DETECTING THE EFFECT OF MALATHION AND IMIDACLOPRID PESTICIDES BY CYTOGENETIC AND HISTOPATHOLOGIC ASSAYS USING LABORATORY MICE

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ABSTRACT

Pesticides are a source of potential risks to the environment and in all parts of the world. Acute poisoning with pesticides has become a major public health problem. The widespread use of pesticides and genetic risks caused by indicating that should be evaluated by using its genotoxicity tests available which include examination of micronucleus formation and mitotic index assay. Cytogenetic changes are among the indicators used widely for the effects of biological factors associated with breaking down the genetic material (DNA). In this research used two types of pesticides that used in agriculture in our country which are malathion and imidacloprid bystudying the cytogenetic and histopathological effects as biological indicator for the detection of environmental pollution with pesticides using laboratory mice by exposing to two concentrations of the pesticide malathion (10 and 20 mg / ml) and pesticide imidacloprid (2.5 and 5 mg / ml) based on the half lethal dose for half (LD_{50}%), which was for malathion (30 mg / 25gm) and for imidacloprid (6 mg /25gm) by oral administration and made cytogenetic and histopathological assay then compared with the control groups that exposed to tap water only. The results showed decreasing in the mitotic index and increasing in micronucleus formation in both concentrations of both pesticides for periods (14 and 30 days) compared with the control groups. Moreover,imidacloprid (5mg/ml) appeared to be was more effective after 30 days of treatment. Furthermore, there is histopathological changes in the liver, kidney and stomach after treatment with both pesticides as inflammatory response against toxic effect of pesticides.

INTRODUCTION

Pesticides have human health concerns, because they are toxic substances, widely released into the environment and major cause of morbidity and mortality especially in developing countries. In addition to their effects, are sometimes found to affect nontarget organisms, including humans (10,11).As increase use of various pesticide compounds, large amounts of these chemical compounds have therefore been released into the environment and affect public health (16).

Malathion is a commonly used organophosphorus insecticide. It has been employed in major eradication programs (9). Toxicity of malathion based on its metabolites and depends on the product purity, route of exposure, amount of protein in the diet and gender. Metabolism of marathon, the metabolites produced by the oxidation of Malaoxon in mammals, insects and plants, is the primary source of malathion's toxicity and it is 40 times more acutely toxic than marathon (12). According to the World Health Organization (WHO), the occurrence of pesticide-related poisonings has doubled over the past ten years due to their widespread use. Symptoms of marathon toxicity include numbness,

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tingling sensation, headache, dizziness, difficulty breathing, weakness, irritation of skin, exacerbation of asthma, abdominal cramps, along with other related symptoms (19).The mechanisms of toxicity in organophosphorus compounds are due to their ability to inhibit cholinesterase enzyme. Malathion binds with acetylcholinesterase (AchE),which is responsible for normal nerve function. AchE inhibition leads to the accumulation of acetylcholine which in turn results in stimulation of cholinergic, muscarinic, and nicotinic receptors leading to common signs and symptoms seen in malathion poisoning (1,25).

Imidacloprid is a new neonicotinoid insecticide which has become an important pest control agent on many crops. The neonicotinoid insecticides are related to nicotine in their structure and action at the nicotinic acetylcholine receptor (23). As the potential environmental impact connected with the introduction and heavy use of Imidacloprid is an agonist at the nicotinic acetylcholine receptor, and as such it is highly effective against many sucking insects (13).It causes a blockage in a type of neuronal pathway that is more abundant in insects than in warm-blooded animals. This blockage leads to the accumulation of acetylcholine, resulting in the insect's paralysis, and eventually death (17). As the use of insecticides has become increasingly widespread throughout the world, additional studies are needed to evaluate the potential toxic risk of insecticides for non-target organisms.

