THE ROLE OF SILYMARIN ON SHORT-TERM ALCOHOL-INDUCED PLASMA AND HEPATIC LIPID PEROXIDATION

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BACKGROUND/AIMS: We studied the role of silymarin on 12-week alcohol- and CCl₄-induced liver injury.

METHODS: Fifty Wistar rats were assigned as control group, ethanol group (E; Liber-Decarli liquid diet), ethanol and silymarin group (ES; silymarin 200 mg/kg/day), CCl₄ group (CCl; 0.75 mL/kg/wk of 40% CCl₄ intraperitoneal injection) and CCl₄ and silymarin group (SCCl).

RESULTS: At 12 weeks, E (34 ± 3 iu/L), ES (44 ± 6 iu/L), CCL (1108 ± 302 iu/L) and CCLS (572 ± 135 iu/L) had higher ALT levels than the control group (23 ± 1 iu/L, p<0.05). E (14.4 ± 0.8, 57.5 ± 1.9), ES (15.1 ± 1.1, 64.4 ± 2.3), CCL (13.0 ± 1.2, 38.2 ± 2.6) and CCLS (14.1 ± 1.5, 45.0 ± 2.5) had lower erythrocyte and hepatic GSH/GSSG ratios than the control group (17.2 ± 1.1, 87.7 ± 2.1, p<0.05). E (64.2 ± 2.5 μg/g), ES (67.6 ± 3.6 μg/g), CCL (48.1 ± 0.5 μg/g) and CCLS (52.2 ± 1.2 μg/g) had lower hepatic vitamin E levels than the control group (93.4 ± 3.6 μg/g, p<0.05). E (11.7 ± 0.3 μM, 4.1 ± 0.2 μM), ES (10.3 ± 0.2 μM, 4.1 ± 0.3 μM), CCL (13.1 ± 0.4 μM, 5.3 ± 0.8 μM) and CCLS (12.4 ± 0.4 μM, 5.2 ± 0.4 μM) had higher plasma and hepatic thiobarbituric acid reactive substances (TBARS) than the control group (8.2 ± 0.4 μM, 2.8 ± 0.2 μM, p<0.05).

CONCLUSIONS: Our data suggest that silymarin may improve hepatic GSH/GSSG ratio but not the plasma ALT levels and lipid peroxidation.

Keyword: alcohol, hepatitis, hepato-protection

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