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# Diazinon residue in liver and kidney with special references to their function in red Baladi rabbits

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## ABSTRACT

The existed residues of diazinon low concentration (DLC) in liver samples after 0, 1, 3, 7, 15 and 21 days from dipping of rabbits were 0.05, 1.2, 3.8, 1.8, 0.87 and 0.0 ppm in kidney and 0.01, 0.9, 2.9, 1.5, 0.31 and 0.0 ppm in liver, respectively. In case of dipping of rabbits in diazinon high concentration (DHC), the residues were elevated and the detected concentrations were 0.07, 4.5, 6.17, 4.62, 1.2 and 0.03 ppm in kidney samples after 0, 1, 3, 7, 15 and 21 days of exposure, respectively. Also the concentrations of diazinon residue in liver were 0.04, 3.01, 4.7, 3.85, 1.1 and 0.02 ppm, respectively, after the same pre-exposure periods. Effect of diazinon on aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), creatinine and urea were investigated in red Baladi rabbits. Seventy-two animals were distributed into three groups, the first dipped in tap water and served as control group. The second and the third groups were dipped in diazinon (0.6 and 3 mg / L water) and served as DLC and DHC, respectively for 10 sec. The previous step was repeated after 10 days. The animals were sacrificed by jugular vein incision after 0, 1, 3, 7, 15 and 21 days following the second dipping of rabbits in diazinon. Both tested concentrations induced elevation of ALT, ALP and urea, while AST values were significantly ($p<0.05$) decreased. Also, a discernable change in creatinine levels. Meanwhile AST, ALT, ALP and urea were fluctuated during the time of treatment, however creatinine values were increased. The consistent correlation between the dizinon residues and the impairment of liver and kidney functions in rabbit's, proof the possible alteration of animal physiology and its production.

## INTRODUCTION

The sheep scab mite *Psoropets ovis* is a serious pest of sheep, causing distress to the animals and economic loss to the farmer. It was effectively controlled in the 1959 by the introduction of sheep dips containing hexachlorocyclohexane. This compound, however, has been banned for use in sheep-dips because of its persistent residues, which could contaminate the animal meat. It has been largely replaced by less persistent compounds such as organophosphates (OP's), propetamphos and diazinon (Blanchflower *et. al.*, 1990). These compounds effectively controlled sheep scab when they were used properly at the recommended levels.

The exposure to OP's can be assessed by a number of methods including determinations of blood cholinesterase levels, residues of intact compounds in blood and tissues, and urinary alkyl phosphate metabolites. Depression of cholinesterase levels has been routinely used as a measure of exposure but has been found to be of low sensitivity and specificity (Bradway *et. al.*, 1977; Shafik *et. al.*, 1973). Moderate depression of cholinesterase is difficult to be attributed to a specific cause, because numerous factors may affect its activity (Kachmar and Moss, 1976). Determination of dialkyl phosphate levels provides a more accurate and sensitive assessment of exposure to OP's (Mount, 1984; Drevenkar *et. al.*, 1991; Brokopp *et. al.*, 1981). Analysis of intact OP's insecticide residues in animal tissues has been a common practice in veterinary diagnostic investigation (Osweiler *et. al.*, 1985). Therefore, the present study was carried out to investigate the possible toxicity of diazinon on some biochemical parameters and to measure its residues in rabbits's tissues.

## MATERIALS AND METHODS

**Diazinon:** *Phosphorothioic acid O,O-diethyl O-[6-methyl-2-(1-methylethyl)-4-pyimidinyl] ester* EC 60%, obtained from Siba Geigy (B.z.n.,Sarolex) All India Medical Co. and other chemicals were obtained from Sigma Chemical Co. (St. Louis, Missouri, USA).

