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ISSN -1817 -2695

# Histopathology of sub chronic toxicity with organophosphate compound coumaphos in Rock dove pigeons (*Columba livia gaddi*)

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Received 23-11-2011, Accepted 14-5-2012

### Abstract

The aim of this study was to investigate sub chronic toxicity of oral administration of organ phosphorus insecticide coumaphos for three month in rock dove pigeons. Thirty birds were divided into three equal groups Each group was treated either with 0.5 mg/bird (group A) or 0.3 mg/bird (group B) or left as control group(group C)..Histopathological changes were revealed degenerate/vacuolated nerve fibers in spinal cord and sciatic nerve, liver congestion and periportal fibrosis. Hyperatrophy of renal cortical tubules and vacuolation of myocardial muscle cells were also noticed.

## 1.Introduction

Coumaphos is an organophosphorus insecticide used to control of a wide variety of livestock insects, including cattle grubs, screw-worms, lice, scabies, flies, and ticks. It is used against ectoparasites, which are insects that live on the outside of host animals such as sheep, goats, horses, pigs, and poultry [1]. It is added to cattle and poultry feed to control the development of fly larvae that exist in manure. It is also used as a dust, dip, or spray to control mange, horn flies, and face flies of cattle [2]

Coumaphos is considered a selective insecticide because it kills specific insect species while sparing other non target organisms.

Coumaphos is one of a class of pesticides referred to as organophosphates. These chemicals act by interfering with the activity of naturally-occurring enzymes called cholinesterases. Cholinesterases are essential for the proper working of the nervous systems in the bodies of both humans and insects.[3]

Figure 1.Molecular structure of coumaphos [4]

# 2. Materials and methods;

# 2.1.Experimental design

A total of 30 rock dove pigeons (Columba livia) were purchased from the local market in Basrah province within body 300-400g. The weight average between birds were divided into three groups. Group A was treated with high dose coumaphos, and group B was treated with intermediate dose of coumaphos ,while group C served as control group. The pigeons were reared in separated cages of 100x100x80 cm<sup>3</sup> at the Poultry Diseases Unit, College of Veterinary Medicine in Basrah University under suitable conditions water and feed were supplied ad libitum.Group A was administrated orally with 0.5 mg of coumaphos .Group B was administrated 0.3 mg of coumaphos

Coumaphos( Bayer AG, Leverkusen) dissolved in distal water to obtain the desired concentration for oral dosing by a gavage needle. The solution was prepared and used immediately. The doses of the Coumaphos were used according to the active ingredients of substance days later all remaining birds were killed by decapitation. Brain, spinal cord, sciatic nerve, liver, kidney, and heart samples were collected for the histopatological examination. Tissue samples were kept in / neutral buffered formalin and treated according to (14) to obtained. 5 µm slides, stained with Haematoxylin and Eosin.

## 3.Results

Histopathological changes of our study were revealed degenerate/vacuolated nerve fibers in spinal cord and sciatic nerve of group A and group B as shown in figure[2,3,4 and 5], hyperatrophy of renal cortical tubules of such groups figure[6 and 7], liver congestion and periportal fibrosis of

both groups as shown in figure[8 and 9], vacuolation of islets of group A while group B revealed slight changes of langerhans as shown in figure [10 and 11], and vacuolation of myocardial muscle cells were also noticed of group A and B in figure[12 and 13].

