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# Effects of Sub Chronic Exposure of Diazinon on Wild Pigeon (*Culumba livia gaddi*) at Basrah City/Southern Iraq

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**Abstract:** A three months as sub chronic study of diazinon on male wild pigeons (*Culumba livia gaddi*) by oral intubation were done. The study consisted of four groups each with six pigeons: A(high dose 0.3 mg), B(intermediate dose of 0.6 mg), C(low dose 1.2 mg) and D(untreated control). Clinical observation of treated pigeons did not show significant changes only the birds appeared to the quite after dosing for short time.

**Key words:** Sub chronic, diazinon, wild pigeons, inflammatory cells, vacculation

## INTRODUCTION

Diazinon, (0, 0-diethyl 0-(2-isopropyl 1-6-methyl 1-4-pyrimidinyl) phosphoorthiate) and several other organophosphorus insecticides exert their toxic effects by inhibiting cholinesterase in many different animals (Davies and Holub, 1980).

A variety of chemicals (pesticides) are repeatedly in our agricultural land and these pesticides can accumulate in animals bodies which concentrate them and pass from pray to predator (Tabassum *et al*., 2003). It was released for experimental evaluation in the early 1950's and today diazinon was used extensively by commercial and home applicators in a variety of formulations to control flies, cockroaches, lice on sheep, insect pests of ornamental plants and food crops (especially corn, rice, onions and sweet potatoes), nematodes and soil insects in turf, lawns and croplands (Berg, 1984).

Waterfowl and other wildlife may acquire diazinon by drinking contaminated water, by absorbing it through legs and feet, by consuming treated grass or grain, or by ingestion of pesticide-impregnated carrier particles (Stone and Gradoni, 1985). The results for examples Kills Canada geese, brunt, mallard, American black duck, other species of waterfowl and songbirds have all been associated with consumption of grass or grain shortly after diazinon application (Stone and Knoch, 1982). Lox (1983) reported that fatal diazinon poisonings have been recorded in humans, while, Sokkar *et al*. (1975) reported that the domestic chickens were killed by diazinon poisonings, domestic ducklings and goslings also killed by diazinon poisonings (Egyed *et al*., 1976). In laboratory monkey of the tamarin and the common marmoset a reported of die by diazinon poisonings (Brack and Rothe, 1982) and honeybee (Anderson and Glowa, 1984).

Mammals seem to be less sensitive than birds to diazinon poisoning (Stone and Gradoni, 1985), but in fishes a sub lethal effects such as reduced hatch, retarded growth and spinal deformities (Allison and Hermantz, 1977). A reduced food consumption and egg production in the ring-necked pheasant (Stromborg, 1977) and behavioral modifications, reduced food intake, alterations in liver enzyme activities, reductions in vitamin concentrations, reduced body temperature and lowered resistance to cold stress in white-footed mice (Montz and Kirkpatrick, 1985). It have been noticed that diazinon concentrations markedly lower than those causing acute mortality and suggested that wildlife partially disabled in the field as a result of diazinon poisoning would be more likely to die of exposure, predation, starvation, dehydration, face behavioral abnormalities learning, impairments and reproductive declines than would similarly treated domestic or laboratory animals (Montz, 1983). Therefore, the aim of the present study was to investigate the toxopathology of diazinon in wild pigeon in many organs at Basrah city/ southern Iraq.

## MATERIALS AND METHODS

**Animals:** Adult wild male pigeons were purchased from local market from Basrah city with average body weight (200-350 gm) and reared in a clean cages (200 x 100 x 80 cm) in poultry unite/college of veterinary medicine/ Basrah university, all pigeons were acclimatized for 10 days before start the experiment.

**Chemicals:** Diazinon 60 EC was applied as a commercial emulsifiable concentrate formulation containing 60% active ingredient, then, it was further diluted in distilled water to obtain the desired concentration. The solution was prepared and used immediately, by oral gavage using disposable syringe (after removing the needle), the doses of diazinon were determine by testing the compound on few pigeons, also the maximum toxic dose used according to the active ingredients of the substance.

