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TOXICITY AND BIOCHEMICAL EFFECTS OF LEAD, CADMIUM, ACETAMIPRID AND THEIR MIXTURES ON MALE MICE

By

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ABSTRACT

The toxicity and biochemical effects of lead (10 mg/kg/day), cadmium (1 mg/kg/day), acetamiprid (34 & 68 mg/kg/day) and their mixtures after 14 days of oral administration on male white albino mice were investigated. Lead and cadmium treatments caused high percent mortalities 55.6% and 66.7 % respectively, while acetamiprid at 67.9 mg/kg/day caused 11.1 % only. The mixtures of acetamiprid with lead and cadmium decreased the percent mortalities of lead and cadmium, this means that acetamiprid (cyanoimin group) had an antagonismic effect to heavy metals. Furthermore, we studied the effect of lead and cadmium treatments on activity of δ-ALAD, AChE and GST enzymes. Both caused significantly decrease of the activity of δ-ALAD (52.12% & 19.94%), AChE (42.57% & 28%) and GST (14.37% & 34.32%), while they caused elevation of serum LDH level (40% & 46.67%), respectively. On the other hand, the mixtures of acetamiprid with lead and cadmium decreased the inhibitory effect of lead and cadmium on δ-ALAD & AChE activity and inhibited the elevation of serum LDH level. A correlation between toxicity and changes of enzymes activities as a result of lead, cadmium treatment and their mixtures with acetamiprid, was quite clear and demonstrated that acetamiprid may act as a protective agent against heavy metals intoxication.

INTRODUCTION

Metals are ubiquitous in the modern industrialized environment. Some metals (lead, cadmium, mercury, etc.) have no beneficial role in humans and there is no known homeostasis mechanism for them. In contrast, other metals such as chromium, copper, zinc, manganese and iron are essential for man. However, these essential trace elements can also be dangerous at high levels (Schumacher *et. al.*, 1994). Toxic effect of lead on humans has been known for centuries. Because lead is abundant, malleable and easily refined it has been employed since antiquity in pigments, pipe for drinking water pipes and in sweetening and preserving wine (WHO, 1987). Recently, significant exposure has been observed primarily in industry and in the use of leaded gasoline. Inhalation of tetraethyl lead from gasoline, once considered to be a major source of lead contamination in the general environment, has been significantly reduced in recent years in countries phasing out leaded gasoline (WHO, 1987). In lead exposed workers, protein damage of red blood cell membranes has been demonstrated and may play a role in the pathogenesis of lead anemia. Lead exposure also, causes changes in some heme biosynthetic pathway parameters, such as inhibition of erythrocyte δ-aminolevulinic acid dehydratse (δ-ALAD) activity, followed by increase of urinary excretion of δ- aminolevulinic acid (ALA-U) (Tabuchi *et. al.*, 1989). During the past three decades, cadmium has attracted, substantial attention, primarily due to abundant evidence of effects on human health in industry and the general environment (Bernard and Lauwerys, 1984). Whereas cadmium metal is mainly employed as an anticorrosive, cadmium compounds are commonly used as pigments in plastics. Mining and metals refining are major industrial emission sources of cadmium. Since only a very minor proportion of cadmium is recycled, it has been referred to as "the dissipated element" (WHO, 1980). Cadmium is transported via blood to other parts of the body. It is predominantly bound to the low-molecular-weight protein metallothionein is induced by cadmium and the highest concentrations are found in the kidneys and liver. Low excretion rates lead to very efficient retention in the body. Because of the exceptionally long biological half-lives of cadmium in the liver and the kidneys, these organs are the main sites of storage (Fitz and Karsa, 1990). On the other

hand, acetamiprid is a new insecticidal compound having cyanoamidine structure. It was discovered and is now being developed by Nippon Soda Co., Ltd. Acetamiprid acts on Acetylcholine receptors (AChRs) receptor of insect central nervous system as an agonist of ACh (Matsuda and Takahashi, 1996). The toxicity of lead and cadmium at maximum limit levels of exposure are well known, but a major concern of today is the possibility that continual exposure to relatively high levels may entail adverse health effect. Also, farm workers are exposed to synthetic agricultural chemicals (i.e: acetamiprid) while working in the fields. Thus, this study was done to investigate the toxicity and some biochemical effects of high doses of lead and cadmium and sub-lethal doses of acetamiprid and their mixtures on the male white albino mice.

