Research Article

Hepatoprotective Role of Gallic Acid on Sodium Fluoride-Induced Liver Injury in Rats

Asma Bouasla[1], Ihcène Bouasla[1], Amel Boumendjel[1], Abdelfattah El Feki[2], Mahfoud Messarah[1]*

[1]Laboratory of Biochemistry and Environmental Toxicology, Faculty of Sciences, University of Badji Mokhtar, BP 12 Sidi Amar, Annaba, Algeria.

[2]Laboratory of Animal Ecophysiology, Faculty of Sciences, Sfax, Soukra road – Km 3.5, BP 802, 3018 Sfax, Tunisia.

***Corresponding author's E-mail:** mahfoud.messarah@univ-annaba.dz

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ABSTRACT

This study was undertaken to evaluate the protective effect of Gallic acid (GA) on oxidative damages in liver of rats intoxicated by sodium fluoride. Rats were randomly divided into three groups of six animals each: group (C): served as a control; group (NaF): treated for three weeks with sodium fluoride (100 ppm in drinking water) and group (NaF+GA) : treated daily and for three weeks also by both of NaF and GA (20mg/Kg orally). It was found that NaF induced liver damages as evidenced by the elevation of plasma amino transferases (ALT, AST), alkaline phosphatase (ALP), lactate dehydrogenase (LDH) activities associated with a decrease in total protein, albumin and bilirubin levels. Hepatotoxicity was objectified by the significant increase of malondialdehyde (MDA) level and a decrease of antioxidant enzyme activities such as catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx) and reduced glutathione (GSH) in liver of NaF treated rats. However, the co-administration of gallic acid caused an amelioration of the previous parameters. This study clearly showed that gallic acid has protective role against the oxidative damage in sodium fluoride intoxicated rats.

Keywords: Gallic acid, Hepatotoxicity, Oxidative stress, Rat, Sodium fluoride.

INTRODUCTION

Sodium Fluoride (NaF) is used in various pesticide formulations, including insecticides and wood preservatives. It can be deposited into soil from several anthropogenic sources, both directly through phosphate fertilizers or indirectly through atmospheric pollution from industrial activities, burning of fossil fuels and from environmental pollutants such as pesticides. In the last years, interest in inorganic fluoride undesirable effects has resurfaced.[1] many studies demonstrated that inorganic fluoride even at low doses can interact with a wide range of cellular processes such as gene expression, cell cycle, proliferation and migration, metabolism, apoptosis, necrosis and oxidative stress.[2]

Numerous investigations indicate that reactive oxygen species (ROS) are implicated in the development and/or progression of cancer and possibly involved in the etiology of many other human diseases.[3] Among its adverse biochemical effects, fluoride causes increased lipid peroxidation in the blood of humans and in the blood and tissues of experimental animals. Over the last decade, numerous antioxidants plants have been discovered.[4] Several studies reveal that the protective effects of plants can be due to the presence of flavonoids, anthocyanins and phenolic compounds.[5] Gallic acid (3,4,5trihydroxybenzoic acid) is a natural product (phenolic acid) from plants which is found particularly abundant in grapes, different berries, fruits as well as tea leaves.[6] It has been reported that gallic acid have a potent free radical scavenging and antioxidant actions, that's why it received much attention in the last years.[7] The aim of our study is the investigation of hepatoprotective role and antioxidant capacity of Gallic acid on sodium fluoride-induced liver injury in rats

MATERIALS AND METHODS

Chemicals

Sodium Fluoride (NaF) and Gallic acid (GA) were purchased from Sigma Chemical Co (St Louis, USA) and all other chemicals used in the experiment were of analytical grade.

Animals and experimental design

Eighteen healthy adult male Wistar rats weighing around 238±2g were obtained from Pasteur Institute (Algiers, Algeria). Animals were acclimated under the same laboratory conditions of photoperiod (12h light:12h dark) with a minimum relative humidity of 40% and room temperature of 23±2°C. Food (standard diet, supplied by the ONAB, Algiers, Algeria) and water were supplied *ad libitum*. After two weeks, rats were randomly divided into three groups of six animals each as follow:

- Group (C): served as a control.
- Group (NaF): treated daily for three weeks with sodium fluoride at a dose of 100ppm in drinking water.
- Group (NaF+GA): was treated daily and for three weeks by both of NaF plus GA (20mg/Kg orally).

Body weight, food and water consumption were monitored during the treatment. The experimental procedures were carried out according to the National Institute of Health Guide- lines for Animal Care and approved by the Ethics Committee of our Institution.