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Effects of *Salvia officinalis* Extract on Carbon Tetrachloride Induced Hepatotoxicity

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Abstract: The objective of this investigation is to evaluate the protective effects of *Salvia officinalis* extract against carbon tetrachloride-induced hepatotoxicity in rats through biochemical assays. Thirty male Wistar rats were randomly divided into five groups. Dried *Salvia officinalis* was slowly boiled in 100 ml of distilled water and heated for 30 minutes. The extracts were then filtered and administered orally to the animals. Group A served as normal control. Group B was a negative control and rats were treated intraperitoneally with a single dose of CCl4. The rats in Groups C, D and E were pretreated with *Salvia officinalis* at dose of 10, 15 and 20 mg/ml orally once daily for 14 days and then they were treated intraperitoneally with CCl4 as group B. On the 15th day, the rats were anesthetized using thiopental and blood was collected from abdominal artery. Biochemical factors, AST, ALT, GSH, SOD, CAT and MDA, were used as the biochemical markers of the hepatic damage. After treatment with CCl4, levels of serum ALT and AST was significantly ($P<0.05$) increased compared to the control. Pre-treatment with extract of three *Salvia officinalis* doses reduced the CCl4-induced elevation of serum ALT and AST activities. Hepatic MDA shows significant increase ($P<0.01$), post CCL4 administration. However pre-treatment with any of the three doses of OS reduced the elevation in hepatic MDA levels associated with AZP treatment alone. In CCL4-treated rats a significant decrease in hepatic SOD and CAT activity ($P < 0.01$) was observed post-treatment. This inhibition was significantly released with pre-treatment with SO. *Saliva officinalis* extract has been found to possess significant reactive oxygen species (ROS) scavenging activity and it seems that neutralizing such radicals could have a hepatoprotective effect.

Key words: *Salvia Officinalis* % Hepatotoxicity % Carbon Tetrachloride % Biochemical Factors

INTRODUCTION

Formation of reactive oxygen species (ROS) is an unavoidable consequence in aerobic organisms during respiration. It has been shown that overproduction of unstable ROS leads to unwanted reactions with other groups or substances in the body, resulting in cell or tissue injury. In addition, numerous studies have revealed that uncontrolled lipid peroxidation is involved in the occurrence of many diseases, including Parkinson's, arthritis, myocardial infarction, Alzheimer's, cancer, cardiovascular disease and liver damage [1]. Therefore, during the last few decades, human nutrition and biochemistry research focused on antioxidants derived from foods that could prevent or diminish ROS-induced damage. Liver damage is a widespread disease which can be caused by reactive oxygen species (ROS) and is characterized by a progression from steatosis to chronic hepatitis, cirrhosis and hepatocellular carcinoma [2]. Several compounds, such as carbon tetrachloride (CC14), acetaminophen, bromobenzene, ethanol and polycyclic aromatic hydrocarbons have been implicated in the etiology of liver diseases [3]. CCl4 is a classical hepatotoxin that causes rapid liver damage progressing from steatosis to centrilobular necrosis [4]. The mechanism of liver injury induced by CCl4 is thought to involve free radicals and lipid peroxidation. CCl4 requires bioactivation by phase I cytochrome P450 system in the liver to form reactive metabolic trichloromethyl radical (CCl3.) and proxy trichloromethyl radical (OOCCl3) [5]. Since free radicals are very unstable, they are immediately neutralized by antioxidants in the cell once they are generated in normal metabolism pathway, so increasing the antioxidant content in cells may play an important role against CCl4-induced liver injury. Due to the risks of synthetic antioxidants, there is a growing interest in the

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