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Hepatoprotective effect of food preservatives (butylated hydroxyanisole, butylated hydroxytoluene) on carbon tetrachloride-induced hepatotoxicity in rat

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ABSTRACT

Carbon tetrachloride (CCl_4), a hepatotoxic agent is widely used to study the toxic mechanisms in experimental animals. This study was carried out to establish the hepatoprotective measures of food preservative antioxidants butylated hydroxyanisole and butylated hydroxytolune (BHA, BHT) when mixed with food towards carbon tetrachloride (CCl_4) intoxication (230 mg/ kg b wt/rat/day) in rat. Biochemical markers like serum glutamate pyruvate tranaminase (AST), serum glutamate oxaloacetate transaminase (ALT), alkaline phosphatase (ALP) and bilirubin content, antioxidant enzymes such as SOD, CAT, GPx, and malondialdehyde (MDA) as the end product of lipid peroxidanion were measured. The results had shown the elevated level of AST (121.16%), ALT (124.68%), ALP (122.41%) an, bilirubin content (57.14%) after CCl_4 treatment. Marked decrease of activity of antioxidant enzymes such as SOD (85.36%), CAT (67.47%), GPx (50.7%) had indicated that the ROS mediated toxicity and pretreatment of BHA and BHT restored the activity of these enzymes. High level of MDA content with reduced GSH value was also observed due to oxidative stress. The hepatic antioxidant status was restored with the food preservative (BHA, BHT) antioxidant treatment which had indicated the significant protective effect against CCl_4 induced hepatotoxicity and finally confirmed by histopathological studies.

1. Introduction

The liver is a vital organ, located in the upper right quadrant of the abdomen below the diaphragm and has a wide range of functions related to metabolism of carbohydrate, protein, lipid and xenobiotics which include gluconeogenesis, glycogenolysis, urea biosynthesis, production of plasma proteins and blood clotting factors, cholesterol biosynthesis, production of triglycerides and bile in addition to detoxification of various metabolites. Reactive oxygen species (ROS) resulting from oxidative stress (OS) are mainly by product of normal cellular metabolism. Altered cellular activities of electron transport system (ETS), cyclooxygenase, oxidases, peroxidases are main factors for production of increased amount of ROS due to an increased OS [1]. Liver is frequently exposed to a variety of xenobiotics, pesticides, organic solvents, anasthetics, and drugs. Carbon tetrachloride (CCl_4) is a xenobiotic compound which produces hepatotoxicity in human beings and animals [2]. It appears in the environment specially in the water of industrial wastes from the manufacturing sector of chlorofluorocarbons, dry cleaning fluids, fire extinguishing agents, etc [3]. Cytochrome p450 enzymes (mostly CYP2E1) of endoplasmic reticulum start its metabolism within the body and generate highly reactive trichloromethyl radical ($CCl_3\cdot$) which rapidly reacts with oxygen to form the highly reactive trichloromethylperoxyl radical ($CCl_3OO\cdot$) [3]. The later molecule rapidly reacts with lipids (particularly PUFA) to form lipid peroxidation products. The free radical mediated lipid peroxidation is one of the main mechanisms of hepatic injury by CCl_4 [4,5]. Butylated hydroxyanisole (BHA) and butylated hydroxyl toluene (BHT) are GARS grade phenolic food preservative; are the structural analog of Vitamin E and most widely applied as synthetic antioxidants. They are mostly used in processed foods like, butter, meat, cereals, chewing gum, baked goods, snack foods, beer etc [6]. BHA, BHT have potential role to inhibit lipid peroxidation (LPO) and OS in many experimental models by restoring the cellular antioxidant enzyme status. Thus the present study was designed to investigate the hepato-protective effect of BHA and BHT on CCl_4 induced oxidative stress.

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