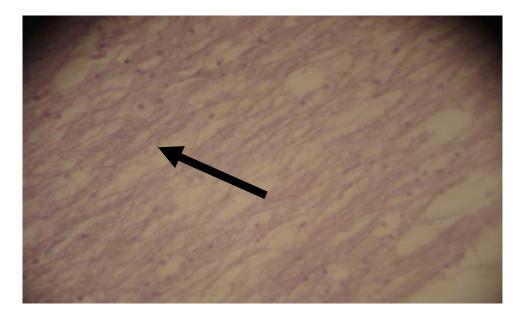


Figure 2 group A ..Spinal cord. Vacuolation of nerve fibers.H &E x200

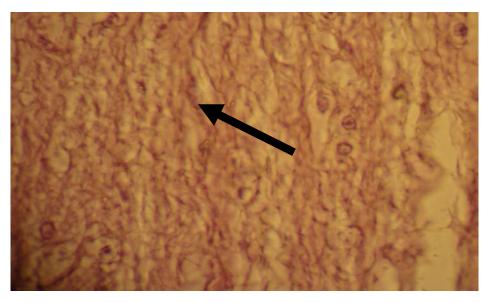


Figure 3. group B. Spinal cord Vacuolation of nerve fibers. H&E  $\,$  X 400  $\,$ 

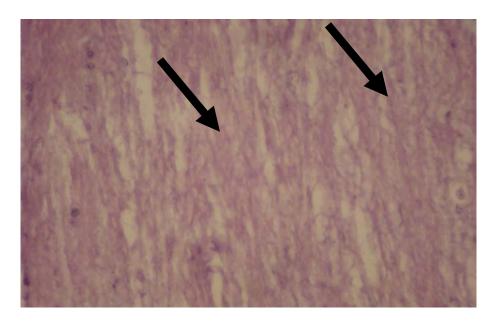


Figure 4.Group A. sciatic nerve. Marked degnerate vacuolation of nerve fibers.H&E  $\,$  X200  $\,$ 



Figure 5.group B. sciatic nerve. Vacuolation of nerve fibers. H&E  $\,$  X 200

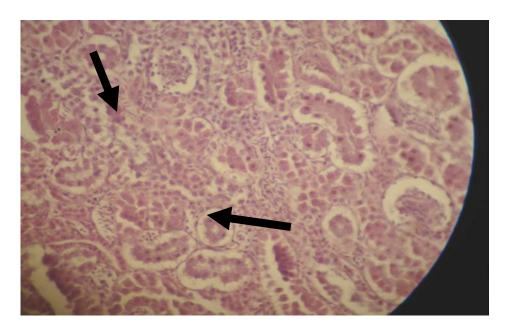


Figure 6.group A. kidney .hyper atrophy of renal cortical tubules. H&E  $\,$  X 200

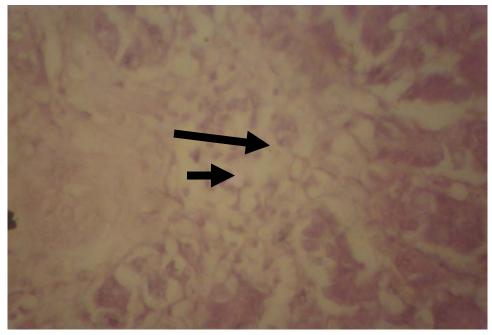


Figure 7. group B ..kidney .degnerate vacuolation of renal tubules. H &E X400  $\,$ 

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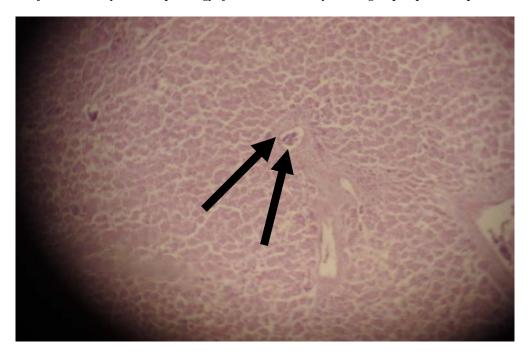


