Biochemical Alterations Induced by Abamectin in Albino Rats, *Rattus norvegicus*

N. A. Hamed and R. K. Abdel-Razik[1]

ABSTRACT

Abamectin is a multipurpose pesticide, used as insecticide, nematicide, acaricide and antiparasitic agent in farm animals and pets. This study examined biochemical evaluation of liver and kidney functions as a result of abamectin toxicity in male albino rats. A single sublethal oral dose of 3.3mg Kg^{-1} body weight (1/3 LD_{50}) was given. Hepatotoxicity was monitored at various times (4, 24 and 336 h) by quantitative analysis of the serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP) activities, total protein, albumin, total bilrubin, triglycerides and total cholesterol levels. Creatinine and urea were used as the biomarkers of kidney damages.

Results showed that abamectin administration caused no clear interaction on total protein level, while AST, ALT and ALP activities, total bilrubin, triglycerides, cholesterol, creatinine and urea levels were increased. Whereas albumin level showed a significant reduction when compared with control group.

In conclusion, the results indicated that changes in body and relative organ weights have been used as indicators of adverse effects of abamectin and also the alteration in tested enzymes activity and other parameters can be used as relevant biomarkers for monitoring toxicity due to abamectin exposure in mammals.

Keywords: Abamectin toxicity; Relative organ weight; Biochemical parameters; Serum; Rats.

INTRODUCTION

Abamectin (ABA) belongs to the family of avermectins, which are the macrocyclic lactones produced by a soil actinomycete, *Streptomyces avemitilis* (Fisher and Mrozik,1989). Abamectin (avermectin B1) is a mixture of two components, with the major component avermectin $B1_a$ 80% of the mixture, and the minor component avermectin $B1_b$ 20% of the mixture, differing by a single methylene group.

Abamectin is currently used in several countries as a pest control agent in livestock and as an active substance of nematicides and insecticides for agricultural use. Abamectin is highly toxic to insects and may be highly toxic to mammals (Lankas and Gordon, 1989). Tests with laboratory animals showed that the half-life of avermectin $B1_a$ in rat tissues averaged 1.2 days and rapidly eliminated from the body via the feces.

Rats given single oral dose of radio-labeled avermectin $B1_a$ excreted most of the dose (69 to 82%) unchanged in the feces (US EBA, 1990 and Thongsinthusak *et al.,* 1990).

Due to its high lipophilic nature, abamectin tends to accumulate in fat tissue, which acts as a drug reservoir and the highest levels of abamectin were found in liver and fat, and the lowest in brain tissue (Gonzalez, 2009). So the detoxification of abamectin may affect the function of hepatocytes although permanent liver damage is not usually revealed immediately (Hsu, 2001). Hepatic disease can be evaluated and diagnosed by determining concentrations of a number of serum analytes which are associated with hepatic necrosis (ALT and AST), cholestasis (ALP), defects in excretion (Bilrubin) and end stage hepatic disease which results in decreased synthetic function (Albumin) Wolf, 1999. The present study aimed to evaluate and followed up the adverse effect of a single sublethal dose of abamectin on some biological and biochemical parameters, consequently these parameters can be used as potential biomarkers of liver and kidney damage caused by abamectin exposure, and whether these effects are temporary or permanent.

MATERIALS AND METHODS

1. Chemicals

Abamectin, technical grade (94%): a mixture containing a minimum of 80% avermectin $B1_a$ (5-O-demethylavermectin A_a) and a maximum of 20% avermectin $B1_b$ (5-O-demethyl-25-de-(1-methylpropyl)-25-(1-1 methylethyl) avermectin A_a) was obtained from Agromen Chemicals Co., Ltd.

2. Animals

Adult male albino rats (Sprague-Dawely), *Rattus norvegicus albinus*, weighting 198±5gm were purchased from Faculty of Medicine; Alexandria University. The animals were allowed to acclimatize under laboratory conditions {12 h light / 12 h dark, 22–26 °C temp., 40–70% humidity} and provided with commercial diet and water *ad libitum* for one week before the initiation of the experiment. All maintenance

[1]Dept. of Mammalian and Aquatic Toxicology, Central Agricultural Pesticides Laboratory (CAPL), Agricultural Research Center (ARC), Alexandria, Egypt

Received August 26, 2015, Accepted September 20,2015

and care were in accordance with the animal welfare guidelines established at the university.