**Treatments:** To study a sub chronic effect of diazinon on pigeons a total Twenty four adult male wild pigeon (*Culumba livia gaddi*) weighting (200-350 gm) were randomly allocated and housed in separate cages of the college of veterinary medicine, Basrah university. The birds equally divided into four groups: A,B.C and D(6 birds in each group). They were poisoned with diazinon for ninety days. Birds of the group A, B and C were orally (a gavage needle) given daily doses of diazinon at the levels of 0.3 mg, 0.6 mg and 1.2 mg respectively, whereas group D was acted as control. All of the birds were supplied with diet with water *and Libitum*. The birds was killed after ninety days and the organs (brain, sciatic nerve, spinal cord, heart, liver, kidney and pancreas) were removed and fixed in 10% neutral buffered formalin for histopathological examination.
After that all pigeons were killed by cervical dislocation, selected visceral organs were fixed in 10% neutral buffered formalin for further histopathological study.

**Histopathological examinations:** Five µm thick paraffin section of 10% neutral buffered formalin as fixative, liver, kidney, spleen, lung, pancreas, brain, spinal cord and sciatic nerve from each pigeon were fixed in formalin, then samples were cut and paraffin blocks were made, slide were cut and stained with Haematoxyline-Eosin (HE), selected histopathological changes were photographed from treated related histopathological changes in comparison to untreated controls, according to the method of Annpreece, 1972.

## RESULTS

The toxologic pathology for diazinon on pigeons were founded this results:

**Liver:** A Liver in group A showed a peripheral fibrosis with mononuclear cells and proliferation in bile ducts (Fig. 1), while, a Minimal vacculation of hepatocytes in centre lobular region as shown in group B (Fig. 2). In group C there was aggregation of mononuclear cells and vacculation in hepatocytes (Fig. 3), as compared with control group (Fig. 4).

**Pancreas:** A vacculation of islet cells of langerhanse were noticed in all groups under this study (Fig. 5).

**Kidney:** A dilated cortical tubules were founded in all groups but with infiltration of mononuclear cells in group B and C and congestion in both B and C groups (Fig. 6, 7, 8, 9) as compared with control (Fig. 10).

**Sciatic nerve:** In group A few and several degenerated, vacuolated nerve fibers (Fig. 11). Several degenerate, vacuolated nerve fibers were founded in group B (Fig. 12), while, in group C a several degenerate, vacuolated nerve fibers (Fig. 13) as compared with control (Fig. 14).

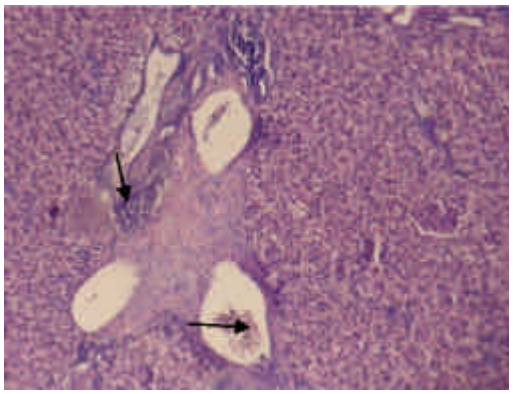

Fig. 1: Liver in group A with peripheral fibrosis and mononuclear cells and proliferation of bile ducts (H&E stain) (40x)

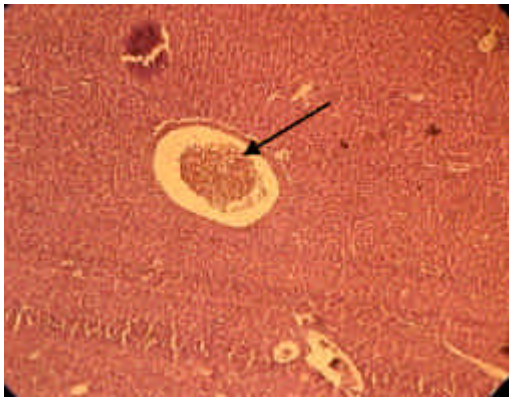

Fig. 2: Liver in group B a Minimal vacculation of hepatocytes in centre lobular region (H&E stain) (40x)

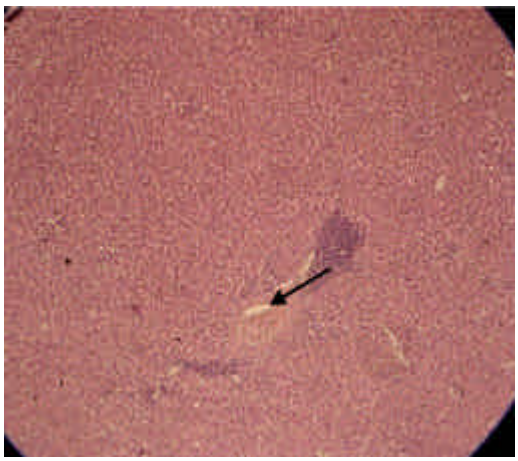

Fig. 3: Liver in group C a vacculation of hepatocytes and aggregation of mononuclear cells (H&E stain) (40x)

