Effect of malathion on chromosomes of mice

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Abstract

This work was done to study the cytogenetic effect of malathion on chromosomes of mice. Malathion is one of organophosphorus pesticides which have alkalyting agents affecting the DNA of the living organisms exposed to them. And we also try to use Nigella sativa oil to minimize the hazards induced by Malathion as it considered one of the greatest healing herbs. In experiment 36 mice of both sexes were used. And the effect of malathion and malathion with Nigella sativa on mitotic index, chromosome abnormalities and micronucleus were examined. **And the following results were obtained** Malathion produces a significant increase in chromosomal aberrations while Nigella sativa has some protective effect when given with Malathion in acute treatment either at 24 or 48 hr. Also Administration of malathion decreases the rate of cell division (mitotic index) of mice bone cells of mice. On the other hand Nigella sativa has protective effect against this effect of malathion hr**.** Moreover exposure to Malathion induces a significant increase in micronuclei formation rate. These results suggested that malathion is powerful mutagenic and/or carcinogenic pesticide.

Introduction

Organophosphorus compounds are a great category of pesticides which are known to react generally with DNA and causing its damage through acting as alkalyting agents **(Xamena et al., 1988).**

Malathion is one of organophosphorus compound, so it acts as alkalyting agents causing DNA damage which appear as increasing in chromosomal aberration, sister chromatid exchanges and cell cycle delay **(Nicholas et al., 1979 and Chen et al., 1981).**

The use of alternative medicine has increased recently and attracted the attention of many researchers allover the world **(Cook and Baisden, 1986, Hunt et al., 2002 and EL-Nahas, 2004).**

Nigella sativa linn seeds are considered to be of the greatest healing herbs as several studies indicated that this plant is antioxidant, anti inflammatory, anticancer, anti-bacterial, antifungal, anthelmintics, antihypertensive, antipyretic, antihistaminic, anti-asthmatic and immune system stimulant. Furthermore, it can be used as hepatoprotective, stomachic, tonic, diuretic, haemostatic, bronchodilator and hypoglycemic **(Houghton et al., 1995, Baskabody and Shobi, 1997 and Saluma, 2005)**.

Material and Methods

I.Material

I.1.Malathion: Malathion (C10 H19 O6 PS2) (Diethyl dimethoxy phosphino thioyl thio butandioate) was obtained from EL Nasr chemical co. Egypt as commercial preparation of malathion (57%) that dissolved in distilled water. Malathion charactrizedly clear, brown to colorless liquid in form & garlic, like odor.

I.2.Nigella sativa (Black seed) oil : Nigella sativa oil obtained from capsules of Baraka (**Product of Pharco pharmaceuticals , Egypt)** each capsule is evacuated and contain 450 ml that contain 450 mg Nigella sativa oil then this oil is diluted with corn oil.

I.3.Experimental animals: Swiss albino mice, musculus 8-10 week old of both sexes, weighting from (14-25 g) were used. They obtained from farm of experimental animals in Helwan. Animals were acclimatized for one week to laboratory conditions before being used. The hygienic condition was kept constant

throughout the experimental period water and balanced ration were offered ad libitum.

I.4.Solutions and Reagents used:

a) Solutions used for cytogenetic studies:

Colchicines powder from sigma chemical co (1 mg / 1ml)

Fixative solution (3 parts methanol + 1 part glacial acetic acid).

Hypotonic solution (potassium chloride 0.075 m).

Fetal calf serum from sigma chemical co.

Geimsa stains solution 4% and 10%.

Dpx.

(II) Methods of cytogenetic studies

II.1. Animal treatment

Six groups of mice were used in this study. First group is used as control not injected with anything, the second group injected intraperitoneal by Nigella Sativa oil 160 mg / kg b.wt. diluted in corn oil **(Aboul Ela, 2002)** the third group injected intraperitoneal by 150 mg /kg b.wt. malathion dissolved in distilled water this dose is 1/20 LD50 (3000 mg /kg b.wt.) based on literature compilation by **(Giri et al., 2002, Dulout, et al., 1983, salvadori et al., 1988)**. Forth group was injected by Malathion intraperitoneal by 300mg/kg b.wt dissolved in distilled water (1\10 of LD50). The fifth group was injected with the 1st treatment of Malathion (150mg /kg b.wt.) in addition to 160mg/kg b.wt. Nigella sativa oil. And the sixth group injected by second treatment (300mg/kg b.wt.) in addition to 160mg /kg b.wt. Nigella sativa oil. One half of mice in each group sacrified after 24 hours and the other half sacrified after 48 hour from injection to collect samples.

