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ORIGINAL ARTICLE

Effect of grapeseed oil on diazinon-induced physiological and histopathological alterations in rats



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Abstract The pollution of environment by toxic chemicals is a global and chronic problem. Human health risk due to exposure to chemical pollutants is constantly increasing. Pesticides form major toxic chemicals in environment. Scientifically, there is an obviously correlation between the exposure to pesticides and appearance of many diseases. Currently, the significance of natural products for health and medicine has been formidable. The present study investigated the effect of grapeseed oil in male rats exposed to diazinon. The experimental rats were divided into five groups. The rats of the first group were served as control. The experimental animals of the second group were exposed to diazinon (DZN). The animals of the third group were supplemented with grapeseed oil and treated with DZN. The rats of the fourth group were supplemented with grapeseed oil. The experimental rats of the fifth group were supplemented with corn oil. Hematobiochemical and histopathological evaluations were chosen as indicators of DZN toxicity and protective role of grapeseed oil. In rats exposed only to DZN, the levels of serum glucose, triglycerides, cholesterol, low density lipoprotein cholesterol, very low density lipoprotein cholesterol, creatinine, urea nitrogen, uric acid, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, creatine kinase and lactate dehydrogenase were statistically increased, while the level of serum total protein was significantly decreased. Moreover, the histopathological evaluations of the liver, kidney and testis showed that DZN causes several severe alterations. Pretreatment with grapeseed oil exhibited a protective role against DZN toxicity which confirmed by the inhibition of hematobiochemical and histopathological changes due to DZN exposure. Additionally, the present study suggests that the effect of grapeseed oil supplementation against DZN toxicity may be attributed to the antioxidant role of its constituents.

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1. Introduction



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The pollution of the environment plays a crucial role in the occurrence of many diseases affecting plants, animals and humans. One of the main factors causing pollution of the

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1319-562X © 2014 Production and hosting by Elsevier B.V. on behalf of King Saud University. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/3.0/). environment is the irrational use of organophosphorus pesticides (Al-Haj et al., 2005). Nowadays, contact with organophosphorus pesticides is an important health problem for agricultural workers (Hurtig et al., 2003). Although rapidly metabolized, they are highly toxic for insects and mammals. Due to easy access to organophosphorous pesticides and their higher degree of toxicity, accidental poisonings and also suicides by using them are wide. So, it is one of the toxic materials causing human poisoning and death worldwide annually (Abdollahi et al., 2004).

Diazinon (DZN), C12H21N2O3PS, is a commonly used organophosphorous insecticide. It has been used since 1956 for the control of soil insects and pests, on ornamental plants, and on fruits, vegetables and field crops. Now it is used to control flies around animal facilities, greenhouses, fairgrounds and other businesses and public places where food or animal wastes might be accumulated (Dikshith and Diwan, 2003). DZN can be highly toxic for animals and human kind (Poet et al., 2004; Sarabia et al., 2009). The main mechanism of action of DZN is acetyl-cholinesterase enzyme inhibition (Kamanyire and Karalliedde, 2004). However, it may induce imbalance in the free radicals production/elimination processes with consequent induction of cellular damage (Kamanyire and Karalliedde, 2004; Gokcimen et al., 2007; Roegge et al., 2008; Cakici and Akat, 2013). Additionally, several studies showed that DZN was capable of inducing histopathological, biochemical and physiological alterations (Al-Attar, 2009; Al-Attar and Al-Taisan, 2010; Al-Attar and Abu Zeid, 2013; Boroushaki et al., 2013; Cakici and Akat, 2013; El-Demerdash and Nasr, 2014).

