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Subchronic Impact of Organophosphorus insecticide Triazophos on Liver, Kidneys and Thyroid in Albino Rats

Ahmed, A. Gh. Farag[1]; Gamila, A. M. Kotb[2]; Amal H Hamza[3] , Rasha Hamed Mahmoud[3,5] **and Manal E. A. Elhalwagy*[3,4]**

[1]Plant Protection Dept., Faculty of Agriculture, Zagazig University, Egypt.
[2]Chemistry Department, Shaqra University, Aldawadmi college of Science and Humanities for Girl's, P. O. Box 1361, 11911, Saudi Arabia.
[3]Department of Biochemistry, Faculty of Science-Al Faisaliah Campus, King Abdulaziz University, P. O. Box 51459, Jeddah- 21453, Saudi Arabia
[4]Mammalian and Aquatic Toxicology Dept., Central Agricultural Pesticides Laboratory, Agriculture Research Center, Giza 12618, Egypt.
[5]Biochmesitry and Nutrition Dep. Faculty of Women, Cairo, Egypt
*Corresponding author: *melhalwagy62@yahoo.com*

Abstract

Objective: The study was designed to identify chronic effect of different doses (1/100LD50= 0.82 mg/kg and 1/50LD50= 1.64 mg/kgm) of Triazophos, O,O-diethyl-1-H-1,2,4-triazol-3-yl phosphorothioate, widely used organophosphorus pesticide (Ops). **Materials and Methods**: oxidative stress and plasma biomarkers of (liver, kidneys and thyroid) organs involved in xenobiotic metabolism. In addition to, histopathological examination. **Results:** The results revealed induction of oxidative stress, as evident by significant increase in malondialdehyde (MDA) level concurrent with reduction in total antioxidant capacity (TAC) in plasma of both TZ intoxicated groups. These observations were recorded following the inhibition of the plasma acetylcholinesterase (AchE) and ATPase enzymes biomarkers of Ops toxicity. Elevation in plasma liver biomarkers alanine (ALT) and aspartate (AST) aminotransferases activities in addition to total plasma protein were recorded in both intoxicated groups. Slight reduction in urea and creatinine plasma kidney biomarkers were recorded; However, significant elevation in plasma tiiodothyronine (T3) at $p<0.05$ and significant reduction in plasma (T4) was recorded in both intoxicated groups at $p<0.01$ and $p<0.001$. Histo-architecture of these organs confirms the above findings. **Conclusion**: The present study concludes that induction of oxidative stress that leading to subsequent alteration in liver, kidney and thyroid biomarkers and their histopathological architectures are an important mechanism in chronic TZ toxicity.

Keywords: organophosphorous pesticides (Ops); Triazophos (TZ); oxidative stress; liver; kidneys; thyroid.

1. Introduction

Pesticides are ubiquitous in the environment and have significant economic, environmental and public health impact. Their usage helps to improve human nutrition through greater availability, longer storage life and lower costs of foods (Weiss et al., 2004). Triazophos, TZ, [O, O – diethyl – O - (1 – phenyl - 1, 2, 4 – triazol – 3 - base) sulfur phosphate], is one of the broad - spectrum, moderately toxic, nonsystemic contact organophosphorus pesticides (OPs). It has been extensively used in agriculture for controlling pests as a stomach and contact poison against a broad spectrum of pest insects, acarids, flies and some nematodes that

damage agricultural, horticultural and forest crops since the late 1970s (Aungpradit *et al.* 2007; Lin and Yuan 2005). Residual levels of triazophos (TZ) in crops, vegetables and water may pose risks to the health of humans and other animals. Severity of TZ intoxication may vary with dose, route and extent of exposure (Dharmender *et al.*, 2014). triazophos (TZ) imposes an important health concern in humans (Kumari and Kathpals ,2009).The primary effect of triazophos (TZ) is neurotoxic leading to accumulation of neurotransmitter acetylcholinesterase in synaptic, cholinergic and neuromuscular effect (Kamanyire and karalliedde, 2004) . Apart from neurotoxicity and neurobehavioral changes in animals, have been shown to induce oxidative stress (OS) by generating elevated levels of reactive oxygen species(ROS) (Dharmender *et al.*, 2014).Oxidative stress (OS) occur when production of reactive oxygen species (ROS) overrides antioxidant capacity in target cells, resulting in the damage of macromolecules such as nucleic acids, lipids and proteins (Agrawal and Sharma, 2010). Rats exposed to triazophos for 30 days have Oxidative stress in blood and significant histopathological alterations in liver (Jain et al., 2010). Organophosphorus insecticides disrupt the endocrine system and they are suspected as triggers for harmful effects on the reproductive system (Oruç, 2010).

The objective of this study was to investigate subchronic impact of low doses of triazophos (TZ) on the function of liver, kidneys and thyroid glands as well as induction of oxidative stress in albino rats.