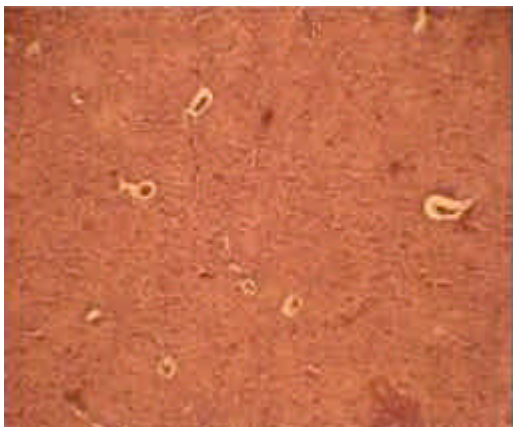

Fig. 4: Liver in group control (H&E stain) (40x)

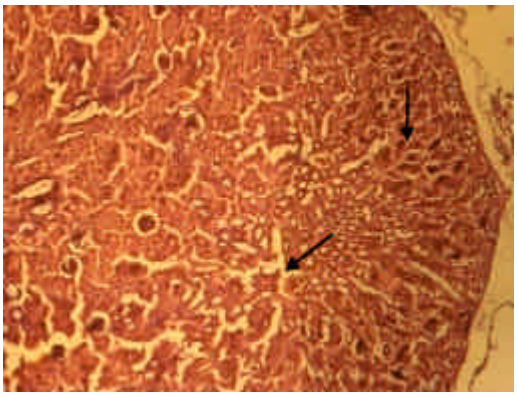

Fig. 7: Kidney in group B: Dilated cortical tubules and infiltration of mononuclear cells (H&E stain) (10x)

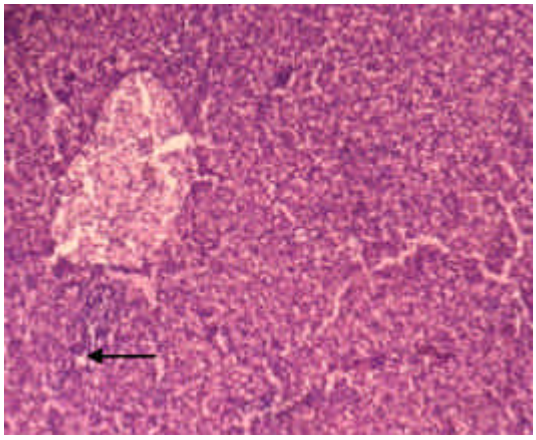

Fig. 5: Pancreas with Vacculation of Islet of langerhanse (H&E stain) (10x)

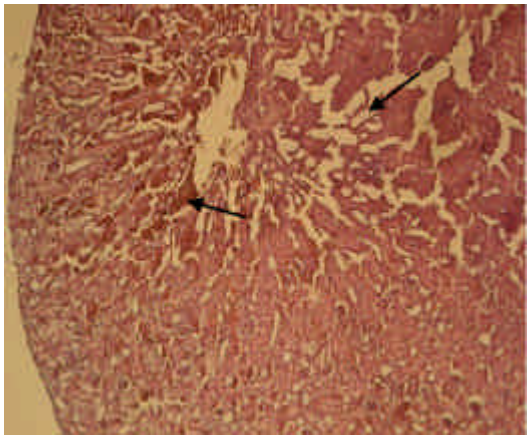

Fig. 8: Kidney in group B: Dilated cortical tubules with congestion (H&E stain) (40x)

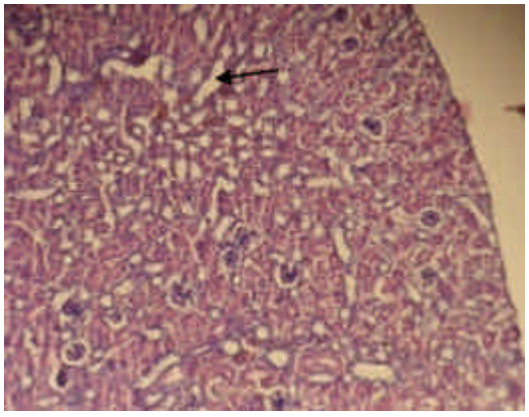

Fig. 6: Kidney in group A: Dilated cortical tubules (H&E stain) (10x)

