

# Wheat germ oil attenuates deltamethrin-induced injury in rat cerebellar cortex: Histological and immunohistochemical study

Original  
Article

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## ABSTRACT

**Introduction:** Deltamethrin, a widely used insecticide, has neurotoxic effects causing apoptosis. Wheat germ oil is a highly valuable part of wheat that proved to have neuro-protective and anti-apoptotic effects.

**Aim of the work:** To evaluate the possible protective role of wheat germ oil in deltamethrin-induced injury in rat cerebellar cortex.

**Materials and Methods:** Four equal groups of adult male albino rats "10 rats, each" were used; control group, wheat germ oil (WGO) group (100 mg/kg), deltamethrin group (0.6 mg/kg), and deltamethrin & wheat germ oil combination group. Daily oral doses were given for consecutive thirty days. Specimens of the cerebellum were processed for histological and immunohistochemical study. Morphometric studies and statistical analysis were performed.

**Results:** In comparison with the control group, the specimens of the deltamethrin group showed disturbed monolayer arrangement of Purkinje cells with loss of the normal shape. Shrunken deeply stained Purkinje cells as well as some lost cells were noticed. Immunohistochemically, a significant decrease in Bcl2 immunoreaction revealed together with a significant increase in Bax immunoreactivity in Purkinje cells. Moreover, significant increase in the GFAP immune-expression was seen in all cortical layers. In contrast, minimal changes appeared in the group treated concomitantly with deltamethrin and wheat germ oil.

**Conclusion:** Wheat germ oil attenuates deltamethrin-induced structural changes in rat cerebellar cortex.

**Received:** 15 January 2018, **Accepted:** 24 February 2018

**Key Words:** Cerebellar cortex, deltamethrin, wheat germ oil.

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**ISSN:** 1110-0559, Vol. 41, No.2

## INTRODUCTION

Most of the agricultural damage is caused by insects and therefore, pesticides are widely used to control this damage<sup>[1]</sup>. Deltamethrin, a pyrethroid insecticide belongs to type II generation, is classified as a safe insecticide with a broad protective activity in agriculture. It protects most crops from many pests as ants, mites, beetles and weevils, but proved to be harmful to human causing pathological changes in many organs<sup>[3, 4]</sup>.

In rats, deltamethrin administration resulted in reproductive toxicity, hepatorenal degeneration and endocrine disruption<sup>[5-7]</sup>. It has damaging effect on liver, immunological system, hematological parameters and neurons<sup>[1, 8]</sup>. Gestational exposure can also affect the development and behavioural parameters of the rat offspring's<sup>[9]</sup>.

Acute occupational exposure to these synthetic pyrethroids may lead to hypersensitivity reactions and even death due to respiratory failure<sup>[10]</sup>. Acute exposure also can lead to neurotoxicity which is manifested by

ataxia, hyperexcitation, loss of coordination, convulsions as well as paralysis. Various cellular, biochemical and molecular mechanisms may be involved in deltamethrin neurotoxicity. Numerous studies suggest the apoptotic role of deltamethrin which is an important key in the neurodegenerative diseases. In context, deltamethrin affects the survival of the neurons of the rat brain by inducing mitochondria mediated apoptosis<sup>[11]</sup>. Deltamethrin accumulation increases reactive oxygen species (ROS) generation in different body systems resulting in oxidative damage as well as cell death<sup>[12, 10]</sup>. Reduced gamma aminobutyric acid level in the cerebellum by deltamethrin was reported in some studies resulting in excitation as well as convulsion<sup>[13]</sup>.

Due to the importance of these synthetic compounds in the agricultural field and due to their dangerous impacts on the human health, it is important to find a way in order to lessen their damaging effects on humans.

Wheat germ oil (WGO) is obtained from germs of wheat kernel which is the most valuable part of the wheat. It is a main nutritious source for essential fatty acids, vitamins and phytochemicals with antioxidant role. It is an important

source for polyunsaturated fatty acids and considered the richest source for  $\alpha$ -tocopherol with high vitamin E<sup>[14-16]</sup>, which is a potent neuroprotective antioxidant vitamin. Subsequently, it protects the brain from oxidative damage and the impairment due to aging process<sup>[17]</sup>. So, WGO has beneficial role in the protection and treatment of diseases with oxidative damaging effect via cell-protection from the free radicals. Moreover, it possesses anti-inflammatory action that helps in the treatment of chronic inflammatory reactions, muscular dystrophies as well as neurological disorders<sup>[18, 19]</sup>. So, we aimed to evaluate the possible role of wheat germ oil in attenuating deltamethrin-induced injury in rat cerebellar cortex.