2. Materials and Methods

2.1.Materials

Triazophos (TZ)formulated form Hostathion 20 % EC was used in this study. The formulation was obtained from Syngenta Ltd., Egypt.

2.2. Animals and experimental design

Male albino rats *Rattus norvegicus* (3–4) months age, weighing between 130–150 g were used. Animals were supplied by the breeding unit of the Egyptian Organization for the Biology and Vaccine Production, Egypt. The animals were housed in plastic cages, fed *ad libitum* and allowed to adjust to the new environment for two weeks before starting the experiment. The rats were housed at 23 ± 2 ^{o}C dark/light cycle. Procedures involving animals were performed in accordance with the guidelines of the standard procedures laid down by (OECD 1992) subchronic oral toxicity rodent 90 days study, the protocol of this study was been approved by department of Mammalian Toxicology, Pesticide Central Laboratory, Agriculture Research Center, Egypt.

Animals were randomly divided into three experimental groups 10 animals each as follows:

Group 1 *control* (Cont) : rats were served as control group and administrated water orally by stomach tube.

Group II *triazophose low dose* (TZLD): Rats were orally given 1/100 LD50 (0.82 mg/kg bw) daily via gastric tube daily for 90 days.

Group III *triazophose high dose* (TZHD): Rats were orally given 1/50 LD50 (1.64 mg/kg bw) daily via gastric tube daily for 90 days

2.3. Samples:

At the end of 90 days, all the rats were put on overnight fasting and the blood samples were collected through retero - orbital plexus vein according to Schalm (1986) in heparinized vials. Plasma samples were separated by centrifugation of the blood samples at 3600 rpm for 15 min in a refrigerated centrifuge at 4 ◦C. Plasma samples were kept at - 40 ◦C till biochemical investigations were carried. animals were sacrificed and samples of the liver ,kidney and thyroid were excised for histopathological studies.

2.4. Biochemical Analysis:

Lipid peroxidation was measured in plasma as malondialdehyde (MDA) level by method of (Ohkawa, et al.,1979). On the other hand ,the total antioxidant capacity (TAC) was determined according to method (Koracevic, et al.,2001). Plasma total ATPase activity was determined as the rate of release of inorganic phosphate by method of (Samson and Quinn,1967). Tri - iodothyronine (T3) and Thyroxin (T4) hormones were performed in plasma using method of (Britton et al.,1975). Acetylcholinesterase (AChE) activity was determined by the method of (Ellmanet al.,1961). Markers for liver and kidney damage were determined using the commercial diagnostic kit of Stanbio Co., Spain. Plasma transaminases (AST and ALT) activities were determined according to (Reitmanand Frankel,1957).Plasma total albumin was carried out according to (Dumas et al., **1971**).Total protein was measured by method of (Bradford,1976). Plasma urea level was determined by the method of (Fawcett and

Scott, **1960),** while, plasma creatinine level was determined by the kinetic method of (Siest et al., 1985).

2.5.Histopathology

Histopathological examination was carried out according to (Drury and Wallington,1980). The liver, kidney and thyroid tissues were dissected and the tissue samples were fixed in 10% formalin solution for 14–18 h, passed in a series of graded ethanol and embedded in paraffin. Paraffin sections were cut with at 5 lm thickness and stained with hematoxylin and eosin for light microscopic examination. The sections were examined and photographed on an Olympus light microscope (Olympus BX51, Tokyo, Japan) with attachment photograph machine (Olympus C-5050, Olympus Optical Co. Ltd., Japan).

2.6. Statistical Analysis:

Data from biochemical analysis were subjected to statistical analysis by analysis of variance (ANOVA) one – way test (**Gad 1999 & 2001**) by SPSS software for Windows version 17 was used to run a Least Significant Differences (LSD) test to identify all biochemical parameters that differed between each treatment and the control at an overall significance level of $p \leq 0.05$, 0.01, and 0.001.

3. Results

3.1.Biochemical Studies:

The depicted results in Figure (1) demonstrated significant inhibition in plasma acetylcholinesterase (AchE) in rats intoxicated with low 1/100 LD_{50} (0.82 mg/kg bw) and 1/50 LD_{50} (1.64 mg/kg bw) of trizophose insecticide for 3months.however, significant inhibition in plasma ATPase enzyme was recorded in high dose intoxicated group only at $p<0.001$. The above changes accompanied with significant elevation in plasma malondialdehyde (MDA) oxidative stress biomarker and significant reduction in total plasma total antioxidant capacity (TAC) at $p< 0.01$ and $p<0.001$ in both intoxicated groups. Figure (2) expressed significant elevation in plasma liver biomarkers alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities in both intoxicated groups this elevation is dose dependent. Meanwhile, remarkable significant increase in total plasma protein was recorded in low dose intoxicated animals only at $p<0.01$. While, the increase in plasma albumin in both TZ intoxicated groups was non-significant. Slight reduction in urea and creatinine plasma kidney biomarkers was recorded in Figure (3). Moreover, significant elevation in plasma tiiodothyronine (T3) at $p<0.05$ and significant reduction in plasma (T4) was recorded in both intoxicated groups at $p<0.01$ and $p<0.001$ Figure (4).