Seventy-two red Baladi rabbit bucks (1.4 ± 0.12 kg live body weight) 6 months of age, were individually housed in universal galvanized wire batteries with feed and water *ad libitum*. A commercial balanced pelleted ration for breeding rabbits containing 18 % crude protein, 14 % crude fiber,

2 % fat and 2600 kcal DE/kg feed was used. Clean fresh tap water was provided all times. Rabbits were distributed randomly into three groups, the first dipped in tap water and served as control group. The second and the third groups were dipped in diazinon (0.6 and 3 mg / L water), and served as DLC and DHC, respectively for 10 sec. The previous step was repeated after 10 days. The animals were sacrificed by jugular vein incision after 0, 1, 3, 7, 15 and 21 days following the second dipping of rabbits in diazinon and blood, liver and kidney tissues were obtained.

**Blood analysis:** Blood samples were obtained by sacrificing the animals by jugular vein and were placed in 12 x 75 $mm^2$ heparinized tubes immediately and allowed to clot at 4°C. Plasma was obtained by centrifuging of blood at 3,000 rpm for 20 min. and then was stored at -20°C until used for analysis. Plasma was used for aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), urea and creatinine detreminations. AST, ALT and ALP were measured according to the method described by Reitman and Frankel (1957), urea and creatinine were determined using commercially available kits obtained from Bio ADWIC, Egypt (Patton and Crouch, 1977; Larsen, 1972).

**Analytical method for determination of diazinon residue:**

**1. Preparation of the standard solution:** A standard solution of diazinon was prepared by weighting and quantitatively transferred into 100 ml volumetric flask using n-hexane to prepare a stock solution. The solution was diluted to make a stock solution of 10 µg / ml using the same solvent. The stock solution was serially diluted using n-hexane to get a working solution (1 ηg/µl) for the quantitative determination. The standard peak of the insecticide appeared at 6.14 min (Nabrawy and Cary, 1988).

**2. Extraction of pesticide residues:** Half gram of each liver and kidney were homogenized in a suitable glass jar for two minutes with 100 ml of acidic acetone [385 ml acetone + 15 ml aqueous $H_2SO_4$ (1:2)] using a homogenizer Polytron (Type / PT45 / 80 Nr 9119). The homogenate was filtered through a filter paper (S & S shark-skin) under suction using a buchner funnel. The jar and filter cake was rinsed twice with 15 ml of acetone. The filtrate was quantitatively transferred into 150 ml conical flask. The acetone was evaporated on the water bath 30°C using a rotary evaporator. The acetone-free sample extract was extracted twice with 50 ml petroleum ether using 250 ml separator funnel and concentrated to a volume

of 1ml using the rotary evaporator The remaining solvent was allowed to evaporate under a fume hood. The residue was dissolved in 10 ml n-hexane and preserved in a freezer for column clean up (Nabrawy and Cary, 1988).

**3. Clean up:** The step was done according to the method adopted by El-Nabarawy and Cary (1988). A chromatographic column (400 x 100mm) was packed to a length of 5mm with glass wool; 4 gm activated florisil; activated for 4 hr at 640°C and kept at 140°C overnight and 2 gm sodium sulphate anhydrous. The column was wetted with 10 ml n-hexane. The residue was quantitatively transferred into the column using 2 - 5 ml portions of n-hexane. Thirty five ml n-hexane was added to the column, then 65 ml of diethyl ether : petroleum ether mixture (1 : 1) v / v were used to elute the residue into 250 ml conical flask.

**4. Determination and recovery:** The eluted portion was evaporated to approximately 1ml on a rotary evaporator, the residue was left under a fume hood to evaporate the remaining solvent. After air dryness, the residue was dissolved in a suitable volume of n-hexane for gas chromatography [Hewlett-Packard Gas Chromatograph, Model 5890 series II equipped with $Ni^{63}$ electron capture detector and integrator 3395 fitted with Hp capillary column (methyl silicon Gum) 30 m x 0.25 mm x µm film thickness. Oven temperature was 220°C and detector temperature 300°C] (Nabrawy and Cary 1988). Untreated samples were fortified by the addition of standard solutions of diazinon at level ranged from 0.1 to 1 ppm. The fortified samples were processed through all steps of the analytical method to validate the assay procedure (Table 1).