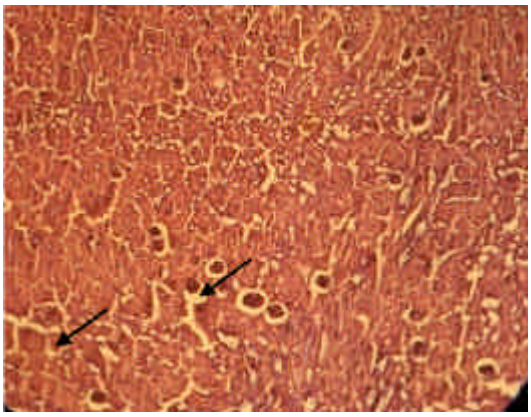

Fig. 9: Kidney in group C: Dilated cortical tubules with congestion and infiltration of mononuclear cells (H&E stain) (40x)

**Spinal cord:** A several degenerate vacuolated nerve fibers were founded in the spinal cord of pigeons in A group (Fig. 15), while, in B group a massive degenerate, vacuolated nerve fibers (Fig. 16). In C group a several degenerate, vacuolated nerve fibers (Fig. 17).

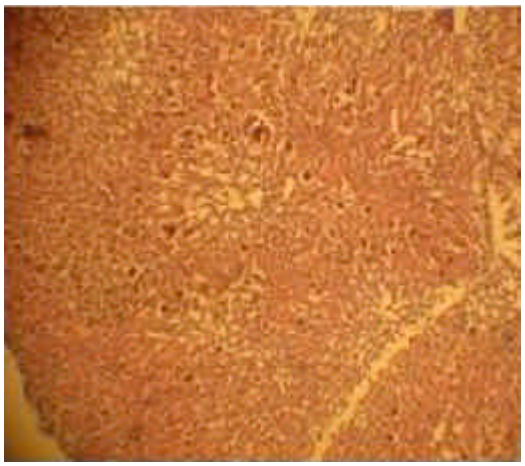

Fig. 10: Kidney control group (H&E stain) (10x)

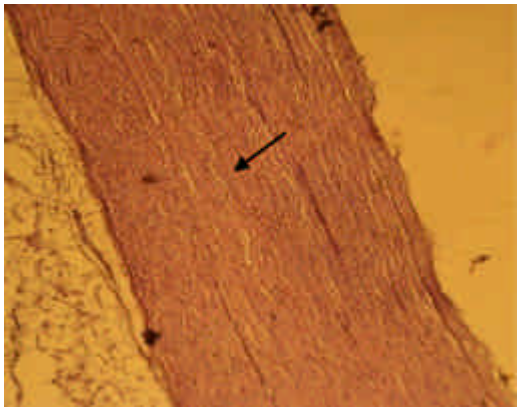

Fig. 11: Sciatic nerve in group A: A few degenerated, vacuolated nerve fibers (H&E stain) (10x)

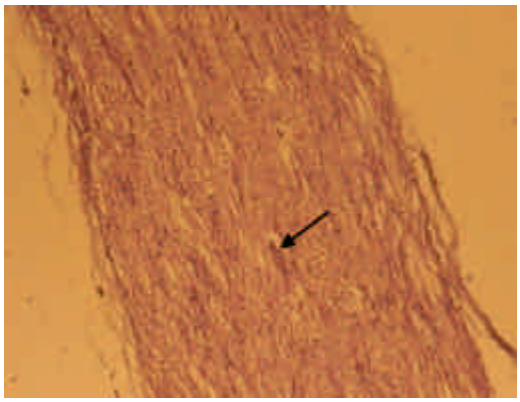

Fig. 12: Sciatic nerve in group B: Degenerate, vacuolated nerve fibers (H&E stain) (10x)

**Brain:** The brain of all pigeons for all groups under this study without any pathological changes, as shown in (Fig. 18).