MATERIALS AND METHODS

Animals :

Six to eight week old (23-31 gm) male white albino mice were used throughout the study. Animals were housed in suspended stainless steel cages and provided with food and water ad lib. All animals were maintained on a 12 hr. light / 12 hr. dark cycle at constant temperature (25 ±1°C) and humidity (approx. 70 %) .

Chemicals :

Acetamiprid (ACP) (iso propsed), 20 % SP formulation from (NIPPON SODA CO. , LTD.) , Ohtemachi Chiyoda- KU Tokyo , JAPAN was used . Lead acetate and Cadmium acetate are pure grade chemical . All the other chemicals were of the highest purity grade available from Sigma , BDH and or Aldrich chemical companies . The preparations were administered orally in the form aqueoues solution .

Experimental Protocol :

The mice were divided randomly into nine groups of ten mice each and orally injected daily for 14 days according to the following protocol : **Group 1:** Control treated with tap water. **Group 2 :** Treated with 1/20 LD_{50} ACP (i.e. 34 mg / kg / day). **Group 3:** Treated with 1/10 LD_{50} ACP

(i.e. 68 mg / kg / day). **Group 4:** Treated with 10 mg / kg / day lead acetate. **Group 5 :** Treated with 1/20 LD_{50} ACP + 10 mg lead acetate. **Group 6:** Treated with 1/10 LD_{50} ACP + 10 mg lead acetate . **Group 7:** Treated with 1 mg / kg / day cadmium acetate. **Group 8:** Treated with 1/20 LD_{50} ACP + 1 mg cadmium acetate. **Group 9:** Treated with 1/10 LD_{50} ACP + 1 mg cadmium acetate . All animals were weighed dialy (for 14 days) to ensure the maximum dose effect . Percent mortality was recorded during the experimental period. In all treatments, percentage mortalities were corrected by Abbott's formula (1925).

Tissue Collection :

After 24 hr. from the last treatment three mice per group were anesthetized with ether and sacrificed. The blood samples were collected from each animal in 5 c.c citrated tubes containing 0.25 ml.of 3.8% sodium citrate. The blood was mixed carefully to avoid the formation of foams, then used freshly for delta-aminolevulinic acid dehydratase (δ-ALAD) assay .Samples used for determining lactate dehydrogenase (LDH) were collected in tubes that did not contain an anticoagulant. Brains and livers were obtained from dissected animals, weighed and then stored at -20°C until biochemical assay were performed (within two weeks).

Biochemical Studies :

Serum preparation : Blood samples were allowed to clott at Room Temperature (R.T.) for 15–30 min. The clot was released from the walls of the tube and allowed to retract overnight at 4°C. Sera were pipetted into clean tubes, centrifuged at 3000 rpm for 10 min. to remove erythrocytes , then used to measure LDH activity.

LDH assay : LDH activity was measured in serum according to the method of McComb (1983) using Na-pyruvate as a substrate and NADH as a co-factor.

δ-ALAD : The erythrocyte enzyme activity was assayed according to the method of Joseph *et al.* (1971).

Acetylcholinesterase (AChE) preparation : The control and treated brains of mice were weighted and homogenized in ten volumes (w/v) of ice-cold of 0.1 M phosphate buffer pH 8 using Tekmar homogenizer for 30 seconds. The homogenate was then centrifuged at 8000 xg for 20 min. at 4°C using Beckman L5-75 ultracentrifuge type 40 rotor. Pellets were discarded and supernatused as the enzyme source for AChE.

AChE assay : AChE activity was assayed by the method of Ellman *et. al.* (1961) using acetylthiocholine iodide as a substrate. Protein content was assayed using the Lowery method (1951).

Glutathione-S-transferase preparation (GST) : The control and treated livers of mice, each was weight and homogenized in four volumes (i.e. 4 ml / g wet. tissue) of 0.1 M sodium phosphate buffer pH 7.5, in Temkar homogenizer. The homogenate was centrifuged for 60 min. at 30000 xg at 4°C using Beckman L 5-75 ultracentrifuge type 40 rotor, and the supernatant was used as crude enzyme fraction (Asao *et al.*, 1977) and Takahashi (1977).