2. Procedure:

a. Chromosomal aberration assay : Cytogenetic analysis of mice bone marrow cell performed according to the technique of **Giri et al., (1986).**

preparation of cells for chromosomal analysis: Mice were injected 2 hour before sample collection with an aqueous solution of colchicines 0.l ml /from 1mg/ml intraperitoneal, in order to accumulate metaphase cells and provide more readily analyzable chromosomes. Colchicine acts as a mitotic inhibitor that inhibits formation of mitotic spindle fibers and thus increases the number of metaphases by preventing their progress to anaphase. The animals were sacrified by cervical dislocation 2 hours after colchicines administration, both femurs are quickly removed, muscle is clean away from bone, and the cartilaginous epiphyses were cutted off.. Bone marrow of one femur was pushed out with about 5ml of 37°c prewarmed kcl (0.075M) solution (hypotonic solution) into small. Clean Petri dishes and minced well by fine scissors and then transferred into conical centrifuge tubes, using a clean syringe, then the tubes were placed into water bath adjusted at 37°c for 20minutes to permit osmotic swelling of bone marrow cells. After 20 minutes had precisely elapsed, the tubes were centrifuged at 800rpm for 5minutes the hypotonic solution was removed. The swollen bone marrow cells were fixed by adding 5ml of ice cold fixative, made freshly immediately before using drop wise with agitation ,the marrow cells were mixed well with fixative by consequent alternative siphoning and blowing of the cell suspension for several times using pasture pipettes. The tubes were centerfugated at 800 rpm for 5 minutes where a clear supernatant fixative was separated from the cell pellets at the bottom of the tubes. Gently, the supernatant was aspirated, leaving a small volume over the cell pellets. About 5ml of cold fresh fixative were added drop wise with agitation followed by resuspension of the cell pellet thoroughly as previously described. The previous steps were repeated respectively 3 or 4 times for proper washing of the bone marrow. After last suspension volume was brought to about 0.5ml with fresh fixative. The cell suspension was dropped on to cleaned wetted slides from about 30cm height and then slides were air-dried. Slides were stained with 4% Gemisa

stain for 15-20minutes, followed by cleaning in distilled water and leave it to dry. Fifty, well spread metaphase plates per mouse were randomly selected for screening to determine any chromosomal aberrations. scanning slides for mitotic spreads was conveniently accomplished with a 40 X .magnification objective. Suitable metaphase spreads were examined and photomic photographed microscopically for study of structural and/or numerical chromosomal aberrations.

b- Mitotic index (M.I): Mitotic index (M.I) for control acute (24 and 48 hrs) were calculated according to the equation **(Brusick, 1980)**

$$M.I = \frac{\text{Number of divided cells}}{\text{Total number of cells}} \times 100$$

C. Micronucleus experiment: The frequency of micro nucleated erythrocytes in femoral bone preparation was evaluated according to procedure described by Schmid, (1975).This test is done on the same animals that used in control and acute treatments. After mice were scarified by cervical dislocation, the bone marrow was flushed out from one femur with 1-15 pre-freezed fetal calf serum into centrifuge tube. The tubes were centrifuged at 1000 rpm for 5 minutes. Discard the supernatant and sediment cells were then smeared onto clean slides, after drying, the slides were fixed with absolute methyl alcohol for 10 minutes and stained with 10% Gemisa. From each mouse 2000 cells were randomly selected for screening to determined number of cells, which contain micronucleus.