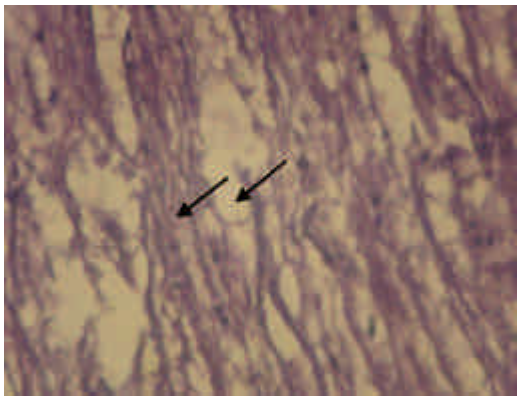

Fig. 13: Sciatic nerve in group C: Several degenerate, vacuolated nerve fibers (H&E stain) (40x)

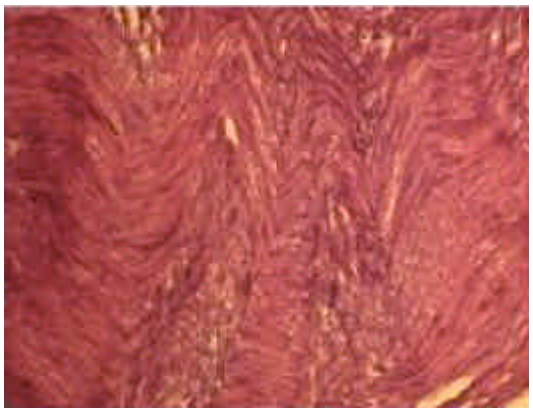

Fig. 14: Sciatic nerve in control group (H&E stain) (40x)

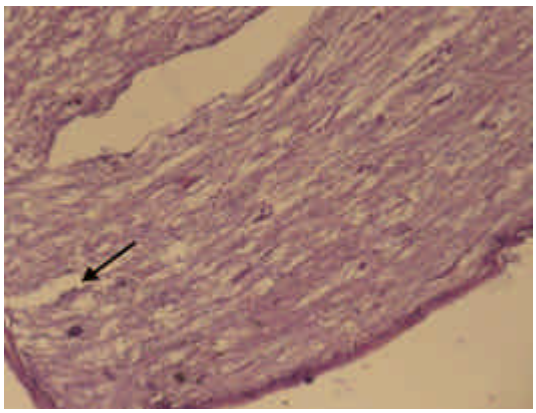

Fig. 15: Spinal cord in group A: Several degenerate, vacuolated nerve fibers (H&E stain) (10x)

## DISCUSSION

Diazinon is a very highly toxic organophosphate compound. Organophosphates are long known and widely applied active ingredients of different insecticides used in the plant protection practice. In animal

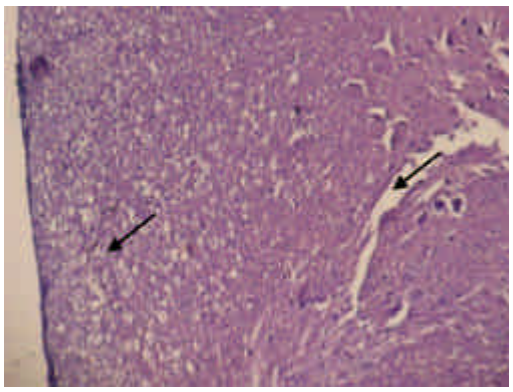

Fig. 16: Spinal cord in group B: Massive degenerate, vacuolated nerve fibers (H&E stain) (10x)

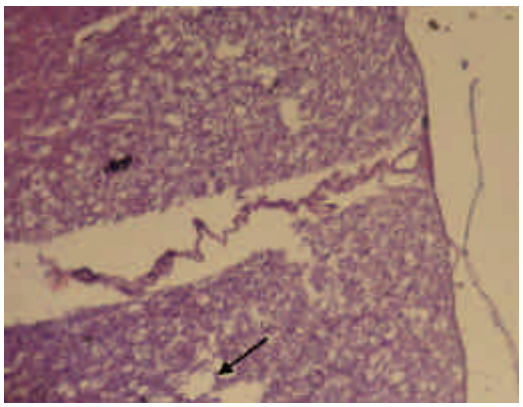

Fig. 17: Spinal cord in group C: Several degenerate, vacuolated nerve fibers (H&E stain) (10x)

