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HISTOPATHOLOGICAL CHANGES ASSOCIATED WITH EXPOSURE OF MALE MICE TO PYRETHROID PESTICIDE (LAMBDA-CYHALOTHRIN)

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ABSTRACT

The male albino rats were administered to sub-lethal concentrations 1/10 LD_{50} = 9.5 mg LCT/kg b. wt., 1/40 LD_{50} = 2.37 mg LCT/kg b. wt., and acceptable daily intake (ADI) = 0.005 mg LCT/kg b. wt. orally twice a week for 30, 60, and 90 consecutive days. Histopathological examination revealed that, tissues were normal in the control group, but in treated group liver showed congestion of hepatic blood vessels, vascular degeneration with necrosis of hepatic cells. The cells within the group oriented toward the base membrane. The hepatic cells showed polymorphism in its shape and size and the nucleus were enlarged with typical or a typical meiotic activities (hepatic carcinoma). Pathological finding in kidney showed congestion of renal blood vessels in both cortex and medulla together with perivascular edema, degeneration and coagulative necrosis and cystic dilation. Pathological finding in spleen showed hyperplasia of the lymphocytes of the white pulp with infiltration of the red pulp with lymphocytes, congestion of splenic sinusoids and hemorrhages with depletion of lymphocytes in white pulp. Pathological finding in brain showed congestion of meningeal blood vessels, lymphocytic aggregation, degenerative changes of the nerve fibers and fragmentation and necrotic changes of some neurons. Generally histopathological examination revealed vascular congestion, hydrophic degeneration and leukocyte infiltration in the affected organs at the initial stages. At the terminal stage of toxics, coagulative necrosis, aggregation, odema, and carcinoid tumors of liver. The degree of changes was obviously at high dose (treatment 1/10 LD_{50} was more effective in changes than 1/40 LD_{50} treatment), it was not able to observe any significant changes at low dose (ADI), it means that (LCT) caused dose dependent, and induces histological aspects of liver which was the most affected organ.

Keywords*:* lambda-cyhalothrin, male mice *(Mus musculus),* histopathology, carcinoid tumors liver, kidney, brain, spleen.

INTRODUCTION

Around the world, approximately three million acute poisoning and 220000 deaths from pesticide exposure have been reported annually. In addition, formers with prolonged exposure, such as, neurobehavioral abnormalities and increased cancer incidence e. g., leukemia, nonhodgkin, Lymphoma and multiple myeloma. The potential utility of biomarkers for monitoring both environmental quality and the health of organism inhabiting in the polluted ecosystems has received increasing attention during the last years Lopes *et al.,* (2001); Torre *et al.,* (2005); Mdegela *et al.,* (2006) and Minier *et al*., (2006). Residual amounts of pyrethroid pesticides have been detected in the soil, water, bodies, vegetables, grain and other food products

Johns *et al.* (2001). Toxicities of pesticides cause adverse effects on many organs. Pesticides affect mitochondrial membrane transportation in rat liver Nakagawa and Moore, (1999). Furthermore, it disturbs cytochrome P450 system in human liver Kappers *et al.* (2001); Sams *et al.* (2003). Meanwhile, OP causes toxic effects on other organisms Keizer *et al.* (1995). Diagnosis and predication of physiological consequences of sub lethal contamination can be obtained thought histopathology Fanta, (1997b), Rodrigues and Fanta, (1998), Rudolph and Boje, (1987), Silva *et al.* (1993). Retention of the pyrethroid in the liver for days or months after intoxication opposes the usual opinion that such pesticides are quickly degraded in nature Ansari and Ansari, (1987a); Murty (1986). This work is important due to the use of pesticide as well as the use of any potentially injurious chemical substance taking into consideration the balance of the benefits that may be expected versus the possible risk of injury to human health or degeneration of environmental quality FAO, WHO (1981). The present investigation aimed to assessment the safety/risk of the chemical under a specific exposure and histopathological study to investigate capability of pesticides to induce cancer or malignant tumors in tissues.

MATERIALS AND METHODS

Animals: 120 male albino mice were used in this investigation, aged 4-5 weeks and of mean weight 20 gram. The animals were randomly housed in appropriate stainless cages in group of 5 animals /cage. The animals were arranged into four groups, they were also monitored daily for abnormal symptom.