### **Material and methods**

#### **Chemicals:**

1- Deltamethrin: Produced by Kafr El Zayat Pesticides and Chemicals Co. (KZ), Egypt.

2- Wheat germ oil: Produced by El Captain Company (Cap Pharm) for extracting natural oils, plants and cosmetics (Egypt).

#### **The study design:**

Four equal groups, 10 rats, each of adult male albino rats (170-200 grams) were enrolled in this work.

Group I (control group): Rats were organized into two equal subgroups; subgroup IA were left untreated, subgroup IB received oral dose of corn oil (1 ml/kg BW), a solvent for deltamethrin.

Group II (WGO group): Rats were given WGO orally at a dose of 100 mg/kg/day<sup>[20]</sup>.

Group III (deltamethrin group): Rats received deltamethrin orally at a dose of 0.6 mg/kg/day<sup>[21]</sup>.

Group IV (deltamethrin and WGO group): Rats received the above mentioned doses of deltamethrin and WGO concomitantly.

All treatments were given once daily and continued for consecutive thirty days. Twenty four hours after the last dose, cerebellum specimens were taken from rats and fixed in 10% neutral-buffered formalin for 24 hours. Thereafter, the specimens were processed by applying the routine protocols. After specimen embedding in paraffin, 5  $\mu$ m sections were stained with hematoxylin and eosin (H&E) and examined by the light microscope for histological study<sup>[22]</sup>.

For immunohistochemistry, formalin fixed, paraffin embedded sections were incubated in a humid chamber with the primary anti-Bax antibody (rabbit polyclonal antibody, 1/50 dilution, Abcam), anti-Bcl2 antibody (rabbit polyclonal antibody, 1/100 dilution, Abcam), and anti-GFAP (Glial fibrillary acidic protein) antibody (mouse monoclonal antibody, 1/100 dilution, Thermo scientific) overnight. After washing in phosphate buffered saline

(PBS), the corresponding biotinylated secondary antibody was applied to the sections for one hour at the room temperature and then washed again in PBS. Streptavidin peroxidase was applied for 10 minutes at room temperature and then washed in PBS. 3, 3'-diaminobenzidine (DAB)-hydrogen peroxide was applied as a chromogen to localize and visualize the immunoreaction. Finally, the immunostained sections were counterstained by adding Mayer's haematoxylin. For negative control sections, the primary antibodies were excluded<sup>[23]</sup>.

#### **Morphometric assessment and statistical analysis:**

For morphometric study, The Image analysis system (Leica Qwin 500 C Image analyzer computer system) at Central Research Lab, Faculty of Medicine, Tanta University, Egypt was used. The color intensity of the positive immunoreaction for Bax and Bcl2 was measured in the DAB-stained slides. Moreover, the area percentage of GFAP positive immunoreaction in the cerebellar cortex was also measured. Ten different microscopic fields from each cerebellum specimen in each animal group were examined.

For analysis of the morphometric data, one-way analysis of variance (ANOVA) followed by Tukey's procedure was done for comparison between the different groups by using statistical package for social sciences statistical analysis software (version 11.5; SPSS Inc., Chicago, Illinois, USA). Mean values  $\pm$  standard deviation were calculated. *P* value < 0.05 was regarded significant.

## **RESULTS**

No deaths were reported during the study period.

#### **Histological results:**

Examination of the cerebellum specimens of the control group as well as WGO group showed the three layers of the cerebellar cortex; superficial molecular, Purkinje cell and deep granular cell layers. The superficial molecular layer composed of scattered cells and fibers, while the deep granule cell layer showed densely packed spherical dark nuclei. Purkinje cell layer was found between the superficial molecular and the deep granular layers and formed of a continuous monolayer (single row) of flask shaped or pyriform cells. These cells had large cell bodies with rounded lightly stained nuclei and prominent nucleoli (Fig. 1).

Examination of the cerebellum specimens of deltamethrin group revealed loss of the normal cerebellar architecture and degenerative changes mainly in Purkinje cell layer. These were in the form of disturbed normal monolayer arrangement of Purkinje cells. Some areas showed many layers of Purkinje cells with loss of their normal shape. Accumulation of the neuroglial cells around

the affected Purkinje cells was noticed in some sections (Figs. 2 and 3). Shrunken deeply stained nuclei and cytoplasm of many Purkinje cells were seen in several areas (Fig. 4). Some Purkinje cells with homogenized cytoplasm and faint nuclei were also noticed. Other cells appeared with vacuolated cytoplasm. Other areas appeared severely affected with focal disappearance of their Purkinje cells. The molecular and granular layers showed minimal affection in the form of appearance of numerous vacuolated areas near Purkinje layer (Figs. 5 and 6).

