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Protective Activities of *Cistanoside* A on CCl_4 Induced Hepatotoxicity in Mice

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***SUMMARY*. To evaluate the protective efficacy of Cistanoside A (C.A), a phenylethanol glycoside isolated from *Cistanche deserticola*, on CCl_4 induced hepatotoxicity in mice, 50 animals were divided into five different protocols, and hepatic functional index were detected by diagnostic kits. Histological changes were compared by H&E stain. Activities of mitochondrial antioxidant enzymes (GST, SOD, and CAT) and respiratory marker enzymes (MDH, SDH, NADH dehydrogenase, and cytochrome c oxidases) were measured. To confirm the effect of C.A on free radical, tests on the free radical scavenging activities were also carried out *in vitro*. We found treatment with C.A (10, 20 mg/kg o.p. for 7 days) could significantly ameliorated the levels of hepatic function indices (AST, ALT, ALP and LDH) ($P < 0.05$). The biochemical results were also confirmed by histopathological examination. C.A treatment decreased the ballooning degeneration, moderated the hepatocytes apoptosis, and alleviated centrilobular and bridging necrosis which were observed in the CCl4 control group. Following experiments revealed that C.A could increase the activities of mitochondrial antioxidant enzymes (GST, SOD, and CAT) and respiratory marker enzymes (MDH, SDH, NADH dehydrogenase, and cytochrome c oxidases) ($P < 0.05$). *In vitro*, C.A exhibited strong scavenging activities for DPPH radical and superoxide anion radical. Our results revealed that C.A possess protective activities on CCl_4 induced hepatotoxicity in mice, which was involved with increasing free radicals clearing activities, alleviating lipid-overoxidation damage, and improving respiratory chain function in mitochondria.**

KEY WORDS: Cistanoside A, CCl_4, Free radical, Hepatoprotection, Respiratory chain.

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