Chemicals: Lambda-cyhalothrin**:** is a restricted use synthetic pyrethroid insecticide. The active ingredient (Lambda-cyhalothrin 99.8 % Agrochemical Co.).

Animal treatment schedule: Randomized groups of rats housed in cages containing saw dust as beading and were allocated into 4 groups (1 control + 3 for tested pesticide) each one contained 30 males, the first group used as a control, while the second, third, and fourth groups were treated with Lambda-cyhalothrin at doses 1/10 LD_{50}, 1/40 LD_{50} and (ADI) through the oral administration for 30, 60 and 90 days. Pesticides were given twice dose weekly.

Sampling: After completion of the treatment period each group were sacrificed by cervical dislocation, the rats were decapitated and liver, kidney, brain, and spleen were removed immediately, washed with sodium phosphate buffer (pH 7.4), histopathological samples were fixed in 10 % neutral buffered formalin and stored at 4°C for histopathological examination.

Histopathological studies: The samples were removed and placed in fresh fixative solution, washed in a running tap water overnight, dehydrated in ascending grades of alcohol, cleared in xylol. Fixed tissue samples were processed routinely by paraffin embedding technique. Liquefied para film, (melting point between 55°C and 60°C) for one and a half hours. After solidification of Para film, wax blocks were cut at section of 5.5 um in

thickness, trimmed with rotary microtome, and every eight sections were collected on slides and stained with haematoxylin and eosin**.**

Staining method: The section were placed in descending grades of alcohol and rinsed in distilled water. The sections were stained in haematoxylin for 1/2 min., and then placed in tap water for 3-5 min., counter staining was done in 1 % solution of eosin for 1 min., followed by washing in distilled water. The sections were dehydrated, cleared in xylol and mounted in Canada balsam. The resulting sections covered with cover slides to be ready for microscopically examination. However, silver impregnation technique was used to show the granules in cytoplasm of carcinoid tumer cells, and distinguish this tumer from cholangiocarcinoma Drury *et al.,* (1980).

RESULTS AND DISCUSSION

Pathological finding in Liver: The liver of rats which sacrified after one month revealed, congestion of hepatic blood vessels and sinusoids (Fig. 1). Other cases revealed congethion of hepatic blood vessels and vacular degeneration of hepatic cells with nuelear changes (Fig. 2). The portal areas showed congestion of blood vessels and aggregation of lymphocytes around the blood vessels and hyperplastic bile ducts proleferation (Fig. 3). After two months, vacular and hydropic degeneration together with focal necrosis of some hepatocytes were noticed in addition to the previously mentioned vacuolation of the hepatic cells, some of cells showed fatty changes with nuclear changes (Fig. 4). After three month, congestion of portal blood vessels and extensive aggregation of lymphocytes together with necrosis of hepatic cells and hyperplasia of the bile ducts were seen (Fig. 5). In some cases the lesion were diffused and the hepatic cells showed dissociation and disorganization with increase miotic activity in the form of condensed chromatics, enlarged and double nuclei in single hepatic cells were recorded (Fig. 6).

Besides to the previously mentioned lesions. The portal areas showed neoplastic cells orginate from the cells of bile ducts epithelium (Fig. 7), and composed from uniform cells with hyperchromatic round or oval nuclei (Fig. 8). The cells were arranged in small group. The cells within the group oriented toward the basemembrane, this type of tumour called liver carcinoma (carlcinoid tumour of liver), silver impregnation was used to show the granules in cytoplasm of carcinoid tumour cells, and distinguish this tumour from cholangiocarcinoma (Fig. 9,10). In addition to the liver carcinoid, other cases showed the hepatic cells under the Glesson's capsule were enlarged (hepatocytomegaly) with disorganization of hepatic architecture and the cells were mostly adjacent to each other (Fig. 11). These changes were mostly subcapsular. The hepatic cells showed polymorphism in its shape and size and the nucleus were enlarged with typical or a typical miotic activities (hepatic carcinoma).

Liver suffered from necrosis after treatment with lambda-cyhalothrin as a toixc materail reached to the liver via the gastro intestinal tract blood supply, therefore, the necrosed aresa mainly appeared around portal tract.