Results and Discussion

1. Chromosomal aberrations

The data listed in table (1.a) revealed that no significant difference between the two control groups (without any treatment and *Nigella sativa*). There was however, a

significant difference between all treated groups and control groups. But there was no significant difference between the groups takes malathion at a dose level of 300 mg/kg b.wt only (35±0.58) and the group take the same dose of malathion with *Nigella sativa* (34±0.57). While there was a significant difference between group take malathion in dose 150 mg/kg b.wt (31.33±0.88) and those take the same dose with *Nigella sativa* (26±0.57).

Table (1.a) Mean of aberrant cells in animals received malathion and/or Nigella sativa in 24 h (acute exposure).

Groups	NO. OF animals/group	No. of examined cell/animal	Aberrant cells (mean±SE)
Control	3	50	19.00±0.58[d]
Nigella sativa	3	50	21.00±0.58[d]
Malathion (150mg/kg b.wt)	3	50	31.33±0.88[b]
Malathion(150mg/kg b.wt) and Nigella sativa oil	3	50	26.00±0.57[c]
Malathion(300 mg/kg B.wt)	3	50	35.00±0.58[a]
Malathion(300 mg/kg B.wt) and Nigella sativa oil	3	50	34.00±0.57[a]

Each value represents least square mean± standard error.
Means having the same letters in the same column don't differ significantly (p> 0.05).

The different types of aberrations of treated and control groups were presented in table (1.b) and figures(2-5). The most prominent types of chromosomal aberrations in malathion and malathion with *Nigella sativa* oil treated groups, were fragment and chromatid deletions. While chromosome breaks was the lowest type of aberration in all treated groups.

Table (1.b) Different types of chromosomal aberrations in animals received malathion and/or *Nigella sativa* 24 h acute exposure.

Groups	Types of aberrations										
	Gap	Fragment	Chromatid deletion	Chromatid break	Chromosome break	Centric fusion	Centric attenuation	End to end association	Ring chromosome	Sickness	Polyploidy
Control	3.33±0.33^{b}	3.00±0.01^{d}	3.33±0.88^{c}	0	0	1.00±0.08^{c}	1.00±0.01^{c}	1.67±0.07^{b}	2.67±0.33^{c}	2.33±0.88^{c}	0.67±0.03^{c}
Nigella sativa (N.S)	3.00±0.58^{b}	3.33±0.6^{d}	3.00±0.53^{c}	0.67±0.03^{c}	0.67±0.03	2.00±0.08^{b}	0.67±0.03^{c}	0.67±0.03^{c}	3.67±0.33^{b}	2.33±0.4^{c}	1.00±0.08^{c}
Malathion (150mg/kg b.wt)	3.00±0.33^{b}	5.00±0.33^{b}	4.67±0.33^{b}	2.00±0.33^{ab}	1.00±0.03	3.00±0.33^{a}	4.00±0.88^{a}	2.67±0.33^{a}	4.00±0.02^{a}	3.00±0.03^{b}	2.00±0.33^{ab}
Malathion (300 mg/kg b.wt)	2.33±0.33^{c}	5.67±0.67^{a}	5.67±0.33^{a}	1.33±0.04^{b}	1.33±0.07	3.00±0.33^{a}	2.00±0.05^{b}	2.33±0.33^{a}	4.33±0.33^{a}	4.67±1.00^{a}	2.33±0.88^{a}
Malathion (150mg/kg b.wt)+ N.S	2.67±0.58^{bc}	4.00±0.33^{c}	4.00±0.33^{b}	1.67±0.33^{b}	1.00±0.07	2.33±0.33^{b}	1.67±0.33^{b}	2.33±0.05^{a}	2.33±0.67^{b}	2.33±0.33^{c}	1.67±0.08^{b}
Malathion (300mg/kg b.wt)+ N.S	4.33±0.33^{a}	5.00±0.33^{b}	5.33±0.33^{a}	3.33±0.33^{a}	0.67±0.07	3.33±0.001^{a}	1.00±0.03^{c}	2.67±0.88^{a}	4.00±0.57^{a}	2.67±0.67^{bc}	1.67±0.03^{b}

The data in table 2.a and figure 1 showed the least square mean±SE of total aberrant cells in two control groups (without any treatment and *Nigella sativa* oil group) as well as the treated groups for 48 h. The results showed that there was a significant difference between the two control groups from one side and between the control and all treated groups from another side. While a significant difference was observed between group take malathion in dose of 300 mg/kg b.wt and that take the same dose with *Nigella sativa*. Although, no significant difference was recorded for group received lower dose of malathion (150 mg/kg b.wt) and that take the same dose with *Nigella sativa*.