**Statistical analysis:** Data were analyzed by general linear model (GLM) SAS, (1995). Significant differences among means were detected using Duncan's Multiple Range Test SAS, (1995).

## RESULTS AND DISCUSSION

**Recovery rates of diazinon from liver and kidney samples of rabbits:** Recovery of diazinon from fortified liver and kidney samples each of 0.5 gm from treated rabbits are shown in Table (1). Recovery percentages were 90.1 and 88.5 % of diazinon from liver and kidney respectively. El-

Nabarawy and Cary, (1988), Gustave *et al.* (1994), Farrag, (1996) and Shalby, (2002), who reported that, the recovery rates of pirimiphos-methy, chloropyriphos-methyl and fenitrothion from fortified liver and kidney samples of white albino rats ranged between 92.1 to 93.13 % of the tested insecticides.

Table (1): Recovery rates of diazinon from liver and kidney samples of rabbits

| Added (ppm) | Liver | kidney |
|---|---|---|
| 0.1 | 90.30 | 87.80 |
| 0.5 | 88.70 | 89.20 |
| 1.0 | 91.30 | 88.50 |
| Average | 90.1 ± 1.31 | 88.5 ± 0.70 |

**Determination of diazinon residues in liver and kidney tissues of rabbits**

The existed residues of diazinon in liver and kidney samples after 0, 1, 3, 7, 15 and 21 days from dipping of rabbits by single and two frequency of diazinon are shown in Table (2). Diazinon residues were 0.05, 1.2, 3.8, 1.8, 0.87 and 0.0 ppm in kidney tissues of treated rabbits by the first and second contact exposure (DLC) after 0, 1, 3, 7, 15 and 21 days. The corresponding residues were 0.01, 0.9, 2.9, 1.5, 0.31 and 0.0 ppm, respectively, in liver tissues after the same periods. In case of dipping of rabbits in DHC, the residues were elevated and the detected concentrations were 0.07, 4.5, 6.17, 4.62, 1.2 and 0.03 ppm, respectively, in kidney samples after 0, 1, 3, 7, 15 and 21 days of exposure. Also, the results in Table (2) showed clearly that the concentrations of diazinon residue in liver were 0.04, 3.01, 4.7, 3.85, 1.1 and 0.02 ppm, respectively, after the same pre-exposure periods.

The significance of the present data can be correlated to the possible consumption of rabbits after dipping in diazinon. It is clearly indicated that liver and kidney could be safely marketed for human consumption just after treatment (zero-time) because the insecticide in this period didn't reach to the organs tissues where those residues were below its permissible limit (0.75 ppm) after 21 days of treatment.

Table (2): Residues of diazinon in liver and kidney after exposure to low and high concentrations

| Days after | DLC | | DHC | |
|---|---|---|---|---|
| | kidney | liver | kidney | liver |
| 0 | 0.05 | 0.01 | 0.07 | 0.04 |
| 1 | 1.20 | 0.90 | 4.50 | 3.01 |
| 3 | 3.80 | 2.90 | 6.17 | 4.70 |
| 7 | 1.80 | 1.50 | 4.62 | 3.85 |
| 15 | 0.87 | 0.31 | 1.20 | 1.10 |
| 21 | ND | ND | 0.03 | 0.02 |

ND: not detected ; DLC and DHC refer to low and high diazinon concentrations.

**The effects of diazinon residues on rabbits liver and kidney functions:**

Biochemical data of Tables (3 and 4) clearly showed a significant changes in the activities of AST and ALT ($p<0.01$) and ALP ($p<0.05$) in rabbits exposed to diazinon. AST activity, showed a significant ($p<0.05$) inhibition in both tested concentrations of diazinon. The mean values of AST activity for the control, DLC, DHC were $40.0 \pm 1.5$, $35.4 \pm 0.72$ and $40.2 \pm 0.96$ respectively. Meanwhile, ALT and ALP activities were elevated in rabbits dipped in DLC and DHC. On the other hand AST, ALT and ALP expressed inconsistent change during the treatment periods (Table 3).

