Potential Effect of Ethylacetate Fraction from Loquat (Eriobotrya japonica) Leaves on Hepatoprotection against Ethanol-induced Toxicity in HepG2 Cells Transfected with CYP2E1

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Chronic alcohol consumption has been shown to stimulate microsomal ROS generation as a result of the induction of cytochrome P-450 2E1 (CYP2E1) and uncoupling between CYP2E1 and NADPH cytochrome c reductase. The elevation of ethanol-induced CYP2E1 activity is suggested to be a major contributor in generating a state of oxidative stress, which results in hepatotoxicity. In the present study, the protective effect of ethylacetate fraction from ethanolic extract of loquat leaves against alcoholic liver damage was investigated in HepG2 cells transfected with human CYP2E1 (HepG2/2E1). The ethanolic extract (100 g) of loquat leaves was suspended in distilled water, and partitioned in the order of hexane, chloroform, and ethylacetate with an equal volume of each, thereby yielding ethylacetate-solubles (EJEE, 4.5 g). Treatment of 200 mM ethanol to HepG2/2E1 cells resulted in approximately 50% of cytotoxicity, primarily due to the increased ROS production. Compared to cells treated with 200 mM ethanol alone, a concentration-dependent increase in cell viability was observed in the cells pretreated with 20 and 40 ug/mL of EJEE (65.6% and 75.9%, respectively). Also, pretreatment with EJEE lead to a decrease in intracellular reactive oxygen species formation and an increase in hepatic antioxidant activity. When compared to the ethanol-alone treated cells, addition of EJEE (20 and 40 ug/mL) strengthened the activities of superoxide dismutase, catalase, glutathione-S-transferase, glutathione peroxidase, and glutathione reductase in a concentration-dependent manner. Furthermore, the amelioration of malondialdehyde levels indicated EJEE’s protective effects against liver damage mediated by alcohol. These results suggest that EJEE attenuates oxidative stress by improving antioxidative potentials, which contribute to this herb’s protective profile against ethanol-induced toxicity in vitro.