GST assay : GST activity was measured according the method of Asaoka and Takahashi (1983) using o-dinitrobenzene as a substrate .Protein content was assayed using the Lowery method (1951).

Analysis of data :

Data were analysed for statistical significance by ANOVA using SAS statistical software .

RESULTS AND DISCUSSION

Toxicity of the nine treatments on the male white albino mice and their effects on the activities of (ALAD , LDH , AChE and GST) enzymes were recorded after 14 days of exposure as illustrated in Tables 1 to 5. The data in Table (1) showed that cadmium and lead treatments were more toxic, since percent mortalities were 66.7 & 55.6 % respectively. On the other hand, ACP at 67.9 mg / kg / day (1/10 LD_{50}) caused 11.1% M.

The Mixtures of lead and cadmium with 1/20 & 1/10 LD_{50} had percentage mortalities between those of Pb & Cd from side and 1/10 LD_{50} in anther side.

Table (1): Toxicity of acetamiprid , lead and cadmium on the male white albino mice after 14 days of oral administration.

Experimental Groups	No. of animals		% Mortality	Corrected
	0 day	14 days	(M)	% M
Control	10	9	10	0
1/20 LD_{50} (ACP)	10	9	10	0
1/10 LD_{50} (ACP)	10	8	20	11.1
Pb	10	4	60	55.6
Pb + 1/20 LD_{50} (ACP)	10	6	40	33.3
Pb + 1/10 LD_{50} (ACP)	10	7	30	22.2
Cd	10	3	70	66.7
Cd + 1/ 20 LD_{50} (ACP)	10	5	50	44.4
Cd + 1/ 10 LD_{50} (ACP)	10	7	30	22.2

Matsuda and Takahashi (1996) mentioned that no damages by ACP at 53.2 mg / kg / day in the diet for male mice. We noticed that sub-lethal doses of ACP decreased the percent mortality of cadmium and lead to animals. The above result may be return to ACP (cyanoimin group) had antigonismic effect to heavy metals.

Delta-Amino Levulinic Acid dehydratase (δ-ALAD) activity of control and treated groups are presented in Table (2). It is obvious from the data that all of the treatments had inhibitory effect on this enzyme except group 9. Cd + 1/10 LD_{50} significantly increased the enzyme activity. Lead treatment (10 mg / kg / day) was more potent inhibitor (52.12%) while the treatment of Cd + 1/20 LD_{50} was less effective (7.72%). We noticed that the mixtures of lead and cadmium with ACP were less effective than lead and cadmium alone ,especillay in the case of cadmium mixtures. δ-ALAD is a sulfhydryl enzyme and is, therefore inhibited by metals such as copper , silver and lead (Tomio *et al.*, 1968).

These data are parallel to that obtained by Masatoshi (1983) , who found that administration of 500 ppm of lead in drinking water to rats produced 82.7% and 74 % inhibition of δ-ALAD activity after 18 and 21 days of gestation. Kira *et al.* (1989) reported that treatment of rats with 500 ppm of lead in the drinking water for 6 weeks caused 82.78 % inhibition of rats erythrocytic δ-ALAD. Also,these data are paralleled to the data obtained by Al-Rajhi (1993) who found that erythrocytic δ-ALAD is significantly inhibited by lead treatments. Dowla *et al.* (1996) found that cadmium at $1.4x10^{-3}$ mM caused 50% inhibition δ-ALAD activity. Hallen. *et al.* (1995) found that δ-ALAD activity was decreased in neonates rats which exposed to lead and an exponential correlation between blood lead

Table (2) : *In vivo* effect of acetamiprid , lead and cadmium on δ amino levulinic acid dehydratase (δ-ALAD) activity after 14 days of oral administration .

Experimental Groups	Activity (μ mole) PBG/ml/RBC/hr.	% change of control
Control	72.45 ± 0.88 b	-
1/20 LD_{50} (ACP)	63.82 ± 3.25 d	11.91 (↓)
1/10 LD_{50} (ACP)	54.95 ± 2.64 f	24.15 (↓)
Pb	34.69 ± 0.97 h	52.12 (↓)
Pb + 1/20 LD_{50} (ACP)	50.70 ± 0.00 g	30.20 (↓)
Pb + 1/10 LD_{50} (ACP)	52.02 ± 1.98 g	28.20 (↓)
Cd	58.00 ± 2.28 e	19.94 (↓)
Cd + 1/20 LD_{50} (ACP)	66.86 ± 1.16 c	07.72 (↓)
Cd + 1/10 LD_{50} (ACP)	81.31 ± 0.00 a	12.23 (↑)

Each value is the mean of 3 replicates ± S.D.

$$\text{One enzyme unit} = OD_{60} - OD_0 \times \frac{100}{HCT} \times \frac{\text{blood dilution}}{\text{blood vol. x 60 min}} \times \frac{1}{0.030} \times 2$$

Significantly different from control at ($p \leq 0.05$) .
Least Significant Difference (LSD) = 2.78
Means with the same letter are not significantly different .
↑ = increase ↓ = decrease

concentration and δ-ALAD activity. Hayashi *et. al.* (1993) found that δ-ALAD activity in erythrocytes in male wistar rats was lower at 20 days after lead treatment. The effect of ACP on the activity of δ-ALAD may be attributed to its rapid absorption and distribution into organs and tissues via blood (Matsuda and Takahashi, 1996). By comparing the data presented in this table with the toxicity in Table 1, a good correlation between toxicity and enzyme inhibition is quite clear. From the above data we can say that ACP may act as a protective agent when mixed with the studied metals, but further studies are needed to confirm these interactions.

Relative changes in the activity of LDH as a result of the nine treatments after 14 days were recorded in Table (3). The enzyme activity is sensitive to a relatively high doses of lead and cadmium (46.67% and 40% activation respectively) since lead and cadmium cause severe muscular contraction which may involve participation of LDH in order to convert excess lactate produced into pyruvate.

Table (3): *In vivo* effect of acetamiprid, lead and cadmium on serum lactate dehydrogenase (LDH) after 14 days of oral administration .

Experimental Groups	Activity (I U / L)	% Increase
Control	447.76 ± .55 c	-
1/20 LD_{50} (ACP)	450.56 ± 089.55 c	00.63
1/10 LD_{50} (ACP)	460.86 ± 051.70 c	02.93
Pb	626.87 ± 089.55 a	40.00
Pb + 1/20 LD_{50} (ACP)	597.01 ± 136.79 a	33.33
Pb + 1/10 LD_{50} (ACP)	537.31 ± 089.55 a	20.00
Cd	656.72 ± 051.70 a	46.67
Cd + 1/20 LD_{50} (ACP)	507.46 ± 051.70 b	13.33
Cd + 1/10 LD_{50} (ACP)	477.61 ± 051.70 c	06.67

Each value is the mean of 3 replicates ± S.D.
Serum LDH = C – T / C – B x 1000 I U / L .
Significantly different from control at ($p \leq 0.05$) .
Least Significant Difference (LSD) = 126.12
Means with the same letter are not significantly different.

These results agree with other reports studying the same parameter. Lead administered at 10 mg / kg caused significant increase in activity of LDH (Randhawa *et al.*, 1995). Madej *et al.*(1988) found that 600 ppm lead acetate on alternative days over 6 weeks caused increased in LDH activity. Also, Khandelwal *et al.*(1991) found that increase in the activity of LDH in rats receiving cadmium at 1 mg / kg. Denizeau and Marion (1990) found that cells of rainbow trout were most sensitive to cadmium, and LDH activity was increased. On the other hand, Eweis and Ibrahim (1996) found that 1 mg/ kg / day lead caused decreased in the activity of LDH in different organs. In the present study, we found that ACP inhibits the elevation of serum LDH level in lead and cadmium induced liver injuries in mice.

Acetylcholinesterase (AChE) activity of control and other experimental groups are presented in Table (4). It is obvious from the data that all of the treatments had inhibitory effect on this enzyme. Lead treatment has the highest effect (42.57%) then cadmium treatment (28%),

Table (4): *In vivo* effect of acetamiprid, lead and cadmium on brain Acetylcholinestrase (AChE) activity after 14 days of oral administration .

Experimental Groups	Specific activity OD λ_{412} / mg protein/hr.	% Inhibition
Control	28.07 ± 1.08 a	-
1/20 LD_{50} (ACP)	26.73 ± 0.95 ab	06.80
1/10 LD_{50} (ACP)	23.16 ± 1.52 cd	15.46
Pb	16.12 ± 0.47 f	42.57
Pb + 1/20 LD_{50} (ACP)	23.39 ± 0.51 d	16.67
Pb + 1/10 LD_{50} (ACP)	25.60 ± 1.38 bc	08.80
Cd	20.21 ± 1.06 e	28.00
Cd + 1/20 LD_{50} (ACP)	22.36 ± 0.93 d	20.34
Cd + 1/10 LD_{50} (ACP)	24.24 ± 1.89 bcd	13.64

Each value is the mean of 3 replicates ± S.D.
Significantly different from control at ($p \leq 0.05$) .
Least Significant Difference (LSD) = 2.03
Means with the same letter are not significantly different .

whereas the two doses of ACP have weak effects 6.8% and 15.46%, respectively. These data are in agreement with other reports studying the same parameter. Lead administered at 500 ppm caused significant decrease in activity of AChE (Al-Rajhi, 1993), while, cadmium administered at 100 ppm caused slight effect on activity of AChE (Al-Rajhi, 1992). Eweis and Ibrahim (1996) found that lead at 1 mg / kg / day caused slightly effect on brain AChE activity of male mice. On the other hand, cadmium at 64 g / liter has no effect on AChE activity in *Daphnia magna* (Guilhermino *et al.*,1996). Also, cadmium did not inhibit AChE in the different marine compartments (water, sediment and living mater) (Bocquene *et al.*, 1995). Also, we noticed the acetamiprid decreased the inhibition effect of lead and cadmium on AChE activity.

Table (5) indicates the effect of ACP, lead and cadmium on GST activity after 14 days of oral administration. The data revealed that the enzyme activity was significantly decreased in all treatments compared with control. The treatments of Pb +1/10 LD_{50} and Cd + 1/10LD_{50} have high percent inhibition 35.75 and 35.51 respectively. While, the treatment of 1/20LD_{50} has low effect on activity of this enzyme (10.69 %). We noticed that the treatment of mixtures had an inhibitory effect of activity of this enzyme more than each treatment alone.

Glutathione-S-transferase is believed to play a physiological role in the detoxification and elimination of toxic and undesirable foreign compound including pesticides (Chasseaud, 1973).

In the present study, we found a reduction of GST activity after exposure to acetamiprid, lead and cadmium. These results are in agreement with those reported by Jordan and Bhatnagar (1990), who found that 80 mg / kg lead acetate and 80 mg / kg cadmium chloride significantly depressed GST activity in the pekin duck after 12 weeks.On the other hand, Sidhu and Nath (1996) found that cadmium at 25 mg / kg / day for 10 weeks caused an induction in GST activity in monkey liver.

Table (5): *In vivo* effect of acetamiprid, lead and cadmium on liver Glutathione-S-transferase (GST) activity after 14 days of oral administration.

Experimental Groups	Specific activity μ mole / mg protein / hr.	% Inhibition
Control	8.42 ± 0.35 a	-
1/20 LD_{50} (ACP)	7.52 ± 0.21 b	10.69
1/10 LD_{50} (ACP)	6.44 ± 0.27 c	23.52
Pb	7.21 ± 0.23 b	14.37
Pb + 1/20 LD_{50} (ACP)	5.80 ± 0.18 d	31.12
Pb + 1/10 LD_{50} (ACP)	5.41 ± 0.17 d	35.75
Cd	5.53 ± 0.24 d	34.32
Cd + 1/20 LD_{50} (ACP)	7.10 ± 0.19 b	15.68
Cd + 1/10 LD_{50} (ACP)	5.43 ± 0.31 d	35.51

Each value is the mean of 3 replicates ± S.D.
Significantly different from control at ($p \leq 0.05$).
Least Significant Difference (LSD) = 0.43
Means with the same letter are not significantly different

It could be concluded that, toxicity of lead, cadmium and their mixtures with ACP after 14 days of oral administration on male white albino mice was paralleled with relative changes of enzymes activities of δ-ALAD, AChE and LDH. The above results may suggest that ACP (cyanoimin group) had an antigonismic effect or acts as a protective agent against heavy metals interactions with the animal biological systems.

REFERANCES

Abbott W.S. (1925). A method of comparing the effectiveness of an insecticides. J. Econ. Entomol. 18: 265-267.