Many studies were carried out concerning the influence of pesticide on ALT and AST activities. Enan *et. al.* (1987) found significant and apparent decrease in ALT activity in serum of rabbits given sub-lethal doses of profenphos. However, the sub-lethal doses of cyanofenphos and profenphos and the acute single dose administration of profenfos resulted in an apparent increase in serum AST activity of intoxicated rabbits. It was observed that hepatic ALT activity was significantly increased in rabbits acutely and sub-chronically intoxicated with these tested insecticides. Also, Enan *et. al.* (1982) recorded a significant increase in serum ALT of rats after the administration of profenphos, parathion-methyl, sulprofos, malathion, dichlorovos and dimethoate. Also, they found a significant inhibition in serum ALT of rats after the administration of leptophos, chlorpyrifos and diazinon.

Table (3): Changes in aspartate aminotransferase (AST ) , alanine aminotransferase (ALT ) and alkaline phosphatase (AL P) in Baladi rabbits treated with diazinon.

| Time (days) | AST (IU/L) | | | ALT (IU/L) | | | ALP (IU/L) | | |
|---|---|---|---|---|---|---|---|---|---|
| | Control | DLC | DHC | Control | DLC | DHC | Control | DLC | DHC |
| 0 | 38.9±5.6 | 33.8±0.48<br>37.3±1.85$^{bc}$ | 39.3±0.48<br>** | 28.3±0.43 | 31.8±0.48<br>38.6±3.69$^{b}$ | 55.8±0.48<br>** | 122±5.40 | 136±0.48<br>181±22.4$^{a}$ | 286±0.41<br>* |
| 1 | 39.6±6.0 | 32.8±0.48<br>37.8±2.12$^{bc}$ | 40.8±0.47 | 31.9±3.40 | 23.8±0.48<br>26.5±1.56$^{d}$ | 23.8±0.47 | 119±18.3 | 137±0.41<br>176±21.2$^{a}$ | 271±0.41 |
| 3 | 38.9±1.1 | 33.0±0.41<br>36.5±0.86$^{c}$ | 37.8±0.47 | 32.4±3.30 | 31.8±0.48<br>40.0±3.50$^{b}$ | 55.8±0.48 | 94±13.1 | 241±0.41<br>158±18.9$^{b}$ | 140±0.41 |
| 7 | 40.5±5.6 | 41.0±0.41<br>43.4±2.05$^{a}$ | 48.8±0.47 | 30.4±0.85 | 26.8±0.48<br>27.0±0.88$^{d}$ | 23.8±0.48 | 109±12.4 | 246±0.41<br>162±18.5$^{b}$ | 131±0.40 |
| 15 | 39.0±0.4 | 32.8±0.48<br>35.2±0.86$^{c}$ | 33.8±0.47 | 33.5±2.10 | 25.8±0.48<br>33.0±1.85$^{c}$ | 39.8±0.48 | 129±2.50 | 301±0.41<br>185±24.7$^{a}$ | 126±0.41 |
| 21 | 42.3±0.9 | 39.3±0.48<br>40.8±0.49$^{ab}$ | 41.0±0.40 | 32.0±0.41 | 62.8±0.48<br>42.5±4.30$^{a}$ | 32.8±0.48 | 123±0.48 | 291±0.41<br>180±23.8$^{a}$ | 125±0.41 |
| Over All mean | 40.0±1.5$^{A}$ | 35.4±0.72$^{B}$<br>** | 40.2±0.96$^{A}$ | 31.4±0.85$^{C}$ | 33.6±2.78$^{B}$<br>** | 38.6±2.78$^{A}$ | 116±4.60$^{C}$ | 225±13.8$^{A}$<br>* | 180±14.6$^{B}$ |

Means with different superscript letters vary significantly (P<0.05) , small letters are used for comparing days; capital letters are used for comparing doses. (* P < 0.05, ** P < 0.01 for comparing main effects).
DLC and DHC refer to low and high diazinon concentrations.

