

Cathepsin B increases apoptosis in fulminant hepatic failure

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The traditional view is that hepatocyte necrosis is the main feature of fulminant hepatic failure, but increasing evidence implicates a dominant role for hepatocyte apoptosis in this pathogenesis. It is not known if cathepsin B-mediated hepatocyte apoptosis is involved in the pathogenesis of fulminant hepatic failure. To ascertain its pathogenic role in hepatic failure, the research examined the protective effect of a cathepsin B inhibitor (CA-074Me) on fulminant hepatic failure in mice.

A research article to be published on March 14, 2009 in the *World Journal of Gastroenterology* addresses this question. The research team led by Prof. Yang in the Department of [Infectious Diseases](#) of the Second Clinical Hospital of Harbin Medical University investigated cathepsin B expression changes in the liver of fulminant [hepatic failure](#). The article further indicated that LPS/D-Gal N-mediated cathepsin B expression initiates hepatocyte apoptosis in fulminant hepatic failure.

Cathepsin B, a lysosomal cysteine protease, is a candidate for an apoptotic mediator originating from acidic vesicles. CA-074me is a selective inhibitor of cathepsin B, and it is highly cell-permeant and can decrease the expression or activity of cathepsin B. The traditional view is that hepatocyte necrosis is the main feature of fulminant hepatic failure, but increasing evidence implicates a dominant role for hepatocyte apoptosis in this [pathogenesis](#). Inhibition of cathepsin B attenuates apoptosis and liver injury, supporting a link between cathepsin B and fulminant hepatic failure, and thus may provide new targets for further understanding of the pathogenesis of fulminant hepatic failure and new

therapeutic targets.

More information: Yan BZ, Wang W, Chen LY, Bi MR, Lu YJ, Li BX, Yang BS. Role of cathepsin B-mediated apoptosis in fulminant hepatic failure in mice. *World J Gastroenterol* 2009; 15(10): 1231-1236
www.wjgnet.com/1007-9327/15/1231.asp

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