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Oxidative and biochemical alterations induced by profenofos insecticide in rats

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ABSTRACT: Profenofos is a persistent and toxic organophosphorous insecticide. Animal's exposure to profenofos occurs via food and water. It is largely known to cause toxicity in various organs, such as the liver and brain. The present study was designed to explore the effect of oral administration of profenofos (26.53 and 53.07 mg/kg body weight /day for 28 days) on lipid peroxidation, endogenous antioxidants (GSH, and catalase), of the liver and brain and serum biochemical changes of male rats. Lipid peroxidation, as measured by thiobarbituric acid reactive substances, (TBARS) was increased, with a decrease in GSH level. A highly increase in catalase activity was observed in the 53.07mg/kg/day dose of profenofos. Furthermore, profenofos exposure were associated with depletion of serum levels of vitamin C, E, A and β- carotene. As compared to the results obtained in control groups the study showed that a lower concentration of serum proteins and albumin were accompanied by decreased globulin alpha 1 and beta along with an increased gamma 2 globulin; and the activity of serum GGT, LDH and concentrations of cholesterol, triglyceride, LDL and VLDL were higher, whereas level of HDL was lower. . [Nature and Science. 2009;7(2):1-15]. (ISSN: 1545-0740).

Keywords: Oxidative; biochemical alterations; insecticide; rat

This study suggests that although profenofos in low concentrations had oxidative stress and induced serum biochemical alteration in male rats.

Introduction

Organophosphorous compounds (OPs) have been widely used for a few decades in agriculture for crop protection and pest control, thousands of these compounds have been screened and over one hundred of them have been marketed for these purposes (Hassall, 1990; Chirions and Geraud-Pouey, 1996 and Geraud-Pouey et al., 1997).

The common use of insecticides in public health and agricultural schedules has caused severe environmental pollution and potential health hazards including severe acute and chronic cases of human and animal poisonings. **(Moghadamnia and Abdollahi 2002 and Abdullahi et al., 2004)**

Toxicities of OP insecticides cause adverse effects on many organs **(Gupta 2006)**. Systems that could be affected by OPs are the immune system, **(Neishabouri et al.,2004),** liver **(Akhgari et al.,2003)**, muscles **(Pournourmohammadi et al., 2005)** urinary system **(Rodrigo et al.,2001),** reproductive system **(Joshi et al., 2003)**, pancreas **(Hagar and Fahmy 2002)** and hematological system **(de Blaquiere et al., 2000)**.

Certain OP`s are also associated with carcinogenesis. Since DNA damage has been correlated with cancer development **(Hagmar et al., 1994),** genotoxicity studies have been carried out on OP`s **(Rupa et al., 1991 and Dolora et al., 1994).** It has been reported that OPs can induce oxidative stress by generating free radicals and altering antioxidant levels of the free radical scavenging enzyme activity **Sharma et al., (2005)**.

Profenofos [0-4-bromo-2-chlorophenyl-0-ethyl S-propyi phosphorothioate] is a broad spectrum organophosphate insecticide and acaricide. Its main physiological effect is the inhibition of cholinesterase (ChE) activity **(Anderson et al., 1977).**

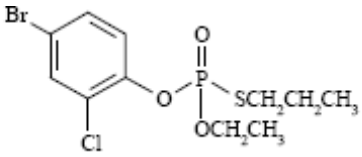
Biochemical Signs of hepatocellular injury and disturbed amino acid metabolism may be of value as markers of exposure to Profenofos, **Gomes et al., (1999).** Moreover, high doses of the profenofos induced tissue vacuolization, haemorrhage and hyperplasia of Kupffer cells in the liver. In addition, swelling of Bowman's capsules and tubular degeneration in the kidney were reported by **Fawzy et al., (2007).**Profenofos can induced oxidant stress which may be earlier diagnostic index in profenofos poisoning **(Lin et al.,2003).**

Therefore, the aim of this study was performed on the oxidative stress and biochemical effects of profenofos as an organophosphorous insecticide.

Materials and Methods

materials

Profenofos: is a pale yellow liquid, was provided by Central Agricultural Insecticides Laboratory (CAPL) Egypt.