Al-Rajhi D.H. (1992). *In vivo* effects of cadmium on some biochemical and haemtological parameters of albino mice. J. Med. Res. Inst. 13: 81-92.

Al-Rajhi D.H. (1993): The interaction of lead in mice: Effects on some biochemical and hematological parameters. J. Med. Res. Inst 14: 45-55.

Asao K., H. Ito, and K. Takahashi (1977). Monkey glutathione s-aryltransferases, Tissue distribution and purification from the liver. J. Biochem. 82: 973-981.

Asaoka K. and K. Takahashi (1983). A colorimetric assay of glutatione s-transferases using O-dinitrobenzene as a substrate. J. Biochem. 94: 1685-1688.

Bernard A. and R. Lauwerys (1984). Cadmium in human population. Experimentia 40: 143-152.

Bocquene G., C. Bellanger, Y. Cadiou and F. Galgani (1995). Joint action of combinations of pollutants on the acetylcholinesterase activity of several marine species, Ecotoxi. 4: 266-279.

Chasseaud L.F. (1973). The nature and distribution of enzymes catalyzing the conjugation of glutathione with foreign compounds. Drug. Metab. Rev. 2: 182-220.

Denizeau F. and M. Marion (1990). Toxicity of cadmium copper and mercury to isolated trout hepatocytes. Cand. J. Fish & Aqu. Sci.. 47: 1038-1042

Dowla H.A., M. Panemangalore and M.E. Byers (1996). Comparative inhibition of enzymes of human erythrocytes and plasma *in vitro* by agricultural chemicals. Arch. Environ. Contam. Toxicol. 31: 107-114.

Ellman G.L., K.D. Courtney, V. Andres and R.L. Featherston (1961). A new and rapid colourimetric determination of acetylcholinesterase activity. Arch. Biophys. 82: 88-95.

Eweis E.A. and N.M. Ibrahim (1996). Lead toxicity in mice. Vet. Med. J. Giza 44: 671-678.

Fitz S. and L. Karsa (1990). Heavy metal concentrations in human. *Comp. Biochem. Physiol.* C 95: 117-123.

Guilhermino L., M.C. Lopes, A.P. Carva and M. Soares (1996). Inhibition of acetylcholinesterase activity as effect criterion in acute tests with juvenile. Daphnis magna, Chemoaphere 32: 727-738.

Hallen I.P., L. Jorhem and A. Oskarsson (1995). Placental and lactational transfer of lead in rats: A study on the lactational process and effects on offspring. Arch. Toxicol. 69: 596-602.

Hayashi M., K. Yamamoto, M. Yoshimura, T. Kishimoto and A. Shitara (1993). Effects of fasting on distribution and excretion of lead following long-term lead exposure in rats. Arch. Environ. Contam. Toxicol. 24: 201-205.

Jordan S.A. and M.K. Bhatnagar (1990). Hepatic enzyme activity after combined adminstration of methylmercury, lead and cadmium in the Pekin duck. Bull. Environ. Contaim. & Toxico. 44: 623-628.

Joseph B.W., B.A. Lipchutz and F.A. Oski (1971). δ-aminolevulinic acid dehydratase activity in circulating blood cells, A sensitive laboratory test for the detection of childhood lead poisoning. New Engl. J. Med. 284: 265-264.

Khandelwal S., N. Agnihotri and S.K. Tandon (1991). Biochmical response to cadmium dose-time effect. Biol. Trac. Element. Ress. 29: 157-164.

Kira M., J.M. Jakoniuk, E. Kulikowska and N. Omielianiuk (1989). The interaction of copper, lead and ethanol in rats, effects on some biochemical parameters of blood. Pol. J. Pharmacol. Pharm. 41: 273-280.

Lowery O.H., N.J. Rosenbourgh, A.L. Farr, and R.J. Randall (1951). Protein measurement with folin phenol reagent. J. Biol. Chem. 193: 265-275.

Madej J.A., G. Radzanowska, S. Klimentowski and M. Zarka(1988). The reaction of some haematological and biochemical indices and the histopathological changes in the organs of hens given lead, selenium or lead plus selenium by mouth. Zessz. Nauk. Akad. Roln. Wroc. Wet. 44: 113-127.

Matsuda M. and H. Takahashi (1996). Mospilon R (acetamiprid, NI-25) a new systemic insecticide. Agrochem. Japan 68: 20-21.

