Epigallocatechin-3-gallate (EGCG) reduced acetaminophen-induced liver injury in rats by increasing its urinary excretion

Chen-Hui Chang and Hsien-Tsung Yao*

Department of Nutrition, China Medical University, Taichung, Taiwan.

The aim of this study was to evaluate the role of EGCG in liver toxicity due to acetaminophen overdose. Twenty-four male rats were divided into four groups, they were: (1) vehicle; (2) acetaminophen; (3) 0.3% EGCG + acetaminophen; (4) 0.6% EGCG + acetaminophen. After four-weeks of EGCG feeding, all the rats were sacrificed 12 hours later after intraperitoneal injection of acetaminophen (1000 mg/kg B.W.). Acetaminophen and its metabolites in the liver, blood, and urine were determined by LC-MS. In addition, plasma alanine aminotransferase (ALT), aspartate aminotransferase (AST), and liver transporters were assessed. Results showed that rats pre-treated with 0.6% EGCG had significantly reduced plasma ALT and AST after acetaminophen treatment. Protein expression of organic anion transporter polypeptide 2 (OATP2), which is mediate the uptake of drugs in liver, was decreased by 0.6% EGCG treatment. Furthermore, concentrations of acetaminophen, acetaminophen-sulfate, and acetaminophen-glucuronate in liver and plasma were not affected by EGCG; however, EGCG administration significantly decreased acetaminophen-glutathione and acetaminophen-cysteine levels in liver. Total urinary excretion (12hr) of acetaminophen was significantly increased after EGCG treatment. These results indicate that EGCG may reduce acetaminophen-induced hepatotoxicity in rats by increasing urinary acetaminophen excretion and decreasing hepatic uptake.

Keywords: EGCG, acetaminophen, hepatotoxicity, rats