In recent years, a considerable emphasis has been focused on the importance of the naturally available botanicals that can be consumed in an individual's everyday diet because of antioxidant and antiinflammatory their properties (Nandakumar et al., 2008). Nature has been a source of medicinal treatments for thousands of years and plant-derived products continue to play an essential role in the primary health care of about 80-85% of the world's population. Despite the trends of molecular biology and chemistry providing fast escalation of synthesized *de novo* drugs, plants still remain a traditional source of medicinal compounds; up to 40% of modern drugs may directly or indirectly be related to natural compounds (Solyanik et al., 2004). Grape (Vitis vinifera) is one of the world's largest fruit crops and grape seed extract is a complex matrix containing approximately 40% fiber, 16% oil, 11% proteins, and 7% complex phenols including tannins, in addition to sugars and mineral salts (Shi et al., 2003). Grapeseed oil as an extract of the grape seed has many uses ranging from cooking (as a food additive), cosmetics and in controlling several diseases and wound healing potential (Shivananda et al., 2011). Nowadays, many scientific researchers have revealed that the grapeseed oil has several health benefits and is considered as a good and potent antioxidant compound for its contents of polyphenols, flavonoids, unsaturated fatty acids and vitamin E (El-Ashmawy et al., 2007; Dos Santos Freitas et al., 2008; Hassanein and Abedel-Razek, 2009; Kikalishvili et al., 2011; Hasseeb et al., 2013). Therefore, the present study was aimed to investigate the effect of grapeseed oil supplementation on physiological and histopathological alterations induced by DZN toxicity in male rats.

2. Materials and methods

2.1. Animals

Thirty healthy male albino rats of the Wistar strain (85.4– 93.8 g) used in this study were obtained from the Experimental Animal Unit of King Fahd Medical Research Center, King Abdulaziz University, Jeddah, Saudi Arabia. The experimental animals were housed 6 per cage in a room with 65% humidity, 12:12 h light: dark cycle at ambient temperature of 20 ± 1 °C. Standard diet, commercial feed pellets and tap water were freely available. The principles of laboratory animal care were followed throughout the duration of experiment and instruction given by King Abdulaziz University ethical committee was followed regarding experimental treatments.

2.2. Experimentation

The experimental animals were randomly distributed into five groups of six each. Animals of group 1 were untreated and served as normal control. Rats of group 2 were orally administrated with 50 mg/kg body weight of DZN in corn oil, daily for 3 weeks. Animals of group 3 were orally given grapeseed oil at a dose of 2 g/kg body weight and after 4 h subjected to DZN at the same dose given to group 2, daily for 3 weeks. Rats of group 4 were treated with grapeseed oil at the same dose given to group 3, daily for 3 weeks. Experimental animals of group 5 were supplemented with corn oil at the same dose given to group 2, daily for 3 weeks. At the end of the experimental period, rats were fasted for 10 h, anesthetized using diethyl ether and blood samples were collected from orbital venous plexus in non-heparinized. For obtaining blood serum, collecting blood tubes were centrifuged at 2500 rpm for 15 min. Serum glucose, total protein, triglycerides, cholesterol, high density lipoprotein cholesterol (HDL), creatinine, blood urea nitrogen (BUN), uric acid, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), creatine kinase (CK) and lactate dehydrogenase (LDH) were measured using Dimension Vista® 1500 System, USA. The level of serum low density lipoprotein cholesterol (LDL-C) was estimated according to the equation of Friedewald et al. (1972).

LDL-C = Total cholesterol-HDL-triglycerides/5

Serum very low density lipoprotein cholesterol (VLDL-C) was evaluated using the following equation:

VLDL-C = Triglycerides/2.175

For histopathological examinations, liver, kidney and testis sections were taken from all groups. The tissues were fixed in 10% neutral formalin, dehydrated with different ethanol solutions and embedded in paraffin, then cut into 4μ thick sections, stained with hematoxylin-eosin and observed under a photomicroscope.

2.3. Statistical analysis

Statistical analysis was performed by one-way analysis of variance (ANOVA) followed by Student's *t*-test. All data are presented as mean \pm standard deviation (SD). Differences below P < 0.05 implies significance.

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