Table (4):Mean squares for the effect of concentration of diazinon, days and their interaction on AST, ALT, ALP, urea and creatinine.

| S.O.V | df | AST | ALT | ALP | Urea | Creatinine |
|---|---|---|---|---|---|---|
| Concentration | 2 | 171.11** | 322.25** | 72446.5* | 453.87** | 2.70** |
| Day | 5 | 111.96** | 562.15** | 1449.2* | 136.21** | 0.47** |
| Concentration × Day | 10 | 24.70 | 567.43** | 21951.1* | 211.06** | 0.22** |
| Error | 54 | 23.02 | 6.79 | 154.89 | 6.21 | 0.007 |

S.O.V. = Source of variation, df = degrees of freedom

The disruption of transaminases from the normal values denotes biochemical impairment of tissue and cellular functions as are involved in the detoxification processes, metabolism, and biosynthesis of energetic macromolecules of different essential functions (Todior and VanHeemstra-Lequin, 1980). Habiba and Ismail (1992) reported that brain muscle ASTs were inhibited in the New Zealand white rabbit fed on clover contaminated with profenphos whereas liver AST was stimulated. Transaminases are important and critical enzymes in the biological processes. They play a role in amino acid catabolism and biosynthesis. ALT transfers the amino group of alanine to α-ketoglutaric acid, forming glutamic and pyruvic acids. Consequently, it is considered as a specific indicator of liver damage. The possible mechanism involved in the elevation of ALT may be due to tissue damage, or due to increased synthesis or decreased catabolism of ALT (Enan *et al.*, 1987).

Significant elevation of ALP in the present study (Table 3 and 4), due to the contact exposure of rabbits to diazinon in both tested concentrations. Diazinon has been recorded to induce ALP in workers of a chemical plant producing dust pesticides (Coker *et. al.*, 2002; Kossmann *et. al.*, 2001). Also, serum level of ALP was increased due to OPs pesticide administration to male albino rats (El-Nabarawy *et. al.*, 2001). The enhancement of the activity of ALP could be related to the influence of glucocorticoides (Murphy, 1966), or could be attributed to its release from ruptured cells due to the effect of pesticide (Shaffi, 1980; Al-Rehiayani *et. al.*, 2002).

OP's are readily absorbed through the skin and biological monitoring is an essential component of any comperhensive assessment of exposure (Cocker *et. al.*, 2002). Significant (P<0.01) changes in blood urea and creatinine levels was found in Baladi rabbits exposed to diazinon (Table 5). Serum levels of creatinine were increased, while there were no significant differences in urea due to dimethoate administration to male albino rats (El-Nabarawy *et. al.*, 2001). Serum level of creatinine was increased in workers exposed to diazinon dust (Cocker *et. al.*, 2002; Kossmann *et. al.*, 2001). Marked elevation in blood urea in rats exposed to OP's pesticides (Rajini and Krishnakumari, 1988) and mice (Zayed *et. al.*, 1993). The results strongly suggest that the bound residues can induce adverse biological effects in rabbits.