Genotoxicity of pesticides for non-target organisms and their influence on ecosystems are of worldwide concern .Studies for genotoxicity have indicated that organophosphate compounds, induce DNA damage. Damage occurs in the form of chromosomal aberrations, mitotic index, and sister chromatid exchanges in both human and animal studies (18). Research performed using genotoxic evaluation in populations with pesticides exposure have clearly indicated a positive genotoxic effect in human blood lymphocytes. Significantly high frequencies of chromosome aberrations have been found in a group of pesticide workers compared to an unexposed control group (24).Pesticides also show organ-specific relationship between its distributions in the internal organs. The greatest accumulation of the pesticide in the liver and kidneys was noted after intravenous administration, followed by an oral and dermal application (8). This study is designed to evaluate the cytogenetic and histopathologic effects of pesticides (malathion and imidacloprid)on the experimental albino mice.

MATERIALS AND METHODS

1- Tested animals: Albino Swiss mice (*Musmusculs*) from the International Center For Medical Monitoring and Researches were acclimatized under laboratory conditions in Al-Nahrein Center For Biotechnology Research and their weight was 23-27 gram. They were divided into 10 groups and 8 mice in each group,each group was kept in a separate plastic cage. The animals were maintained at a temperature of 20-25 °C, and they had free excess to food (standard pellets and water during all experiments).

2- Pesticides and dosing: Two types of pesticides widely used in Iraq which are malathion and imidaclopride were used. Two concentrations of malathion(10 and 20mg / ml) and imidacloprid (2.5 and 5 mg / ml) were taken based on the lethal dose for half (LD_{50}%) , which was for malathion (30 mg / 25gm) and imidacloprid (6 mg /25gm) using orally administration for periods of (14 and 30 days) . Four groups of mice for each pesticide and two groups as control which was taken tap water for the same periods.

3-Cytogenetic study

3-1 Mitotic index assay: Experimental animals were injected (i.p.) with colchicine (4mg/kg) 1.5h prior to sacrifice. Animal sacrifice was performed using a chamber. Both femora were dissected out and cleaned of any adhering muscle. Bone-marrow cells were collected from both femora by flushing KCl (0.075M, at 37ºC), and incubated at 37ºC for 25 min. Collected cells were centrifuged at 2000 × g for 10 min, and fixed in fixative solution (acetic acid: methanol, 1:3, v/v). Centrifugation and fixation were repeated five times at an interval of 20min. The cells were resuspendedin a small volume of fixative, dropped onto chilled slides, and allowed to dry. They were stained the following day with freshly prepared 2% Giemsa stain for 3–5 min, and washed in distilled water to remove excess stain (2).Mitotic Index (MI %) was calculated as follows: MI % = (number of divided cells/total number of cells counted) x 100.

3-2 Micronucleus test: The frequency of micronucleated erythrocytes was evaluated based on a technique developed by (26).The animals were sacrificed by cervical dislocation. The femoral bone marrow was flushed out using 1 ml of fetal calf serum and centrifuged at 2000 *g* for 10 min. The supernatant was discarded. Smears were prepared for each animal, fixed in methanol and stained with 5% Giemsa in Sorensen buffer. Smears were screened at magnification of 1000X, using a light microscope. The erythrocytes with one or more micronuclei were counted in at least 2000 erythrocytes per animal. Micronucleus frequency (MN%) was calculated as follows: MN%=(number of cells containing micronucleus/total number of cells counted) x 100.

4- Histological examination: After sacrificed animals, liver, kidney and stomach were carefully separated and washed by distilled water. Small pieces of organs were fixed in 10% neutral buffer formalin and in Bowan's fixative. The fixed samples were dehydrated in ethyl alcohol (70 ,80,90,100 and100%) each one for 1hr and xylene for 1/2 hr, processed and embedded in paraffin blocks. Sections of 5 – 7 µ were prepared and stained with heamatoxelin and eosin (5).

5- Statistical analysis: Statistical significance between the control and experimental data were subjected to analysis by (SPSS) version (16).

RESULTS AND DISCUSSION

Mitotic Index

In the present study, evaluation of the genotoxic potential of malathion and imidacloprid were illustrated by using mitotic index (MI), and DNA damage as toxicological endpoints. The results indicated significant increases in the cytogenetic damage in the bone marrow cells associated with exposure to these pesticides orally were significantly different compared to the control groups.