3. Experimental designs

Experimental animals were divided into two groups each of fifteen rats.

Group I: received corn oil 1 ml. kg $^{-1}$ b.w., used as control. Group II was received a single oral dose of 3.3 mg kg^{-1} b.w., of abamectin which represent 1/3 LD_{50} (the oral median lethal dose LD_{50} was 10 mg. kg^{-1} body weight) Tomlin, 1994. Five animals from each group were scarified at 4, 24 and 336 h after dosing. The animals were dosed orally via stomach gavage tube. The body weights of control and treated animals were recorded daily. At the end of the tested periods the animals were sacrificed and dissected, then the livers and kidneys from both control and treated animals were removed and immediately washed with physiological saline (0.9%NaCl) and weighed individually and the relative organ weight was calculated (organ weight : body weight).

4. Blood sample collection

Blood samples were collected by cardiac puncture from each animal into clean dry centrifuge tubes. The blood were left to clot at room temperature for about 20 min. and then centrifuged at 3000 rpm for 15 minutes. Serum samples were drown in dry clean-capped tubes and kept in deep freezer at -20 ^{o}C until conducting the biochemical analysis.

5. Biochemical parameters

Sera samples were used to estimate the activities of aspartate and alanine aminotransferase (AST& ALT) according to the method of (Retiman and Frankel, 1957), whereas alkaline phosphatase (ALP) activity was determined by the method of (Belfield and Goldberg, 1971). Total protein**,** albumin, total bilirubin, urea and creatinine concentrations were determined according to the methods of (Barawill and David, 1949; Doumas *et al*., 1971; Walter and Gerade, 1970; Fawcett and Scott, 1960; Schirmeister *et al*., 1964 respectively). Cholesterol and triglycerides levels were determined by the methods of Richmond, 1973; Fassati and Prencipe, 2007 respectively.

6. Statistical analysis

Data obtained from the experiments were expressed as mean ±standard error (SE). Significant differences of measurement traits were analyzed using one-way analysis of variance (ANOVA) followed by the Student-Newman-Keuls Test. The criterion for statistical was set at $p<0.05$.These test were performed using a computer software CoStat program.

RESULTS

1. Body and relative organ weights

The physiological status of control and treated animals was noticed as the change in body and relative organ weights. Male rats orally administrated with a single dose of abamectin (3.3 $mg.kg^{-1}$ b.w) have shown no mortality occurred during the experimental period (2 weeks). Table (1) showed a decrease in rat's weight after 4 and 24 h compared to their controls. A marked increase in the relative liver weights was observed at 336 h, while the kidney weights showed a significant reduction at (4&336 h), compare with control.

2. Biochemical studies

2.1 Liver function parameters:

AST, ALT and ALP activities: AST, ALT and ALP were considered to be sensitive indicators of hepatocellular damage (Peng *et al.,* 2007).

Data in Fig. 1 showed a significant ($p<0.05$) increase in the activity of AST at 24 and 336 h, which are almost twice as much as the activity of control and elevation in ALT activity has been reported at all tested times compared to control. The ALP activity showed gradually elevation in time dependent manner.

Total protein, albumin and total bilrubin levels: Fig. 2 revealed that both of albumin and total bilrubin levels were changed in time dependent manner. Albumin exhibit significant reduction ($p<0.05$) at 24 and 336h after treatment compared to control. Conversely, the total bilrubin was gradually increased at 4, 24 and 336h. In addition, total protein levels were oscillating and didn't revealed a significant changes.