Table (5): Changes in urea and creatinine in Baladi rabbits treated with diazinon.

| Time (days) | Urea (mg/dl) | | | Creatinine (mg/dl) | | |
|---|---|---|---|---|---|---|
| | Control | DLC | DHC | Control | DLC | DHC |
| 0 | 19.3±2.93 | 20.4±0.02<br>24.5±2.17$^{bc}$ | 33.7±0.58<br>** | 0.70±0.09 | 1.14±0.06<br>0.82±0.08$^{cd}$ | 0.63±0.01<br>** |
| 1 | 24.7±3.24 | 42.9±0.78<br>31.8±2.61$^{a}$ | 27.9±0.54 | 0.70±0.07 | 1.01±0.07<br>0.77±0.06$^{de}$ | 0.63±0.01 |
| 3 | 24.7±1.50 | 25.7±0.32<br>23.0±1.60$^{c}$ | 18.6±0.41 | 0.86±0.03 | 2.07±0.04<br>1.28±0.17$^{a}$ | 0.91±0.01 |
| 7 | 22.2±1.64 | 27.6±0.56<br>26.0±0.98$^{b}$ | 28.2±0.45 | 0.87±0.03 | 0.78±0.01<br>0.73±0.04$^{e}$ | 0.55±0.01 |
| 15 | 21.1±0.93 | 24.0±0.45<br>29.8±3.14$^{a}$ | 44.4±0.46 | 0.81±0.04 | 1.28±0.03<br>0.90±0.09$^{b}$ | 0.61±0.01 |
| 21 | 19.1±0.70 | 29.9±0.17<br>25.5±1.40$^{b}$ | 27.4±0.34 | 0.61±0.01 | 1.41±0.08<br>0.88±0.12$^{bc}$ | 0.61±0.01 |
| Over All mean | 21.8±0.88$^{C}$ | 28.4±1.49$^{B}$<br>** | 30.0±1.63$^{A}$ | 0.76±0.03$^{B}$ | 1.28±0.09$^{A}$<br>** | 0.66±0.02$^{C}$ |

**Means with different superscript letters vary significantly (P<0.05), small letters are used for comparing days;capital letters are used for comparing doses. (* P < 0.05, ** P < 0.01 for comparing main effects). DLCand DHC refer to low and high diazinon concentrations.**

Recent studies have confirmed that exposure to pesticides can cause significantly different biological effects if the evaluation considers only the active ingredients of the pesticides and/or their activated metabolites in urine as individual bio-markers for systemic and contact exposure (Lewalter and Leng, 1999). Indeed, animal components and products should be free from any insecticide residues. It is well known that nutritional value of animal organs necessitates the absence of any insecticide residues due to the harmful effects expected on humans. A fact which determines the economic value of the food commodities.

## REFERENCES

Al-Rehiayani, S.; M.A. Al-Doghairi, K.A. Osman and E.A. El-Hag (2002). Toxicity and biochemical effects of some insecticides on the fruit stalk borer, *Oryctes elegans*. J. Pest. Cont. & Environ. Sci., 10: 23-35.

Blanchflower, W.J.; R.J. MeCracken, D.A. Rice, and A. Clements (1990). Survey of levels of propetamphos and diazinon used to control sheep scab in Northern Irelan. Vet. Record, 126: 263-265.

Bradway, D.E.; T.M. Shafik and E.M. Lores (1977). Comparison of cholinesterase activity, residue levels, and urinary metabolite excretion of rats exposed to organophosphorus pesticides. J. Agric. Food Chem., 25: 1353-1358.

Brokopp, C.D.; J.L. Wyatt and J. Gabica (1981). Dialkyl phosphates in urine samples from pesticide formulators exposed to disulfoton and phorate. Bull. Environ. Contam. Toxicol., 26: 524-529.

Cocker, J.; H.J. Mason, S.J. Garfitt and K. Jones (2002). Biological monitoring of exposure to organophophate pesticides. Toxicol. Lett., 134: 97-103.

Drevenkar, V.; Z. Radic, Z. Vasilic and E. Reiner (1991). Dialkyl phosphorus metabolites in the urine and activities of esterases in the serum as biochemical indices for human absorption of organophosphorus pesticides. Arch. Environ. Contam. Toxicol., 20: 417-422.