Also, inflammatory cells were aggregated in portal tracts and present as differential foci in the liver parenchyma. They act as a defence mechanism due to irritation of toxic material and necrosed tisse for the same reason the kupfer cells were activated (Abd-Allah, 1987). In high dose of pesticides subcapsular haemorrhage was observed in the liver of the treated albino mice. This ocurred due to damge of endotheliallining of blood vessels by the tested insecticides, with chronic intoxication of the cytoplasm near the nucleus.

Liver lession were observed by many investigator (Chu *et al*., 1986 and Abd-Allah, 1987) who noted that liver suffered from severe lesions after treating the experimental animals with some pesticides. Moreover, haemorrhage was evident intertubular or subcapsular, this happened as a sequale of liver lessions which leading to lack of clotting factors. Also, observed severs toxicicty led to necrosis of renal tubules which were replaced with inflammatory cells. This findings were confirmed with results of (Gupta *et al.,* 1981 and Kehrer *et al.,* 1986).

Pathological finding in Kidney: The kidney of the sacrified rats after one month showed congestion of renal blood vessels in both cortex and nedulla together with perivascular edema (Fig. 12). Perivascular and periglomerular infiltration of lymphocytes and macrophages were noticed (Fig. 13). Shrinkage of some glomerular tuft due to edema of Bowman's capsule (Fig. 14). Cystic dilatation of some renal tubules (Fig. 15), degeneration and coagulative necrosis in other renal tubules were also detected . After two months the kidney of scarificed rats showed wide spread lesions represented by congestion of renal blood vessels together with interstetial hemorrhage. Aggregation of lymphocytes perivascular, perigloneular and among the degenerated renal tubuls were detected (Fig. 16). Some renal tubules showed hyoline and cellular casts (Fig. 17). After three months the kidneys of scarified reats revealed that severe wide spread congestion of renal blood vessels with perivascular edema and heamorrhages (Fig. 18). Shrinkage of large number of glomeruli together with perivascular, periglomerular and interstitial aggregation of lymphocytes were also seen. Large number of renal tubules showed cystic diltation which lined by flat epilhelium (Fig. 19). Some slides showed focal medulary hemorrhages and edema, sever cangested glomerular tuft and few round cells could be observed (Fig. 20).

The glomerular tubles of the kidney were vaculated due to edema, with excessive toxicity concentration and destruction of the glomerular tubules occurred which may be due degenerative changes. Degeneration of renal tubules resulted from collection of albuminous material lining during its excretion in the urine (Chu *et al.,*1986, and Nebbia and Fogliato, 1987). Methyl parathion exposure caused glomerular atrophy and vascular dilatation, and after 7 weeks, necrosis and edema were observed in the kidney tissues (Suna Kalender *et al.,* 2007).

Pathological finding in Spleen: Histopathological changes of the spleen of sacrified rats after one month showed hyperplasia of the lymphocytes of the white pulp together with infiltration of the red pulp with lymphocytes Fig. 21). After two months the spleen showed congestion of splenic sinusoids and heamorrhages with depletion of lymphocytes in white pulp (Fig. 22). After

three months the spleen showed depletion of lymphocytic elements (Fig. 23). Extensive proleferation of reticuloendothelial cells mainly macrophages (Fig. 24), other cases showed increasing of the number of megaterrocytes with extensive heamorrrhages (Fig. 25), and haemosiderosis (Fig. 26). The toxic effect of lambda-cyhalothrin on hepatic lession leading to congestion and hemorrhage of spleen. Also lymphocytes occurred, which may be affected the immunity.

Pathological finding in Brain: The brain of rats which sacrificed after one month showed congestion of meningeal blood vessels in addetion to lymphocytic aggregation (Fig. 27). Degenerative changes of the nerve fibers represented by deep eosinophilic stain and fragmentation. Also necrotic changes of some neurons (Fig. 28), and pericellular and perivascular edema (Fig. 29) were detected. In addation to demylenation of nerve fiber (Fig. 30), and focal areas of encephalomalacia were seen (Fig. 31). After two moths, wide spread area of encephalomalacia in cerebrum could be noticed (Fig. 32). Neuronal degeneration, neuronophagia and satillitosis (Fig. 33), and focal aggregation of microglial cells (focal gliosis) (Fig. 34) were observed. Degeneration and necrosis of purkinje cells of cerebellum (Fig. 35), together with pyknosis or karryolysis of the nuclei (Fig. 36) were seen. After three months the lesions in the brain were as the previously mentioned lesions but wide spread and more severe as aggregation of lymphocytes among the nerve fibers.