In the deltamethrin and WGO group, the sections appeared with mild disorganization of the Purkinje cell monolayer. Few shrunken and deeply stained Purkinje cells were observed in between the normal cells. The molecular and granular layers appeared more or less as the control group (Fig. 7).

**Immunohistochemical results:**

**Bcl2 antigen immunostaining:** Bcl2-immunostained cerebellum sections of the control group (group I) showed strong positive cytoplasmic brown immunoreaction for Bcl2 in Purkinje cells (Fig. 8). Deltamethrin group (group III) showed that numerous Purkinje cells expressed weak positive cytoplasmic immunoreaction for Bcl2 (Fig. 9), while deltamethrin and WGO group (group IV) expressed moderate positive cytoplasmic immunoreaction for Bcl2 in Purkinje cells (Fig. 10).

**Bax antigen immunostaining:** Bax-immunostained cerebellum sections of the control group (group I) showed weak positive brown cytoplasmic reaction in few Purkinje cells (Fig. 11). In deltamethrin group (group III), a strong positive cytoplasmic reaction for Bax was observed in many Purkinje cells (Fig. 12), while in deltamethrin and

WGO group (group IV), a moderate positive cytoplasmic reaction for Bax was detected only in few Purkinje cells (Fig. 13).

**GFAP antigen immunostaining:** GFAP-immunostained cerebellum sections of the control group (group I) expressed mild positive cytoplasmic reaction in the cell bodies and processes of the astrocytes in the three cortical layers (Fig. 14). In deltamethrin group (group III), an intense GFAP immune-expression was observed in the three cerebellar cortical layers (Fig. 15), while deltamethrin and WGO group (group IV) showed a moderate GFAP immune-expression (Fig. 16).

**Morphometric and statistical results (Table 1):**

The mean color intensity of Bcl2-positive immunoreaction in the deltamethrin group (group III) showed a significant decrease (7.47±0.354) compared with the control group (9.599±0.499), whereas the deltamethrin and WGO group (group IV) showed a non-significant decrease (8.795±0.676) compared with the control.

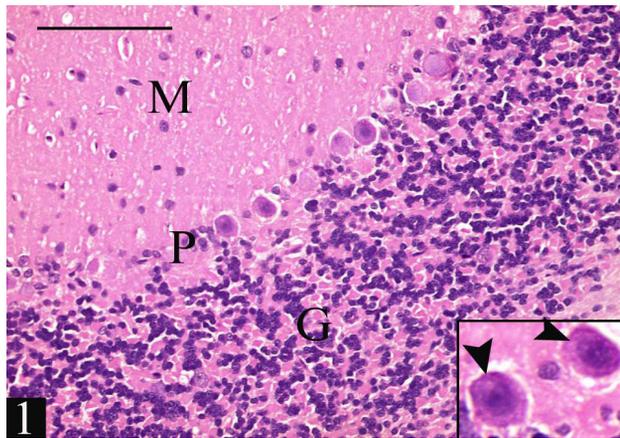
The mean color intensity of Bax-positive immunoreaction in the deltamethrin group (group III) showed a significant increase (9.942±0.474) compared with the control group (2.771±0.564), but the deltamethrin and WGO group (group IV) showed a non-significant increase (3.475±0.423) compared with the control.

The mean area percentage of GFAP-positive immunoreaction in deltamethrin group (group III) showed a significant increase (30.464±1.06) compared with the control group (9.514±0.658). Moreover, the deltamethrin and WGO group (group IV) showed a non-significant increase (10.038±0.428) compared with the control.

**Table 1:** Summarizes the mean the color intensity of Bcl2 and Bax immunoreaction and the mean area percent of GFAP immune-expression (mean ± standard deviation) for different rat groups.