3.2.Histopathological Examination

Photomicrograph (1) depicted liver of rats chronically treated with different doses of triazophos; photo (a) showed normal histological structure of hepatic lobule of liver tissue of control group.Portal edema and congestion of hepatoportal blood vessel in addition to necrosis of sporadic hepatocytes were expressed in liver of rats intoxicated with low 1/100 LD_{50} (0.82 mg/kg bw) of TZ (photos b&c). liver of high dose intoxicated rats 1/50 LD_{50} (1.64 mg/kg bw) TZ showed sinusoidal leucocytosis, also kupffer cells activation and portal infiltration with leucocytes (photo d).changes in kidney tissues architecture was demonstrated in Photomicrograph (1) control rat revealed normal histological structure of renal parenchyma of kidneys photo(a) Tz intoxication induced congestion of renal blood vessels in low dose group photo(b). Whereas, high dose treated group showed lobulation and congestion of glomerulertuft, intertubular blood capillaries and atrophy of some glomeruler tuft photo (c).

Photomicrograph (3) photo (a) showed normal histological structure of the follicle with cuboidal lining epithelium and eosinophilic secretion in the follicular lumen in thyroid of control animals. On the other hand, there was vacuolization in the follicular lining epithelium while other follicles had desquamated cells in the lumen with interacinar leucocytic cells infiltration in low dose TZ photo (b).sever degeneration in thyroid tissue was depictes in high dose TZ where The follicles showed cystic dilatation with flattened lining epithelium while other had desquamation in the lining epithelium with cast formation in the lumen and the third showed disorganization in the histological structure of the epithelial lining cells photo (c &d).

4. Discussion

Extensive application of pesticides is usually accompanied with serious problems of pollution and health hazards. It is established that many pesticides, in common use, can produce some toxic and adverse effects on the liver, kidney and thyroid gland

