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Epigallocatechin-3-gallate (EGCG) reduced acetaminophen-induced liver injury in rats by inhibiting cytochrome P450.

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The aim of this study was to evaluate the role of EGCG in liver toxicity due to acetaminophen overdose. Eighteen male rats were divided into three groups, they were：(1)vehicle；(2)acetaminophen；(3)0.54% EGCG + acetaminophen. After one week 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), liver CYP3A4 and CYP2E1 activity were assessed. Results showed that rats pre-treated with 0.54% EGCG had significantly reduced plasma ALT and AST after acetaminophen treatment. EGCG intake inhibited CYP3A4 and CYP2E1 activity. Furthermore, concentrations of acetaminophen, acetaminophen-sulfate, and acetaminophen-glucuronate in liver, urine and plasma were not affected by EGCG; however, EGCG administration significantly decreased acetaminophen glutathione conjugate in plasma and liver. These results indicate that EGCG may reduce hepatotoxicity of APAP in rats by inhibiting CYP-mediated APAP metabolism in liver.