Table (2.a) Mean of aberrant cells in animals received malathion and/or *Nigella sativa* in 48 h (acute exposure).

Groups	NO. OF animals/group	No. of examined cell/animal	Aberrant cells (mean±SE)
Control	3	50	19.03±.0.58[d]
Nigella sativa (N.S)	3	50	22.00±0.57[c]
Malathion (150mg/kg b.wt)	3	50	30.33±0.33[b]
Malathion (150mg/kg b.wt) and Nigella sativa oil	3	50	28.00±0.57[b]
Malathion(300 mg/kg B.wt)	3	50	37.00±0.57[a]
Malathion (300 mg/kg B.wt) and Nigella sativa oil	3	50	31.00±0.57[b]

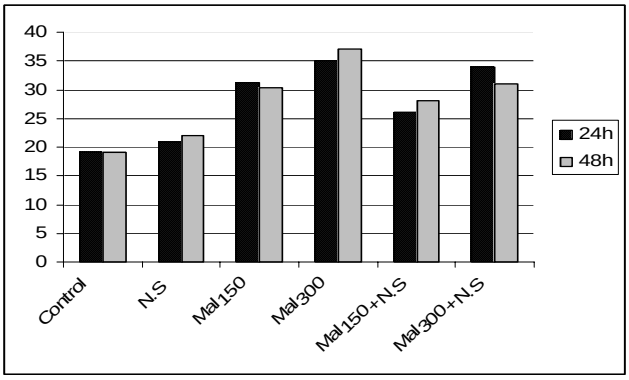
Each value represents mean± standard error
Means having the same letters in the same column don't differ significantly (p> 0.05).

The data listed in table 2.b, illustrated that the most prominent type of chromosomal aberrations was fragments in all treated groups except the group given malathion in dose of (150 mg/kg b.wt) with *Nigella sativa* (5.0 ± 0.57, 6.67 ± 0.67 and 5.0± 0.34 respectively) while the highest type in group received malathion in dose of (150 mg/kg b.wt) with *Nigella sativa* was gaps (5.33±0.57). On the other hand the lowest aberration type in two groups received malathion only at two dose level were chromosome breaks (0.6 7± 0.03). And in groups given malathion with Nigella sativa were chromosome breaks and polyploidy (0.33 ± 0.03, 1.33 ± 0.007, 0.33 ± 0.03 and 1.0 ± 0.03). Malathion produced a significant increase in chromosomal aberrations and no change in this effect appeared between 24 h than 48 h of exposure. On the other hand, *Nigella sativa* had some protective effects when given with malathion in acute exposure either at 24 or 48 h tables (1.a, 2.a) and figure (1).

Table (2.b) Different types of chromosomal aberrations in animals received malathion and/or *Nigella sativa* for 48 h acute exposure.