Mitotic index significantly decreased with both concentrations of both pesticides. The mitotic index is defined as the proportion of cells undergoing mitosis (cell division) compared to the total number of cells. Hence, the mitotic index is very critical in determining the rate of cell division. Significant decreases in the mean of MI values in both concentrations of both pesticides especially after 30 days at 20 and 5 mg/ml of malathion and imidacloprid respectively were

7.00 ± 0.316% and 6.64 ± 0.365% respectively compared to the control group (Table 1) , Fig. (1).

Table 1: Mitotic in albino mice after treatment with pesticides

Group	Dose mg/ml	MI% 14 days	MI% 30 days
Control		13.40 ± 0.484a	13.00 ± 0.316a
Malathion	10	9.32 ± 0.365b	8.64 ± 0.658b
Malathion	20	8.64 ± 0.183b	7.00 ± 0.316c
Imidacloprid	2.5	9.00 ± 0.316b	7.64 ± 0.365c
Imidacloprid	5	8.00 ± 0.316b	6.64 ± 0.365c

*Different letters: significant difference (p≤ 0.05) between means ;*Values are presented as means ± S.E.

Micronuclei consist of DNA material, which is lost from the cell nucleus during mitosis. They are generated by chromosomal breakage or by dysfunction of the spindle apparatus.Imidacloprid and malathion induced increases in micronucleus frequency (Table 2). Both doses of these pesticides significantly increased the micronucleus frequency but imidacloprid (5 mg/ml) was more effective as the result illustrated in Table 2 (2.70 ± 0.20% and 2.82 ± 0.241% after 14 and 30 days respectively)when compared to the control group. The induction of micronucleated erythrocytes due to exposure to both imidacloprid and malathion indicates that they have a potential for clastogenicity.

Table 2: Micronucleus formationin albino mice after treatment with pesticides

Group	Dose mg/ml	MN% 14 days	MN% 30 days
Control		0.82 ± 0.489a	0.90 ± 0.00a
Malathion	10	1.40 ± 0.144b	1.24 ± 0.081b
Malathion	20	1.44 ± 0.174b	2.14 ± 0.329c
Imidacloprid	2.5	1.74 ± 0.081b	1.94 ± 0.174b
Imidacloprid	5	2.70 ± 0.20c	2.82 ± 0.241c

* Different letters: significant difference (p≤ 0.05) between means; *Values are presented as means ± S.E.

The reported genotoxicity of malathion might, therefore be a consequence of its metabolic biotransformation to malaoxon, formed by oxidation or the presence of malaoxon or isomalathion, formed by isomerization, as well as other unspecified impurities in commercial formulations of malathion (6). Impurities of malathion and imidacloprid such as isomalathion and various trimethylphosphorothioate esters, present in the technical grade or formed during storage, can induced toxicity and are responsible for other effects, including DNA lesions (7,15).

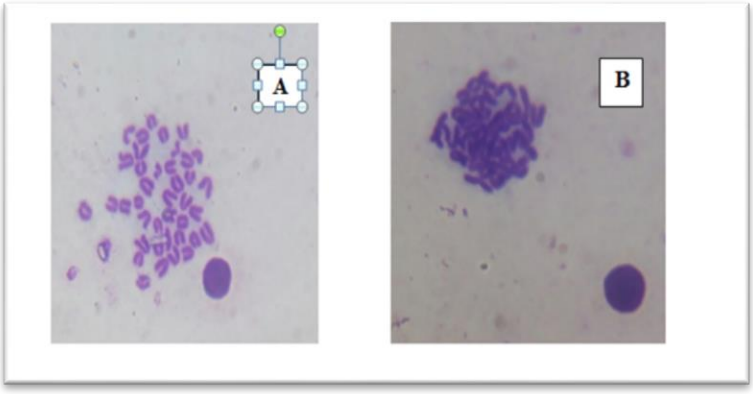

Fig.1: Mitotic Index of albino mice bone marrow (100X) treated by (A) Malathion and (B) Imidacloprid