In conclusion, histopathological examination revealed vascular congestion, hydrophic degeneration and leukocyte infiltration in the affected organs at the initial stages. At the terminal stage of toxics, coagulative necrosis, perivascular/periductal fibro cellular reaction along with mononuclear cellular infiltration in the liver, mucosal eruptions with inflammatory reaction in the gastrointestinal tract and hyalinization of the tubular epithelium of the kidneys were observed. High degree of changes was found at high dose (treatment 1/10 LD_{50} was more effective in changes than 1/40 LD_{50} treatment), while it was not able to observe any significant changes at low dose (ADI), it was mean (LCT) caused dose dependent, and induced histological aspects of liver which was the most affected organ (Luty *et al.,* 1998).

Liver was the most affected organ followed by the stomach, intestine, kidney, spleen, and brain. The liver showed excessive hepatocellular damage. Also vaculated nuclei were evident periportal coagulation necrosis as well as necrosis distributed throught the liver. The hepatic cells showed polymorphism in its shape and size and the nucleus were enlarged with typical or a typical miotic activities (hepatic carcinoma). The renal tubules suffered fron hyalin degenerate. Infiltrayion of inflammatory cells in between the degenerated renal tubules was observed emphysima and haemorhage. The brain affected by neuron degenerate.

This findings were confirmed with results of (Lakkawar *et al.,* 2004, Kazuhito Yokoyama 2007, and Ferah Sayim *et al.,* 2007) pesticides caused dose related histopathological changes such as mononuclear cell infiltration, congestion, an enlargement of the veins and sinusoids, hepatocellular damage, necrotic changes, an increase in the number of Kupffer cells,

cytoplasmic vacuolization and degeneration in nuclei in the liver of exposed rats. Also, Binukumar B.K. *et al.,* (2010) noted that, dichlorvos caused liver dysfunction.

In addition to the appearance of these cytotoxic lesions, there were hyperphostic lession in parenchymal cells. Bannash *et al.,* (1982) described the foci of cellular alteration as proliferative lesions prossessing an increased miotic index and postulated that they appear regularly in the early stage of hepatocarcinogensis. According to Gopinath *et al.,* (1987) necrosis is a morphological changes associated with death of liver cells whilst still part of liver tissues is viable,. Toxic liver necrosis is viewed as a disorder in the control of intracellular calcium homeostasis. The ability and oval cell proliferation were described also by Bulter *et al.,* (1981), Gopinath et al., (1987) reported that the origin of oval cells is a much discussed subject, with some reports proposing bile ducts, while others sugegested stem cells, with the ability to differentiate into transitional forms and hepatocytes. Solivan and Krieger (1992) concluded that the mechanism of tumor production has not been determined. Carcinogenesis seems to involve a two step process of induction, followed by a long latent period during which neoplasm develops. Most chemical carcinogens act as initiators by causing structural damage to DNA. Balli *et al.,* (1996) pesticides induced histopathological changes in the liver, kidney and brain such as necrosis, congestion, increase of the mitotic activity, enlargement of the sinusoids and polymorphisms of the hepatocytes, were detected in liver tissues for both exposure groups.

Also, Piramanayagam *et al.,* (1996) stated that, malathion included congestion and hemorrhage in the liver, kidneys, brain, lungs and epicardium, and hyperemia of the kidney, spleen brain and lung. Other changes were micro granuloma formation in the liver, kidney and lungs; lymphoid depletion with reticular cell hyperplasia in the spleen; focal edema, per vascular cuffing and neuronal degeneration in the brain. Sahu C. R, and Ghatak S. (2002) Showed abnormal features in the formation of different vital organs, the liver and kidney were severely affected by the dimecron. Dermal application of deltamethrin in the rat, nerve cell lossed Seyed Khosrow Tayebati *et al.,* (2009).