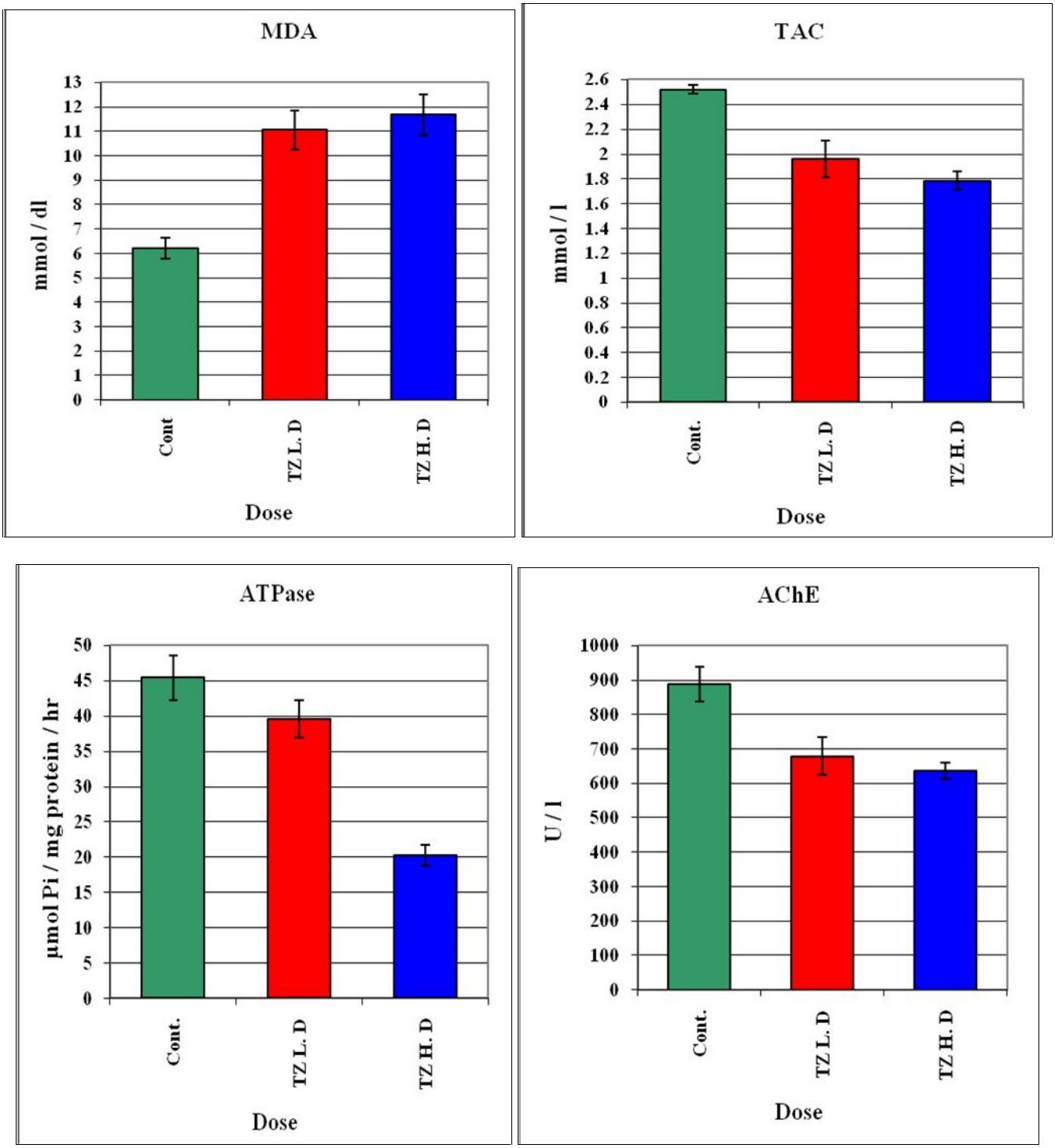

Fig. (1): Subchronic Effect of Different Doses of Triazophos (TZ) on Plasma Oxidative Stress and Neurotransmission Biomarkers of Male Rats.

All data were expressed as mean + MSE. ** Significant differences versus control at $p \leq 0.01$.
*** Significant differences versus control at $p \leq 0.001$.