Histological Test

Table 3: Histopathological changes in albino mice after treatment with alathion

14 Days		
Organ	**Malathion 10 mg/ml**	**Malathion 20 mg/ml**
Liver	**Necrosis in some parts of tissue and seem to be lysis (karyolysis),single cell necrosis ,congestion of blood vessels and cuffing with inflammatory cells.**	**Necrosis of some liver cells and increase kuphercell, congestion of central vein ,inflammatory cell infiltration cuffing wall of blood vessels with chronic inflammatory cells. In some part of liver degenerative changes cells and hyper plasia of epithelial cells lining bile duct.**
Kidney	**Degenerative changes in epithelial cells lining renal tubules,haemorhage in the interstitial tissue with inflammatory cells.**	**Degenerative changes in epithelial cells lining renal tubules,infiltration of inflammatory cells ,enlargement of some glomeruli, hemorrhage and congestion of blood vessels.**
Stomach	**Metaplastic changes in epithelial cells layer, alter to keratinized layers and some parts show sluphing of epithelial layer.**	**Metaplastic changes in epithelial cells layer , alter to keratinized layers,some parts show sluphing of epithelial layer**
30 Days		
Liver	**Degenerative changes represented by pyknotic neucleus, single cell necrosis, cuffing the wall of blood vessels within chronic inflammatory cells.**	**Single cell necrosis, congestion and inflammatory cuffing of blood vessels, pyknotic nucleus in some part of liver tissue.**
Kidney	**Shrinkage in some glumerul, increase number of mesengeal cells, infiltration of chronic inflammatory cells,congestion of some glomerule, haemorrhage in interstitial tissue and odema in the renal tubules.**	**Degenerative changes represented by vacuolization of cells lining renal tubules, congestion of blood vessles haemorrhage in interstitial tissue, chronic inflammatory infiltration, singl cell necrosis of cells lining renal tubule represented by pyknotic nucleus, necrosis and shrinkage of some renal glomeruli.**
Stomach	**Metaplastic changes in epithelial cells layer, alter to keratinized layers,some parts show sluphingof epithelial layer.**	**Metaplastic changes in epithelial cells layer, alter to keratinized layers, some parts show sluphingof epithelial layer.**

Table 4 :Histopathological changes in albino mice after treatment with Imidacloprid

14 Days		
Organ	**Imidacloprid 2.5 mg/ml**	**Imidacloprid 5 mg/ml**
Liver	**Aggregation of chronic inflammatory cells,congestion and cuffing of blood vessels. Degenerative changes near portal area represented by pyknotic of nucleus and single cell necrosis.**	**Single cell necrosis, aggregation of chronic inflammatory cells, degenerative changes include pyknotic of nuclei, congestion of blood vessels and increasing in kupher cells.**
Kidney	**Degenerative changes in epithelial cells lining renal tubule, infiltration of inflammatory cells,enlargement of some glomeruli, haemorrhage and congestion of blood vessels.**	**Degenerative changes in epithelial cells lining renal tubules, haemorrhag in interstitial tissue, infiltration of inflammatory cells and shrinkage of some glomeruli.**
Stomach	**Metaplastic changes in epithelial cells layer , alter to keratinized layers,some parts show sluphing of epithelial layer.**	**Metaplastic changes in epithelial cells layer, alter to keratinized layers,some parts show sluphing of epithelial layer.**
30 Days		
Liver	**Degenerative changes represented by pyknotic nucleus, single cell necrosis, cuffing the wall of blood vessels within chronic inflammatory cells.**	**Single cell necrosis, aggregation of chronic inflammatory cells, congestion of blood vessels, increasing cell division, cuffing the blood vessels with necrosis and degenerative changes (vaculation of cells)**
Kidney	**Shrinkage in some glumeruli, increase number of mesengeal cells, infiltration of chronic inflammatory cells,congestion of blood vessels and degenerative changes in some renal tubules.**	**Degenerative changes in epithelial cells lining renal tubules, chronic inflammatory cell infiltration, patch of fatty degeneration in some parts of renal tissue, necrosis in glomeruli and increasing in number of mesengeal cells.**
Stomach	**Metaplastic changes in epithelial cells layer, alter to keratinized layers,some parts show sluphing of epithelial layer.**	**Metaplastic changes in epithelial cells layer alter to keratinized layers,some parts show sluphing of epithelial layer.**

