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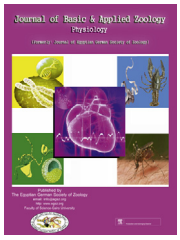

Ameliorative effect of antioxidants (vitamins C and E) against abamectin toxicity in liver, kidney and testis of male albino rats

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Abstract This study evaluated the effect of vitamins C and E as antioxidants on the physiological and histopathological changes induced by abamectin pesticide in liver, kidney and testis of male albino rats. Thirty male albino rats were divided into five groups of 6 rats each. First group served as control, while the second group received 10 mg/kg b.wt of abamectin orally, the third group received abamectin daily and 160 mg/kg b.wt of vitamin C two times per week. The fourth group received abamectin daily plus 50 mg/kg b.wt of vitamin E two times per week, while the fifth group received abamectin daily plus vitamins C and E two times per week. The experiment was conducted for six weeks. Abamectin was found to induce, hepato renal and testicular toxicity in rats, since the biochemical parameter of liver function (i.e. alanine amino transferase (ALT), aspartame amino transferase (AST), acid phosphatase (AP), glucose, total protein, albumin) and kidney function (i.e. creatinine, urea, uric acid, cholesterol and triglycerides) were highly affected. These effects were demonstrated by histopathological examination of liver, kidney and testis tissues. These observations were much reduced in the vitamin-treated groups.

In conclusion, it appears that vitamins C and E, or in combination (as antioxidants) ameliorate the hepato-renal and testicular toxicity of abamectin, but are not completely protective, especially in liver tissue.

Introduction

Abamectin (ABM) is a macrocyclic lactone product derived from the soil microorganism Streptomyces avermitilis. It is a mixture of avermectin containing about 80% avermectin B1a and 20% avermectin B1b (Burg et al., 1979; Fisher and Mrozik, 1989). These two components, B1a and B1b have similar biological and toxicological properties (Lankas and Gordon,1989). ABM is used as an insecticide and acaricide in many parts of the world.

Abamectin is nearly insoluble in water and has a strong to bind to soil particles. In the environment, ABM is quickly degraded (half life time 4–12 h.) by oxidative and

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photo-oxidative mechanisms when exposed to light in water or as thin film on biological surfaces (e.g. leaves) or when it binds to the soil particles and then exposed on glass plates (Wislockii et al., 1989). ABM is highly toxic to insects and fishes and may be highly toxic to mammals (Moline et al., 2000; Jençiç et al., 2006). Additionally, as a safe chemical in mammals, abamectin has been used as an anthelmintic agent in both animals and humans (Kaplan et al., 1994). Intoxication of abamectin may affect the function of hepatocytes although the permanent liver damage is usually not revealed immediately. In rats, abamectin led to an elevation in serum AST and nitric oxide (Hsu et al., 2001). It is known that the detoxification of the toxic materials which enter the body occurs mainly in the liver (Baisterri and Shaw, 1987). Therefore, liver can be used as an index for the toxicity of abamectin in vertebrate animals.

Previous study has shown that abamectin caused a significant increase in serum aspartate aminotransferase (AST), alanina aminotransferase (ALT), alkaline phosphatase (ALP) and acid phosphatase (AP) in rats treated with sub-acute dose (9.83 mg/kg b.wt for 21 days) and sub-chronic dose (5.93 mg/kg b.wt for 57 days). (Soliman et al., 2009). A dose of 1/10 or 1/100 of LD50 of abamectin led to an increase in the activity of AST and AP, whereas it caused a decrease in ALT activity, total protein, albumin and glucose concentration in serum of treated male rats in a dose-dependent manner, but ALP activity and cholesterol concentration remained unaltered (Eissa and Zidan, 2010).

Abamectin at a dose of 2–13 mg/animal/day for 14, 28 and 42 days was found to cause a significant increase in the glucose count and levels of AST and ALT in the liver of male and female albino rats (Khaldoun-Oularbi et al., 2013).

Histological examination of liver treated with 1/10 or 1/100 of LD50 of abamectin showed excess portal tract infiltration and a focus of dysplasia with cytological atypia in male albino rats (Eissa and Zidan, 2010). It has been postulated that treatment of male and female rats with abamectin caused vascular degeneration, hemorrhage, cellular infiltration, sinusoidal dilatation and foamy cytoplasm in the hepatocytes in the male albino rats (Khaldoun-Oularbi et al., 2013).

