Weiwei Cui et al., J Nutr Food Sci 2017, 7:1 (Suppl)
http://dx.doi.org/10.4172/2155-9600.C1.038

Research on the protective effect of gallic acid-phospholipid complex on chronic alcoholic liver injury model in mice

Weiwei Cui, Xiangqun Wu and **Ya Liu**
Jilin University, China

Background: With the increasing consumption of alcohol, the Alcoholic Liver Disease (ALD) caused by alcohol abuse has become a serious public health problem that seriously endangers human health. Gallic Acid (GA) is well known for its antioxidant and hepatoprotective activity. Its phospholipid complex (GA-P) is more bioavailable as compared to GA owing to increase the lipophilicity. GA-P could enhance the capacity to cross the lipid rich membranes of the small intestine enterocytes and finally overcoming the disadvantage of poor absorption. In this study, GA-P was prepared and the hepatoprotective effect of it was investigated on chronic alcoholic liver injury model in mice.

Methods: Sixty healthy male C57BL/6J mice were purchased from Changsheng Experimental Animal Company (Liaoning, China). After a period of two weeks, the animals were divided into 6 groups of 10 mice each. Mice in Group I served as the normal control and were given physiological saline only; Animals in Groups II –VI were given orally alcohol infusions for 4 weeks to induce liver damage. Group II served as the bifendate control, and the animals were orally administered bifendate. Group III served as the alcohol-induced liver injury model and treated with alcohol only. In addition to alcohol, mice in groups IV were also intragastric administrated GAP daily. At the end of the 4th week, blood was taken from heart and samples were collected into EP tube.

Results: (1) The repeated oral administration caused total mortality in 50/60 mice, among which 2/10 mice in Group I, 1/10 mice in Group II, 3/10 mice in Group III, and 2/10 mice in Group IV respectively; (2) The mice body weight decreased in almost all the alcohol treated animals compared with Group I; (3) A remarkable promotion of the content of the four substances (the level of TG, GHO, ALT and AST in serum) was observed in the alcohol-induced liver injury model group when compared with the normal control. Conversely, animals treated with bifendate had a significant reduction of their compounds compared with those in the hepatic injury model group. Groups treated with GAP also showed a decrease in the amount of these four substances; (4) Compared with normal control, mice in Group III which had hepatic injury induced by alcohol caused significant decrease in liver Superoxide dismutase (SOD) and a significant increase in liver malondialdehyde (MDA) content.

Conclusions: These findings suggest that GA-P is an efficacious treatment for alcohol-induced liver damage in mice. Based on our data, GA-P should be regarded as a new and promising drug that may be useful for the prevention of liver injury and even liver fibrosis.

Biography

Dr. Weiwei Cui, Department of Nutrition and Food Hygiene, School of Public Health, Jilin University, China.

cuiweiwei@jlu.edu.cn

Notes: