

BIOCHEMICAL EFFECTS OF PROFENOFOS IN THE NEW ZEALAND WHITE RABBIT.

R.A. Habiba¹ and S.M. Ismail²

1 Department of Food Science & Technology, 2

Department of Plant Protection, Faculty of Agriculture,

Suez Canal University, Ismailia, Egypt 41522

Received 3/12/1991 Accepted 27/1/1992.

ABSTRACT

The in vivo effects of drifted profenofos residues on acetylcholine esterase (AchE), transaminases (glutamic-oxaloacetic transaminase, GOT and glutamic-pyruvic transaminase, GPT), and the phosphatases (Acid phosphatase, Apase and alkaline phosphatase, Alkpase) in different organs (brain, liver, heart, muscles, and kidney) of the New Zealand white rabbits exposed to one-day feeding and continuous feeding on clover contaminated with the drifted pesticide were studied. The drifted profenofos was more potent to inhibit liver AchE than brain AchE. The studied transaminases and phosphatases showed different level of inhibition or stimulation depending on the type of organ and time of exposure to the investigated pesticide. The disruption of enzymes from the normal values denote biochemical impairment and lesions of tissues and cellular functions because these enzymes are involved in the detoxification processes, metabolism, and biosynthesis of energetic macromolecules needed for different essential functions. Therefore, It is necessary to take the drifted profenofos problem into consideration and to restrict its use to nonedible crops.

INTRODUCTION

The relative hazards of pesticide drift to nontarget organisms have been a subject of prime concern. Several reports showed direct evidence of drift damage to invertebrates from ground application of pesticides (Dobson, 1986, and Payne et al., 1988).

profenofos (Curacron), [O -(4-bromo-2-chlorophenyl)O -ethyl s-propyl phosphorothioate]], is a widely used organophosphorus insecticide in Egypt. It has been used to control various caterpillars, white fly and mites in various vegetable crops (Enan et al., 1981). The probability of contamination or exposure to profenofos through direct and indirect ways, as well as, possible toxicity are potential hazards. Minute amounts of this insecticide may reach the body and affect the physiological mechanisms involved in normal behavior of tissues. A few attempts have been made to understand the possible mechanisms of action. Enan et al.(1981) and Leader and Casida (1982) have reported that profenofos causes inhibition of cholinesterase. Ghazal et al.(1984) concluded that profenofos exerts an inhibitory effect on both adrenergic and cholinergic transmission. Besides, the authors added that profenofos posses a direct inhibitory effect on the smooth muscles. On the other hands, reports on the toxicity of spray drift deposited on adjacent crops that are used as animal feed are scarce. As indicated in a previous publication (Ismail et al., 1991), that with such small acquisition of agriculture land at Ismailia Governorate, problems of pesticide drift in adjacent field are enormous. The study showed that profenofos residues have the ability to persist in clover and different organs of the New Zealand white rabbits fed on clover contaminated with drifted profenofos. In addition, considerable levels of profenofos were able to resist heat treatments normally used in food processing.

The aim of the present work was to study the effect of drifted profenofos on some enzymes in different organs of the New Zealand white rabbits exposed to one-day and continuous feeding on clover contaminated with drifted profenofos resulted from the application of such pesticide on an adjacent tomato field.

MATERIALS AND METHODS

Materials:

Formulated profenofos (72% EC) with the trade mark "Selecron 720 EC" was applied at both the manufacturer (Ciba-Geigy) and the Egyptian Ministry of Agriculture recommended rate (0.75 liter/feddan). A blower sprayer fitted with one nozzle boom was used to treat tomato field which was adjacent to clover field. The clover contaminated with the drifted profenofos sprays was fed to male New Zealand white rabbits each weighing 0.8-1.0 kg. The rabbits were first adapted to the clover ration, then divided into 3 groups. The first group was exposed to one-day feeding on clover contaminated with the drifted profenofos (400g) followed by uncontaminated clover feeding for 7 more days. After 1, 2, 4, and 8 days, rabbits were killed by decapitation. The dorsal muscles, liver, heart, brain, and kidney were dissected and homogenized in 10 volumes (w/v) of ice-cold physiological saline using "Virtus 23" mechanical homogenizer. The homogenates were centrifuged at 6000 x g for 20 minutes at 4°C. The supernatants were kept at -20°C until used for enzyme assays. The second group of rabbits was continuously fed on the clover contaminated with drifted profenofos (400g daily) for 12 days. After 1, 3, 6, and 12 days, the rabbits were decapitated. The dorsal muscles, liver, heart, brain, and kidney were taken for enzyme assay as in the first group. The third group of rabbits was fed on uncontaminated clover and taken as control.

Methods:

1. Determination of profenofos residues. - Profenofos residues in the contaminated clover as well as in different rabbit organs (dorsal muscles, liver, heart, brain, and kidney) were taken from our previous work (Ismail et al., 1991).
2. Assay of enzymes - AchE was determined according to Ellman et al. (1961). GOT and GPT were assayed according to the Reitman and Frankel (1957) method. Apase was determined according to Fishman and Lerner (1953), and Alkpase was determined according to the method of Bessey et al. (1946) using a commercial available kit from Boehringer Mannheim GmbH Diagnostica.

Protein content of the enzyme extracts was determined using the dye binding method "Bio-Rad protein assay" according to Bradford (1976).

RESULTS AND DISCUSSION

1. In vivo effect of drifted profenofos on brain and liver acetylcholine esterase (AChE) in the New Zealand white rabbit after one-day feeding on profenofos-contaminated clover.

AChE activity was determined in brain and liver. The obtained results of untreated rabbits indicated a higher specific activity (56.75 u/mg protein) in the brain as compared to that of liver (11.50 u/mg protein). As shown in Table (1), AChE specific activity in the brain and liver of the New Zealand white rabbit was inhibited when the rabbits were exposed to one-day feeding on contaminated clover. The level of inhibition was more noticed in the liver than in the brain. The percent of AChE inhibition in the brain decreased from 19.85% after one day of exposure to 0.8% after 8 days. However, the rate of % inhibition in liver was slower than that of the brain. The % of AChE inhibition in the liver; as compared to control; was 69.25, 58.1, and 46.9% after 1, 4, and 8 days, respectively. It was also noticed from residue studies (Table 2) that the degradation rate of profenofos in liver was slow. These results are in agreement with those of Enan et al. (1981) who reported in-vivo inhibition of liver and brain AChE of white male rats by sublethal dose of three organophosphorus insecticides. The authors added that profenofos was not a potent AChE inhibitor. In another study, on common carp fish, El-Gendy et al. (1990) reported the effect of; two organophosphorus compounds; Pyrazophos and Glyphosate on brain AChE and found that they caused 78.36 and 49.71% inhibition by half LC50 and 80.73 and 50.5% inhibition by LC50, respectively.

2. Accumulated effect of drifted profenofos on brain and liver acetylcholine esterase (AChE) in the New Zealand white rabbit during continuous feeding on profenofos-contaminated clover.

As shown in Table (3), the inhibition of AChE activity in the brain of continuously fed rabbits on profenofos contaminated clover increased from 19.85% after one day to 77.2% inhibition after 6 days of exposure, then, the inhibition relatively decreased to 69% at the end of 12 days of continuous feeding on clover from the same drifted profenofos field. This is may be due to the dilution of the studied pesticide residues (Table 4) governed by clover growth and environmental factors. Liver AChE was also inhibited by the drifted profenofos, but the level of inhibition was slightly

varied (69.25 - 72.05%) during the studied 12 days of continuous feeding. This could be due to the balance between the pesticide intake and its degradation by the biochemical detoxification processes occurred in liver. However, profenofos residues detected in liver (Table 5) showed accumulation of the investigated pesticide in the liver indicating the inability of liver to degrade such pesticide and supporting the results of Enan et al. (1981) that profenofos had a moderate effect on liver AchE. It is also reported that the organophosphorus compounds produce their acute toxic action by inhibiting cholinesterase (O'Brien, 1969 and Aldridge, 1971). Therefore, the inhibition of AchE in rabbits exposed to drifted profenofos may serve as an indicator of hazard due to the application of this pesticide in the environment. El-Gendy et al.(1990) reached similar conclusion from a study on common carp using the sublethal doses of two organophosphorus compounds.

3. In vivo effects of drifted profenofos on Transaminases and phosphatases in different organs of the New Zealand white rabbits after one-day feeding on profenofos-contaminated clover.

3.1 Transaminases

It was evident from the obtained results (Table 6) that for the untreated rabbits, liver contained the highest activities of GOT and GPT, followed by the heart. Muscles showed the least activity for GOT, whereas, the brain showed the least activity for GPT.

In the treated rabbits, the GOT specific activity decreased in the brain, muscles, and kidney after one-day feeding on clover contaminated with drifted profenofos. This effect was continued until the end of the studied 8 days. However, the specific activity of GOT in the heart decreased after the first 2 days, then, the enzyme was stimulated. The liver GOT specific activity was slightly induced at the beginning, then the normal activity was restored. Results of profenofos residues in muscles (Table 2) paralleled to the level of GOT activity in the muscles. Moreover, the decrease in profenofos residues in liver (Table 2) was paralleled to the enzyme activity in the same tissue (Table 6).

GPT specific activity decreased in the kidney, brain and heart of the New Zealand white rabbits exposed to one-day feeding on contaminated clover with the drifted profenofos. Kidney GPT specific activity decreased until the end of the tested period, while the enzyme in the brain and heart was stimulated after 4 days.

Table (1). In vivo effects of drifted profenofos on brain and liver acetyl cholinesterase (AChE) in New Zealand white male rabbits after one-day feeding on profenofos-contaminated clover

Day(s) after treatment	% Inhibition of AChE activity	
	Brain	Liver
1	19.85 $\pm$ 2.65	69.25 $\pm$ 2.75
2	12.80 $\pm$ 4.40	60.90 $\pm$ 5.60
4	12.70 $\pm$ 4.50	58.10 $\pm$ 2.80
8	0.80 $\pm$ 0.10	46.90 $\pm$ 2.80

Specific activities of AChE in brain and liver of untreated rabbits were 56.75 $\pm$ 7.80 and 11.50 $\pm$ 1.38 micromole hydrolyzed acetylthiocholine iodide/mg protein /hr., respectively. Results are expressed as mean $\pm$ standard deviation of 2 experiments each performed in triplicate.

Table (2) Profenofos residues in liver of white New Zealand rabbit after one-day feeding on clover contaminated with drifted profenofos

Time (days)	Profenofos residues (ng/g)
	"mean $\pm$ standard deviation"
1	35.6 $\pm$ 1.42
2	27.3 $\pm$ 1.37
4	25.6 $\pm$ 1.02
8	22.8 $\pm$ 1.14

Table (3) Effects of drifted profenofos on brain and liver acetyl cholinesterase (AChE) in New Zealand white male rabbits during continuous feeding on profenofos-contaminated clover

Day (s) of treatment	% Inhibition of AChE activity	
	Brain	Liver
1	19.85 $\pm$ 2.65	69.25 $\pm$ 2.75
3	48.30 $\pm$ 2.05	66.45 $\pm$ 5.55
6	77.20 $\pm$ 3.80	67.15 $\pm$ 0.45
12	69.00 $\pm$ 5.20	72.05 $\pm$ 5.50

Specific activities of AChE in brain and liver of untreated rabbits were 56.75 $\pm$ 7.80 and 11.50 $\pm$ 1.38 micromole hydrolyzed acetylthiocholine iodide/mg protein /hr., respectively. Results are expressed as mean $\pm$ standard deviation of 2 experiments each performed in triplicate.

Table (4) Drifted profenofos residues in clover (*Trifolium alexanderinum*) at different intervals from spraying of adjacent field.

Time (days)	Profenofos residues (ng/g) "mean $\pm$ standard deviation"
zero	11.48 $\pm$ 0.69
1	5.29 $\pm$ 0.26
2	3.63 $\pm$ 0.18
4	3.56 $\pm$ 0.14
8	2.13 $\pm$ 0.13
16	0.86 $\pm$ 0.05
32	n.d.*

* not detected

Table (5) Accumulated Profenofos residues in liver of New Zealand white rabbit at different intervals of continuous feeding on clover contaminated with drifted profenofos

Time (days)	Profenofos residues (ng/g) "mean $\pm$ standard deviation"
1	35.6 $\pm$ 1.42
3	47.5 $\pm$ 2.38
6	58.1 $\pm$ 2.91
12	63.1 $\pm$ 3.79

