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Effect of α tocopherol and selenium on antioxidant status, lipid peroxidation and hepatopathy induced by malathion in chicks

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Abstract

The objective of the present study was to investigate the role of α tocopherol and selenium on malathion induced hepatic damage, and antioxidant defense in chicks. The chicks were divided into three groups. First group received malathion 10 mg/kg BW, orally for 60 days, the second group received the same dose of malathion but was supplemented with α tocopherol and selenium for 60 days and the third group served as the control. A compromised antioxidant capacity as evidenced by increased levels of erythrocytic lipid peroxidation and decreased concentration of vitamin E and decreased activity of glutathione peroxidase was observed in chicks following the administration of malathion. An improved antioxidant status was observed in chicks of second group with α tocopherol and selenium supplementation including higher concentration of vitamin E, increased activity of glutathione peroxidase and lower levels of lipid peroxidation. Histopathological studies of liver in the chicks which received malathion exhibited, moderate to severe degenerative and necrotic changes in the hepatocytes. The correlation of decreased antioxidant status of chicks with degenerative changes in liver suggests that lipid peroxidation may be one of the important mechanism in the chronic toxicity of malathion. The results indicate that α tocopherol and selenium were effective in partially alleviating degenerative changes induced by malathion in the liver of chicks by attenuating processes leading to lipid peroxidation.

Keywords: Malathion; Lipid peroxidation; Chicks; Oxidative damage; Vitamin E; Selenium; Antioxidants; Hepatopathy; α Tocopherol

1. Introduction

Malathion is an organophosphorus insecticide and has a wide range of use in agriculture, veterinary medicine and public health. However, the unregulated use and its aerial application over large agricultural and urban areas has caused severe environmental pollution and potential health hazards [1]. The exposure of low levels of malathion over an extended period of time would have a more serious impact on animal and human health. Previous studies from our laboratory focused on immunomodulatory effects of chronic doses of organophosphorus insecticides such as quinalphos and malathion [2–5]. Reported toxicity of malathion might be attributed to its metabolite malaxon, which unlike its parent compounds can damage DNA [6]. These compounds have been suggested to cause oxidative damage and cellular injury due to the involvement of free radicals [7].

Living organisms have a complex antioxidant (enzymatic and non-enzymatic) system to protect against the deleterious effects of free radicals. Vitamin E is the most effective chain breaking lipid soluble antioxidant in the biological membranes and protects cellular structures against damage from oxygen free radicals and reactive products of lipid peroxidation. Selenium is a constituent of cytosolic enzyme glutathione peroxidase and facilitates the action of vitamin E in reducing peroxy radicals [8]. In chicken, absorption of vitamin E is impaired by severe selenium

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deficiency and selenium alleviates vitamin E deficiencies by permitting higher levels of vitamin E to be absorbed [9]. Most studies concerning such supplementation have focussed on the preventive and curative properties in diseases of poultry [10–15]. In addition, studies have been reported which showed that these nutrients alleviate metal toxicity [16–20] and mycotoxin toxicity [21,22].

In the present study the ability of potent antioxidants, α tocopherol and selenium to modulate hepatic damage induced by malathion is studied. To assess effects induced by malathion, alterations in the endogenous antioxidant defenses, vitamin E and activity of glutathione peroxidase have been studied. In addition cellular accumulation of lipid peroxides or by products such as malonyl dialdehyde (MDA) was studied along with histopathological alterations in hepatic tissues.

2. Materials and methods

2.1. Experimental design

Day old broiler chicks ($n = 45$) were procured and maintained in the Department of Animal Nutrition, GADVASU. The chicks were acclimatized for 7 days before the start of the experiment. These were vaccinated against New Castle disease at 7 days of age using an F_1 vaccine and at 14 days of age against infectious bursal disease using an intermediate strain. They were supplied each day with fresh water and feed, *ad libitum*. Proper hygiene conditions were maintained in the cages where chicks were kept. At 1 week of age, the chicks were divided randomly into three groups of 15 chicks each. The chicks in group I were given 10 mg/kg body weight of malathion orally each day for 60 days. The chicks of group II received the same dose of malathion plus–α tocopherol selenium combination (α tocopherol 150 IU/kg feed and selenium 0.1 mg/kg feed) daily for a period of 60 days. The chicks in group III served as controls. At 20 day intervals, blood samples were collected from five chicks in each group by cardiac puncture for various biochemical estimations. At the end of experiment, Five chicks from each group were sacrificed and liver was collected for histopathological studies.

2.2. Histopathological examination

Representative samples from the liver tissues were collected in 10% neutral buffered formalin. After prolonged washing in running water, the pieces of tissues were dehydrated in ascending grades of alcohol and acetone and then cleared in benzene. Paraffin sections, 4.5 mm thick, were cut and stained with haematoxylin and eosin [23].

2.3. Biochemical estimation

Hemolysate was prepared for estimation of lipid peroxidation and activity of Glutathione peroxidase. Blood was collected in a heparinized graduated centrifuge tube upto the marked level. Plasma was separated and erythrocytes were washed and centrifuged thrice with normal saline solution, then distilled water was added to erythrocyte pellet slowly with constant stirring up to the marked level which was stored in aliquots at −20 °C. Haemoglobin concentration in hemolysate was estimated [24]. Estimation of product of lipid peroxidation, malonyldialdehyde (MDA), was done in hemolysate by the method of Placer et al. [25]. Glutathione peroxidase (GPX) activity was measured by the method of Hafeman et al. [26]. Vitamin E was estimated by the method of Kayden et al. [27].

2.4. Statistical analysis

Statistical analysis was done by the methods described by Snedecor and Cochran [28] using Student's '*t*' test at the 5% level of significance.

3. Results

3.1. Biochemical findings

The plasma lipid peroxidation level of control chicks (group III) did not vary significantly during 1, 20, 40 and 60 days of experiment (Table 1). The lipid peroxidation level was higher in chicks of group I which had received malathion. The supplementation of α tocopherol and selenium in chicks of group II brought down the lipid peroxidation to the level comparable to chicks of control group.

The erythrocytic GPX activity (Table 2) did not vary significantly in control chicks (group III) during 1, 20, 40 and 60 days of experiment. The mean activity of GPX decreased

Table 1
Progressive changes (means ± SD) in lipid peroxidation of erythrocytes (nmol MDA produced/g Hb) in chicks

DPA	Group I	Group II	Group III
1	62.60 ± 1.32	58.77 ± 2.69	61.90 ± 4.23
20	152.56 ± 3.64*	63.53 ± 4.51	60.13 ± 3.60
40	179.35 ± 5.12*	68.69 ± 3.68	62.34 ± 3.72
60	231.43 ± 5.70*	65.32 ± 4.62	59.29 ± 4.79

Group I: chicks administered malathion, group II: chicks administered malathion and supplemented with vitamin E and selenium, group III: control chicks, DPA: days post administration.
* Significantly different from control ($p < 0.05$).

Table 2
Progressive changes (means ± SD) in erythrocytic glutathione peroxidase activity (U//mg Hb) in chicks

DPA	Group I	Group II	Group III
1	14.91 ± 0.39	14.83 ± 0.35	14.96 ± 0.49
20	10.37 ± 0.42*	15.16 ± 0.57	14.72 ± 0.38
40	9.32 ± 0.32*	15.36 ± 0.52	14.86 ± 0.42
60	9.10 ± 0.50*	16.64 ± 0.61	14.98 ± 0.32

Group I: chicks administered malathion, group II: chicks administered malathion and supplemented with vitamin E and selenium, group III: control chicks, DPA: days post administration.
* Significantly different from control ($p < 0.05$).