Table 1. Body and Relative Organ Weights of Rats after Various Time of Treatment with a Single Sublethal Dose of Abamectin (3.3 mg. Kg^{-1} body weight)

Time after Treatment (h)	Body Weight (g)		Relative Organ Weights (%)	
	Control	Treated	Liver	Kidney
0	217.67±1.46^{c}	217.67±1.46^{b}	3.55±0.05^{a}	0.80±0.06^{b}
4	193.00±1.73^{a}	190.33±1.20^{a}	3.50±0.02^{a}	0.67±0.09^{a}
24	194.67±0.34^{a}	186.77±1.25^{a}	3.63±0.03^{a}	0.77±0.04^{b}
336	206.67±1.46^{b}	217.67±0.88^{b}	4.21±0.04^{b}	0.69±0.04^{a}

Values are expressed as means (5 rats) ± standard errors

Values in column with different letters are significantly different at ($p < 0.05$).

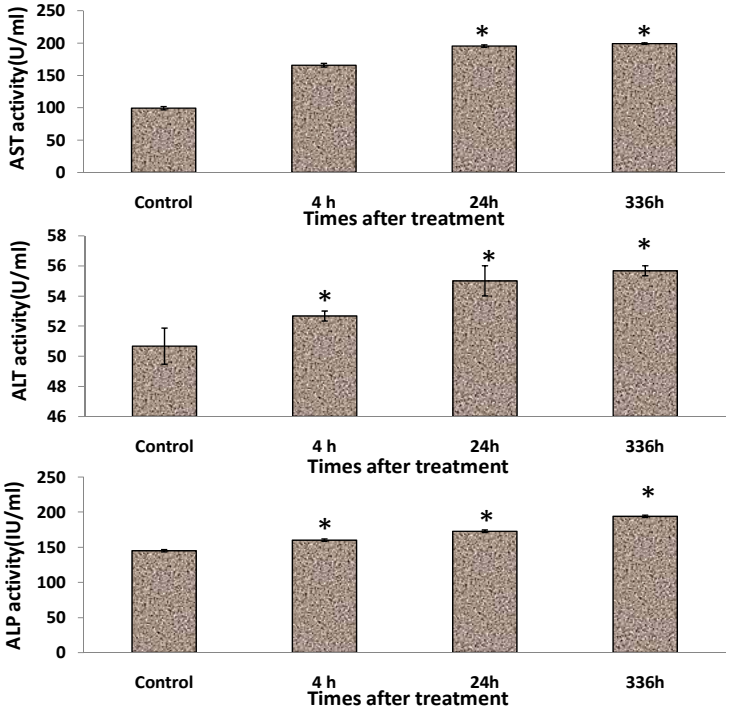

Figure 1. AST, ALT and ALP activities on sera of male rats after treatment with a single sublethal dose of abamectin (3.3 mg Kg^{-1} body weight). Values represent the mean± SE of five rats. *Significantly different from the control value at ($p < 0.05$)

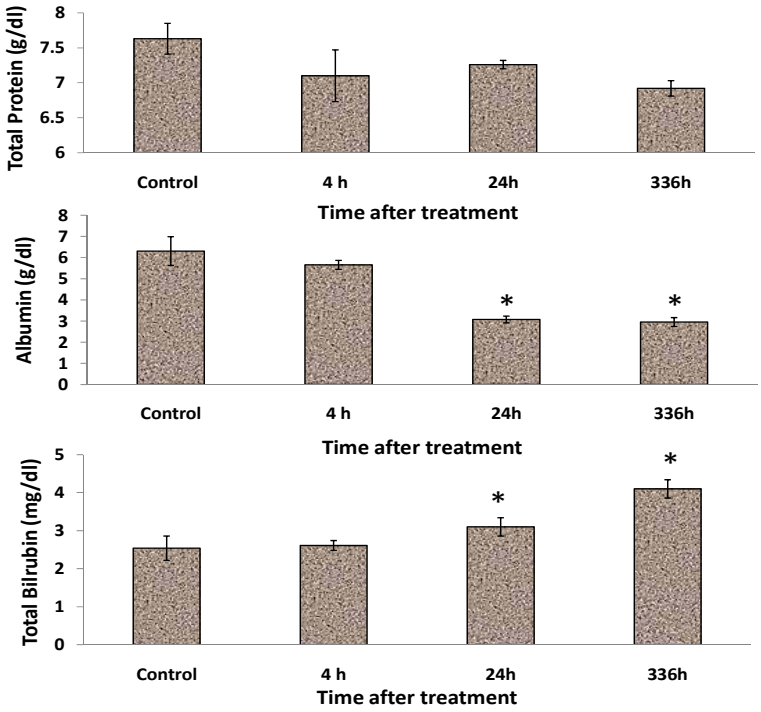

Figure 2. Total protein, albumin and total bilrubin on sera of male rats after treatment with a single sublethal dose of abamectin (3.3 mg Kg^{-1} body weight). Values represent the mean± SE of five rats. *Significantly different from the control value at ($p < 0.05$)