Enzyme	Organs	Specific activity (means $\pm$ S.D.)				
		Control	1	2	4	8
GOT	Brain	55.8 $\pm$ 0.3	30.0 $\pm$ 0.3	22.2 $\pm$ 0.5	39.5 $\pm$ 0.4	23.0 $\pm$ 0.5
	Liver	440 $\pm$ 13.2	480 $\pm$ 12.8	449 $\pm$ 12.5	456 $\pm$ 14.7	430 $\pm$ 8.2
	Heart	93.6 $\pm$ 2.8	53.2 $\pm$ 1.6	42.4 $\pm$ 1.6	156 $\pm$ 5.2	208 $\pm$ 6.8
	Muscle	28.8 $\pm$ 0.6	22.0 $\pm$ 0.5	17.5 $\pm$ 0.5	10.0 $\pm$ 0.7	0.7 $\pm$ 0.1
	Kidney	49.5 $\pm$ 2.5	17.5 $\pm$ 0.8	12.4 $\pm$ 0.8	31.0 $\pm$ 1.5	10.6 $\pm$ 0.4
GPT	Brain	3.0 $\pm$ 0.2	2.7 $\pm$ 0.1	3.0 $\pm$ 0.2	3.8 $\pm$ 0.4	11.2 $\pm$ 0.4
	Liver	15.6 $\pm$ 0.4	20.7 $\pm$ 0.3	24.1 $\pm$ 0.3	15.0 $\pm$ 0.3	16.8 $\pm$ 0.6
	Heart	14.9 $\pm$ 0.5	13.3 $\pm$ 0.1	12.4 $\pm$ 0.8	12.8 $\pm$ 0.3	16.3 $\pm$ 0.5
	Muscle	9.3 $\pm$ 0.1	10.2 $\pm$ 0.2	11.9 $\pm$ 0.2	7.2 $\pm$ 0.10	8.1 $\pm$ 0.3
	Kidney	13.9 $\pm$ 0.3	7.0 $\pm$ 0.2	9.1 $\pm$ 0.5	10.6 $\pm$ 0.4	6.8 $\pm$ 0.3
Apass	Liver	10.1 $\pm$ 0.5	10.4 $\pm$ 0.3	13.3 $\pm$ 0.4	13.9 $\pm$ 0.5	9.8 $\pm$ 0.3
	Kidney	17.5 $\pm$ 0.5	16.2 $\pm$ 0.6	12.6 $\pm$ 0.4	14.6 $\pm$ 0.6	13.8 $\pm$ 0.5
Alkpase	Liver	21.6 $\pm$ 0.6	10.8 $\pm$ 0.3	19.2 $\pm$ 0.6	21.0 $\pm$ 0.6	19.2 $\pm$ 0.6
	Kidney	110.8 $\pm$ 4.5	114 $\pm$ 4.2	118.2 $\pm$ 4.0	131.8 $\pm$ 3.1	140 $\pm$ 4.8

GOT (Glutamic-oxaloacetic transaminase).- units/liter

GPT (Glutamic-pyruvic transaminase) - units/liter

Apase (Acid phosphatase) - units/mg protein

Alkpase (Alkaline phosphatase) - units/mg protein

Table (7). In vivo accumulated effect of drifted profenofos on some enzymes in different organs of New Zealand white male rabbits during continuous feeding on profenofos-contaminated clover

