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Hepatoprotection of silymarin against thioacetamide-induced chronic liver fibrosis.

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Abstract

BACKGROUND: Liver fibrosis is chronic liver damage usually caused by alcohol, viruses or other toxins and is characterised by an excessive accumulation of extracellular matrix proteins such as collagen. The aim of this study was to establish an animal model of chronic liver damage and investigate molecular mechanisms of silymarin hepatoprotective effects.

RESULTS: Thioacetamide (TAA; 100 mg kg(-1) intraperitoneal (i.p.) injection three times weekly) effectively induced chronic liver fibrosis in male ICR mice. Then 24 ICR mice were randomly divided into four groups: (1) saline (i.p.) + water (gavage); (2) saline (i.p.) + 150 mg kg(-1) silymarin (gavage); (3) 100 mg kg(-1) TAA (i.p.) + water (gavage); (4) 100 mg kg(-1) TAA (i.p.) + 150 mg kg(-1) silymarin (gavage). Eight weeks of TAA treatment resulted in lower body weight, serum cholesterol and triglycerides as well as increased liver size, ALT, AST and LDH values ($P < 0.05$). These TAA-induced effects were attenuated by silymarin ($P < 0.05$); therefore silymarin also ameliorated TAA-induced liver lesions. Effects of silymarin on TAA-induced chronic liver damage may be attributed to down-regulation of hepatic MMP-2, MMP-13, TIMP-1, TIMP-2, AP-1, KLF6, TGF-β1, α-SMA and COL-α1.

CONCLUSION: A mouse model of chronic liver fibrosis was successfully established by injecting 100 mg kg(-1) TAA three times weekly in male ICR mice. Meanwhile, silymarin showed hepatoprotection against TAA-induced damage.

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