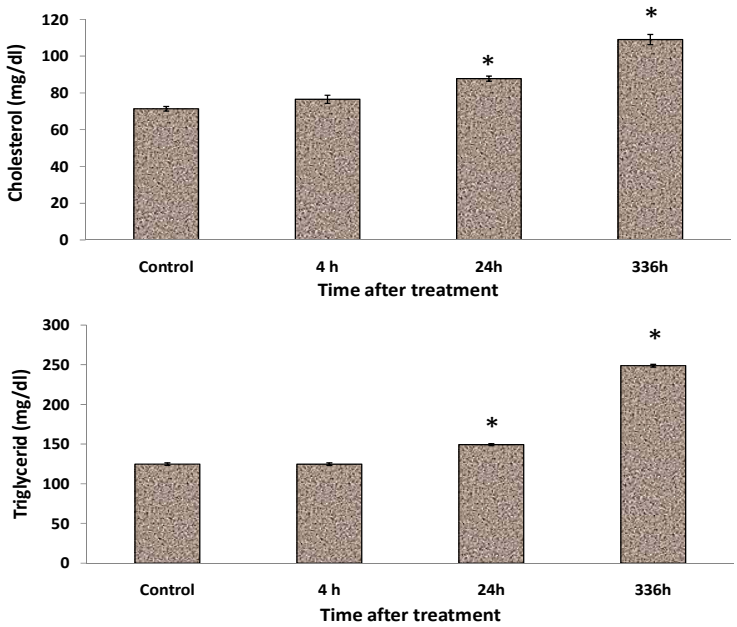

Figure 3. Cholesterol and triglyceride on sera of male rats after treatment with a single sublethal dose of abamectin (3.3 mg Kg^{-1} body weight). Values represent the mean± SE of five rats. *Significantly different from the control value at ($p < 0.05$)

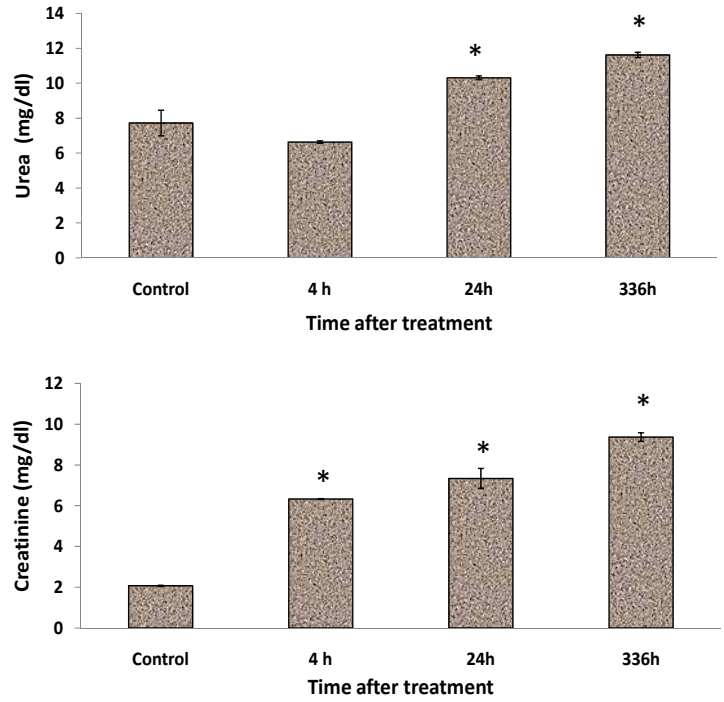

Figure 4. Urea and creatinine concentrations on sera of male rats after treatment with a single sublethal dose of abamectin (3.3 mg Kg^{-1} body weight). Values represent the mean± SE of five rats. *Significantly different from the control value at ($p < 0.05$)

Changes in cholesterol and triglycerides levels: abamectin administration caused significant alterations in the tested serum lipids; an enhancement in the total cholesterol concentration at 24 and 336 h and a drastic increase in level of triglycerides at 336 h after treatment compared to control were showed in Fig. 3.

