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ATCHAREEYA CHANAWIRAT: PROTECTIVE EFFECT OF
THUNBERGIA LAURIFOLIA EXTRACT ON ETHANOL-INDUCED
HEPATOTOXICITY IN MICE. THESIS ADVISORS: CHAIVAT TOSKULKAO,
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Thunbergia laurifolia Linn (Thunbergiaceae), commonly known as 'Rang Jert', is used as an antidote for several poisonous agents in Thai traditional medicine. It was reported that an aqueous extract of dried *Thunbergia laurifolia* leaves could protect rats against the toxic effects of ethanol. However, there have been no reports on the protective mechanisms of *Thunbergia laurifolia* leaves extract (TLE) on ethanol-induced hepatotoxicity. Therefore, the present study was designed to investigate the possible protective mechanisms of TLE on ethanol-induced hepatotoxicity in mice. Mice were pretreated with TLE at a dose of 200 mg/kg BW (i.p.) 1 hour prior to an intraperitoneal administration of ethanol 9 g/kg BW. Animals were sacrificed at 15 hours after ethanol administration. Liver damage was determined by quantifying plasma activities of glutamic oxaloacetic transaminase (PGOT) and glutamic pyruvic transaminase (PGPT) and also liver triglyceride content. The potential hepatoprotective effect of TLE was also confirmed by histopathological examinations. Additionally, the protective effect of TLE pretreatment on ethanol-induced hepatotoxicity was investigated at various time intervals after ethanol administration. TLE at a dose of 200 mg/kg BW presented significant hepatoprotective effects in these experimental situations. It significantly reduced the activities of PGOT (1.3 folds) and PGPT (1.9 folds), liver triglyceride (1.4 folds) and the severity of hepatic injury of mice pretreated with TLE and then treated with ethanol, compared to mice treated with ethanol alone. However, this study demonstrated that TLE significantly decreased the loss of righting reflex caused by the high doses of ethanol but did not prevent ethanol-induced lethality. The result of acute toxicity study of *Thunbergia laurifolia* roots extracts (TRE) and TLE demonstrated that TRE was more toxic than TLE at the same dose. The following studies were to investigate the possible mechanisms of TLE on the protective effect of ethanol-induced hepatotoxicity by using hepatic lipid peroxidation, blood ethanol concentration as well as hepatic alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH) activity as the indicators. All indicators of hepatic response due to an acute ethanol intoxication such as an increase in hepatic lipid peroxidation, blood ethanol concentration and a diminution of ADH and ALDH activities, were reverted by TLE treatment. These results suggested that TLE might be useful for counteracting the effects of alcohol and might be effective for treating hepatic injury.