How did glycine significantly decrease liver injury?

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The nonessential amino acid glycine has been shown to be anti-inflammatory in several animal injury models. Recent studies demonstrated that dietary glycine protected both the lung and liver against lethal doses of endotoxin in rat or other animals and improved graft survival after liver transplantation. The influence of dietary glycine on oxidant-induced or cholestatic liver injury was not known.

A research article to be published on October 21, 2008 in the *World Journal of Gastroenterology* addresses this question. The research team led by Prof. Thurman from the University of North Carolina used a dietary and cholestatic model thru BDL in rats to address this question. They could demonstrate that hepatic injury due to BDL is significantly reduced by dietary glycine in rats. Moreover, the data indicated that glycine decreases liver injury under the conditions of experimental cholestasis thru a direct effect on hepatocytes. Surprisingly, Kupffer cells did not appear to play a major role in the pathological changes caused by cholestasis.

It is best known that Kupffer cells, the resident macrophages of the liver, are involved in several disease states, such as endotoxin shock, alcoholic liver diseases, and other toxicant-induced liver injury by releasing eicosanoids, inflammatory cytokines, and free radical species. Furthermore, in previous studies of the research team, a glycine-dependent chloride channel on the cell membrane of Kupffer cells and other macrophages that influence the activation process of these cells could be detected. But in the actual used cholestatic model no significant

influence of this cell line on liver injury could be detected.

Source: World Journal of Gastroenterology