2.2 Kidney function parameters:

Results in Fig. 4 showed a significant increase in urea concentration at times 24 and 336h.at the same manner creatinine levels were markedly elevated by 3, 3.5 and 4.5 fold at 4, 24 and 336 h. respectively, compared to control.

DISCUSSION

It's known that abamectin is a biocide widely used in agriculture and veterinary against pests. But it's toxicity to mammals has a great debate. So we carried out this research to investigate the adverse effect of one fixed dose at short time and after recovery period of exposure to abamectin.

In toxicological studies body and organ weights are important criteria for the evaluation of toxicity. Our Results showed decreasing in the body weight, and this may be attributed to a decreased food intake as a result of diarrhea and food avoidance which observed after 4h and 24h of the abamectin administration. It has been reported that changes in either absolute or relative weight of an organ after administering a chemical or drug is an indication of the toxic effect of that chemical (Simons *et al*.,1995). The increase in relative liver weight in rats exposed to abamectin seems to be due to its toxic effect, and this is in agreement with those obtained by Eissa and Zidan, 2009.

The increase in liver weight may be attributed to the increased circulation as a result of increased demands for the detoxification of toxic compounds (Okuna *et al*., 1976). Some researchers reported that liver enlargement may be related to the maintenance of the liver normal functional capacity (Robinson and Yarbrough, 1978). Others explained that, the liver enlargement could be due to the accumulation of abnormal amounts of fat, predominately triglyceride, in the parenchymal cells. Triglyceride accumulation is a result of an imbalance between the rate of synthesis and the rate of release of triglyceride by the parenchymal cells into the systemic circulation (Plaa, 1980). In contrast; another study stated that liver enlargement is not necessarily considered toxic lesions, since this effect is observed in a large number of compounds (Bhatnagar and Jain, 1986). On the other hand, Abamectin exhibited significant decrease in the relative weight of treated male rat's kidney (4&336h) compared to control rats. Changes in kidney weight may reflect renal toxicity, tubular hypertrophy or chronic progressive nephropathy (Sellers *et al*., 2007).

Liver is often the primary target for the toxic effects of xenobiotics. It is known that the detoxification of the toxic materials which enter the body occurs mainly in the liver (Balistrei and Shaw, 1987). Therefore; liver can be used as an index for the toxicity of xenobiotics. The elevation in the liver enzyme activities may be due to liver dysfunction with a consequent reduction in enzyme biosynthesis and altered membrane permeability permitting enzyme leakages into the blood (Mansour and Mossa, 2010). Obtained results revealed significant increase of AST, ALT and ALP activities, which may be due to damage of liver cells under the effect of abamectin biocide (Ismail *et al*., 2013). In addition, the increases of serum AST and ALP activities may be referred to diffusion of these enzymes from the intracellular sites due to the damage caused by the pesticide on the sub cellular level (Kalender *et al*., 2005). As certain hepatic damage is considered pathologically irreversible (Helling *et al*., 1995), the elevation of AST may render the liver to be more susceptible to other pathogen/toxicants (Nayak *et al*., 1996). The magnitude of the ALP elevation is of some help in the differential diagnosis of hepatobiliary disorders such as primary or secondary liver malignancy and extrahepatic cholestasis (Tomlin , 1994).