Studies with pesticides showed an existing relationship between its distribution in the internal organs and the route of administration. The greatest accumulation of the pesticide in the liver and kidneys was noted after intravenous administration, followed by an oral and dermal application (22). In this study the administration of malathion and imidacloprid was orally caused degenerative changes ,necrosis, chronic inflammatory cells and cuffing of blood vessels of the liver (Fig.2,3,4and 5) in all concentrations after 14 and 30 days but these changes were increasing in malathion 20 mg/ml and imidacloprid 5mg/ml after 30 days compared with control group (Fig.13).As the liver is the natural position for detoxification therefore it is consider as the important organ which affected by the toxin lead to disturbance of it is normal function such as degeneration swelling inflammation ending by necrosis and complete distraction of the cells and this changes depending on intensity of the toxic effect of pesticide (20). Most of organophosphorus pesticides were compound diffused easily cross the hepatocytes membranes and effect on this membranes function and increase its permeability to water and then hepatocytes swelling (3,14).

In the kidney, there were histopathological changes represented by degeneration in epithelial cells lining renal tubules, chronic inflammatory cell infiltration and necrosis were noted (Figs.7,8 and 9) in all concentrations of pesticides but imidacloprid 5 mg/ml after 30 days caused fatty degeneration in

some parts of renal tissue (Fig.6) as compared with control group (Fig.12). Several factors affected the toxicity of the pesticide that interferes with its effecting on renal tissue such as type of pesticide, its concentration and dosage. These toxins lead to district the renal tissue depending on the dosage and mentioned the necrosis in different tissue appeared after the appearance of degeneration (4,21).

In the stomach, the histopathological changes were the same for both pesticides at all concentrations which caused metaplastic changes in epithelial cells layer, alter to keratinized layers and some parts show sluphing of epithelial layer (Fig.11) compared with control group (Fig.10).These changes as a result of orally administration of pesticides and directly affecting the epithelial layer and the toxic effect on the blood vessels increased its permeability and exit the liquid to outside of vessels and induce odema then lead to sluffing the mucous epithelial layer .The effect of organophosphorous pesticides, including malathion, on protein lipid membranes was shown that these compounds change physical and chemical properties of the membranes. According to the authors, this is a manifestation of the toxic effect on cells, because the integrity of protein-lipid membranes ensures the normal functioning of the cells (27).

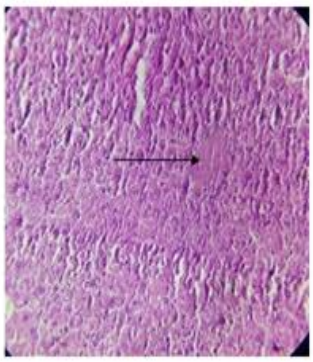

Fig.2: Liver: Necrosis in some part of liver tissue (40X). Malathion 10 mg/ml (14 days)

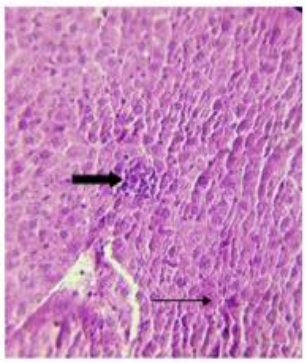

Fig.3 Liver: Pyknotic nuclei → , Aggregation of chronic inflammatory cells and single cell necrosis ➡ (40X). Malathion 10 mg/ ml (30 days)

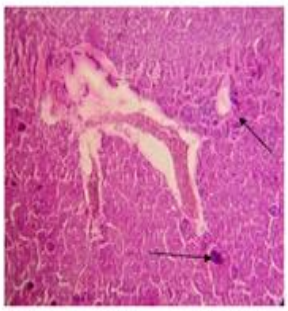

Fig.4:Liver: Cuffing of blood vessels with chronic inflammatory cells (40X). Imidacloprid 2.5 mg/ml (30 days)

