

Hepatoprotective effects of Silymarin on TNF- α /D-GaIN induced Liver Damage

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Silymarin is a polyphenolic compound derived from *Silybum marianum* [L.] Gaertner, and is also known to have pharmacological activities as diverse as being a hepatoprotective, antioxidant, anti-inflammatory, anticancer, and cardioprotective substance [1]. This flavonoid has been shown to exert a protective effect upon the liver, an organ particularly vulnerable to poisoning by several hepatotoxic substances, including carbon tetrachloride (CCl₄), and thioacetamide [2]. The present study seeks to examine the acute hepatoprotective effects of silymarin on TNF- α /D-GaIN-induced liver damage in Balbc mice. Each of the 3 experimental groups was composed of 7 mice. The 0,5 ml saline-treated control group and the group receiving 15 μ g/kg TNF- α /700 mg/kg D-GaIN were sacrificed only 4 hours later. As to the final group, which had already been given 15 μ g/kg TNF- α /700 mg/kg D-GaIN in the beginning, were injected silmarin of 100 mg/kg at the 4th hour of the experiment. The mice in this final group were sacrificed at the 15th hour in order that their livers could be analysed for histopathological changes. It was concluded that the normal liver structure showed similarity in Groups 1 and 3. When compared with Group 1, such destructive changes as mononuclear cell infiltration, rupturing of the endothelium of some central veins, apoptotic hepatocytes, necrotic areas, sinusoidal dilatation, sinusoidal congestion in sinusoids around some central veins and necrotic areas were observed in the liver of the mice in Group 2. However, a comparison of Groups 2 and 3 showed that there were degenerative changes in the liver of the D-GaIN/TNF-a mice (Group 2), while those in Group 3, which were additionally treated with silymarin, showed either none or minimal liver damage. In conclusion, our microscopic evaluations indicate that Silymarin could play a protective role in liver injury due to D-GaIN/TNF-a.

Reference

- [1] Valenzuela A, Garrido A. Biochemical bases of the pharmacological action of the flavonoid silymarin and of its structural isomer silibinin. *Biol Res* 1994; 27: 105-112
- [2] Mourelle M, Muriel P, Favari L, Franco T. Prevention of CCl₄-induced liver cirrhosis by silymarin. *Fundam Clin Pharmacol* 1989; 3: 183-191.

Figures

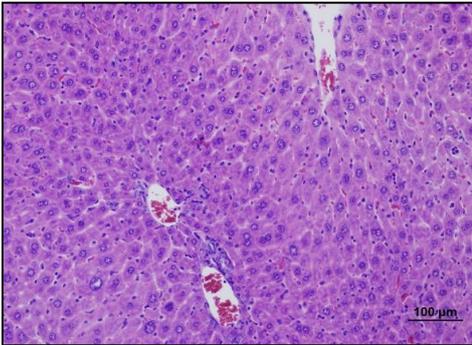


Figure 1. Hematoxylin-eosin stained normal liver tissue of Balbc mouse (scale bar: 100μm)

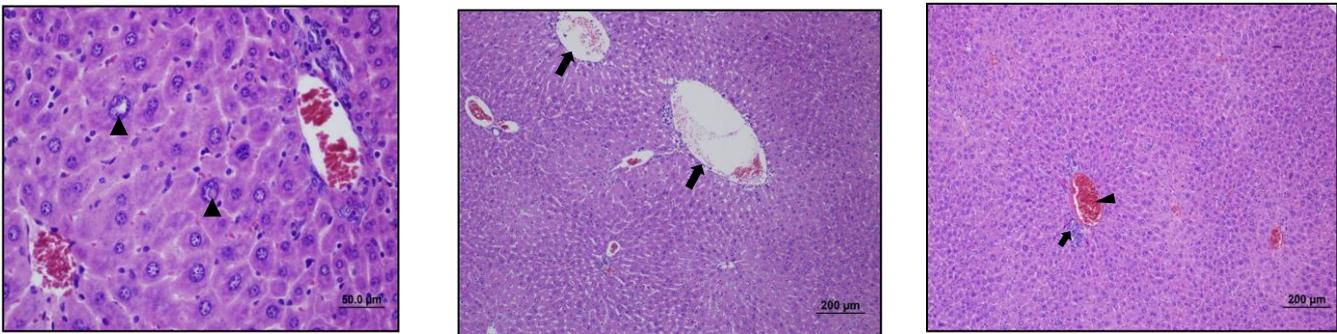


Figure 2. Hematoxylin-eosin stained liver tissue of TNF- α /D-GaIN administered mice for 4 hours. A; Degeneration in some hepatocyte's nuclei and atypical nucleus (►) (scale bar: 50μm), B; Dilatation in the veins (→) (scale bar: 200μm), C; Congestion in the veins ► and partial inflammation (→) (scale bar: 200μm).



Figure 3. Hematoxylin-eosin stained liver tissue of silibinin administered (for 11 hours) mouse after TNF- α /D-GaIN application for 4 hours (scale bar: 100μm).