Albumin plays a major role in binding bilirubin, fatty acids, hormones such as thyroxin, triiodothyronine, cortisol and aldosterone; it also binds calcium and magnesium. Qualitative and quantitative disturbance of protein synthesis is a consequence of impaired hepatic function (Celia and Wilkinson, 1973). Our results showed a significant reduction in albumin levels (Hypoalbuminemia), which thought to be a consequence of decreased hepatic synthesis of albumin and these results are in agreement with those reported by (Burtis and Ashwood, 1994). Present work showed that total bilrubin level was elevated which is in accordance with (Perlstein *et al.,* 2008) who found that a high serum total bilirubin level may protect neurologic damage due to stroke. Others reported that serum bilirubin significantly contributes to total antioxidant capacity. It was discovered that bilirubin had anti-

inflammatory effects as well as acts as scavengers of reactive oxygen species (Ndisang and Jadhav, 2009).

Many clinical trials has demonstrated the relationship between coronary heart diseases and arteriosclerosis with hypercholesterolemia, elevation in cholesterol and triglycerides levels (Kannel *et al*., 1979). Our data exhibited a significant elevation of cholesterol and triglyceride levels in treated rats which demonstrate the ability of abamectin to influence liver metabolism towards increased synthesis of lipids which in line with other pesticides (Padma *et al.* 2012).

Significant increases of sera urea and creatinine post abamectin treatments in the present study may be due to damage of kidney cells and/or failure of kidney under the toxic effect of abamectin which in agreement with those reported by (Eissa and Zidan, 2009).

CONCLUSION

The present study demonstrated that sublethal oral administration of abamectin at 1/3 LD_{50} for 4, 24 and 336h, caused cytotoxic changes in the hepatic and renal biochemical markers, which reflects hazardous effects at various levels to non-target organisms. Hence these biomarkers might be used to monitor the non target organism's exposure to pesticides to take effective measures to avoid serious adverse health effects.

REFERENCES

Balistrei, W. F. and Shaw, L.M. (1987). Liver function. In: Tietz NW, ed. Fundamentals of clinical chemistry.3rd ed. Philadelphia: WB Saunders.729-761.

Barawill, C. J. and David, M.M. (1949). Total protein was determined spectrophotometerically. J. Biol. Chem.177:751.

Belfield, A. and Goldberg, D.M. (1971). Revised assay for serum phenyl pho sphatase activity using 4-aminoantipyrine.Enzyme.12: 561- 573.

Bhatnagar, P. and Jain, N. (1986). Morphofunctional changes in the liver of male mice after chronic treatment with Phosphamidon. Bull. Environ. Contam. Toxicol. 37: 767 – 773.

Burtis, C.A. and Ashwood, E.R. (1994).Tietz Textbook of Clinical Chemistry, 2nd ed. Philadelphia: W. B. Saunders.

Celia, M.H. and Wilkinson, J.S. (1973). Liver function. Aust. Vet. J. 49:163-169.

Doumas, B.T., Watson, W.A. and Biggs, H.G. (1971). Albumin standards and measurement of serum albumin with bromcresol green. Clin. Chim. Acta. 31 (1): 87–96.

Eissa, F.I. and Zidan, N.A. (2009). Haematological, biochemical and histopathological alterations induced by abamectin and *Bacillus thuringiensis* in male albino rats. Australian J. of Basic & Applied Sci.3 :(3): 2497-2505.

Fassati, P. and Prencipe, L. (1982). Serum triglycerides determined colorimetrically with an enzyme that produces hydrogen peroxide. Clin. Chem.28: 2077.

Fawcett, J.K .and Scott, J.E. (1960). Determination of urea (urease modified Berthelot reaction). J. Clin. Pathol. 13 (2): 156–159.

Fisher, M.H. and Mrozik, H. (1989). Chemistry. In: Ivermectin and Abamectin .WC. Campbell, ed. Springer-Verlag, New York, 363 pp; P.1-23.

Gonzalez, A., Sahagun, A., Diez, M.J., Fernandez, N., Sierra, M. and Garcia, J.J. (2009). The pharmacokinetics and metabolism of ivermectin in domestic animal species. The Veterinary Journal.179: 25–37.

Helling, T.S., Wogahn, B.M., Olson, S.A., Evans, L.S., Reddy, B.R. and VanWay, C. (1995). The effect of prostaglandin E1 on liver adeninenucleotides and cytoplasmic enzymes in a porcine model of normothermic hepatic ischemia. Hepatology. 22: (5): 1554–1559.