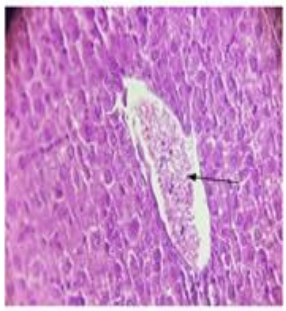

Fig.5: Liver: Congestion of blood vessels (40X). Imidacloprid 5 mg/ml (30 days)

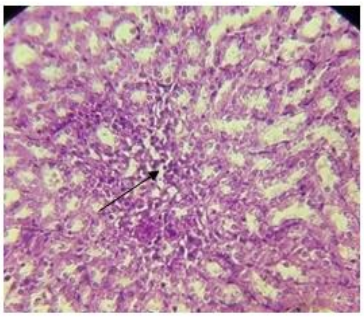

Fig.6 :Kidney: Fatty degeneration in renal tubules (40X).Imidacloprid 5mg/ml(30 days).

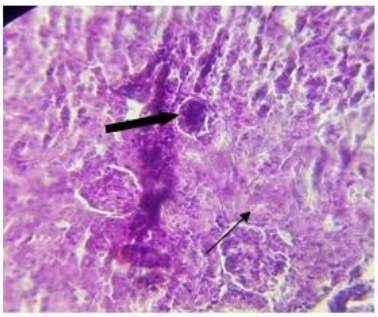

Fig.7: Kidney: Shrinkage of glomeruli ➡ necrosis of epithelial lining renal tubules ⟶ (40X). Imidacloprid 5mgl/ml (30 days).

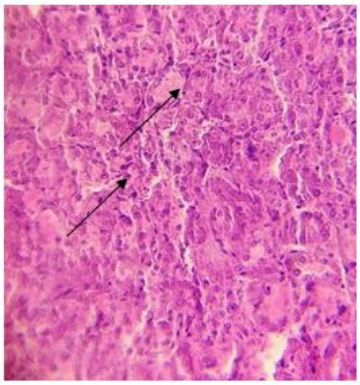

Fig.8: Kidney: Odema with infiltration chronic inflammatory cells (40X). Malathion 10 mg/ml (14 days).

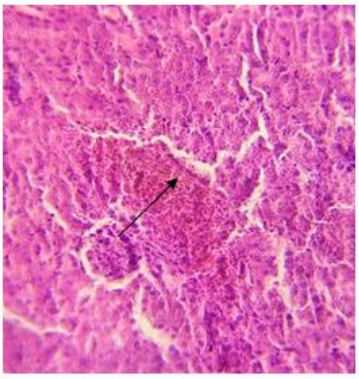

Fig.9:Kidney: Congestion of blood vessels, infiltration of inflammatory cells (40X). Malathion 20 mg/ml (30 days).

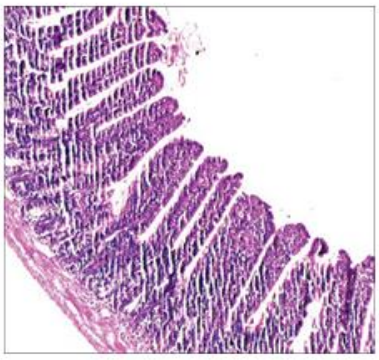

Fig.10: Control of stomach tissue (40X)

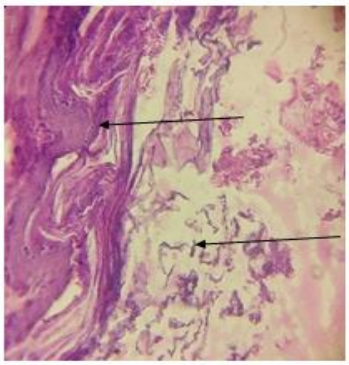

Fig.11:Stomach: metaplasia, stuphing of epithelial lining stomach (40X). Imidacloprid 5mg/ml (30 days).