Groups	Types of aberrations										
	Gap	Fragment	Chromatid deletion	Chromatid break	Chromosome break	Centric fusion	Centric attenuation	End to end association	Ring chromosome	Sickness	Polyploidy
Control	2.33±0.33^{c}	2.67±0.33^{d}	2.00±0.07^{d}	1.00±0.001^{b}	0	1.00±0.07^{b}	1.33±0.33^{b}	2.00±0.07^{c}	3.67±0.88^{a}	1.67±0.33^{c}	0
Nigella sativa (N.S)	3.00±0.33^{c}	4.33±0.33^{c}	2.33±0.33^{d}	1.67±0.33^{b}	0.67±0.03^{b}	1.67±0.33^{b}	1.33±0.33^{b}	3.00±0.004^{b}	3.00±0.005^{b}	0.67±0.03^{d}	0.33±0.03
Malathion (150mg/kg b.wt)	3.33±0.57^{bc}	5.00±0.57^{b}	3.67±0.57^{c}	2.00±0.06^{a}	0.67±0.03^{b}	2.33±0.88^{a}	3.00±0.57^{a}	3.67±0.33^{ab}	4.00±0.15^{a}	1.33±0.07^{c}	1.00±0.07
Malathion (300 mg/kg b.wt)	6.00±0.33^{a}	6.67±0.67^{a}	5.67±0.67^{a}	2.67±0.33^{a}	0.67±0.03^{b}	1.33±0.08^{b}	1.33±0.33^{b}	4.67±0.33^{a}	3.33±0.88^{ab}	2.67±0.33^{ab}	0
Malathion (150mg/kg b.wt)+ N.S	5.33±0.57^{a}	4.67±0.33^{bc}	4.67±0.33^{b}	1.33±0.07^{b}	0.33±0.03^{b}	1.00±0.03^{b}	1.00±0.08^{b}	2.67±0.33^{bc}	2.33±0.88^{c}	2.33±0.33^{b}	0.33±0.03
Malathion (300mg/kg b.wt)+ N.S	4.00±0.57^{b}	5.00±0.34^{b}	3.67±0.33^{c}	2.33±0.006^{a}	1.33±0.007^{a}	3.00±0.33^{a}	3.33±0.57^{a}	3.00±0.57^{b}	3.67±0.33^{a}	3.33±0.57^{a}	1.00±0.03

Each value represents least square mean± standard error.
Means having the same letters in the same column don't differ significantly (p> 0.05).



Mal=malathion
N.S=Nigella sativa

Fig (1): Mean of aberrant cells in both acute exposures for 24 and 48 h

These results agree with in vitro results which obtained by **Nicholas et al., (1979)** when exposed human fibroblast to malathion. Also with the finding of **Walter et al., (1980)** who did their experiment on cultured human lymphocytes where they found that malathion increased RNA and DNA fractions in culture. The same results obtained by **Nishio & Uyeki, (1981)** on cultured hamster ovary cells and they found that malathion induce sister chromatid exchange in culture. Also

Garry et al., (1990) obtained results agree with the present results on cultured human lymphocyte and they illustrated that Malathion significantly increase the chromosomal aberrations and sister chromatid exchanges.

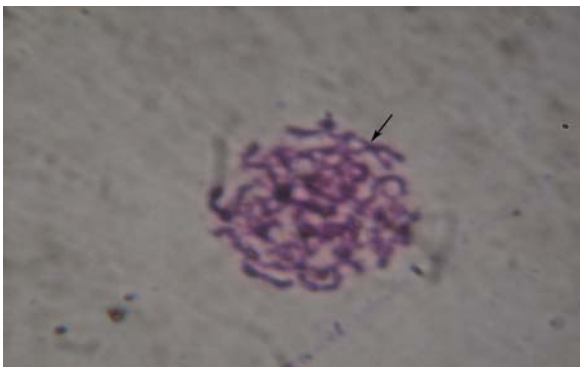
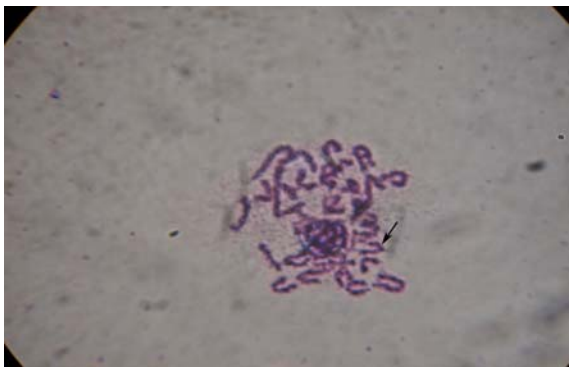


Fig (2) chromosomes of mice bone marrow showed centric fusion



Fig(3) chromosomes of mice bone marrow showed deletion

Also in vitro results obtained by **Balaji & Sasikala, (1993)** illustrated that malathion increased chromosomal aberrations and sister chromatid exchange in cultured cells.

Moreover, the present results agree with the in vivo results of **Dulout et al., (1983)** who reported that malathion induced chromosomal aberrations (gaps, breaks, and chromatid exchanges) when injected I/P in mice in doses 115, 230 or 460 for 24 h treatment. Also **Amer et al.**, **(1996)** investigated mice injected I/P with malathion in a dose of 300 mg/kg b.wt and sacrificed them after 6, 24, 48 h. he found a significant increase in chromosomal aberration after 24 h more than 6,48 h and this agrees with the present findings.