Hsu, D.Z., Hsu, C.H., Huang, B.M. and Liu, M.Y. (2001). Abamectin effects on aspartate aminotransferase and nitric oxide in rats. Toxicology.165 (3):189-193.

Ismail, N.H.A., Suliman, S.E., Bulldan, A.G.A. and Seri, H.I. (2013). Haematological, Biochemical and Histopathological Alteration Induced by Ivermectin in Donkeys (*Equus asinus*) .Sudan Journal of Science and Technology.14: (2):1-14.

Kalender, S., Ogutcu, A., Uzunhisarcikli, M., Acikgoz, F., Durak, D. and Ulusoy, Y. (2005). Diazinon-induced hepatotoxicity and protective effect of vitamin E on some biochemical indices and ultrastructural changes. Toxicology. 211: (3): 197–206.

Kannel, W.B., Casteli, W.B. and Gordon, T.L. (1979). Cholesterol in the Prediction of atherosclerotic disease. New perspectives based on the Framingham study. Ann Intern Me. 90:85-91.

Lankas, G.R. and Gordon, L.R. (1989). Toxicology in W.C. Campbelln (ed.). Ivermectin and Abamectin. Springer-Verlag, NY.

Mansour, S.A .and Mossa, A.H. (2010). Oxidative damage, biochemical and histopathological alteration in rat exposed to chlorpyrifos and the role of zinc as antioxidant. Pest Biochem.Physiol. 96: (1): 14–23.

Nayak, N.C., Sathar, S.A., Mughal, S., Duttagupta, S., Mathur, M. and Chopra, P. (1996). The nature and significance of liver cell vacuolation following hepatocellular injury-an analysis based on observations on rats rendered tolerant to hepatotoxic damage. Virchows Archiv. 428: (6): 353–365.

Ndisang, J.F. and Jadhav, A. (2009). Up-regulating the hemeoxygenase system enhances insulin sensitivity and improves glucose metabolism in insulin-resistant diabetes in Goto-Kakizaki rats. Endocrinology.150:2627-2636.

Okuna, K.T., Kohdo, H. and Mtyamoto, J. (1976). Mammalians toxicological study of permethin, 3 phenoxy benzyl(±) astrans –2,2- dimethyl-3-(2, 2 dichlorvinyl) – cyclopropane –1-carboxylate. Botyukgoku. 41: 143-151.

Padma, V.V., Lalitha, G., Shirony, N.P. and Baskaran, R. (2012). Effect of quercetin against lindane induced

alterations in the serum and hepatic tissue lipids in wistar rats**.** Asian Pac. J. Trop. Biomed. 2 :(11): 910-915.

Peng, D., Chen, S., Ruan, L., Li L, Yu. Z. and Sun, M. (2007). Safety Assessment of transgenic *Bacillus thuringiensis* with VIP insecticidal protein gene by feeding studies. Food Chem. Toxicol. 45:1179–1185.

Perlstein, T.S., Pande, R.L. and Creager, M.A. (2008). Serum total bilirubin level prevalent stroke and stroke outcomes. NHANES 1999-2004. Am. J. Med. 121: 781-788.

Plaa, G.L. (1980). Toxic responses of the liver. In: Casarett and Doull's toxicology: the basic science of poisons. 2 ed., J. Doull, C.D. Klaassen and M.O. Amdur (Eds.). Macmillan, New York.

Reitman, S. and Frankel, S. A. (1957) Colorimetric method for the determination of serum glutamic oxalacetic and glutamic pyruvic transaminases. Am J Clin Path. 28 (1): 56–60.

Richmond, W. (1973). Preparation and properties of a cholesterol oxidase from Nocardia sp. and its application to the enzymatic assay of total cholesterol in serum. Clin. Chem. 19:1350–1356.

Robinson, K.M. and Yarbrough, J.D. (1978). Liver response to oral administration of Mirex in rates. Pest. Biochem. Physiol. 8 :(1): 65-72.