Profenofos [O-(4-bromo-2-chlorophenyl)-O-ethyl-S-propyl phosphorothioate]

Laboratory animals:

Sixty adult male albino rats weighting (150 ± 10 g) were obtained from the farm of General Organization of Serum and Vaccine (Helwan Farm). The animals were housed in plastic cages in an air conditioned room where regular alternate cycles of 12 hr light and darkness were maintained and supplied with pelleted diet and tap water ad libitum. Animals were observed and signs of intoxication were recorded**.**

Experimental design:

The rats were divided into three groups of 20 rats each. Group (1) served as control and was given tap water only. Group II and III were given profenofos (72 EC, trade name: "Ictacrone") at a dose of 26.53 and 53.07 mg/kg body weight in 0.4 ml tap water through oral intubation. Dosages represent 1/8 and 1/4 LD, 50 respectively. LD, 50 value of profenofos (217.15 mg/kg b.W.) was determined orally (per os) according to **Weil, (1952).** The treatment was carried out for 28 days, the dose schedule being four days a week. Body weight was monitored twice a week and the dose was adjusted accordingly.

Sampling:

Individual blood samples were obtained after 28days from rats of each group, left to clot, sera were separated and kept at - 40°C for biochemical analysis Then animals were sacrificed and autopsy performed immediately; Brain, liver and kidney tissues were removed and washed with saline solution, then minced and homogenized (10% w/v) in ice-cold normal saline. The homogenate was centrifuged at 10,000xg for 20 min at 4C° and the resultant supernatant was used for antioxidant assay **(Chitra et al., 1999)**.

Biochemical Analysis

The biochemical assays of serum gamma glutamyle transferase (GGT) and lactic dehydrogenase (LDH) activities were determined according to methods of **(Szase et al., 1976)**. Triglyceride **(Wahlefeld, 1974),** cholesterol **(Watson, 1960)**, high density lipoprotein (HDL), low

density lipoprotein (LDL), **(Peace and Kaplan, 1987)**. Vitamin E, A, C and β carotene were performed according to **Henry et al., (1974).** Estimation of serum total protein and electrophoretic pattern were carried out after **SonnenWirth and Jaret (1980) and Davis (1964) ,**respectively.

Catalase activity; lipid peroxidation (TBARS) and reduced glutathione **(**GSH**)** in tissue were determined according to **Aebi, (1974); Ohkawa et al., (1979)** and **Ellman, (1959)**, respectively. The activity of catalase was expressed as IU per mg protein which estimated by **Bradford, (1976)**.

The obtained data were statistically analyzed using t`test after **Petrie and Watson (1999).**

Results and discussion

The present study has indicated the manner of organophosphorus poisoning in the experimental animals. Profenofos caused different symptoms of toxicity and revealed some biochemical changes especially in the enzymes activity of the liver and brain following two sublethal doses of profenofos in mice **(Saeed et al., 1995).** Animals dosed with 26.53 and 53.07 mg/kg body weight /day for 28 days showed significant signs of poisoning with in high dose.

It is reported by **Malkovics, (1995**) that OP, besides their inhibitory effect on AChE, also induce changes characteristic of oxidative stress (induced free radical)**.** Insecticides have been reported to induce production of reactive oxygen species and oxidative tissue damage **(Bagchi et al., and 1995**). All the major biomolecules like lipids, proteins, and nucleic acids may be attacked by free radicals, but lipids are probably the most susceptible **(Cheeseman and Slater1992)**. In this study, (table1) have shown that profenofos treatment result in a significant increase in MDA concentrations but a significant decrease was obtained in glutathione (GSH**)** levels of brain and liver tissues. The activity of CAT also increased in the brain, and liver (table 1), its increase was also remarkable in the brain of the rats as compared with control rats. These results were in agreement with **(Fortunato et al.,2006**and **Güney et al.,2007).**

Levels of MDA, a major oxidation product of peroxidized polyunsaturated fatty acids, have been considered as an important indicator of lipid peroxidation **(Kalender et al., 2004**).

Table 1: Effect of profenofos insecticides on some anti-oxidant parameter of liver and brain tissues of male rats.

	Malondiahyde (mM/100g)		GHS (µmol/mg protein)		Catalase (IU/mg protein)	
	Liver	brain	Liver	brain	liver	brain
G1	0.79± 0.27	1.18± 0.22	408.22± 29.9	589.3± 18.16	5.19 ± 0.41	5.14 ± 0.73
G2	1.63± 0. 26*	2.10± 0.35*	315.31± 24.2*	501.88± 15.22**	6.26 ± 0.19*	8.45 ± 1.01
G3	3.21± 0.42***	4.64± 0.63***	240.5± 29.3***	394.4± 17.57***	8.65± 0.93**	10.14± 1.02***

Results are expressed as means ± SEM (n =5), student 't' test

* P < 0.05 ** P < 0.01 *** P < 0.001

Glutathione is the cell's natural antioxidant, which destroys free radicals formed in cells. Significant dose-dependent depletion of GSH levels confirmed the potential of the profenofos to induce oxidative stress in brain and hepatic tissue**.** (**Rajeswary et al., 2007). Lin et al., (2003)** reported that profenofos was increased the antioxidase activities (SOD, CAT and GSH-Px,) earlier than the decease of ChE activity. They suggested that profenofos can result in the increases of the antioxidant enzyme activities which may be earlier diagnostic index in profenofos poisoning.