El-Nabarawy, I.M.; M.M.H. Osfor; A.B. Okab and D.E.A. Shebi (2001). Effect of contaminated honey with insecticide-residues on some natritional and biochemical parameters in male, albino rats. Egypt. J. Food Sci., 29: 183-195.

El-Nabarawy, I.M. and W.F. Cary (1988). Improved method for determination of chlorothalonil and its related residue in cranberryies. J. AOAC., 71:358-360.

Enan, E.E.; I.G. Berberian; S. Al-Fiki; M. El-Masry and O.H. Enan (1987). Effects of two organophosphorus insecticides on some biochemical constituents in the nervous system and liver of rabbits. J. Environ. Sci. Health B22: 149-170.

Enan, E.E.; A.H. El-Sebae, O.H. Enan and S. El-Fiki (1982). *In vivo* interaction of some organophosphorus insecticides with different biochemical targets in white rats. J. Environ. Sci. Health B17: 549-570.

Farrag, A.H. (1996). Ultrastructural and histopathological studies and residue determination of insecticide profenophos in the albino rat. M.Sc. Thesis, Institute of Environmental Studies and Research. Ain Shams University.

Gustave, A.B.; K. Singh, S.U. Khan, M.H. Akhtars, S. Kacew and N.D.G. White (1994). Fate of wheat bound malathion residues in rats during gestation. Chemosphere, 29:451-455.

Habiba, R.A. and S.M. Ismail (1992). Biochemical effects of profenphos in the New Zealand white rabbit. J. Pest. Control & Environ. Sci., 4: 15-29.

Kachmar, J.F., and D.E. Moss (1976). Enzymes. In Fundamentals of Clinical Chemistry. Tiez, N.W., Ed.; Saunders, Philadelphie, pp. 645.

Kossmann, S.; J. Tustanowski and B. Kolodziej (2001). Renal dysfunction in chemical plant workers producing pesticides. Med. Pr., 52:253-256.

Larsen, K. (1972). Colorimetric method for creatinine determination. Clin. Chem. Acta., 41:209.

Lewalter, J and G Leng (1999) Considration of individual susceptibility in adverse pesticide effects Toxicol Lett., 107: 131- 144

Mount, M.E. (1984). Measurement of dialkyl phosphates in urine as an aid to recognize exposure to organophosphate insecticides. Proc. Ann. Meet. Am. Assoc. Vet. Lab. Diagn., 27: 383-402.

Murphy, S.D. (1966). Liver metabolism and toxicity of triphosphate insecticides in mammals, avian, and piscine species. Proc. Soc. Exp. Biol Med., 123: 393.

Osweiler, G.D.; T.L. Carson, W.B. Buck and G.A. Van Gelder (1985). Clinical Diagnostic Veterinary Toxicology, Kendall Hunt Publishing. Dubuque, IA, pp 495.

Patton, C.J. and S.R. Crouch (1977). Clorimetric determination of urea. 49:464-469.

Rajini, P.S. and M.K. Krishnakumari (1988). Toxicity of pirmiphos-methyl: Effect of dietary feeding on blood and urine constituents in albino rats. J. Environ. Sci. Health B 23: 145-158.

Reitman, S. and S. Frankel (1957). A colorimetric method for the determination of serum glutamic oxaloacetic and glutamic pyruvic transaminases. Am. J. Pathol., 28: 56-63.

SAS (1995). SAS user guide: Statistics Version 5th ed. SAS institute Inc., Cary, NC.

Shaffi, S. (1980). Thiodon toxicity. Non-specific phosphonosterases in nine fresh water toleosts. Toxicol. Lett., 6: 399.

Shafik, T.; D.E. Broadway, H.F. Enos, A.R. Tobs (1973). Human exposure to organophosphours pesticides. A modified procedure for the gas-liquid chromatographic analysis alkayl phosphate metabolites in urine. J. Agric. Food Chem., 21: 625-628.