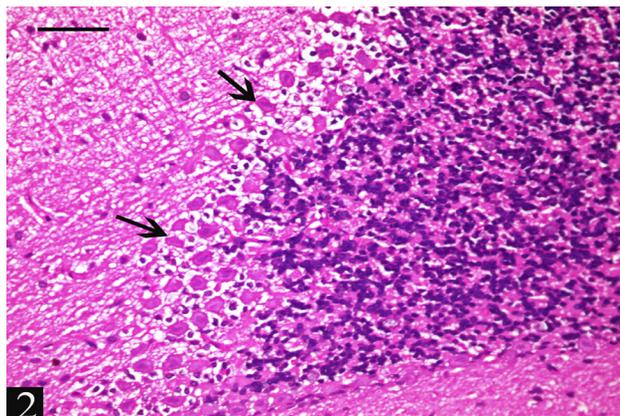
Parameters	Group I	Group II	Group III	Group IV
Mean color intensity of Bcl2	9.599±0.499	9.57±0.555	7.47±0.354 *	8.795±0.676
Mean color intensity of Bax	2.771±0.564	2.549±0.498	9.942±0.474*	3.475±0.423
Mean area percentage of GFAP	9.514±0.658	9.506±0.651	30.464±1.06*	10.038±0.428

\*P < 0.05 is significant versus control (group I).



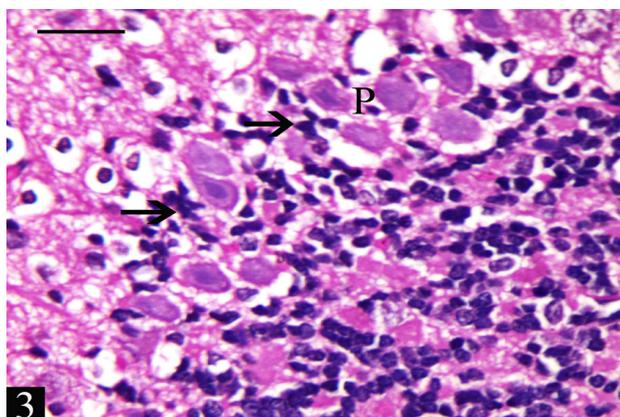
**Fig. 1:** A photomicrograph of a section in the cerebellar cortex of control rat showing the three layers; molecular (M), Purkinje cell (P), and granular cell layers (G). The inset shows Purkinje cells with large pale stained nuclei and prominent nucleoli (arrow heads). Notice the arrangement of Purkinje cells (P) in one row between the other two layers.

(H&E. X400; inset X1000; Scale bar = 25µm)



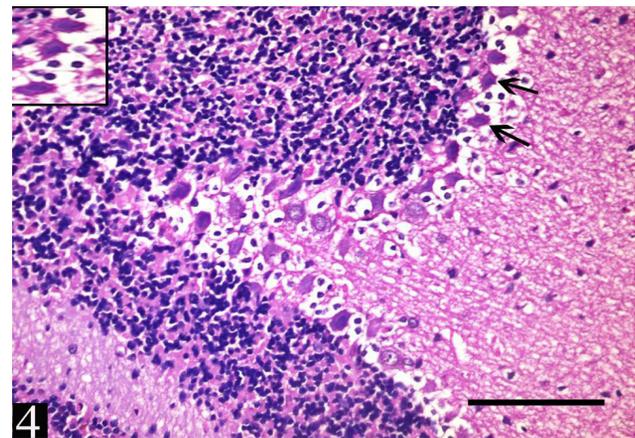
**Fig. 2:** A photomicrograph of a section in the cerebellar cortex from delta-methrin group (group III) showing many layers of Purkinje cells with loss of their normal shape (arrows).

(H&E. X400; Scale bar = 50µm)



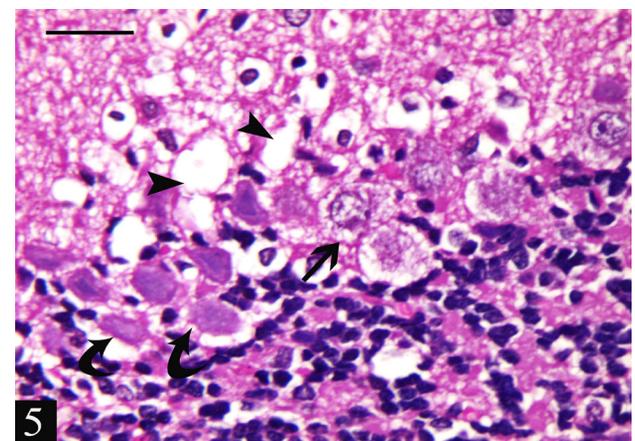
**Fig. 3:** A photomicrograph of a section in the cerebellar cortex from delta-methrin group (group III) showing disturbed monolayer of Purkinje cells (P) with interposition of many neuroglial cells (arrows) in between and around the Purkinje cells.