Vitamin E and A (Vit E and A) is the primary liposoluble antioxidant, which may have an important role in scavenging free oxygen radicals and in stabilizing the cell membranes, thus maintaining its permeability **(Bjørneboe *et al.*, 1990, Navarro *et al.*, 1999).** Vitamin E and A may also affect oxidative changes which occur in other cell organelles **(Ibrahim et al., 2000).** Vitamin C is a potent scavenger of free oxygen radicals and it has been shown that marginal Vit C deficiency results in intracellular oxidative damage in the animal **(Hudécová and Ginter 1992, Nagyová *et al.*,1994, Tatara and Ginter 1994).** Our results showed that profenfose intoxication decreased the concentration of Vit C, E, A and b-carotene as compared to control animals (table .2).

Table 2: Effect of profenofos insecticides on serum vitamin C, E, A and β- carotene of male rats.

	Vit. c (µg/dl)	Vit. E (µg/dl)	Vit. A (µg/dl)	β- carotene (µg/dl)
G1	0.77±0.09	501.47±18.12	43.41±3.04	22.17±2.63
G2	0.56± 0.11	420.61± 14.77**	40.01±3.13	19.74 ± 1.22
G3	0.38± 0.08**	376.93± 17.22***	33.15 ±3.81*	12.97± 2.62*

Results are expressed as means ± SEM (n =5), student 't' test

* P < 0.05 ** P < 0.01 *** P < 0.001

The observed depletion of serum levels of vitamin C, E and A can be explained by impairment of liver function and peroxidative processes caused by profeofose **(Rajeswary et al., 2007).**

Prolonged exposure of rats to profenofos was also shown by this study to cause a significant increase in γ-glutamyltransferase (GGT) and lactate dehydrogenase (LDH) Activities, compared with the control group (table 3). This finding agreed with those of **Irfan et al., 2002**. The significant elevations in enzymes activities of GGT and LDH indicate damage to any or all organs producing these enzymes such as liver or kidneys injuries **(Amacher, 2002 and Ncibi et al., 2008).**

At the same table (3) profenofos significantly increased the levels of serum cholesterol, triglyceride (TG), low density lipoprotein (LDL) and very low density lipoprotein (VLDL), while the level of high density lipoprotein (HDL) decreased. These results were in agreement with **Yousef et al., (2006)** and disagree with **Young and Koplovitz (1995).**

Table 3: Effect of profenofos insecticides on some serum enzymes and lipid profile of male rats

	G1	G2	G3
GGT u/l	4.48 ±0.42	6.51 ±0.45**	8.50 ±0.49***
LDH u/l	559.7±14.95	604.3±13.22*	711.5±16.45***
Cholesterol mg/dl	94.52± 6.47	118.54±5.21*	130.57±5.43**
Triglyceride mg/dl	130.36± 7.81	157.33±6.95*	175.91±8.11**
HDL mg/dl	60.05± 6.01	44.12± 2.98*	37.27±3.11**
LDL mg/dl	60.62± 8.11	105.86± 8.92***	127.37±8.47***
VLDL mg/dl	26.13±2.12	31.44± 1.98	35.07± 3.01*

Results are expressed as means ± SEM (n =5), student 't' test

* P < 0.05 ** P < 0.01 *** P < 0.001

Paraoxonase (PON) has been found to hydrolyzes various organophosphorus compounds **(Yamada et al., 2001)** and protect LDL and HDL from oxidation **(Mackness et al., 1998b; and Cao et al., 1999).** Organophosphorous inhibits PON activity in serum (**Ellenhorn, et al., 1997**). the activity of PON1 have been correlated with HDL-C and apoA-I levels, **Durrington, et al., (2001),** and therefore this may decrease the protective ability of PON to secure against free radicals **(Mackness et al., 2000)** Supporting the present data, the relationship between Op and the alteration of serum lipoprotein.

The electrophoretic pattern of serum protein (table 4) pointed out that profenofos provoked a significant lower serum protein concentration with higher of gamma-globulins and lower albumins and therefore A/G decreased.