On the other hand our results disagree with, Tos-Luty *et al.,* (2003) noted that dermal application of malathion, in a small dose did not cause histopathological changes in the liver, kidneys, heart and lungs of the animals, while the administration of a higher dose resulted in changes only in the liver. Also, dermal application of alpha-cypermethrin was assessed in rats; the preparation resulted in slight histological changes in liver, kidney, lung and brain Luty *et al.,* (1998).

REFERENCES

Abd-Allah G.H. (1987): Effect of dimetoate and decamethrin on reproductive performance an on some haematobiochenical characteristics in male rabbits. Ph.D. Thesis, Fac. of Agric, Alexandria Univ.

Ansari and Kumar (1987a): Malathion toxicity. Pathological changes in the liver of zebra fish, *Brachidanio rerio* (cyprinidae), Bol. Fisiol. Anim. Univ. S. Paulo 11: 27-34.

Balli S., Yuksel E., Ozmen M., (1996): Cytogenetic and clinicopathologic changes associated with exposure of male Baladi goat to low dose of organophosphorus pesticide selecron. Regulatory Toxicology and Pharmacology, 6(4): 416-421.

Bannash P., Morre F., Klinek and Zerban H., (1982): Biological makers of pre-neoplastic foci and neoplastic nodules in rodent liver. Toxicol. Pathol. 10:19-36.

Beutler, E., Duron, O., and Kelly M. B., (1981): Improved method for the deyermination of blood glutathione. J. Lab. & Clin. Med. 61: 882-888.

Binukumar B.K., Amanjit Bal, Ramesh kandimalla, Aditya Sunkaria and Kiran Dip Gill (2010): Mitochondrial energy metabolism impairment and liver dysfunction following chronic exposure to dichlorvos. Toxicology, Vol. 270, PP. 77-84

Chu I.D. C., Villeneeuve C. W., Sun V., Secours B., procter E., Arnold D., Clegg L., Reynolds and Valli V. E. (1986): Toxicity of toxaphene in the rat and beagle dog. Fund. Appl. Toxicol. 7 (3): 406-418.

Drury R. A. B., and Wallington E. A., (1980): Evaluation of oxidative stress responses and neurotoxicity potential of diazinon in different tissues of Cyprinus carpio. Environmental Toxicology and Pharmacology, 23: 48-55

Fanta (1997b): Organophosphorus: Bone para an agricultural, runis para os peixes. Germinis Bolm. Inf, Conis. Fed. Biol. (1987): 3-4.

FAO/WHO (1981): Evaluation of some pesticides in food. WHO Food Additive Serious No, 42. World Health Organization Geneva.

Ferah Sayim (2007): Dimethoate induced biochemical and histopathological changes in the liver of rat's.Experimental and Toxicologic Pathology, 59: 237-243.

Gopinath, C., Prentice D.E., and Lewis D.J. (1987): Atlas of Experimental. Toxicological Pathology. 13 of Current Histopathology. 77-90 MTP Press Ltd. Lancater, Boston. The Haque Dordrecht.

Gupta R. C., Singh N., Paul B. S. and Kwatra M. S. (1981): Role of residual estimation and clinic-biochemical and pathological changes in diagnosis of toxicity in bubals caused by malathion. Indian. J. Anim. Sci. 51 (6): 616-622.

Johns S., Kale M., Rathore N., Bhamagar D., (2001): Protective effect of Vitamin F in directorate and malathion induced oxidative stress in rat erythrocytes. J. Nutr. Biochem. 12, 500-504.

Kappers W.A., Fdwards R.J., Murray S., Boobis A.R. (2001): Diazinon is activated by CYP2C19 in human liver. Toxicol, Appl Pharmacol, 177, 68-76.

Kehrer J.P., Klenin-Szanto A. P., Thurston D. E., Lindenschmidt R. C., and Wotschi H. R. (1986): O, S, S, trimethyl phosphorodithioate induced lung damage in rats and mice. J. Toxicol. Appl. Pharmacol. 84: 480-492.

Keizer J., Agostino G., Nagel R., Volpe T., Gnemi P., Vitrozzi L., (1995): Enzymological differences of AchE and diaznon hepatic metabolism correlation of in vitro data with the selective toxicity of diazinon to fish species, Sci. Total Environ. 171, 213-220.