Figure 8.group A. liver. Periportal fibrosis and congestion. H&E  $\,$  X 200

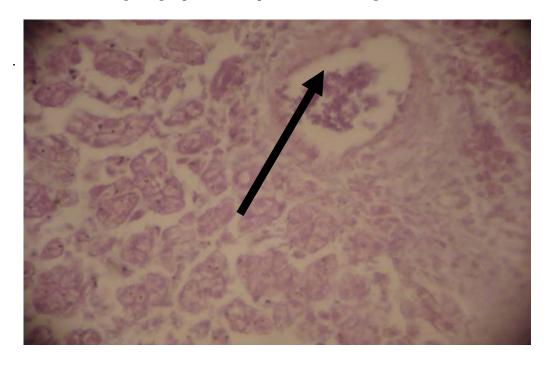


Figure 9. group B .liver .congestion.  $H\&E \times X200$ 

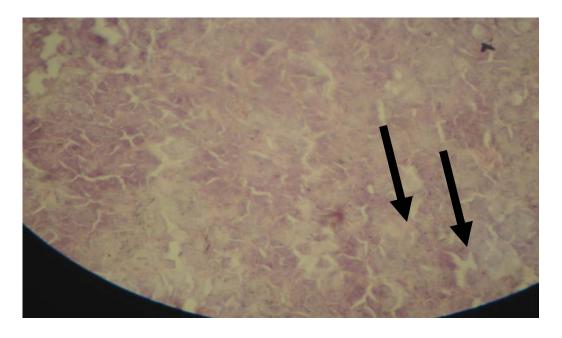


Figure 10.group A. Pancreas.Vacuolation of islets. H&E X 200

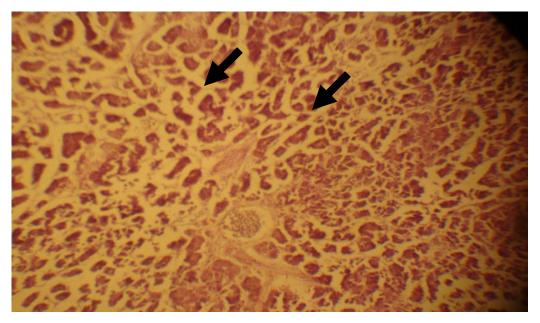


Figure 11 .group B . Pancreas within normal limits, arrow for islet  $\ .H\&E\ X\ 200$ 

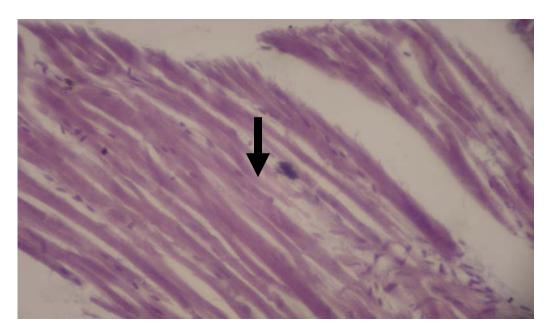


Figure 12. Group A. Heart. Vacuolation of cardiac muscle cells .H & E X 200

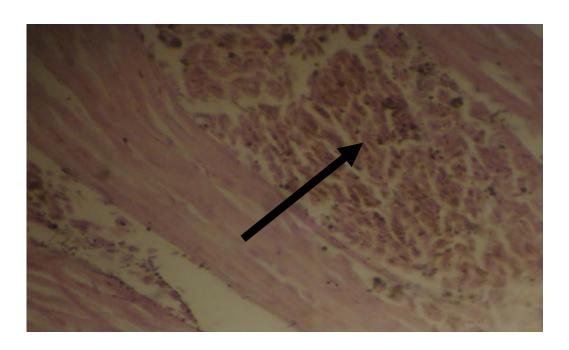


Figure 13. Group B. Heart congestion H&E X 200

#### Discussion

neurotoxic effects of Acute organophosphorus insecticides are well documented. In recent years there is widespread concern over exposure to low levels of organophosphates in the diet over a longer period of time. There reports which suggest organophosphorus insecticides manifest enhanced their toxic effects bv production of reactive oxygen species (ROS) which is a major cellular source of oxidative stress[5,6 and 7]