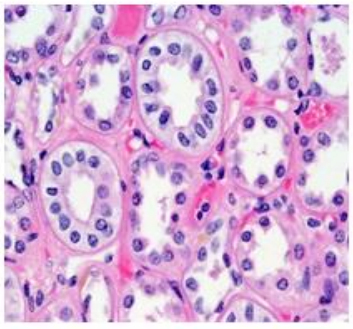

Fig.13: Control of kidney tissue (40X)

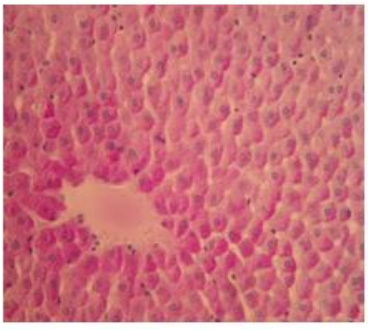

Fig.12:Control of liver tissue (40X)

CONCLUSIONS AND RECOMMENDATIONS

-Malathion and Imidacloprid have genotoxic effects as they decreased mitotic index and increased micronucleus formation which lead to the DNA damage of the cell.

-Malathion and Imidacloprid caused histopathological changes in liver, kidney and stomach as an inflammatory response the toxic effect of these pesticides so they can impair the organs functions.

Recommendations: Further studies are needed to reveal the cytogenetic effects of pesticides in human and used more advanced parameters for histopathological examination.

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الكشف عن تأثير مبيد المالاثيون والاميداكلوبراید في تطبيق التحليلات الوراثية الخلوية والنسيجية المرضية باستخدام الفئران المختبرية

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الملخص

المبيدات هي مصدر للمخاطر المحتملة على البيئة وفي انحاء العالم كافة جميعها فالتسمم الحاد بالمبيدات، اصبح مشكلة صحية عامة رئيسية. يشير انتشار استخدام المبيدات والمخاطر الوراثية التي تسببها الى انه ينبغي تقويم السمية الجينية لها باستخدام الفحوص المتاحة والتي تشمل فحصاً لتكوين النواة الصغيرة ودراسة الانقسام الخلوي. تعد التغييرات الوراثية الخلوية من المؤشرات المستخدمة على نطاق واسع للتأثيرات البايولوجية المرتبطة بعوامل تحطيم المادة الوراثية DNA. في هذا البحث تم استخدام نوعين من المبيدات الحشرية الشائعه الاستعمال في الزراعه داخل بلدنا هما المالاثيون والاميداكلوبراید ودراسة التأثيرات الوراثية الخلوية والنسجية المرضية فيها مؤشراً بايولوجياً للكشف عن التلوث البيئي بالمبيدات باستخدام الحيوانات المختبرية والتي تم تعريضها الى تراكيز من مبيد المالاثيون (10 و20ملغم/مل) ومبيد الاميداكلوبراید (2.5و5ملغم/مل) بالاعتماد على التركيز القاتل للنصف للمالاثيون (30ملغم/25كغم) والاميداكلوبراید (6 ملغم/25كغم) باستخدام طريقة التجريع الفموي ثم مقارنتها مع مجموعة السيطرة المجرعة بالماء الطبيعي فقط وذلك للحصول على قاعدة بيانات يمكن الاستعانة بها مستقبلا لتحديد نوع الملوث الذي يتم التعرض اليه. اظهرت النتائج نقصان في معامل الانقسام الخلوي وزيادة في تكون النوى الصغيرة لكلا التركيزين للمبيدين ولمدتي التعريض 14 و30يوم مقارنه مع مجاميع السيطرة ولكن الاكثر تأثيراً كان مبيد الاميداكلوبراید (5ملغم/مل) للمدة 30يوم. كما اظهرت النتائج تغييرات نسيجية مرضية واضحه في الكبد والكلى والمعدة لكلا المبيدين نتيجة الاستجابة الالتهابية للتأثير السمي في هذين المبيدين.

وزارة العلوم والتكنلوجيا، بغداد، العراق.