Abamectin may affect the kidney function parameters. It has been reported that administration of 1/4 LD50 of abamectin orally in male albino rats led to a significant increase in plasma levels of urea, uric acid, and creatinine, while that of glutathione-s-transferase(GST) and catalase enzymes were significantly decreased (El-shafey et al., 2011). Also it has been postulated that the administration of dietary dose of abamectin equivalent to 1/10 or 1/100 of the LD50 values for 30 days caused a significant increase in uric acid and creatinine concentrations of serum in the male albino rats (Eissa and Zidan, 2010). Administration of 30 mg/kg b.wt for 30 days three times per week or 10 mg/kg b.wt for 210 days once a week orally of abamectin in male albino rats resulted in a significant increase in the levels of plasma urea and creatinine but, a significant decrease in the levels of plasma albumin, total proteins and RNA was observed (Abd-Elhady and Abou-Elghar, 2013).

Recent studies revealed that administration of 30 mg/kg b.wt of abamectin orally for 30 days caused a decrease in the level of protein content, the activities of antioxidant enzymes and alkaline phosphatase (ALP) in male Wistar rats (Nasr et al. (2016).

Histopathological examination of the kidney tissue from male albino rats exposed to 1/10 or 1/100 of LD50 of abamectin for 30 days showed interstitial nephritis (Eissa and Zidan, 2010). The kidney tissue from male albino rats which was exposed to 1/10 or 1/30 of LD50 of abamectin orally showed marked necrosis of tubular cells, atrophy of the glomeruli and areas of interstitial infiltration of round cells (Abd-Elhady and Abou-Elghar, 2013).

Abamectin caused a significant increase in the level of plasma testosterone, but sperm count and sperm motility were significantly decreased in males albino rats (Eissa et al., 2003). It has been reported that subacute and sub chronic exposure of abamectin for 30 and 210 days respectively, resulted in a significant reduction in male albino rats that the number of springs was significantly reduced (Abd-Elhady and Abou-elghar, 2013).

Histopathological evaluation of the testes of albino rats which were ingesting abamectin at a dose of 1.19, 1.87 and 2.13 mg/animal/day for six weeks revealed several abnormalities including infiltration with congested blood vessels with marked hemorrhage and a significant accumulation of connective tissue surrounding the seminiferous tubules (Elbetieha and Da'as, 2003). The administration of low dose (1 mg/kg/day) for 1 week and high dose (4 mg/kg/day) for 6 weeks orally in male albino rats, resulted in disruption of spermatogenesis in a way that a tubule arrested in round spermatid stage consisting of immature germ cells with halo appearance in their nuclei, tubule with disrupted spermatogenesis comprising abnormal gametes and consisting of multinuclear giant cell (Celik-Ozenci et al., 2011). Oral administration of abamectin at a dose of 30 mg/kg b.wt three times a week for 30 days and 10 mg/kg b.wt for 210 days, once a week showed degeneration of spermatogonia cells lining seminiferous tubules and lumen contains fewer spermatozoa, necrosis of spermatogonia cells lining seminiferous tubules associated with peritubular edema and the lumen contains a decreased number of spermatogenesis elements (Abd-Elhady and Abou-elghar, 2013).

The body has several mechanisms to counteract the damage caused by free radicals. The basic and the most important defense mechanism of the body are antioxidant agents (Abdollahi et al., 2004). The term antioxidant is any substance that delays, prevents or removes oxidative damage to a target molecule (Halliwell, 2007). Pesticides have been extensively studied for their toxic potentials. Pesticide induced oxidative stress has been the focus of toxicological research for over decade as a possible mechanism of toxicity. Studies have established oxidative stress in humans and animals result from various agents in the group and are associated with their toxic manifestation (Uchendu et al., 2012).

It has been established that organophosphate pesticides which have been widely used in agriculture to enhance food production a public health to control nuisance bests may cause oxidative stress through excessive production of reactive oxygen species resulting in an imbalance between the production of free radicals and cellular antioxidants (Milatovic et al., 2006). So, it should be noted that the information about the toxic effects of abamectin induced oxidative stress in the literatures are very rare. It has been stated that antioxidant may ameliorate, protect and remove the oxidative damage to a target organ or molecule (El-Shenawy and Al-Ghamdi, 2014).

The major natural antioxidant which are derived from the natural sources by dietary intake are vitamins A, C, E and carotenoids (Heistad,2006). Accordingly, interest has recently grown in the role of the natural antioxidant as a strategy to