(H&E. X1000; Scale bar = 25µm)



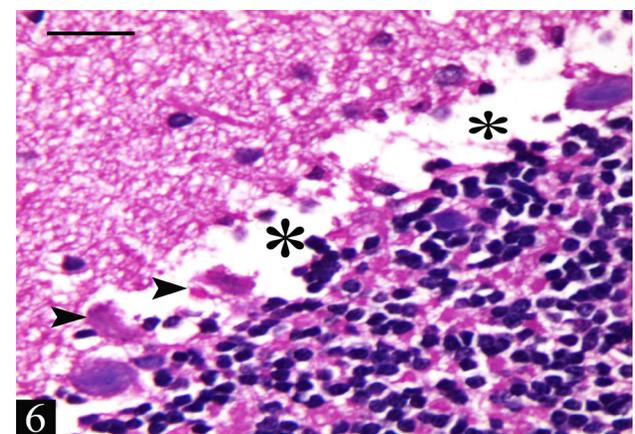
**Fig. 4:** A photomicrograph of a section in the cerebellar cortex from delta-methrin group (group III) showing shrunken deeply stained nuclei and cytoplasm of many Purkinje cells (arrows).

(H&E. X400; inset X1000; Scale bar = 25µm)



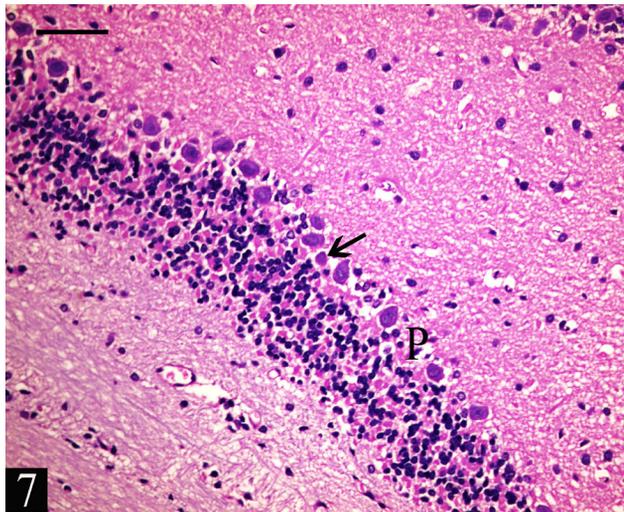
**Fig. 5:** A photomicrograph of a section in the cerebellar cortex from delta-methrin group (group III) showing some Purkinje cells with homogenized cytoplasm and faint nuclei (curved arrows). Other cells appeared with vacuolated cytoplasm (arrow). Notice vacuolation within the nearby molecular layer (arrow heads).

(H&E. X1000; Scale bar = 25µm)



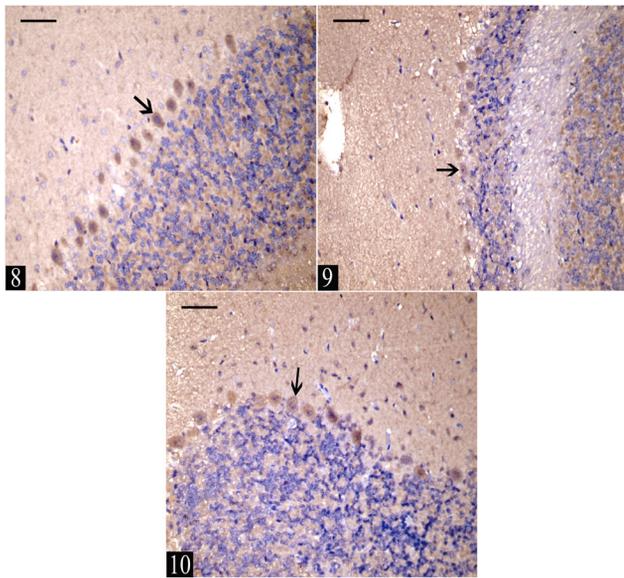
**Fig. 6:** A photomicrograph of a section in the cerebellar cortex from delta-methrin group (group III) showing disappearance of Purkinje cells from focal areas (\*) and presence of remnants of cells (arrow heads).

(H&E. X1000; Scale bar = 25µm)



**Fig. 7:** A photomicrograph of a section in the cerebellar cortex from deltamethrin & WGO group (group IV) showing monolayer arrangement of Purkinje cells (P) inbetween the other two layers. Notice few affected Purkinje cells (arrow) in between the normal cells.