Enzyme	Organs	Specific activity (means $\pm$ S.D.)				
		Control	1	3	6	12
GPT	Brain	3.0 $\pm$ 0.20	2.7 $\pm$ 0.1	1.3 $\pm$ 0.04	1.2 $\pm$ 0.10	0.4 $\pm$ 0.0
	Liver	15.6 $\pm$ 0.4	20.0 $\pm$ 0.8	7.4 $\pm$ 0.25	4.9 $\pm$ 0.15	2.4 $\pm$ 0.1
	Heart	14.9 $\pm$ 0.5	13.2 $\pm$ 0.1	7.8 $\pm$ 0.20	7.3 $\pm$ 0.25	7.8 $\pm$ 0.2
	Muscle	9.3 $\pm$ 0.60	10.2 $\pm$ 0.2	2.3 $\pm$ 0.10	2.0 $\pm$ 0.06	2.1 $\pm$ 0.1
	Kidney	13.9 $\pm$ 0.30	7.0 $\pm$ 0.2	3.7 $\pm$ 0.12	4.2 $\pm$ 0.10	3.8 $\pm$ 0.1
GOT	Brain	55.8 $\pm$ 0.35	30.0 $\pm$ 0.3	49.5 $\pm$ 1.5	160 $\pm$ 4.6	51.0 $\pm$ 1.6
	Liver	440 $\pm$ 13.20	480 $\pm$ 12.8	480 $\pm$ 14.5	240 $\pm$ 7.5	78.5 $\pm$ 3.0
	Heart	93.6 $\pm$ 2.81	53.2 $\pm$ 1.6	42.6 $\pm$ 1.5	34.2 $\pm$ 1.1	23.0 $\pm$ 0.9
	Muscle	28.8 $\pm$ 0.60	22.0 $\pm$ 0.5	19.8 $\pm$ 0.8	140 $\pm$ 4.2	16.0 $\pm$ 0.5
	Kidney	49.5 $\pm$ 2.50	17.5 $\pm$ 0.8	23.0 $\pm$ 0.8	20.7 $\pm$ 0.9	21.0 $\pm$ 0.8
Apass	Liver	10.15 $\pm$ 0.35	10.3 $\pm$ 0.3	8.3 $\pm$ 0.3	12.6 $\pm$ 0.5	10.8 $\pm$ 0.3
	Kidney	17.5 $\pm$ 0.52	16.2 $\pm$ 0.6	14.8 $\pm$ 0.6	19.1 $\pm$ 0.8	17.5 $\pm$ 0.7
Alkpase	Liver	21.6 $\pm$ 0.60	10.8 $\pm$ 0.3	15.0 $\pm$ 0.4	10.2 $\pm$ 0.3	15.6 $\pm$ 0.4
	Kidney	110.8 $\pm$ 4.5	114 $\pm$ 4.2	174 $\pm$ 5.2	164.4 $\pm$ 4.8	158 $\pm$ 4.5
GOT. (Glutamic-oxaloacetic transaminase).- units/liter						
GPT (Glutamic-pyruvic transaminase) - units/liter						
Apass (Acid phosphatase) - units/mg protein						
Alkpase (Alkaline phosphatase) - units/mg protein						

GOT. (Glutamic-oxaloacetic transaminase).- units/liter

GPT (Glutamic-pyruvic transaminase) - units/liter

Apass (Acid phosphatase) - units/mg protein

Alkpase (Alkaline phosphatase) - units/mg protein

Liver and muscle GPT specific activities were induced after the first 2 days, then the enzyme normal activity was restored by the end of the 8 days (Table 6). These results are in agreement with those of Enan et al. (1981) who studied the effects of sublethal doses of organophosphorus insecticides on the transaminases in male white rats.

The disruption of transaminases from the normal values denote biochemical impairment and lesions of tissues and cellular function because they are involved in the detoxification process, metabolism, and biosynthesis of energetic macromolecules needed for different essential functions (Tordior and Van Heemstra-Lequin, 1980).

3.2. Phosphatases

As shown in Table (6), the acid phosphatase specific activity was induced in the liver of the New Zealand white rabbits exposed to one-day feeding on clover contaminated with the drifted profenofos, then decreased after 8 days to about normal activity. This trend, induction of Apase, is supported by similar observations (Sexena and Sarin, 1980 and El-Sebae et al., 1987). With regard to kidney Apase specific activity, the enzyme showed a gradual decrease with time. These results are in agreement with those of Micol et al. (1980).

Liver Alkpase specific activity was inhibited as the rabbits exposed to the contaminated clover, then the enzyme normal activity was restored. However, the kidney Alkpase specific activity showed an expected trend of stimulation as reported by Enan et al. (1981). The site of detoxification processes of toxic chemicals entering the body is mainly in liver (Hinderer and Menzer, 1976). Therefore, the deviation of the liver enzymes from their normal values is due to the toxic effect of profenofos.

4. In vivo effects of drifted profenofos on Transaminases and phosphatases in different organs of the New Zealand white rabbits during continuous feeding on profenofos-contaminated clover.

4.1 Transaminases

As shown in Table (7), GPT specific activity was inhibited in all tested organs. The inhibition effect was increased as the rabbits continued to eat the contaminated clover. The same trend was noticed for the GOT specific activity except that the GOT inhibition was lower than that found for GPT.

