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Moringa oleifera Lam. leaf extract mitigates carbon tetrachloride-mediated hepatic inflammation and apoptosis via targeting oxidative stress and toll-like

receptor 4/ nuclear factor kappa B pathway in mice

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## Impact Factor: 2.455

## ISSN: 2213-4530

Carbon tetrachloride (CCl<sub>4</sub>) is a hepatotoxin that triggers liver damage. This study aimed to evaluate the protective effect of phytochemicals detected in Moringa oleifera Lam. leaf extract (MOLE) on CCl4-induced hepatotoxicity in mice. Phytochemicals, total phenolics, and total flavonoids were detected in MOLE. MOLE markedly decreased the elevation of serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) in consistence with the ameliorating effect on CCl4-induced histopathological abnormalities. Moreover, MOLE significantly alleviated the decrease in the antioxidant defense mechanism induced by CCl4. The suppressing effect of MOLE on the boosted inflammatory pathway triggered by CCl4 was detected by measuring the protein levels of nuclear factor kappa-light-chain-enhancer of activated B-cells (NF- $\kappa$ B-p65), toll-like receptor 4 (TLR4), tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), interleukin (IL)-6, IL-1 $\beta$ , and IL-8 as well as the relative expressions of nuclear factor kappa B (NF- $\kappa$ B), TNF- $\alpha$ , IL-1 $\beta$ , and TLR4 genes. Apoptosis and genotoxicity induced by CCl4 were significantly alleviated by MOLE. MOLE co-administration modulated TLR4/NF-kB pathway as presented by the suppressed gene expression of TLR4 and NF- $\kappa$ B as well as by the reduced protein expression of TLR4 and NF-kB-p65. In conclusion, MOLE has a multifarious protective role against hepatotoxicity through control of oxidative stress and modulation of TLR4/NF-κB.

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