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IN VIVO ASSESSMENT OF MAMMALIAN TOXICITY OF PESTICIDES

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Abstract

Pesticides are a class of chemicals considered to kill or control pests that threaten crops, livestock, and human health. Organophosphates, cholinesterase- inhibiting pesticides, are widely used throughout the world in many different habitats. Because they are toxic to many species of pest. However, they are also toxic to many non-target species including human, and wildlife. The assessment of pesticide toxicity in mammals is crucial for understanding their potential effects on human health and the environment.

Current study aimed to analyze the histopathologic and biochemical effects caused by an organophosphate pesticide, dimethoate by using in vivo mammalian model. The subchronic toxicity of dimethoate administered orally at sublethal doses to a mammalian species, *Rattus norvegicus* was evaluated biochemical and histopathological point of view.

It is revealed that dimethoate caused differences in biochemical parameters and organ weights, and dose-related degenerative histological changes in the liver, testis and ovary of rats.

Keywords: *Pesticides; Dimethoate; Subchronic Effect; Histopathology; Mammalian Toxicity*

1. Introduction

Pesticides are a class of chemicals considered to kill or control pests (rodents, insects, or plants) that may affect agricultural crops, livestock or carry diseases like malaria and typhus. Organophosphates, cholinesterase-inhibiting pesticides, are widely used throughout the world in many different habitats including forests, agricultural land, rangeland, wetlands, residential areas, and commercial areas. Because they are toxic to many species of pest. However, they are also toxic to many non-target species, and when these pesticides are applied over large areas, non- target plants, fish, and wildlife can become poisoned [1].

Cholinesterase-inhibiting pesticides are known to disrupt the nervous system by inhibiting an important neuro-transmitter (cholinesterase) and prevent normal transmission of nerve impulses in the nervous system. These chemicals that are foreign to a biological system are known to induce metabolic disorders, teratogenesis, mutagenesis and carcinogenesis in non-target organisms, and cause environmental pollution. The assessment of pesticide toxicity in mammals is crucial for understanding their potential effects on human health and the environment.

Current study aimed to analyze the histopathologic and biochemical effects caused by an organophosphate pesticide, dimethoate by using in vivo mammalian model.

2. Materials and Methods

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Dimethoate, a widely used organophosphate pesticide, was administered orally at doses of 2, 8, 20 mg/kg body weight/day to Wistar Albino rats, *Rattus norvegicus*, for 13 weeks. At the end of the exposure period blood samples, liver, testes and ovarian tissues of each experimental animal were taken; these organ weights were recorded and relative organ weights were calculated. Plasma and liver cholinesterase (ChE) activities and total protein levels were determined by spectrophotometric assay. Histopathological changes in the liver, testis and ovary were determined by light microscopy

3. Results and Discussion

Treatement with dimethoate resulted in a significant decrease in liver (Table 1), testes and ovarian weight of rats [2, 3].

The biochemical results showed that dimethoate caused an increase in liver total protein level (Table 2), decrease in plasma total protein level; decrease in both liver (Table 3) and plasma cholinesterase activities [3].

Histological examinations revealed mononuclear cell infiltration (Fig. 1), congestion Fig. 2), an enlargement of the central veins and sinusoids, hepatocellular damage, an increase in the number of Kupffer cells, and in mitotic activity in the liver of treated rats. Vacuolized cytoplasm and degenerated nuclei in hepatocytes, and necrotic changes were observed in the liver sections from dimethoate treated rats. [3].

Sections from testis revealed that dimethoate caused dose-related testicular damage characterized by moderate to severe seminiferous tubules degeneration (Fig. 3) and by partial arrest of spermatogenesis. It was also detected that seminiferous tubules showed irregular arrangement of spermatogenic cells. Dimethoate also caused germ cell degeneration and depletion, sloughing of germ cells into the tubular lumen (Fig. 4) [2].