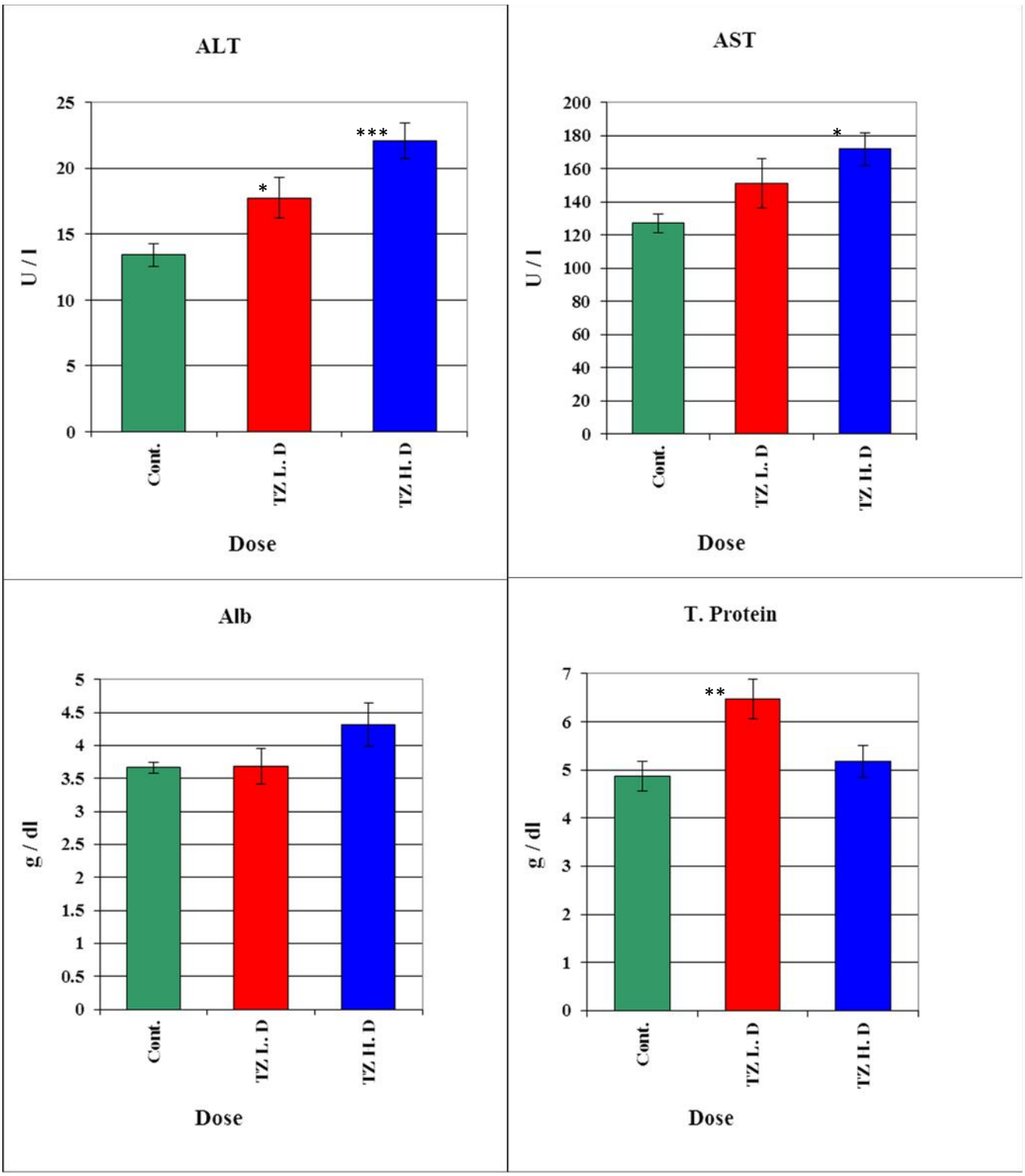

Fig. (2): Subchronic Effect of Different Doses of Triazophos (TZ) on Plasma Liver Biomarkers of Male Rats. All data were expressed as mean + MSE.

* Significant differences versus control at $p \leq 0.05$.** Significant differences versus control at $p \leq 0.01$.*** Significant differences versus control at $p \leq 0.001$.

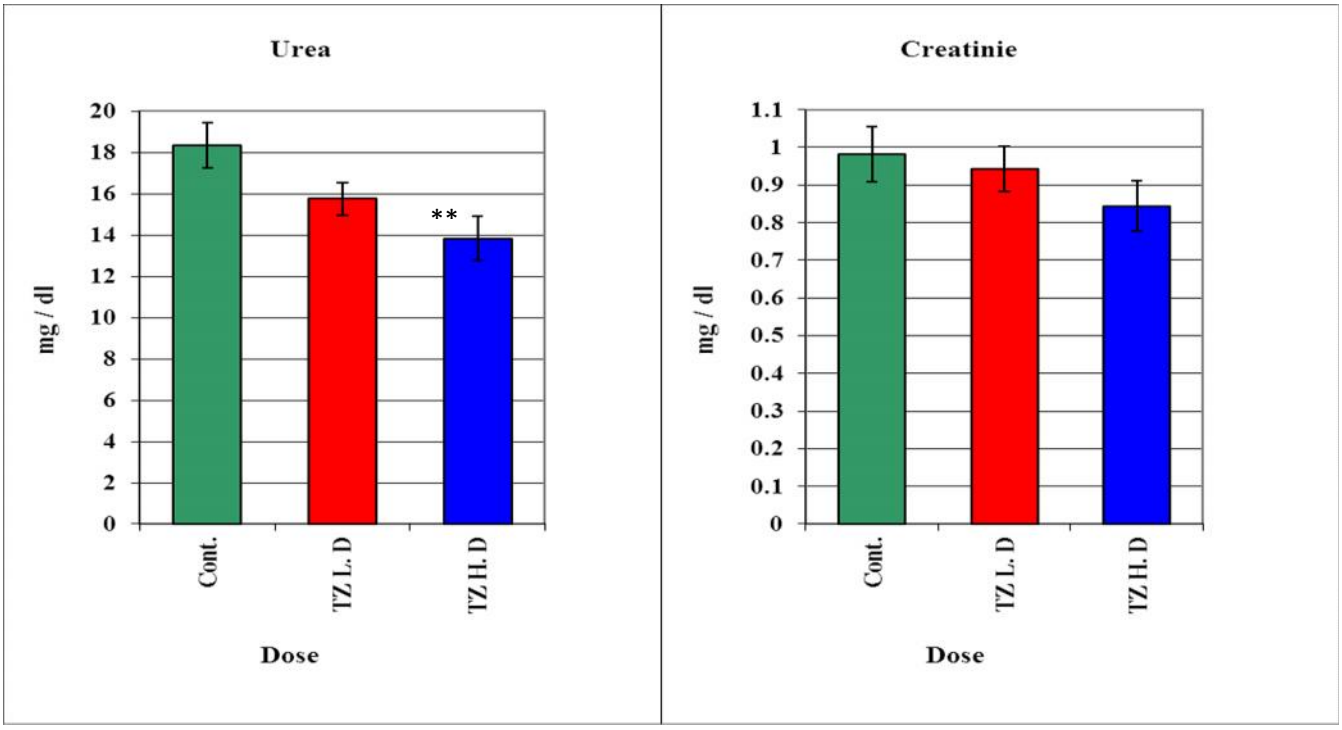

Fig. (3): Subchronic Effect of Different Doses of Triazophos (TZ) on Plasma Kidney Biomarkers of Male Rats. All data were expressed as mean + MSE.

* Significant differences versus control at $p \leq 0.05$.** Significant differences versus control at $p \leq 0.01$.*** Significant differences versus control at $p \leq 0.001$.