Pigeons dosed with 0.5mg and 0.3 mg/ bird of coumaphos were showed changes histopatholoical either peripheral nerves (sciatic nerves) or in spinal cord as degenerate/ vacuolated nerve fibers, as shown in fig [2,3,4 and 5], these results were in line with that of Abu donia who found that the neurotoxic effects of coumaphos when applied orally or dermally in the adult hen. Dermal administration of single (50-500 mg/kg) or daily (100 mg/kg) doses resulted in delayed neurotoxicity in hens, similar to that caused by other delayed organophosphorus neurotoxic compounds .Degeneration of axons and myelin in the spinal cord was the most consistent histopathologic alteration and was identical to that reported for other delayed neurotoxic organophosphorus esters. Only one hen showed peripheral nerve degeneration.[8]

Our results are also in accordance with that of [9] who found that ten day old chick sympathetic ganglia cultured in a microslide assembly were treated with a selected group of organophosphate pesticides to evaluate their cytotoxicity ranges, and the usefulness of such a model for screening pesticides include methylparathian, diazinon, paraoxon, mevinphos, diisopropylfluorophosphate,

tri-o-tolyl phosphate and its mixed isomers to a  $1 \times 10^{-3}$ M (intermediate) for malathion, leptophos, coumaphos, monoand dicrotophos. Some or no effects were evident at  $1 \times 10^2$ -M for O'ethyl-O**p**-nitrophenyl phenyl phosphonothioate, tri-m-tolylphosphate, chlorpyriphos and triphenyl phosphate. In all instances, nerve fibers were more sensitive than neurons or glial cells to insecticides. All cellular growth was inhibited at  $1 \times 10^{-1}$ <sup>2</sup>M (except triphenyl phosphate).. The abnormalities secondary included decreased cellular migration, diffuse cellular growth pattern, increased vacuolization, nerve fiber swelling and cellular degeneration).

The results of our study exhibited degenerate/vacuolated of nerve fibers in spinal cord and sciatic nerves related to that of [10], were noted the ducklings treated daily with cyanofenphos or with leptophos at different dose levels for 90 days, or until they died, or became paralyzed.. The observed clinical signs were confirmed by histological changes found in the spinal cords of the treated birds. These changes were of the type associated with organophosphorus-induced delayed neuropathy (OPIDN). These results demonstrate that wild mallard ducklings are susceptible to OPIDN and this avian species can be used in screening organophosphorus compounds for such effect ,OPIDP is a rare toxicity caused by certain organophosphorus compounds (OP) characterized by degeneration of some long axons in the central and peripheral nervous system that appear about 2-3 weeks after exposure.

These results conflicted with that of [11]. Who noticed histological alterations in the spinal cord of paralyzed one hen

after oral administration with organo phosphorous leptophos included axon and myelin degeneration in the ventral, lateral and posterior columns. This result might be attributed to the metabolic variation of the avian species, using of different doses . In the present investigation, it was observed that organ phosphorus insecticide, coumaphos caused histopathological changes in the liver and kidney of the rock dove pigeons. The liver is the centre for detoxifying any foreign compounds entering the body. So, it uniquely exposed to a wide variety of exogenous and endogenous products. These include environmental toxins and chemicals present in food or drinking.

The present investigation indicated administrated that pigeons with coumaphos caused significant alterations in the liver and kidney which include liver congestion and periportal fibrosis, and hypertrophy of renal cortical tubules. These results were in line with that of [12] who found that malathion and spinosad insecticides led to degenerative changes of hepatocytes, vacuolation of hepatocytes and hepatic necrosis in rat. In kidney, degenerative changes of epithelial lining of renal tubules were associated with occlusion of the lumen and necrotic changes with desquamation of epithelial lining.

These results were also related to that of [13] who exhibited

the high dose of diazinon decreased body weight significantly. Damage in the liver and kidney tissues .VitE partially counteracts the toxic effect of DZN and repairs tissue damage in the liver and kidney, especially when supplemented to 1/4 LD<sub>50</sub> intoxicated animals. Histopathological changes in liver and kidney were observed only in 32.5 mg/kg DZN given group.