Schirmeiste, J. (1964). Determination of creatinin in blood spectrophotometerically. Dtsch. Med. Wsch. 89:1940.

Sellers,R.S., Morton, D., Michael, B., Roome, N., Johnson, J.K. and Yano, B.L.(2007). Society of Toxicological Pathology Position Paper: Organ Weight Recommendations for Toxicology Studies *Toxicologic Pathology*. 35:751–755.

Simons, J.E., Yany, R.S. and Berman, F. (1995). Evaluation of the nephrotoxicity of complex mixture containing organics and metals. Advantages and disadvantages of the use of real- world complex mixture. Environ. Health Prospect.103: (supple. 1): 67-71.

Thongsinthusak, T., Ross, J., Fong, H.R., Formoli, T. and Krieger, R.I. (1990). Estimation of exposure of persons in California to pesticide products that contain abamectin. California Dept. of Food and Agriculture, Div. of Pest Management, Environmental Protection and Worker Safety, Worker Health and Safety Branch, Sacramento, CA; June 5, H1567.

Tomlin, CDS. (1994). The Pesticide Manual - World Compendium. 10th ed. Surrey, UK: The British Crop Protection Council. p. 4 **PEER REVIEWED**

U.S. Environmental Protection Agency. (1990). Pesticide Fact Sheet Number 89.2: Avermectin B1. US EPA, Office of Pesticide Programs, Registration Div. Washington, DC. July 20.

Walter, M. and Gerade, R.W. (1970). Bilirubin direct/total. Microchem. J. 15:231–233.

Wolf, P.L. (1999). Biochemical diagnosis of liver disease. Indian Journal of Clinical Biochemistry.14 (1):59-90.

الملخص العربي

التغيرات البيوكيميائية الناجمة عن الأبامكتين في الجرذان البيضاء، الجرذ النرويجي

نادية على حامد، رضا خميس عبد الرازق

الأبامكتين من المبيدات متعددة الأغـراض، وتـستخدم كمبيد حشرى، نيماتودى، أكاروسى ومضاد للطفيليات فـي حيوانات المزرعة والحيوانات الأليفة. قامت هذه الدراسـة بتقييم التغيرات البيوكيميائية لوظائف كلا من الكبد والكلـى كنتيجة لسمية الأبامكتين في ذكـور الجـرذان البيـضاء. أعطيت جرعة واحدة تحت المميتة عن طريـق الفـم ٣،٣ ملجم كجم$^{-1}$ وزن الجسم ١/٣ LD_{50} (ثلث الجرعة المسببة لموت نصف الأفراد المعاملة، تم رصد السمية الكبدية فـي أزمنة مختلفة(٤، ٢٤، ٣٣٦ ساعة) من خلال التحليل الكمي لـسيرم اسـبارتات امينوترانـسفيراز (AST)، ألانـين امينوترانـسفيراز (ALT)، الفوسـفاتيز القلويـة (ALP)، البروتين الكلي، الألبيومين، البيليـروبين الكلـي، الـدهون الثلاثية ومجموع مستويات الكوليسترول في الدم كمؤشرات حيوية على الإضرار بالكبد. استخدم الكرياتينين واليوريـا كمؤشرات حيوية على الإضرار بالكلى.

لم تظهرالمعاملة بلأبامكتين أى تغيـر واضـح فـى مستوى البروتين الكلي، بينما زاد نشاط كل مـن ALP & AST, ALT وكذلك مستويات البيليروبين الكلي والـدهون الثلاثية والكوليسترول، والكرياتينين واليوريا في حـين أن مستوى الألبيومين أظهر انخفاضا كبيرا.

ونستخلص من هذا البحث أنه يمكن استخدام التغيـرات في وزن الجسم والأوزان النسبية للأعضاء كدالـة علـى الآثار السلبية للأبامكتين وايضا حدوث تغيـر فـي نـشاط

الانزيمات والثوابت الأخرى المختبرة، يمكـن أن تـستخدم كمؤشرات حيوية لرصد سمية الأبامكتين في الثدييات.

Keywords: Abamectin toxicity; Relative organ weight; Biochemical parameters; Serum; Rats