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Determination of malondialdehyde, raftlin, and sphingosine 1-phosphate levels in hepatic ischemia-reperfusion injury in rats

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Hepatic ischemia–reperfusion (HIR) injury involves inflammation and oxidative stress resulting from paradoxical cellular stress upon reperfusion. This study investigated the protective effects of resveratrol (RSV) against HIR injury using oxidative stress, inflammation, and cellular signaling biomarkers. Twenty-eight male Wistar albino rats were randomly divided into four groups: Control, HIR (30 min ischemia + 30 min reperfusion), Vehicle (HIR + 0.9% NaCl 100 mg/kg/day), and RSV (50 mg/kg/day) administered three days prior to the procedure. Liver tissues were analyzed for malondialdehyde (MDA), raftlin, sphingosine-1-phosphate (S1P) levels, and histopathological assessments. Statistical analyses employed Kruskal-Wallis and Mann-Whitney U tests ($P<0.05$). MDA and raftlin levels were highest, and S1P levels lowest, in the HIR group ($P<0.001$). RSV significantly attenuated HIR-induced biochemical changes compared to HIR and vehicle ($P<0.05$). Histopathologically, RSV reduced vascular congestion and inflammation while preserving cellular architecture. This study is the first to demonstrate RSV's protective potential against HIR injury via modulation of oxidative stress, inflammation, and cellular signaling. Additionally, raftlin was identified as a potential biomarker for inflammation in HIR, highlighting its relevance for future research. These findings provide novel insights into the mechanisms of HIR injury and suggest RSV as a candidate for further preclinical investigation.

Keywords: oxidative stress, inflammation, cellular signaling, resveratrol

Introduction