The present study was indicated damage of the liver and kidney of pigeons supporting the earlier reports of [14] who explained that malathion intoxication led to severe effects on the structures of the liver and kidney including the capsular presence of fine sub infiltrations, diffused parenchymatous degeneration of single hepatocytes parenchymatous degeneration of the cells of renal tubules and hyperemia of the cortical part of the kidney, especially of renal glomeruli, as well as infiltrations were noted.. However, different studies showed that malathion and other pesticides induced liver and kidney histopathological alterations experimental animals(14). Other changes in this study included vacuolation of myocardial muscles.

It may be concluded from the present study that subchronic oral administration of rock dove pigeons with organ phosphorus compound coumaphos was caused histopathological changes in such organs as well as exhibited in our study.

# References;

1-Toxnet.. National library of medicine's toxicology data network. Hazardous Substances Data Bank (HSDB). Public Health Service. National Institute of Health, U. S. Department of Health and Human Services. Bethesda, MD: NLM. (1975-1986).

2- Environmental Protection Agency. U. S. EPA chemical profile : coumaphos. Washington, DC. 1985.

3- Environmental Protection Agency US. Pesticide Fact Sheet: 1989 . Coumaphos (Fact Sheet Number 207). US EPA, Washington, DC.

4-Symptoms: Recognition and Management of Pesticide Poisoning, 5th edition, U.S. EPA, Chapter 4.

5- M. Abdollahi, A. Ranjbar, S. Shadnia, S. Nikfar, A. Rezale, 2004 . Pesticides and oxidative stress: a review, Med. Sci. Monit. 10 (6) RA141–RA147 .

6 Altuntas, N. Delibas, M. Demirci, I. Kiline, N. Tamer .2002 .. The effects of methidathion on lipid peroxidation and some liver enzymes: role of vitamin E and C, Arch. Toxicol. (76)470–473

7-. Altuntas, N. Delibas,. 2002 . Effects of organophosphate imedicide fenthion on lipid peroxidation and antioxidant enzymes in rat erythrocytes: the role of vitamins E and C, Bio-med Res. 13.43–47

8-Abou-Donia MB, Makkawy HA, Graham DG- .1982 . Journal of <u>Toxicol</u> <u>Environ Health.</u>; Coumaphos: delayed neurotoxic effect following dermal administration in hens. 10,87-99 .

9-. Obersteiner E. J and R. P. Sharma. 1978 .Evaluation of Cytotoxic Responses Caused by Selected Organophosphorus Esters in Chick Sympathetic Ganglia Cultures. Canadian Journal Comparative Medicine.; 42,80–88.

10-Soliman SA, Ahmed NS, el-Gendy KS, el-Bakary AS, el-Sebae AH. 2001. Delayed neurotoxicity in the wild mallard duckling caused by the organophosphorus insecticides cyanofenphos and leptophos.(abstract). 11-Hussain MA, Oloffs PC. 1979.

eurotoxic effects of leptophos (PhosvelR) in chickens and rats following chronic low-level feeding. Environ Sci Health B.;14,367-82.

12- Sameeh A Mansour, Tarek M Heikal, Abdel-Tawab H Mossa.. 2001. histopathological Biochemical and containing effects of formulations Malathion and Spinosad in rats. Environmental Toxicology Research Pesticide (ETRU), Chemistry Department, National Research Centre, Dokki, Cairo, Egypt

13-Nahla S. EL-

SHENAWY, Rasha A. A.

EISA, Fawzia ELSALMY, Omema SAL AH. Prophylactic effect of vitamin E against hepatotoxicity, nephrotoxicity, haematological indices and histopathology induced by diazinon insecticide in mice Prophylactic effect of vitamin E against hepatotoxicity, nephrotoxicity, haematological indices and histopathology induced by diazinon insecticide in mice.

14- Atef M. Al-Attar. 2010 . Physiological and Histopathological Investigations on the Effects of  $\alpha$ -Lipoic Acid in Rats Exposed toMalathion. Journal of Biomedicine and Biotechnology ., Article ID 203503, 8 ,