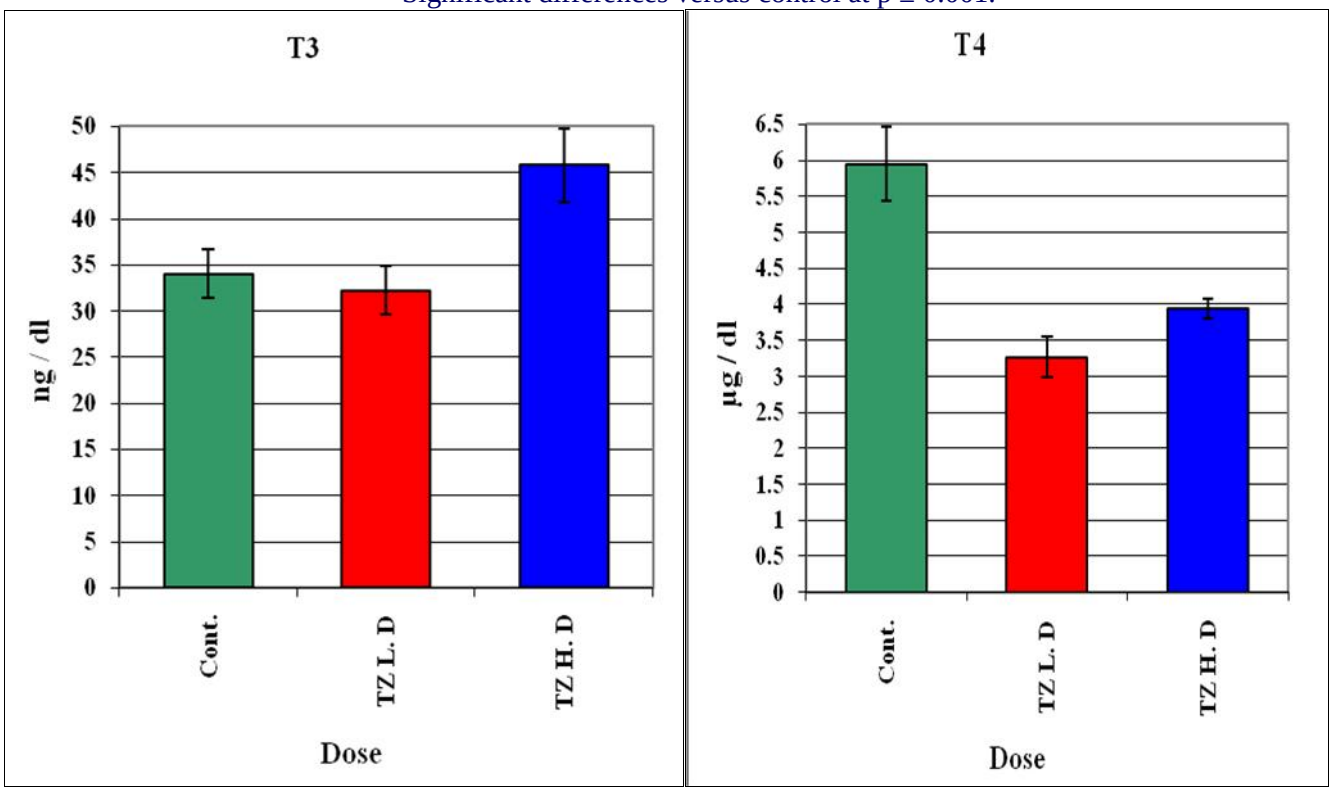

Fig. (4): Subchronic Effect of Different Doses of Triazophos (TZ) on Plasma Hormonal Thyroid Biomarkers of Male Rats.

All data were expressed as mean + MSE. * Significant differences versus control at $p \leq 0.05$
** Significant differences versus control at $p \leq 0.01$. *** Significant differences versus control at $p \leq 0.001$.