(H&E. X400; Scale bar = 50µm)



**Fig. 8:** A photomicrograph of cerebellum section from control group showing strong positive immunoreaction for Bcl2 in the cytoplasm of Purkinje cells (arrow).

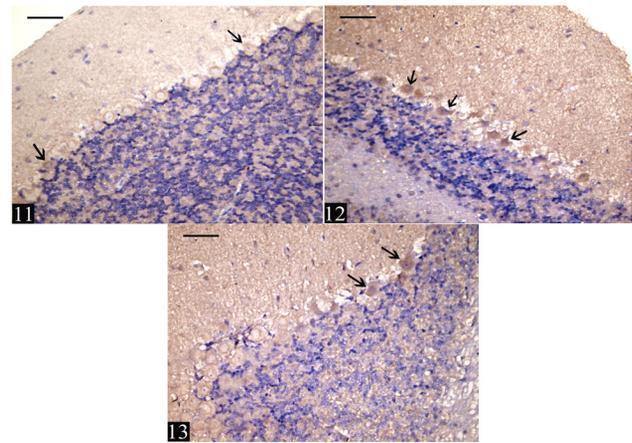
(Bcl2 immunostaining, X400; Scale bar = 50µm)

**Fig. 9:** A photomicrograph of a section in the cerebellar cortex from deltamethrin group (group III) showing weak positive immunoreaction for Bcl2 in the cytoplasm of Purkinje cells (arrow).

(Bcl2 immunostaining, X400; Scale bar = 50µm)

**Fig. 10:** A photomicrograph of a section in the cerebellar cortex from deltamethrin & WGO group (group IV) showing moderate positive immunoreaction for Bcl2 in the cytoplasm of Purkinje cells (arrow).

(Bcl2 immunostaining, X400; Scale bar = 50µm)



**Fig. 11:** A photomicrograph of cerebellum section from control group showing weak positive immunoreaction for Bax in the cytoplasm of few Purkinje cells (arrows).

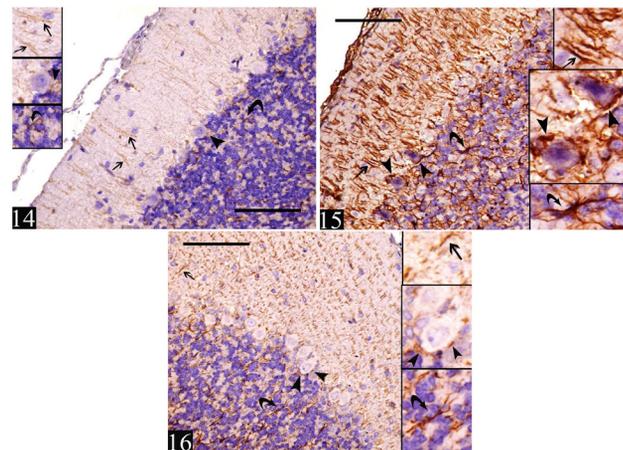
(Bax immunostaining, X400; Scale bar = 50µm)

**Fig. 12:** A photomicrograph of a section in the cerebellar cortex from deltamethrin group (group III) showing strong positive immunoreaction for Bax in the cytoplasm of many Purkinje cells (arrows).

(Bax immunostaining, X400; Scale bar = 50µm)

**Fig. 13:** A photomicrograph of a section in the cerebellar cortex from deltamethrin & WGO group (group IV) showing moderate positive immunoreaction for Bax in the cytoplasm of few Purkinje cells (arrows).

(Bax immunostaining, X400; Scale bar = 50µm)



**Fig. 14:** A photomicrograph of cerebellum section from control group showing mild GFAP immune-expression in the molecular (arrows), Purkinje cell (arrow heads) and granular (curved arrows) layers.

(GFAP immunostaining, X400; inset, X1000; Scale bar = 25µm)

**Fig. 15:** A photomicrograph of a section in the cerebellar cortex from deltamethrin group (group III) showing an intense GFAP immune-expression in the molecular (arrows), Purkinje cell (arrow heads) and granular layers (curved arrows).

(GFAP immunostaining, X400; inset, X1000; Scale bar = 25µm)

**Fig. 16:** A photomicrograph of a section in the cerebellar cortex from deltamethrin & WGO group (group IV) showing moderate GFAP immune-expression in the molecular (arrows), Purkinje cell (arrow heads) and granular (curved arrows) layers.