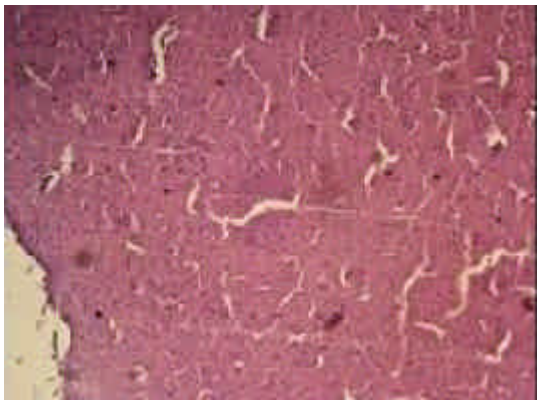

Fig. 18: Brain without any changes (H&E stain) (10x)

production these compounds are used to control a variety of ectoparasites such as mites and lice. In the living organism, organophosphates inhibit the enzyme acetylcholinesterase, causing an accumulation of acetylcholine, a neurotransmitter involved in impulse transmission and leading to an over-stimulation of the parasympathetic nerves (Farage-Elawar, 1989). Poisoned animals show salivation, lachrymation, diarrhoea and convulsions followed by depression, prostration, ataxia and cyanosis and then death usually ensues within a short time. Avian species are more susceptible to the toxic effects of diazinon than are mammals. Poisoned chickens often exhibit only respiratory distress, lachrymation and salivation before death and therefore the suspicion of an acute respiratory infection may also arise (Brown *et al*., 2003).

Diazinon is rapidly metabolized in mammals and is excreted principally through the urine. It is metabolized *in vivo* by four enzyme systems, which include mixed function oxidases, hydrolases or phosphatases, glutathione-dependent transferases and non-specific esterases. Most *in vivo* animal studies have demonstrated the production of diazoxon, hydroxydiazinon, isohydroxy-diazinon and a propylenediazinon metabolite. Diazinon does not bioaccumulation in tissues or organs. The mode of action of diazinon, as with other organophosphate insecticides, is inhibition of the enzyme cholinesterase (VDH, 2001).

There was a pathological changes in different organs of pigeon under this study; in liver and pancreas founded a vacculation in hepatocytes and in islet of langerhanse, fibrosis in many parts and in bile duct and infiltration of mononuclear cells. This is because that which could be due to the toxic effects of diazinon on liver cells due to the metabolic mechanisms of the liver cells and the vacculation of cytoplasm of hepatocytes may be from extensive lipid infiltration. This result agree with many studies on different animals by Anees, 1978; Sastry and Sharma, 1981.

Anthony *et al*. (1986); Jacqueson *et al*. ( 1977) noticed that the liver of male wistar rats chronically treated with sublethal doses of diazinon sustain a form of hepatic injury characterized by cellular lipid accumulation and this is because a toxic agents as carbon tetrachloride, phosphorus and chlorinated hydrocarbon insecticides.

Fazekas *et al*. (2008) reported that diazinon cause an demonstrated the signs of circulatory disturbances in the inner organs of geese. VDH (2001) reported that diazinon is highly toxic to birds and the acute oral LD50 (mg/kg) for technical diazinon is: 6.81 for turkey; 40.7 for chicken; 14.7 for goose; 2.75 for gosling. The sub acute dietary LC50 (ppm) for technical diazinon is: 191 for mallard ducks; 245 for bobwhite quail; 244 for ring-necked pheasant; 47 for Japanese quail.

In kidney the main pathological signs was a dilated cortical tubules and aggregation of mononuclear cells, this may be that diazinon cause a toxicity in renal system by their metabolism of this compound and the immune system make a good role for defending against foreign particles.

In sciatic nerve and spinal cord the main changes was vacculation and degenerate in fibers this may be due that diazinon cause a neurotoxic effects on these organs.

Diazinon exposure of pregnant laboratory animals in tests has demonstrated that this insecticide can cause a variety of reproductive problems, including damage to the developing nervous system, delays in sexual development, stillbirths, death of newborn offspring and birth defects. But the effects on the developing nervous system are most significant (NCAP, 2000).

Diazinon toxicity varies widely within and among species and is modified by organism age, sex and body size, climatic conditions, pesticide formulation, chemistry of the environment and other factors (Montz, 1983). Diazinon has a potential for causing acute avian poisoning episodes (Schafer *et al*., 1983).

Fazekas *et al*. (2008) reported that a histopathological examination of Geese with diazinon poisoning was demonstrated changes indicative of acute circulatory disturbance, passive congestive hyperaemia and occasionally, mild interstitial edema was observed in the brain, liver, kidney, heart and pancreas.

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