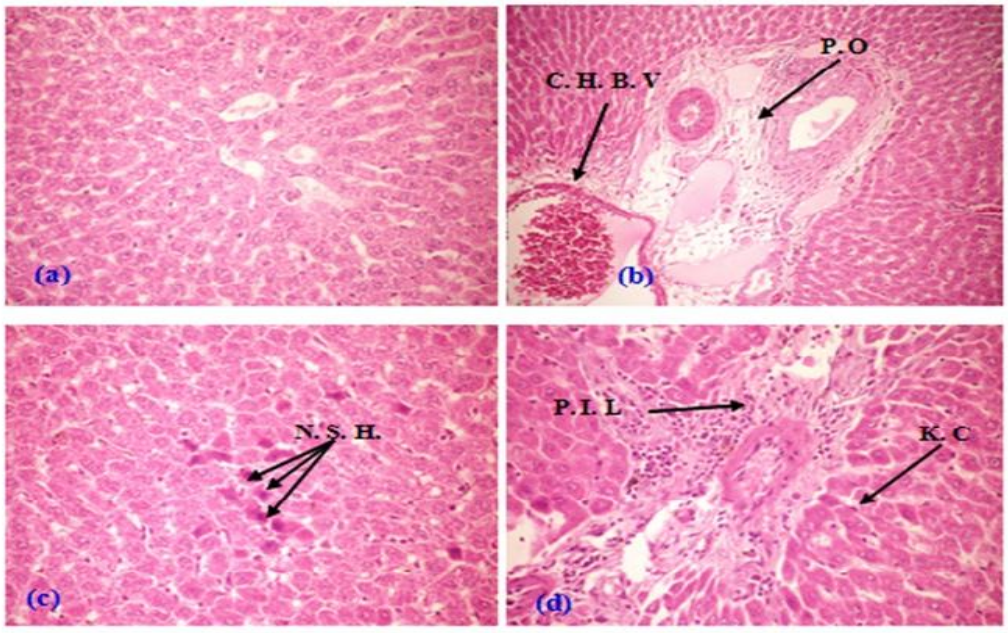

Photo micrograph (1) of rat liver (H&EX 400)from:

Photo(a): control group showing the normal histological structure of hepatic lobule.
Photo (b) Low dose of triazophos group showing portal oedema (P.O) and congestion of hepatoportal blood vessel(C,H,B,V).
Photo (c) Group treated with low dose of triazophos showing necrosis of sporadic hepatocytes (N.S.H)
Photo (d) Treated group with triazophos at high dose showing kuffer cells activation(K.C) and portal infiltration with leucocytes (P.I.L).

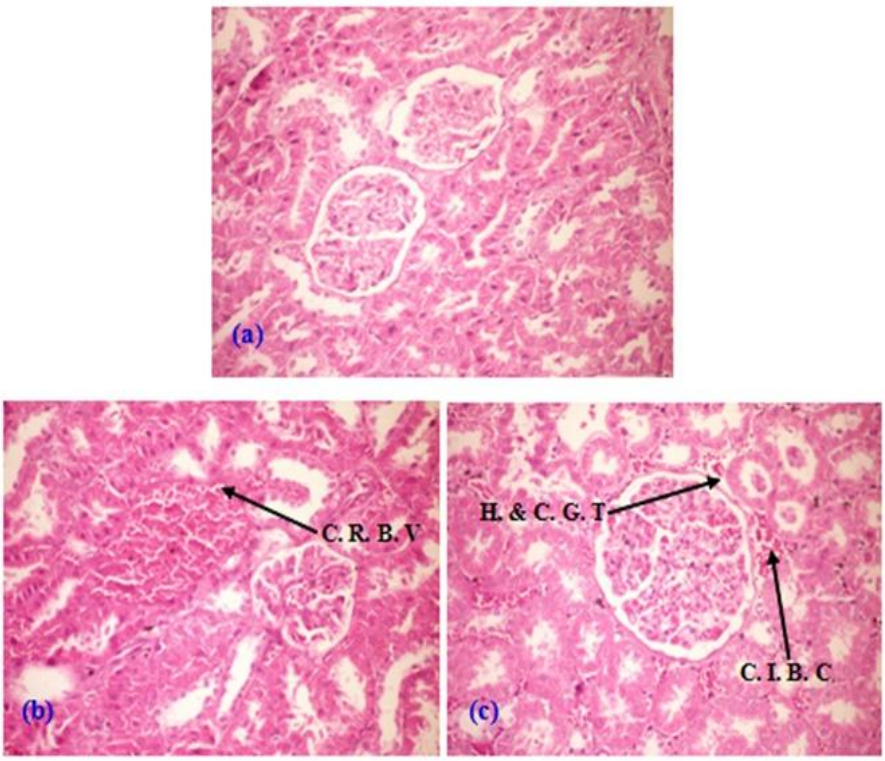

Photo micrograph (2) of rat Kidney (H&E X 400)from:

Photo(a): Untreated group showing the normal histological structure of renel parenchyma.
Photo (b) Low dose group of triazophos showing congestion of renel blood vessel(C,R,B,V).
Photo (C) High dose group of triazophos showing, hypertrophy and congestion of glomerular tuft. (H.&C,G,T) and ongestion of intertubular blood capillaries (C.I.B.C).