(GFAP immunostaining, X400; inset, X1000; Scale bar = 25µm)

## DISCUSSION

Our histological and immunohistochemical results demonstrated that deltamethrin administration to albino rats induced degenerative changes and apoptosis in Purkinje cells of the cerebellar cortex. These results coincided with previous reports indicating that deltamethrin has a neurotoxic effect<sup>[21, 11]</sup>. Neurotoxins are considered a main risky factor involved in the pathogenesis of the chronic neurodegenerative disorders. Inflammation, oxidative stress and apoptosis have been implicated in the induction of this neurodegeneration<sup>[24]</sup>.

Numerous studies investigated the molecular mechanisms involved in deltamethrin induced neurotoxicity. It is well known that pesticide intoxication results in oxidative stress in mammals due to free radicals overproduction and/or defective antioxidative defense processes. This leads to disturbed cellular physiology and even apoptotic cell death<sup>[25]</sup>.

Deltamethrin was reported to cause neuronal damage through free radicals overload. These oxidative hazards lead to brain injury. Deltamethrin causes elevated lipid peroxidation, nitric oxide (NO) concentration and DNA fragmentation. Moreover, it reduces the antioxidant capacity. The brain contains large amounts of polyunsaturated fatty acids which are the main target for reactive oxygen species (ROS) leading to lipid peroxidation. Nitric oxide is a neurotoxic agent as it is a potent free radical that reacts with superoxide and also causes highly reactive radical peroxynitrite generation. The hydrophobic lipophilic property of deltamethrin is responsible for its accumulation in tissues, ROS generation, and DNA damage. DNA fragmentation is a characteristic feature for apoptotic cell death and caused by induction of the proteolytic enzymes by oxidative stress. In addition, this oxidative stress leads to depletion of the mitochondrial energy through Na, K ATPase inhibition. Such results of reduced energy supply in neurons and subsequent neuronal damage<sup>[21]</sup> are consistent with the literature<sup>[26, 6]</sup>.

Previous studies have assumed that apoptosis plays an important role in deltamethrin neurotoxicity and also in the pathogenesis of the neurodegenerative disorders. This occurs through altered expression of caspases, P53, Bax, and Bcl2 which play a critical role in induction of apoptosis. Bcl2 and Bax are proteins belong to Bcl-2 family and play anti- and pro-apoptotic role, respectively<sup>[27-30]</sup>. This was evident in this study by significant decrease in Bcl2 immuno-expression and significant increase in Bax immunoreaction in Purkinje neurons of deltamethrin treated rats.

Another mechanism for apoptosis induction by deltamethrin is altered expression of cytochrome c leading to mitochondrial mediated apoptosis of the neurons in rat

brain. Deltamethrin can also interact with Na channels resulting in calcium overload, induction of ER stress pathway, and apoptosis<sup>[30, 31]</sup>.

In this work, deltamethrin induced disturbance in the histological structure of the cerebellar cortex particularly in Purkinje cell layer. They appeared arranged in many layers in some focal areas rather than the monolayer arrangement. Previous researcher reported that prolonged neuronal insult may results in adaptive mechanism in the form of Purkinje cell crowding in these focal areas. This is a trial to reestablish synapsis with the other nerve cells to be able to achieve their functions. Moreover, shrunken Purkinje cells with dark stained cytoplasm and unidentified nuclei were observed and considered by some authors as a sign for chromatolysis and gliosis. Gliosis or increased neuroglial cells could be explained as a rapid response to brain injury that is regulated by the soluble mediators of the neurons or by cell-cell contact. Other researchers considered the dark shrunken neurons as a reflection for certain apoptotic phase with markedly condensed cytoplasm as well as nucleoplasm<sup>[32, 33]</sup>.

The vacuolation appeared within the molecular and Purkinje cell layers were described by some authors as spongiform changes<sup>[34]</sup>. This increased vacuolation was attributed by others to loss of the cellular components within the cerebellar cortex<sup>[35]</sup>.

Concerning GFAP cytoplasmic immuno-expression in the astrocyte's cell body and processes, a significant increase was observed in the deltamethrin-treated rats compared to the control group. GFAP is the main protein of the intermediate filament of the mature astrocytes, subsequently, is considered as an astrocyte specific marker. Previous reports assumed that any degenerative brain insult induce astrocyte proliferation and hypertrophy with increased GFAP production resulting in severe astrogliosis. The appearance of these reactive astrocytes due to neuronal damage may be considered as a compensatory process following neurodegeneration. In addition, the up regulation of GFAP immunoreaction may be attributed to the production of NO. These activated astrocytes play an important beneficial role in tissue repair after injury by monitoring the PH, ion homeostasis, and extracellular water. However, astrocytes over activation may lead to detrimental effects<sup>[33, 36]</sup>.