Regarding the histopathological evaluation of the ovary, there was an increase in the size and the number of corpora lutea, and the number of atretic follicles of treated rats when compared with the that of control animals [4].

It is revealed that dimethoate caused dose-related differences in organ weights, biochemical parameters, and also histopathological damages in the liver, testes and ovary of the rats.

Because of causing serious toxic effects on non-target organisms, pesticides should be applied appropriately by trained and licensed pesticide applicators and diluted according to the prospectus to prevent wildlife toxicity. Whenever possible pesticides should not be used in areas widely occupied by wildlife, especially during the reproductive season. People should make sure to use pesticides that are more specific to the target organisms, pests and less toxic to non-target organisms and wildlife.

Table 1. Relative liver weights of experimental rats. *Significantly different from control ($p \leq 0.05$)

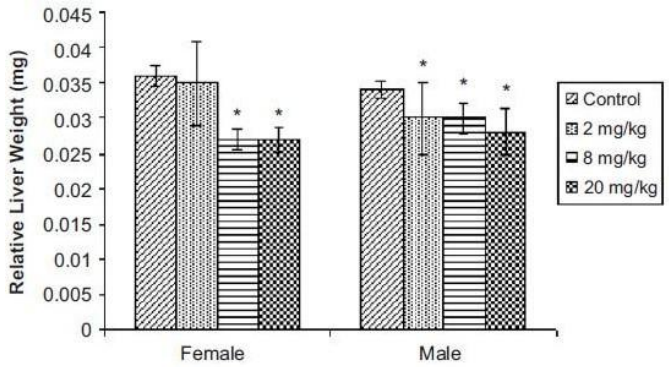

Table 2. Liver total protein values of experimental rats. *Significantly different from control ($p \leq 0.05$)

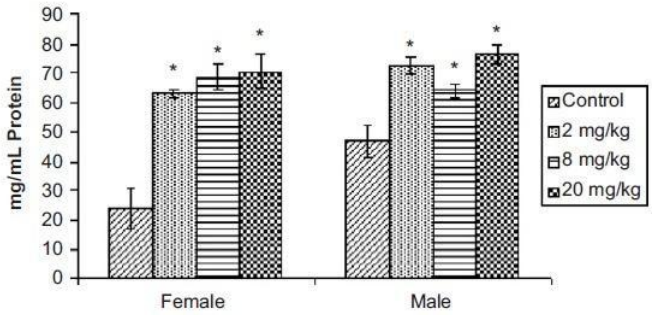

Table 3. Liver cholinesterase enzyme activities of experimental rats. *Significantly different from control ($p \leq 0.05$)

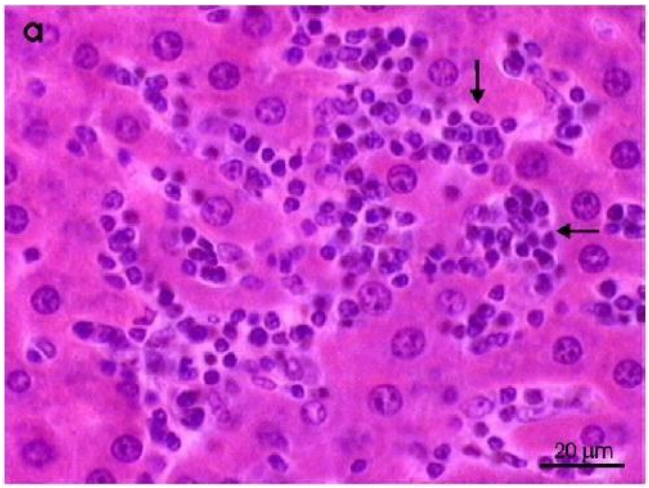

Fig. 1. Photomicrograph of mononuclear cell infiltration in parenchymatous tissue of the liver of 8 mg/kg dimethoate-exposed groups. Stain:H&E