4.2. Phosphatases

Liver acid phosphatase (Apase) specific activity of the New Zealand white rabbits decreased after 3 days of continuous feeding on contaminated clover. Then, the enzyme activity was stimulated after 6 days. The normal enzyme activity was restored after 12 days (Table 7). The same trend was observed for the kidney Apase. Similar results were reported by Enan et al. (1981) who studied the effect of profenofos on male white rats. However, in this work the time needed to restore the enzyme normal values was longer than that of Enan and his coworkers. This may be due to the accumulation effect resulted from the continuous feeding of rabbits on the contaminated clover. Other studies have shown that inhibitors of protein synthesis prevent the elevation of rat liver phosphatases induced by poisoning with toxic compounds (Murphy, 1965 and Greengard et al., 1963).

Liver alkaline phosphatase (Alkpase) specific activity was inhibited as the rabbits continue to eat the contaminated clover. The rate of inhibition decreased by time. However, the kidney Alkpase specific activity showed gradual increase over the 12 days. The highest level of Alkpase was noticed after the third day of continuous feeding on clover contaminated with the drifted profenofos. As mentioned before, the detoxification of toxic materials occurs mainly in liver (Hinderer and Menzer, 1976 and Guyhrle and Hodgson, 1987). Thus, the toxic effect of profenofos could affect the liver and kidney cells and consequently disturb their functions.

The above results and the previous conclusion of Ghazal et al. (1984) that profenofos exerts an inhibitory effect on both adrenergic and cholinergic transmission as well as its direct inhibitory effect on the smooth muscles lead to the conclusion that direct and indirect contamination of edible crops by profenofos may alter the normal metabolism and cause elevated concentration of undesirable compounds in edible animal tissues. Therefore, there is an increasing demand to minimize the adverse effects of these chemicals on environmental quality. Profenofos and similar pesticides should be restricted to non-edible crops. Besides, implication of a reasonable buffer zone in case of the necessity of using such pesticide may reduce the possible adverse effects of the drift.

REFERENCES

- Aldridge, W.N. (1971). The nature of the reaction of organophosphorus compounds and carbamate with esterases. *Bull.WHO* 44:25-30.
- Bessey, A.D.; O.H. Lowry; and M.I. Borck (1946). Determination in serum with p-nitrophenylphosphate. *J. Biol. Chem.* 146: 321-329.
- Bradford, M.M. (1976). A rapid and sensitive method for the quantitation of microgram quantities of protein utilizing the principle of protein-dye binding. *Analytical Biochem.* 72:248-254.
- Dobson, C. M. (1986). Insecticidal drift from sprayers and the effect on beneficial arthropods in winter wheat. Ph.D. thesis, University of London, England.
- El-Gendy, K.S; N.M. Ali; N.S. Ahmed; and A.H. El-Sebae (1990). Comparative toxicity of some pesticides to common carp and their effects on biochemical targets in living tissues. *Pest. Cont. Env. Sci.* 2:29-41.
- Ellman, G.L.; K.D. Courteny; V.Jr. Andress; and R.M. Featherstone (1961). A new and rapid colorimetric determination of acetyl cholinesterase activity. *Biochem. Pharmacol.* 7: 88-95.
- El-Sebae, A.H.; N.S. Ahmed; K.S. El-Gendy; A.S. El-Bakary; and S.A.Soliman (1987). Methamidophos (Tamaron): A delayed neurotoxic compounds to man, but negative to chickens. 2nd Nat. Conf. of Pests and Dis. of veg. and fruits, Ismailia, Oct. 474-485
- Enan, E.E.; O.H.Enan; and A. El-Sebae (1981). Biochem. targets affected by sublethal doses of organophosphorus insecticides. *International Pest Control* Sept./Oct. 1981.
- Fishman, W.H.; and F. Lerner (1953). A method for estimating serum acid phosphatase of prostatic organ. *J. Biol. Chem.* 200: 89-97.
- Ghazal, A.; M.M. Mohy El-Din; and F.M. Sharabi (1984). Inhibitory action of profenofos on myoneural transmission. *J. Egypt. Soc. Toxicol.* 1: 101-114.
- Greengard, O.; M.A.Smith; and G. Smith (1963). ACS: Relation of cortisone and synthesis of ribonucleic acid to induced and developmental enzyme formation. *J. Biol. Chem.* 238: 1548.
- Guyhrle, F.E.; and E. Hodgson (1987). Elimination of toxicants. In *Modern Toxicology*. (Hodgson, E. and P.E. Levi, Eds.) Elsevier Science Publishing Co., Inc., New York, pp. 123-131.