Table 4: Effect of profenofos insecticides on serum total protein and electrophoretic pattern of male rats

	G1	G2	G3
T.protein	7.6 ±0.11	7.26 ±0.09*	7.02 ±0.1***
Albumin	2.46±0.22	1.98±0.12	1.81±0.14*
α. globulin	2.28 ±0.11	2.42±0.09	2.35±0.07
α_1	0.33 ±0.07	0.6±0.07*	0.71±0.08**
α_2	1.95 ±0.1	1.82±0.21	1.64±0.12*
β. globulin	1.56± 0.12	1.47± 0.1	1.46± 0.13
B1	0.98±0.09	0.77±0.06	0.68±0.04**
B2	0.58 ±0.05	0.7 ±0.04	0.78 ±0.06*
γ .globuli	2.28 ±0.11	2.42±0.09	2.35±0.07
γ_1	1.95 ±0.1	1.82±0.21	1.64±0.12*
γ_2	0.33 ±0.07	0.6±0.07*	0.71±0.08**
T.globulin	5.14 ±0.21	5.28 ±0.15	5.21 ±0.12
A:G ratio	0.48 ±0.03	0.37 ±0.02**	0.35 ±0.02***

Results are expressed as means ± SEM (n =5), student 't' test

* P < 0.05 ** P < 0.01 *** P < 0.001

Such changes in t. protein and albumin reflect hepatocellular injury and disturbed amino acid metabolism induced by profenofos **(Gomes et al.,1999 and Yousef et al.,2006).** As a matter of fact, free radicals can damage DNA and proteins, either through oxidation of DNA bases (primarily guanine via lipid peroxyl or alkoxyl radicals) or through covalent binding to DNA resulting in strand breaks and cross-linking. Reactive oxygen species can also induce oxidation of

critical Sulfhydryl (SH) groups in proteins and DNA, which will alter cellular integrity and function (**Fatemeh-Teimouri**, **2006**). Exposure to organophosphorus insecticides has been shown to inhibit all the cytoplasmic proteases and some of the lysosomal proteases in the liver tissue, the major site for insecticide metabolism. **(Mantle, 1997)**.

The previous study added that α2, β1, γ1 content were decreased while α1, β2, γ2 globulins were increased **(Kossmann and Magner-Krezel 1992 and Gupta et al., 1994).** These findings may be related to impact of profenofos towered the hepatic cells and immune system **(Yousef et al., 2006).** Organophosphate-induced immunosuppression was associated with severe cholinergic stimulation **(Pruett et al., 1992).**

The immunosuppression may result from direct action of acetylcholine upon the immune system or it may be secondary to the toxic chemical stress associated with cholinergic poisoning **(Zahran et al., 2005).** The increase in α1-globulin (alpha-1antitirypsin) might be attributed to tissue destruction and inflammatory reaction as mentioned by **Peasce and Kaplan, (1987).**

From this study we can conclude that treatment with profenofos induces oxidative stress, alteration in some biochemical parameters and the changes in anti-oxidant enzymes indicate a situation of enhanced oxy-radicals generation. The brain was the most sensitive organ to the oxidative stress induced by porfenofose.

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التغيرات التاكسديةو البيوكيميائية المصاحبة لاستخدام مبيد للبيروفنيوفوس في الجرذان

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**المعمل المركزي للمبيدات- الدقي

الملخص العربي

يعتبر البروفينوفوس احد المركبات الفسفورية العضوية السامة، تتعرض الحيوانات لمثل هذه المركبات عن طريق المآكل والمشرب وهى تحدث سميتها في كثير من الأعضاء مثل الكبد والمخ.

وقد أجريت هذه الدراسة لاستبيان تأثير تعاطي البروفينوفوس عن طريق الفم (26.53 &53.07 ملجم/كجم من وزن الجسم/يوم لمدة 28 يوما) على المالونالدهيد و مضادات الأكسدة (الكتاليز و الجلوتاثيون في خلايا الكبد والمخ وبالإضافة إلى التغيرات البيوكيميائية في مصل ذكور الجرذان.

وقد حدث ارتفاع حاد في تركيز الدهون المؤكسدة مع نقص في معدل الجلوتاثيون و أظهرت النتائج زيادة معنوية ملحوظة في مستوى إنزيم الكتاليز بالنسبة إلى الجرعة 53.07 ملجم/كجم من البروفينوفوس.

التعرض للبروفينوفوس يصاحبه نقصا في معدل الفيتامينات ا, هـ, سى, البيتا كاروتين فى مصل الدم. وبالمقارنة مع المجموعة الضابطة وجد نقص معنوي في معدل البروتين الكلي والالبيومين مصاحبة لنقص فى جلوبيولنات الالفا-1 والبيتا مع ارتفاع في معدل جاما-2 جلوبيولن ونشاط كل من GGT و LDH , الكوستيرول ,التراى جلسريد,VLDL, LDL بينما وجد نقص في HDL.

وقد بينت هذه الدراسة ان البوفينوفوس في الجرعات القليلة له أثر مؤكسد ويتسبب في التغيرات البيوكيمائية في ذكور الجرذان.