Our results demonstrated the neuroprotective effect of WGO against deltamethrin induced cerebellar cortical damage. This finding is supported by previous reports which stated that WGO possesses a neuroprotective effects through its anti-inflammatory and antioxidant actions<sup>[37]</sup>. WGO significantly attenuates the severity of lipid peroxidation and enhances the antioxidant enzyme activity. This effect is attributed to its high content of  $\alpha$ -tocopherol or vitamin E, a potent anti-oxidant vitamin. This vitamin protects polyunsaturated fatty acids that are found in the

membrane phospholipids. It is also a potent peroxy radical scavenger protecting the biological cell membranes from free radical damage<sup>[3, 16, 38]</sup>. It neutralizes the elevated ROS production and modulates DNA oxidative damage in mammals. WGO intake leads to a rapid increase in vitamin E level in different body systems as in the brain, heart, liver, lung, kidney and spleen<sup>[19]</sup>. Moreover, WGO contains also carotenoids that contribute in this anti-oxidant effect<sup>[39]</sup>. Beyond this antioxidant effect, a previous study on WGO showed its anti-apoptotic effect through down regulation of the pro-apoptotic genes in irradiated rats<sup>[40]</sup>.

## DISCUSSION

Deltamethrin is a neurotoxic agent that induced degeneration and apoptosis in Purkinje neurons of the cerebellar cortex of rats. On the other hand, WGO exerted a neuroprotective effect against deltamethrin toxicity in rat cerebellar cortex through its anti-oxidant and anti-apoptotic activities.

## CONFLICT OF INTEREST

There are no conflicts of interest.

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## الملخص العربي

### زيت جنين القمح يخفف من الإصابة الناجمة عن دلتاميثرين في قشرة مخيخ الجرذ : دراسة هستولوجية وهستوكيميائية مناعية

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**المقدمة:** دلتاميثرين هومبيد حشرى يستخدم على نطاق واسع في مجال الزراعة. وله آثار عصبية سامة تسبب موت الخلايا المبرمج. زيت جنين القمح هو جزء ذو قيمة عالية من القمح التي ثبت أن لها آثار عصبية واقية ومضادة لموت الخلايا المبرمج.

**الهدف من البحث:** تقييم الدور المحتمل لزيت جنين القمح في تخفيف الإصابة الناجمة عن الدلتاميثرين في قشرة مخيخ الجرذ.

**مواد وطرق البحث:** استخدمت أربع مجموعات متساوية من ذكور الجرذان البيضاء البالغة كمجموعة ضابطة، ومجموعة زيت جنين القمح (100 ملجم / كجم)، ومجموعة دلتاميثرين (0.6 ملجم / كجم)، وكل من مجموعة الدلتاميثرين وزيت جنين القمح. أعطيت الجرعات عن طريق الفم يوميا لمدة ثلاثين يوما متتالية. تم معالجة عينات من المخيخ للدراسة النسيجية والهستوكيميائية المناعية. وأجريت دراسات مورفومترية وتحليل إحصائي للنتائج لجميع المجموعات.

**النتائج:** وبالمقارنة مع المجموعة الضابطة، أظهرت عينات من مجموعة دلتاميثرين ترتيب أحادي الطبقة المضطرب للخلايا بركنجى مع فقدان شكلها الطبيعي. لوحظت خلايا بركنجى منكشمة وداكنة مع اختفاء بعض الخلايا. وقد أظهرت الدراسة الهستوكيميائية المناعية وجود انخفاض له دلالة احصائية فى التفاعل المناعى لل بي-سى-ال 2 وكذلك وجود زيادة فى التفاعل المناعى لل باكس فى خلايا بركنجى. كما لوحظ وجود زيادة فى التفاعل المناعى للبروتين الليفى الحامضى فى كل طبقات القشرة. وفي المقابل، ظهرت تغييرات ضئيلة في المجموعة المعالجة بالتزامن بكلا من دلتاميثرين وزيت جنين القمح.

**الإستنتاج:** يمكن استنتاج أن زيت جنين القمح يخفف من التغيرات التركيبية التي يسببها الدلتاميثرين في قشرة مخيخ الجرذ.