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Codium Fragile Pretreatment Ameliorates Hepatic Ischemia–Reperfusion Injury In Mice.

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Background : codium fragile (CF) is a complex of sulfated polysaccharides derived from marine green seaweeds. CF demonstrated antioxidant as well as potential anti–inflammatory properties in previous studies. Ischemia–reperfusion injury (IRI) is a major critical event that commonly occurs after liver transplantation and resection. Reactive oxygen species –mediated release of related inflammatory factors have important roles in hepatic IRI. In this study, we investigated whether CF extract protects against IRI–induced acute liver injury in mice.

Methods : Partial (70%) hepatic IRI was induced in male C57BL/6 mice by portal triad pedicle occlusion for 45 min followed by reperfusion for 6 h. CF extract (300 mg/kg body weight [BW], oral) was administered 5 days before the IRI.

Results : Treatment with CF extract significantly decreased serum alanine aminotransferase (sALT), serum aspartate aminotransferase (sAST) and serum lactate dehydrogenase (LDH) as well as liver histological changes. CF extract also prevented hepatic glutathione (GSH) depletion, increased malondialdehyde (MDA) and HO–1 levels induced by IRI. Western blotting indicated that the expression of the ERK, C–JUN, and iNOS were significantly decreased in the CF extract treatment group after IRI. RT–PCR indicated that CF extract significantly attenuated the Toll–like receptor 2/4 (TLR2/4) protein levels after IRI. The expression of tumor necrosis factor (TNF)–α, and interleukin (IL)–1β was significantly decreased in the CF extract treatment group.

Conclusions : CF improved the acute hepatic IRI by reducing oxidative damage, and inflammation. These findings suggest that CF is a promising agent against acute IR–induced hepatic damage

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