枸杞地黃熱水萃取物對於四氯化碳誘發老鼠肝傷害之影響

Effect of hot water extracted Lycium barbarum and Rehmannia glutinosa on carbon tetrachloride-induced liver injury in rats

中文摘要

本研究探討以熱水粗萃取枸杞與地黃中多醣之成分，餵予雄性 Sprague-Dawley 老鼠，並以四氯化碳(CCl4)誘發肝傷害，探討對肝臟病理切片、血漿中肝功能指標、脂質代謝與抗氧化能力、發炎反應及纖維化的影響。將老鼠隨機分成四組 (每組 10 隻) ：正常飲食＋腹腔注射橄欖油、正常飲食＋注射 CCl4、CCl4＋一倍 (1×)劑量組 (1×HE，分別添加枸杞地黃熱水萃取物各 0.05% (w/w) 於飼料中) 、CCl4＋三倍 (3×) 劑量組 (3×HE，分別添加枸杞地黃熱水萃取物各 0.15% (w/w) 於飼料中)。一倍劑量與三倍劑量組於誘發肝傷害前一週即開始給予枸杞地黃熱水萃取物，之後每週注射一次 CCl4，實驗爲期八週。結果顯示給予枸杞地黃熱水萃取物一週後，可顯著降低血漿三酸甘油酯。八週期間，ー倍與三倍劑量均可顯著降低血漿 GOT、GPT 活性與三酸甘油酯含量，且三倍劑量可降低肝臟總膽固醇濃度。病理切片結果顯示一倍與三倍劑量均可抑制由 CCl4 所造成肝細胞壞死、發炎細胞聚集與纖維化之情形。在發炎反應方面一倍與三倍劑量均可抑制肝臟中 tumor necrosis factor-α (TNF-α)、interleukin-1 (IL-1β)含量，一倍劑量可提升肝臟中抗發炎細胞激素 interleukin-10 (IL-10)之含量。且一倍與三倍劑量具抑制肝臟導致纖維化的重要因子 transforming growth factor-β1 (TGF-β1)之含量的功能，且可減少肝臟中膠原蛋白之前驅物羥基脯胺酸(hydroxyproline) 含量，對於肝纖維化具有抑制的功能。但血漿總膽固醇含量、肝臟中脂肪堆積與三酸甘油酯含量、總抗氧化狀態與脂質過氧化產物無差異。因此，給予枸杞地黃熱水萃取物可降低四氯化碳誘發肝傷害老鼠血漿 GOT、GPT 活性，並藉由降低發炎反應，達到抑制肝臟損傷及纖維化之功能。

英文摘要

This study investigated the effects of hot water extracted Lycium barbarum and Rehmannia glutinosa (HE) on hepato- pathological examination, liver functions, lipid metabolism, antioxidative function, as well as inflammation and fibrosis in male Sprague Dawley rats with carbon tetrachloride (CCl4)-induced liver injury. Rats (n = 10 per group) were randomly divided into: normal diet + peritoneal injection of olive oil (normal), normal diet+CCl4 injection (CCl4), 1 HE (0.05% HE for each)+CCl4 (1? HE), and 3 HE (0.15% HE for each)+CCl4 (3? HE) groups. Hot water extracted Lycium barbarum and Rehmannia glutinosa were given in the 1 HE and 3 HE groups a week prior to the induction of liver injury. Rats were injected with CCl4 once a week for 7 weeks. The results showed that plasma triglycerides

decreased significantly after rats were given HE for one week. Both 1 and 3 HE treatments for 8 weeks decreased not only plasma GOT and GPT activities, but also triglyceride contents. The 3 HE treatment reduced liver cholesterol contents. The pathological examination showed both 1 and 3 HE diminished necrotic hepatocytes, chemoattraction of inflammation cells, and fibrosis. Both 1 and 3 HE treatments reduced tumor necrosis factor-α (TNF-α) and interleukin-1 (IL-1 β) . The 1? HE treatment increased interleukin-10 (IL-10) contents. Both 1? and 3? HE treatments suppressed transforming growth factor-β1 (TGF-β1) concentraction. However, HE did not affect plasma cholesterol, hepatic fat accumulation, hepatic total antioxidant status, and lipid peroxides. Therefore, HE can decrease CCl_4-induced liver injury rats plasma GOT and GPT activities, and prevent liver injury and fibrosis by down-regulation of inflammation.