El-Nahas & El-Ashmaway, (2004) reported that single I/P injection of malathion in a dose of (400 mg/kg b.wt) in mice caused a significant increase in chromosomal aberrations.

2. Mitotic index:

The data listed in table **3** and figure 6 showed the mitotic index of control and 24 and 48 hour treated groups. There were significant differences between treated

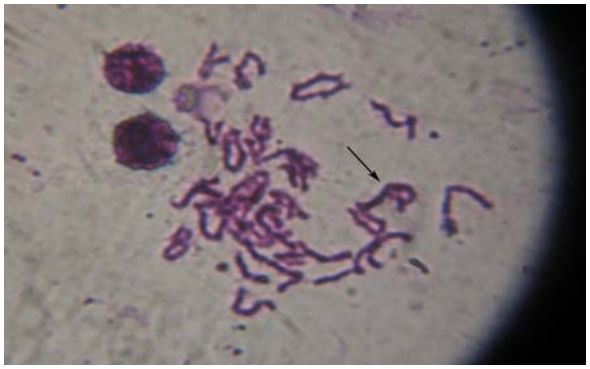


Fig (4) chromosomes of mice bone marrow showed End to end association

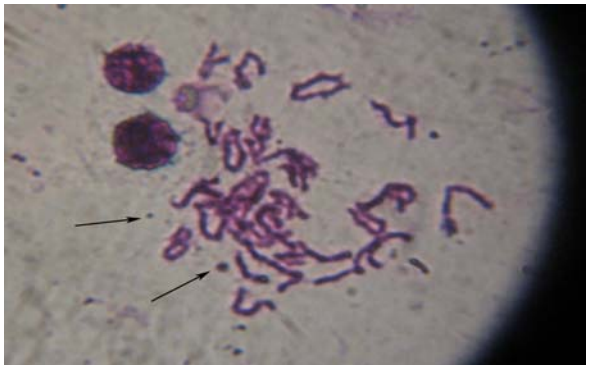


Fig (5) chromosomes of mice bone showed chromosomal Fragment

groups and control groups in both 24, and 48 h. Also there were significant differences between group takes malathion only and those take malathion in the same doses with *Nigella sativa* in both 24 and 48 h after single injection of malathion.

These results suggest that malathion decreases mitotic index of bone marrow cells of mice and *Nigella sativa* had a protective effect against the effect of malathion. These results agreed with results obtained by **Walter et al., (1980)** who found that malathion decreases the cell survival in human lymphocyte cell culture.

Also **Salavadori et al., (1988)** suggested that malathion decreases the mitotic indices in bone marrow when single dermal applied to mice in doses (500, 2000 mg/kg b.wt). Moreover, **Balaji & Sasikala, (1993)** reported that malathion significantly decreases the mitotic index in human peripheral leukocyte cell cultures treated with malathion in doses (0.02, 0.2, 2 and 20 µg/ml) added to culture and index was examined after 0, 24, 48 and 72 h from culture initiation.

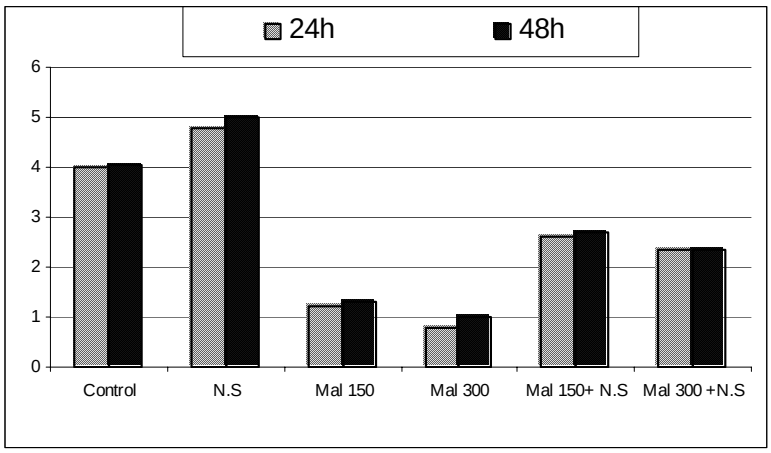
3-Micronucleus test:

The data listed in table (4) and figure (7) showed that there were significant differences between all treated groups in both 24 and 48 h and control groups. On the other hand there were no significant differences between the groups received

Table (3) Mitotic indices of control and 24 and 48 hrs treated groups.