- Ismail, S.M; M.T.Ahmed; and R.A. Habiba (1991). Drifted profenofos on clover and its residues in fresh and processed tissues of the New Zealand white rabbit (*Oryctolagus cuniculus*). Fourth Arab Congress of plant Protection, Cairo 1-5 Dec., 1991
- Leader, H.; and J.E. Casida (1982). Resolution and biological activity of the chiral isomers of O-(4-bromo-2-chlorophenyl) O-ethyl S-propyl phosphorothioate (profenofos insecticide. *J. Agric. Food Chem.* 30: 546.
- Micol, Y.B.; F. Roux; F. Decoitre; and E.P. Fournier (1980). Liver enzyme induction in lindane and captan treated rats. *Food Cosmet. Toxicol.* 4: 377.
- Murphy, S.D. (1965). Mechanism of the effect of acrolein on rat liver enzymes. *Toxicol. Appl. Pharmacol.* 6 : 355.
- O'Brien, R.O. (1969). Biochemical effects phosphorylation and carbamylation of cholinesterase. *New York Academic Science* 160:204-214.
- Payne, N. J.; B.V. Helson; K.M.S. Sundarain; and R.A. Fleming (1988). Estimating buffer widths for pesticide applications. *Pestic. Sci.*, 24, 147-161.
- Reitman, S.; and S. Frankel (1957). A colorimetric method for the determination of serum glutamic oxaloacetic and glutamic pyruvic transaminases. *Amer. J. Clin. Pathol.* 28: 56-63.
- Sexena, A.K.; and K.Sarin (1980). Histopathological and biochemical changes in the liver and testes of desert gerbil after repeated exposures of Thimet (Phorate). *Toxicol.* 18: 133.
- Tordior, W.F.; and E.A. VanHeemstra-Lequin (1980). Field studies monitoring exposure and effects in the development of pesticides. Elsevier, Amsterdam, Oxford, New York., p. 207.

الملخص العربي

التأثيرات البيوكيميائية لمبيد البروفينوفوس في الأرنب النيوزيلندي الأبيض

تم تقدير نشاط الإنزيمات استيايل كولين استريز، ترانس أمينيز (جلوتاميك اكسالو اسيتيك ترانس أمينيز وجلوتاميك بيروفيك ترانس أمينيز) والفوسفاتيز (اسيد فوسفاتيز والكالاين فوسفاتيز) في عدة أنسجة من الأرنب النيوزيلندي الأبيض. كذلك تم تقدير تأثير متبقيات مبيد البروفينوفوس (داخل الأنسجة) على نشاط الإنزيمات السابقة وذلك عقب تناول الأرنب برسيم ملوث بالمبيد المذكور نتيجة رش هذا المبيد على حقل طماطم مجاور. شملت الدراسة كل من ١. تأثير تعرض الأرنب الى جرعة واحدة من البرسيم الملوث، ٢. تأثير التعرض المستمر للأرنب للبرسيم الملوث وقد أوضحت النتائج تغير نشاط الإنزيمات التي درست نتيجة للتعرض للبروفينوفوس وقد زادت هذه التغير في حالة التعرض المستمر للمبيد.

تغير نشاط الإنزيمات السابق ذكرها بالزيادة أو النقصان عن قيمها الطبيعية نتيجة للتعرض للبروفينوفوس يدل على اختلال الأنظمة الحيوية بالخلايا والأنسجة، لأن تلك الإنزيمات تشترك في كثير من العمليات الحيوية الهامة بالجسم من إزالة المواد السامة أو بناء المواد الغنية بالطاقة المرتبطة بالعمليات الميتابوليزمية المختلفة وبالتالي فإنه لابد من الاهتمام بمشكلة التلوث بالبروفينوفوس وتحديد استعماله فقط مع المحاصيل التي لا تؤكل (القطن على سبيل المثال..).