PROTECTIVE EFFECTS OF 4-4’-DICHLORO-DIPHENYL DISELENIIDE IN A HYPERLIPIDEMIC MODEL IN RATS

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Introduction: Hyperlipidemia may cause fatty infiltration of the liver and contributes to the manifestation and development of atherosclerosis and coronary heart diseases. Triton WR-1339, a non ionic detergent, has been used widely to produce acute hyperlipidemia in animals. The purpose of this study was to investigate the possible hepatoprotective and antihyperlipidemic effects of p-chloro-diphenyl diselenide (p-ClPhSe)2 in the hyperlipidemic model induced by triton-WR 1339 in rats.

Material and Methods: Simvastatin (20 mg/kg) or (p-ClPhSe)2 (10 mg/kg) were administered to male Wistar rats by intragastric (i.g.) route during seven days. Thirty minutes after the last treatment, all animals were given a saline (vehicle) or triton (400 mg/kg) injection. Animals were fasted for 18h. Subsequently, blood samples were collected and the rats were killed by decapitation to extract the livers. Plasma biochemical parameters and hepatic oxidative stress parameters were determined. The experiments were approved by Committee on Care and Use of Experimental Animal Resources of the UFSM, Brazil (509850115).

Results and Discussion: The levels of total cholesterol (TC), triglycerides (TG), non-HDL-cholesterol (non-HDL), and coronary risk index (CRI) were significantly increased in rats treated with triton. The administration of (p-ClPhSe)2 resulted in a significant decrease in plasma lipid levels. Different from simvastatin, (p-ClPhSe)2 was effective to reduce TG levels and to increase HDL levels altered in rats treated with triton. Triton induced an increase in alanine (ALT) and aspartate aminotransferase (AST) activities and (p-ClPhSe)2 was effective in normalizing these enzyme activities. Triton increased the levels of reactive species in the rat liver but (p-ClPhSe)2 was not effective in counteracting this increase.

Conclusions: Our findings suggest that (p-ClPhSe)2 can help developing new therapeutic approaches to prevent and treat liver damage caused by fat excess or toxic agents and hyperlipidemia.

Keywords: Triton WR-1339, organoselenium, hepatotoxicity.

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