Groups	24 hours		48 hours	
	NO of divided cells/group	(Mean±SE) %	NO. of divided cells/group	(Mean±SE) %
Control	240	4.00±0.5[a]	242	4.03±0.48[a]
Nigella sativa (N.S)	290	4.80±0.73[a]	300	5.00±0.57[a]
Malathion (150 mg/kg.bwt)	72	1.20±0.06[c]	78	1.30±0.05[c]
Malathion (150 mg/kg.bwt)+N.S	136	2.60±0.5[b]	166	2.70±0.15[b]
Malathion(300 mg/ kg b.wt)	48	0.80±0.05[d]	62	1.00±0.09[C]
Malathion(300 mg/ kg b.wt)+	140	2.33±0.18[bc]	140	2.33±0.2[b]

- Each group contain 3 animals and 2000 cell from each animal were examined



Mal =Malathion N.S=Nigella sativa

Fig (6) Mitotic indices of control and 24 and 48 hrs treated groups.

malathion alone in dose (300 mg/kg b.wt) and that received malathion in the same dose with *Nigella sativa* in both 24 and 48 h. Moreover there was a significant difference between the group received malathion in dose (150 mg/kg b.wt) alone and those treated with the same dose with *Nigella sativa* in both 24 and 48 h interval after single injection.

The present data suggested that malathion increases the rate of micronucleus production and the *Nigella sativa* has some protective effect against this effect of malathion. These results agree with **Dulout et al., (1982**) They found that there was significant increase in micronucleus in mice exposed to malathion at doses of 120,240, and 480 mg/kg b.wt. in relation to control.

Titenko-Holland et al., (1997) suggested that malathion induced a significant increase in micronucleus formation in vitro when applied malathion to lymphocyte cell culture in doses 5 to 100 µg/ml for 48 h. this matched with our results.

Table (4): Mean ±SE of micronucleus for control and 24 and 48 hrs treated groups.

Groups	24 hours		48 hours	
	NO of aberrant cells/group	Aberrant cells (Mean±SE)	NO. of aberrant cells/group	Aberrant cells (Mean±SE)
Control	556	92.67±5.04^{c}	530	90.00±12.67^{c}
Nigella sativa(N.S)	512	85.33±10.7^{c}	508	84.67±7.90^{c}
Malathion (150 mg/kg.bwt)	1028	186.33±16.85^{a}	1012	185.33±9.21^{a}
Malathion (150 mg/ kg b.wt) + N.S	958	159.67±3.18^{b}	956	159.00±0.88^{b}
Malathion(300 mg/ kg b.wt)	1200	200.00±4.04^{a}	1186	197.67±1.45^{a}
Malathion (300 mg/kg b.wt) + N.S	1080	180.00±5.8^{ab}	1076	179.00±3.5^{ab}

When **Garaj-Vrhovac & Zeljezic, (2001)** did an experiment on Croatian workers who occupationally exposed to malathion for 8 months and the same workers reexamined after 8 months from removal from exposure to malathion. Their results suggested that malathion increased the micronucleus frequency in exposed workers either before or after removal from exposure which came higher rates than control unexposed persons. This result indicates that long term exposure to malathion induces a potent mutagenic effect. Furthermore, **Garaj-Vrhovac &**

Zeljezic, (2002) on other exposed workers during the production of malathion, that long term of exposure to malathion cause significant high increase in micronucleus formation that indicate genome damage induced by malathion.

On the contoraly **Titenko-Holland et al., (1997)** found that examining of blood samples collected from malathion occupationally exposed workers indicated that there was no change in micronucleus formation level compared with unexposed control persons. These difference in results may be due to the period of exposure of these persons were unknown.