Lakkawar A. W., Chattopadhyay S. K. Somvanshi R. (2004): Experimental cypermethrin toxicity in rabbits - a clinical and patho-anatomical study. Medical and Veterinary Entomology, 85:187-191.

Lopes P.A., Pmheiro T., Santos M.C., Mathias M.L., Coltares-Pereira M.J., Viegars-Crespo A.M. (2001): Response of antioxidant enzyme in fresh water fish populations (Leucalburnoides complex) to inorganic pollutants exposure, Sci., Total Environ, 280, 153-163.

Luty S., Latuszynska J., Halliop J., Tochman A., Obuchowska D., Przylepa, E. Korczak E. (1998): Lymphocyte DNA damage in rats exposed to pyrethroids: effect of supplementation with Vitamins E and C. Toxicology, 203: 17-26.

Mdegela R., Myburgli J., Correia D., Braathen M., Ejobi F., Botha C., Sandvik M., and Skaare J. U. (2006): Evaluation of the gill filament-based EROD assay in African sharp tooth catfish (Clarus gariepinus) as a monitoring tool for waterborne PAH-rype contaminates. Ecotoxicology 15, 51-59.

Minier C., Abarnou A., Jaouen-Madoulet A., and Le Guellec A.M. (2006): A pollution- monitoring pilot study involving contaminant and biomarker measurements in the Seine Estuary, France, using zebra mussels. Environ. Toxicol. Chem. 25, 112-119.

Murty A. S., (1986): Toxicity of pesticides to fish. Handbook of human Toxicology of Pesticides CRC Press, Inc., N.W., Boca Raton, Florida.

Nakagawa Y., and Moore G., (1999): Role of mitochondrial membrane permeability transition in p-hydroxybenzoate ester-induced cytotoxicity in rat hepatocytes, Biochem, Pharmacol, 58, 811-816.

Nebbia C.G., and Fogliato T.G. (1987): Diagnosis of paraquat poisoning in the dog objective. Document Veterinary 8 (6): 49-52.

Piramanayagam S., Manohar B. M., Sundararaj A. (1996): Histopathological changes induced by pesticide in rats. Proceedings of the Academy of Environmental Biology, 3: 119-123.

Rodrigues E. L., and Fanta E. (1998): Liver histopathology of the fish after acute exposure to sub lethal levels of the organophosphate dimethoate 500. Rev. Bras. Zool,15:441-450.

Rudolph P., and Boje R. (1987): Histopathological changes associated with some pesticides. Okotoxicologie, Ecomed, Landsberg (1987).

Sahu C. RX., Ghatak C. R. (2002): Effects of Dimecron on Developing Chick Embryo: Malformations and other Histopathological Changes. Journal of Veterinary Medicine Series C, 31(1):15–20.

Sarabia L., Maurer I., and Bustos-Obregón E. (2008):Melatonin prevents damage elicited by the organophosphorous pesticide diazinon on mouse sperm DNA. Ecotoxicology and Environmental Safety, 72: 663-668.

Sams, C., Cocker, J., and lennar, M.S., (2003): Metabolism of chlorpyrifos and diazinon in human liver microsomes. Toxicol. Let 144, 146.

Seyed Khosrow Tayebati, Maria Antonietta Di Tullio, Alberto Ricci and Francesco Amenta (2009): Influence of dermal exposure to the pyrethroid insecticide deltamethrin on rat brain microanatomy and cholinergic/dopaminergic neurochemistry. Brain Research, 1301: 180-188.

Silva H.G.S., Medina, E. Fanta and Bacila, M., (1993): Sub-lethal effects of the organophosphate Folidol 600 (methyl parathion) on Callichthys callichthys (Pisces, Teleosteri). Comp. Biochem. Physiology. C 105: 197-201.

Solivan, P. K. and Krieger S., (1992): Induction of preneoplastic liver lesions in mice by a single prenatal exposure to aflatoxin. Indian Journal of Verterinary-Pathology, 16 (2): 124.

Suna Kalender, Yusuf Kalender, Dilek Durak, Ayse Ogutcu, Meltem Uzunhisarcikli, Bekir Sitki Cevrimli and Murat Yildirim (2007): Methyl parathion induced nephrotoxicity in male rats and protective role of vitamins C and E. Pesticide Biochemistry and Physiology, 88:213-218.