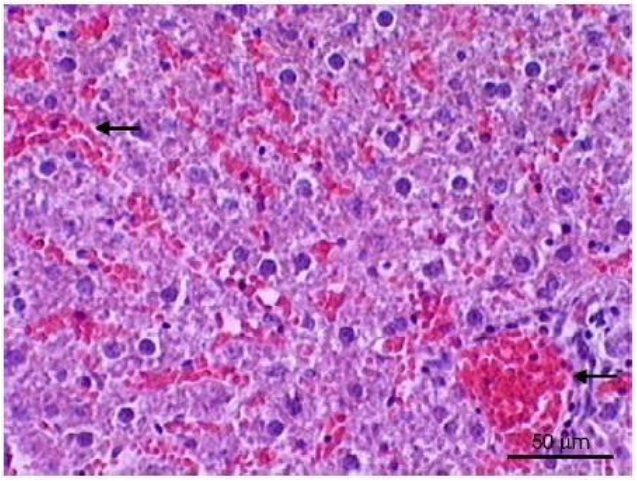

Fig. 2. Photomicrograph of congestion and enlargement at sinusoids in the liver of 8 mg/kg dimethoate-exposed group. Stain: H&E

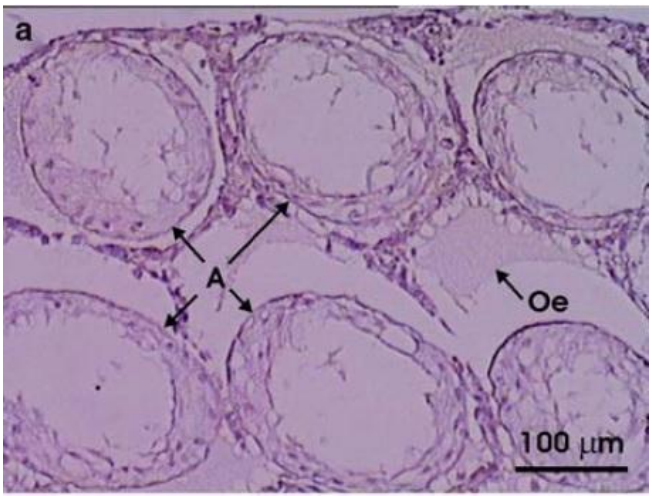

Fig. 3. Testicular sections of rats treated with 20 mg/kg dimethoate, which show further seminiferous tubule degeneration characterized by severe atrophy of tubules. Note the loss of all germ cells and, the enlargement of interstitial spaces due to tubular atropy and oedema. Atrophic tubules (A), oedema (Oe)

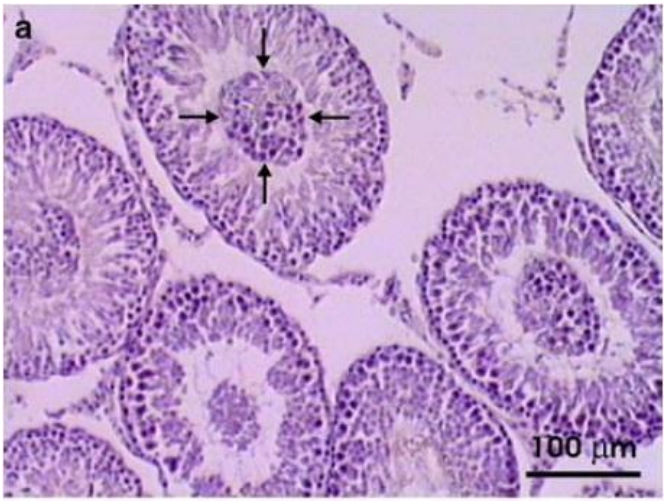

Fig. 4. Testicular sections of rats treated with 8 mg/kg dimethoate showing seminiferous tubule degeneration. Note the sloughing of germ cells into the tubular lumen.
Arrows point to the sloughing

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