McComb R.B. (1983). The measurement of lactate dehydrogenase, In: Clinical and analytical concepts in enzymology, Hombrger, H.A. (eds.). Academic Press, New York, pp. 157-171.

Masatoshi H. (1983). Lead toxicity in the pregnant , The effect of high level lead on δ-aminolevulinic acid dehydratase activity in maternal and fetal blood or tissues. Environ. Res. 30: 152-160.

Randhawa S.S., G. Singh, D.C. Nauriyal and R.S. Brar (1995). Comparative evalution of therapeutictrials in experimentally induced lead toxicosis in crossbred calves. Bovi. Practi. 29: 136-138.

SchumachM., J., L. Domingo, J.M.L. Lobet and J. Corbella (1994)., Cadmium,chromium, copper and zinc in rice and field soil from southerm catalonia. Spain.Bull. Environ. Contam. Toxicol. 53: 54-60.

Sidhu M. and R. Nath (1996). Induction of hepatic glutathione s-transferase activity in rhesus monkey by cadmium. J. Trac. Element. Experi. Med. 9: 41-46.

Tabuchi T., A. Okayama, Y. Ogawa, K. Miyajima, M. Hirata, T. Yoshida, K. Sugimoto and K. Monrimoto (1989). A new HPLC flurimetric method to monitor urinary delta-aminolevulinic acid levels in workers exposed to lead. Int. Arch. Environ. Health 61: 297-302.

Tomio J.M., V. Tuzman and H. Grinsteine (1968). δ-aminolevulinic acid dehydratase from rat harderian gland. Purification and properties. Eurp. J. Biochem. 6: 84-87.

World Health Organization (1980). Recommended health-based limits in occupational exposure to heavy metals. Techn. Rep. Ser. 647.

World Health Organization (1987). Air quality guidelines for Europe. WHO Reg. Publ., Europ. Ser. 23.

الملخص العربى

السمية والتأثيرات البيوكيماوية لكل من الرصاص والكادميوم ومبيد الاستيامبيرد ومخاليطهما على ذكور الفئران

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قسم وقاية النبات – كلية الزراعة – جامعة الملك سعود

الرياض – المملكة العربية لسعودية

تم دراسة سمية وكذلك التأثيرات البيوكيماوية لكل من الرصاص(١٠مجم/كجم/يوم) والكادميوم (١ مجم/كجم/يوم) ومبيد الاستيامبيرد (٣٤ ، ٦٨ مجم/كجم/يوم) ومخاليطهما وذلك بمعاملة ذكور الفئران عن طريق الفم لمدة ١٤ يوم. أظهرت النتائج ارتفاع نسبة كل من الرصاص و الكادميوم بالجرعات السابقة حيث بلغت نسبة لموت ٥٥,٦٪ ، ٦٦,٧٪ لكل منها على التوالى بينما أدت الجرعه المرتفعة من الاسيتاميبرد (٦٨ مجم/كجم/يوم) الى نسبة موت بلغت ١١,١٪ فقط.

أدت مخاليط الرصاص والكادميوم مع مبيد الاستياميبرد الى خفض النسب المئوية للموت لكل من الرصاص و الكادميوم كل على حدة وهذا يعنى ان هناك فعل تضادى من هذا المبيد تجاة تأثير كل من الرصاص و الكادميوم.

تم دراسة تأثير المعاملات السابقة من الرصاص والكادميوم على انزيمات AIAD، AChE، GST، وبينت الدراسة قدرة كل من الرصاص والكادميوم على تثبيط جزئى لنشاط هذة الانزيمات كما أدت فى نفس الوقت الى ارتفاع مستوى انزيم LDH فى السيرم، أما مخاليط هذة المعادن مع مبيد الاستيامبرد فقد أدت الى خفض القدرة التثبيطية لكل من الرصاص والكادميوم على تلك الاهداف الانزيمية بما فى ذلك الزيادة التى حدثت فى مستوى انزيم LDH.

كذلك وضحت النتائج ان هناك توافق بين السمية والتغير فى نشاط الانزيمات نتيجة لتأثير المعاملات المختلفة مما يعكس إمكانية قيام مبيد الاستيامبرد بدورة كجوهر حماية ضد التأثيرات الضارة التى تحدثها المعادن الثقيلة.