Torre, F.R., Fenari, L., Salibian, A., (2005): Biomarkers of a native fish application to the water toxicity assessment of a peri-urban poliuted river of Argentina, Chemosphere, 59: 577-583.

Tos-Luty, S. Obuchowska-Przebirowska, D. Latuszynska, J. Tokarska-Rodak, M. Haratym-Maj, A. (2003): Toxicity of dermally applied alpha-cypermethrin in rats. Toxicology Letters, . 104: 111-116.

دراسات هستوباثولوجية لاختبار قدرة مبيد اللامباداسيهالوثرين على أستحداث ألاورام السرطانية

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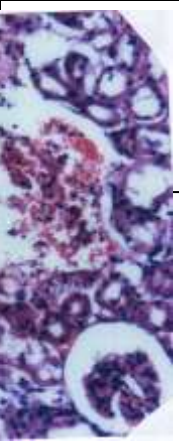
**** قسم المبيدات - كلية الزراعة - جامعة المنصورة**

يؤدى زيادة أستخدام المبيدات الى مشاكل بيئية وصحية خطيرة للانسان والحيوان . وتكمن الخطورة فى أن بعض المبيدات الحشرية لها القدرة على أحداث تلف واضرار بالمادة الوراثيه للخلايا الحية بصورة مباشرة أو غير مباشرة. وتختص الدراسة الحالية فى أختبار ثلاث جرعات من مبيد حشرى من مجموعة البيروثريدات وهو مبيد اللامباداسيهالوثرين على ذكور الفئران البيضاء.

وقد أجرى هذا البحث لدراسة:- الضرر الخلوى فى الخلايا الجسدية والمصاحبة لنمو الأورام ودراسة القدرة على أستحداث الأورام السرطانية فى الانسجة وذلك بدراسة الخصائص الهستولوجية المرضية فى خلايا الكبد، الكليه، المخ، والطحال فى ذكور الفئران البيضاء.



وقد أستخدم فى دراسة هذا الاختبار120 فأر من ذكور الفئران البيضاء حيث قسمت عشوائيا الى 4 مجموعات متساوية (1 مجموعه كنترول + 3 مجموعات للمبيد تحت ألاختبار) وخصص لكل مجموعه 30 فأر. وأجريت المعاملة بتجريع الفئران عن طريق الفم بجرعات 10/1، 40/1 من الجرعة المميتة النصفية LD_{50}، (ADI) لمبيد اللامباداسيهالوثرين ولمدة 30، 60، 90 يوم مع الاستمرار فى المعاملة مرتين أسبوعيا.

وقد أوضحت النتائج المتحصل عليها ما يلى :

أدت المعاملة بالمبيد تحت ألاختبار الى حدوث زيادة شديدة فى تكسر الخلايا الكبدية مع تحلل وتكسر فى النواة وحدوث تجويف وتقعر بين الخلايا الكبدية وظهور فجوات داخل الانوية، وكذلك زيادة فى عدد وحجم الخلايا الكبدية مع ظهور الخلايا السرطانية بشكل وأضح، وأيضا أنتشار الخلايا ألالتهامية فى المنطقة البابية، مع أحتقان فى ألاوعية الدموية وتجمع خلايا الدم حول ألاوعية الدموية والقنوات الصفراوية.

أما فى الكلية فقد حدث أنزفة دموية مع تجمعات الخلايا حول الاوعية الدموية وحول مصفاة الكلية مع أرتشاح مائى فى محفظة بومان، مع أنتشار ألاوديما، وكذلك موت وتحلل الانوية فى الخلايا حول القنوات البولية.

وفى خلايا الطحال وجد أحتقان وأنزفة مع أرتشاح مائى وأيضا زيادة عدد الخلايا فى منطقة اللب ألابيض, مع ظهور الفجوات بين الخلايا.

وفى المخ ظهرت الانزفة الدموية مع زيادة فى عدد خلايا الدم مع أرتشاح مائى حول ألاوعية وكذلك أحتقان فى أغشية المخ ، وتجمع خلايا الدم حول ألاعصاب والنسيج العصبى وكذلك ظهور بؤر واسعة حول العصب

قام بتحكيم البحث

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أ. د/ محمد ابراهيم عبد المجيد — كلية الزراعة – جامعة عين شمس