kg) of 21 days or in combination with Pb, once daily for 42 days. Working and reference memory was assessed using an Eight-arm radial water maze (8-ARWM). The changes in brain lead level, the neuronal nitric oxide synthase (nNOS) activity, and the level of neuroinflammatory markers such as tumour necrosis factor-alpha (TNF-α) and Interleukin 1 Beta (IL-1β) were determined. Immunohistochemically, astrocyte expression was evaluated. The results showed that the brain level of lead was increased significantly in lead-exposed rats. The expression of astrocytes increased in the CA3 and CA1 regions of the hippocampus, and the levels of brain TNF-α and IL-1β increased in lead-exposed rats. Lead impaired reference and working memory by increasing reference memory errors and working memory incorrect errors in lead-exposed rats. However, quercetin treatment effectively improved memory and inhibited neuroinflammation by reducing astrocytes' expression and the levels of TNF-α and IL-1β. The expression of astrocytes and the levels of TNF-α and IL-1β correlated with memory function. The possible explanation for quercetin's anti-neuroinflammatory effect is that it modulates the activity of cellular proteins involved in the inflammatory response; inhibits the transcription factor of nuclear factor-kappa B (NF-κB), which regulates the expression of proinflammatory molecules; inhibits kinases required for the synthesis of Glial fibrillary acidic protein (GFAP) and modifies the phosphorylation of some proteins, which affect the structure and function of intermediate filament proteins; and, lastly, induces Cyclic-AMP Response Element Binding (CREB) activation and neurogenesis as a compensatory mechanism for memory deficits and neuronal cell death. In conclusion, the levels of neuroinflammatory markers negatively correlated with memory function. Thus, quercetin may be a promising therapy in neuroinflammation and memory dysfunction in populations prone to lead exposure.

Keywords : lead, quercetin, neuroinflammation, memory
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