Shalby, Sh.E.M. (2002). Determination of pesticide residues in and on tomato fruits and their effects on experimental rats. Ph.D. Thesis, Fac Agric Mansoura University

Todior, W.F. and E.A. VanHeemstra-Lequin (1980). Field Studies Monitoring Exposure and Effects in the Development of Pesticides. Elsevier, Amsterdam pp. 207.

Zayed, S.M.; S.M. Amer, M.F. Nawito, M. Farghaly, H.A. Amer, M.A. Fahmy and F. Mahdy (1993). Toxicological potential of malathion residues in stored soybean seeds. J. Environ. Sci. Health B 28: 711-729.

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# متبقيات الديازينون في كبد وكلية الأرانب البلدي وعلاقتها بوظائفها

إبراهيم متولي النبراوى، صباح جابر البنا، على بسيوني عقاب و أحمد مرسى عطية
قسم وقاية النبات بالمركز القومي للبحوث - القاهرة
و قسم الدراسات البيئية بمعهد الدراسات العليا و البحوث - جامعة الإسكندرية

بينت نتائج هذه الدراسة أن متبقيات الديانينون في كبد الأرانب بعد المعاملة مباشرة ، وبعد يوم وثلاث و سبع وخمسة عشر وإحدى وعشرون يوما من المعاملة بالتركيز المنخفض هي ٠,٠٥، ١,٢، ٣,٨، ١,٨ ، ٠,٨٧، ٠,٠ جزء في المليون بينما في الكلية فكانت هي ٠,٠١، ٠,٩، ٢,٩، ١,٥، ٠,٣١، ٠,٠ جزء في المليون في الكبد. أما في حالة غمر الأرانب في التركيز العالي من الديازينون أدت إلي زيادة متبقيات المبيد وكانت التركيزات ٠,٠٧، ٤,٥، ٦,١٧، ٤,٦٢ ، ١,٢ ، ٠,٠٣ جزء في المليون في الكلية وذلك بعد المعاملة مباشرة ، وبعد يوم وثلاث و سبع وخمسة عشر وإحدى وعشرون يوما من المعاملة أيضا كانت التركيزات في الكبد ٠,٠٤، ٣,٠١، ٤,٧ ، ٣,٨٥ ، ١,١ ، ٠,٠٢ جزء في المليون عند نفس المدة.

تم دراسة تأثير الديازينون على الأسبارتيت أمينو ترانسفيريز (AST)، الألانين أمينو ترانسفيريز (ALT)، الفوسفاتيز القلوي (ALP)، الكيرياتينين و اليوريا وذلك في الأرانب البلدي. قسمت ٧٢ من الحيوانات إلي ثلاث مجموعات، المجموعة الأولى تم غمرها في مياه الشرب واعتبرت كمجموعة ضابطة. المجموعة الثانية و الثالثة تم غمرها في مبيد الديازينون (EC 60%) بتركيز ٠,٦ و ٣ مجم/ لتر ماء على التوالي وذلك لمدة ١٠ ثوان. تم تكرار الخطوة السابقة بعد ١٠ أيام. ثم تم ذبح الحيوانات مباشرة، وبعد يوم وثلاث و سبع وخمسة عشر وإحدى وعشرون يوما وذلك بعد غمر الأرانب في الديازينون للمرة الثانية. أوضحت النتائج أن كلا التركيزين من الديازينون أدى إلي زيادة مستوى إنزيم ALT و الفسفاتيز القلوي بينما انخفض مستوى إنزيم AST. أيضا بينت النتائج حدوث تغيرات في مستوى الكيرياتينين و حدوث تذبذب في مستويات إنزيمات AST، ALT، الفسفاتيز القلوي واليوريا وذلك خلال فترة المعاملة. ومن ناحية أخرى انخفضت مستويات الكرياتينين. العلاقة المترابطة بين متبقيات الديازينون والتأثير على وظائف كبد وكلية الأرانب تبرهن إمكانية إعاقة وظائف الحيوان وبالتالى إنتاجيته.