MALATHION-INDUCED HYPER-ADRENAL ACTIVITY IN WLH CHICKS

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ABSTRACT

The effect of malathion (75 and 150 mg/kg, orally) given daily for 30 days on the adrenal cortical and medullary activity in white leg horn chicks has been investigated. Significant reduction in the levels of adrenal ascorbate, cholesterol and catecholamines viz. adrenaline and noradrenaline were observed after 30 days of treatment, at both the dose levels. However, the level of adrenal corticosterone increased significantly. Malathion did not produce any significant changes in the levels of any of the parameters after 15 days of treatment.

INTRODUCTION

The use of organophosphorus insecticides has greatly increased because of their low and brief persistence, high versatality and mild toxicity to man and animals. Their indiscriminate use has, however, resulted in the pollution of our ecosystem causing hazards to non-target organisms including man, animals and birds. Definite signs of toxicity such as retardation of growth, slow feather development, soft droppings, weakness of legs and paralysis due to malathion have been reported in birds¹.

Exposure to malathion has been demonstrated to induce hypercortical and hypermedullary activity in rats from this laboratory^{2,3}. Gablicks et al⁴ also reported that continuous use of pesticides even in normal recommended doses produces stimulation of adrenal cortex and medulla and interferes with immune responses. Subtoxic levels (5, 50, or 500 ppm) of technical grade DDT have been reported to inhibit adrenal corticosterone synthesis in chicken⁵. The apparent effects of acute and chronic exposure of some body systems to insecticides are on record, but little attention has been paid to examine the influence of these chemicals, on toxicity to sympathoadrenal activity which is the first line of defence in case of any stressful situation in birds. Keeping this in view, the present investigation was undertaken to study the effects of malathion—an extensively used pesticide in poultry industry, on adrenal gland activity in WLH chicks.

MATERIALS AND METHODS

The study was carried out on WLH chicks of either sex (6-10 weeks of age) procured from the Department of Animal Breeding of this University. The birds were

acclimatized in the animal house of the department for 3-4 days before starting the experiment. Feed and water were provided ad lib.

Malathion [O, O-dimethyl-S-(1, 2 dicarboethoxy ethyl) phosphorodithioate] of technical grade (97.2%) was dissolved in arachis oil and administered orally to the birds. The study was carried out with six groups of birds.

Birds of group I were given arachis oil and of group II and III were given malathion at the dose rate of 75 mg and 150 mg/kg orally, corresponding to 1/20th and 1/10th of LD-50, respectively for 30 days continuously. Birds of groups IV, V and VI were administered arachis oil and malathion (75 mg and 150 mg/kg) respectively, orally continuously for 15 days.

The birds of group I, II and III were sacrificed by decapitation on the 31st day and of group IV, V and VI on the 16th day of treatment. After opening the peritoneal cavity, adrenal glands were taken out for estimating ascorbic acid⁶, cholesterol⁷, corticosterone⁸ and catecholamines⁹. The experimental data were analyzed by applying paired *t*-test to determine the statistical significance according to Snedecor and Cochran¹⁰.

RESULTS AND DISCUSSION

Malathion at the dose rate of 75 mg or 150 mg/kg for 15 days produced no significant effect on any of the parameters studied. When this treatment was given for 30 days at the same dose level, significant decrease in the levels of adrenal ascorbic acid and cholesterol was observed (table 1). This shows that malathion treatment for 15 days was inadequate to have any effect on adrenal cortical activity. Decrease in adrenal content of ascorbic acid or cholesterol indicates that these pre-

Table 1 Effect of daily oral administration of malathion on adrenal components.

Adrenal components	Group		
	I Arachis oil (control)	II Mal	III athion
		75 mg/kg	150 mg/kg
Ascorbate	3.17 ± 0.09	2.30**	
(mg/g)	_	± 0.09	$2.06** \pm 0.07$
Cholesterol (mg/g)	54.83 ± 4.54	36.65* ± 2.23	29.09** ± 1.45
Corticosterone (µg/100 mg)	17.84 ± 1.47	25.96* ± 2.68	32.16** ± 2.49
Adrenaline	2.34 ± 0.18	1.92 ± 0.04	1.30** ± 0.06
(μg/g) Noradrenaline (μg/g)	1.60 ± 0.07	1.19* ± 0.10	0.72** ± 0.02

Values are mean \pm sE of four birds. * p < 0.05; ** p < 0.01

cursors have been used up in the enhanced biosynthesis of corticosterone as reported earlier. Similar observation was reported when rats were sacrificed after two hours of malathion administration at the dose level of 170 mg/kg. This observation agrees with the results of many other workers 4-16, who reported significant depletion of ascorbic acid and cholesterol from adrenal glands after applying various physical stimuli like cold, temperature, haemorrhage, burns, trauma and pains and chemicals e.g. morphine and organophosphates.

A significant increase in adrenal corticosterone levels was observed after 30 days of malathion (75 mg or 150 mg/kg) treatment (table 1). This also confirmed the above findings that the decrease in adrenal cholesterol and ascorbic acid was due in fact to enhanced synthesis of corticosterone. Similar increase in plasma corticosterone level was reported after administration of single dose of insecticides like paraquat and diquat^{17,18}. Thus a decrease in the content of adrenal cholesterol and ascorbic acid and increase in the content of corticosterone indicates that prolonged administration of malathion resulted in hypercortical activity.

The levels of both adrenaline and noradrenaline declined significantly after 30 days of malathion (150 mg/kg) administration (table 1). However, there was no effect of malathion when administered at the dose rate of 75 mg/kg for 15 days or 150 mg/kg for 15 days, on either of the catecholamines studied. The decrease in adrenal medullary catecholamines indicates that these amines are released in the blood to

enable the organisms to adapt to the stressful situation created by prolonged administration of malathion. Similar findings were reported by us² in rats after exposing them to malathion at the dose level of 46 mg/kg (i.p.) for 15 days.

The present results indicate that malathion administration for 30 days stimulates adrenal medulla as well as adrenal cortex. This gland is known to be stimulated by a number of other stressors like cold¹⁹, heat²⁰ and pesticides e.g. paraquat and diquat ^{17,18} and drugs like morphine²¹ and diazepam²² etc. Selye²³ described that in all stressful situations, it is the adrenal cortex which is primarily responsible for adapting to the stressful situation. But the present study reveals that both adrenal cortex and medulla are involved in adapting the birds to stressful situation produced by prolonged administration of malathion.

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