While **Windham et al., (1998)** found that malathion induced a significant increase in micronucleus formation level in occupationally exposed workers compared to unexposed control persons. They suggested that malathion had relatively low risk of genotoxic damage. The present results showed some protection for *Nigella sativa* in acute exposure against the increase in the rate of micronucleus formation .

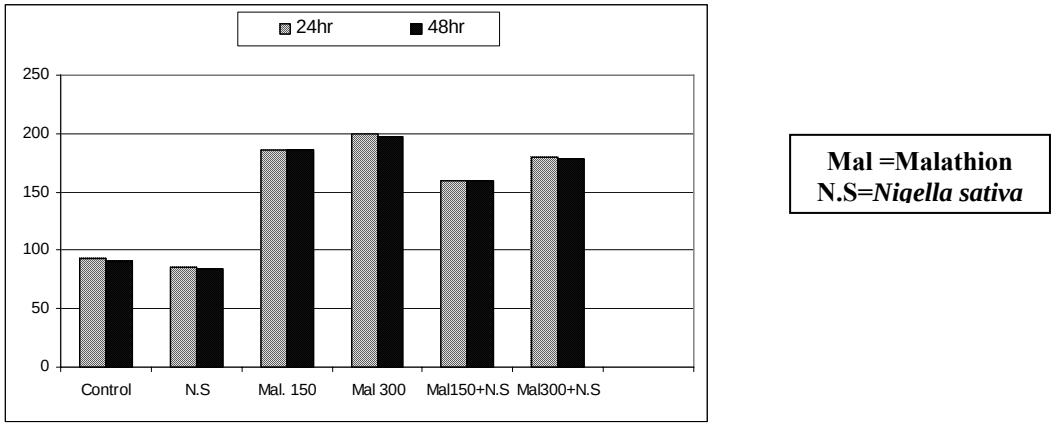


Fig (7): Micronucleus for control and 24 and 48 hrs treated groups.

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تأثير المالاثيون على الكر وموسومات في الفئران

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الملخص العربي

هذا العمل اجري من اجل دراسة التأثير الوراثى الخلوي للمالاثيون احد المبيدات العضوية الفسفورية التي تسبب تأثيرا على المادة الوراثيه الخاصة بالكائنات الحية التي تتعرض لتلك المبيدات و كذلك دراسة تأثير زيت حبه البركة في تقليل والحماية من تأثيرات ذلك المبيد الحشري حيث أنها معروفه بتأثيرها الواقي.

تم استخدام ٣٦ فأر تجارب من كلا الجنسين وقسمت هذه الفئران إلى ست مجموعات. التجربةفي هذه نصف الحيوانات المستخدمة في كل مجموعه ذبحت وأخذت العينات منه بعد ٢٤ ساعة من المعاملة. والنصف الآخر ذبح بعد ٤٨ ساعة من المعاملة. و ثم تم دراسة تأثير المالاثيون منفردا و المالاثيون مع حبه البركة على معدل انقسام الخلايا والشذوذ الكروموسومى وكذلك على معدل تكوين الانويه الصغيرة. **و تم الحصول على النتائج يمكن تلخيصها كالأتي** إعطاء المالاثيون في إحداث زيادة كبيرة الصور مختلفة من الشذوذ الكروموسومى مما يدل على أن المالاثيون له تأثير مطفر كبير وربما يكون مسرطن . المالاثيون تسبب في تسبب وكذلك تقليل كبير في معدل انقسام خلايا النخاع العظمى للفئران عندما أعطى سواء لفترات قصيرة. المالاثيون في زيادة معدل تكوين الانويه الصغيرة بشكل واضح. السابقة يتضح أن للمالاثيون تأثير كبير سئ على المادة الوراثيه والذي يعطى احتماليه عن انه قد يكون للمالاثيون تأثير مسرطن وكذلك للمالاثيون تأثير ضار على الكبد . وكذلك يمكن من النتائج السابقة استخلاص أن لزيت حبه البركة تأثير وقائي فعال ضد التأثيرات الضارة للمالاثيون التي تم تناولها في هذه الدراسة.