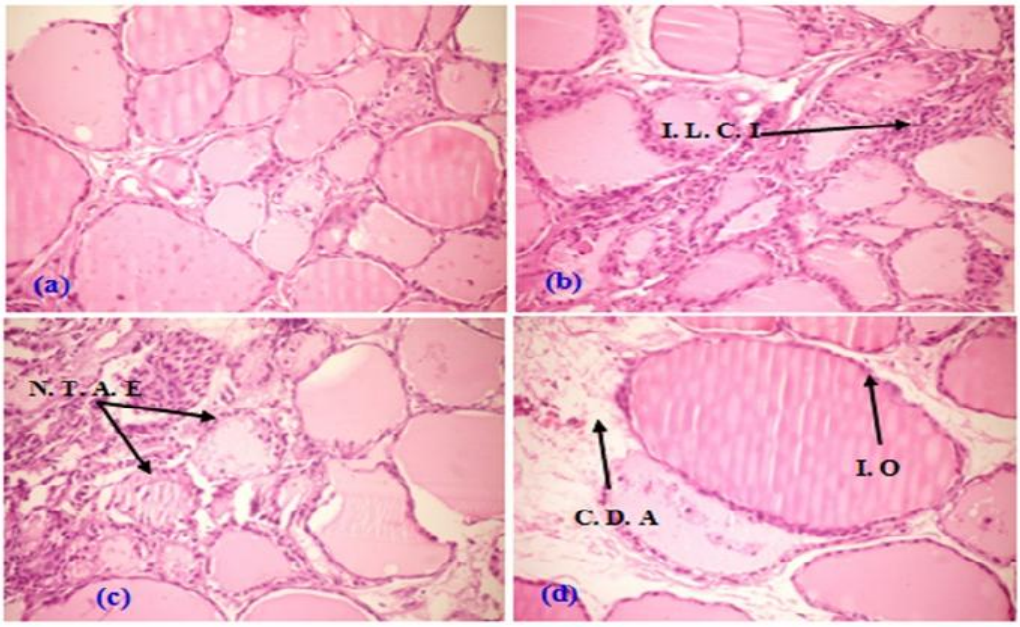

Photo micrograph (3) of rat Thyroid gland (H&E X 400)from:
Photo(a): control group showing no histological changes.
Photo (b) Low dose group of triazophos showing intercinar leucocytic cells infiltration vessel(I.L.C.I).
Photo (c) High dose of triazophos showing necrosis of thyroid acini epithelium (N.T.A.E)
Photo (d) Group treated with high dose of triazophos showing cystic dilation acini (C.D.A) and intracinar oedema (I.O).

and other biological systems when tested on various types of experimental animals through their mode of action or by production of free radicals that damage all cell component (Khan,2006) . Regarding the plasma cholinesterase (ChE) activity, a standard biomarker of organophosphate poisoning, the present study revealed that Triazophose intoxication at both doses induces the classical inhibitory effect of organophosphorus insecticides. Inhibition of ChE, an enzyme that restricts the activity of acetylcholine (ACh) in space and time, causes an increase in ACh content at sites of cholinergic transmission in the body. The inhibition of ChE is the most plausible explanation for much of the symptomatology following OP intoxication (Yamashita elal., 1997). Significant inhibition in plasma ATPase enzyme was recorded in high dose TZ intoxicated group Na+, K+ - ATPase is an enzyme present in all animal cell membranes and plays essential roles for the maintenance of neuronal excitability. (Inhibition of Na+, K+ - ATPase by an insecticide may be due to its binding with the ATPase molecule at or near the ouabain site (Duchnowicz, et al., 2005 and **Desaiah, 1980)** or may be a result of its interaction with the dephosphorylation step of the phosphoryl intermediate of the enzyme (**Bansal and Desaiah, 1982)**. As well as inhibition of ATPase implicates that the utilization of energy derived from hydrolysis of ATP, by the enzyme, is less in hypertension as in normotensive conditions. This phenomenon might be of physiological importance especially in conditions of increased intracellular ATP requirements under conditions when the heart is working against the increased systemic resistance (**Vrbjar *et al.*, 2002**).The previous mechanism of TZ toxicity generate free radicals, represented by increase of MDA lipid peroxidation biomarker in both intoxicated groups. OPs, apart from neurotoxicity and neurobehavioral changes in animals, have been shown to induce oxidative stress (OS) by elevated levels of reactive oxygen species, ROS, (Dharmender et al., 2014). Oxidative stress (OS) occur when production of reactive oxygen species (ROS) over rides antioxidant capacity in target cells, resulting in the damage of macromolecules such as nucleic acids, lipids and proteins (Agrawal and Sharma, 2010). These findings explain the recorded inhibition in plasma total antioxidant capacity (TAC) in the present work Antioxidant capacity is an important factor for all physiological standards in animals (Prior and Gao, 1999). Decreased total antioxidant activity of plasma in TZ treated rats is also in agreement with number of studies where OPs exposure leads to decreased total antioxidant activity (Hundekari et al., 2011; Aita et al., 2012).Chronic intoxication with TZ showed significant increase in alanine animotransferase (ALT) ,aspartate aminotransferase (AST). Total protein and

albumin as plasma liver biomarkers These results coincide with previous studies (Patil *et al.*, 2003) that showed a significant increase in liver enzymes in rats and humans exposed to organophosphorus insecticide, (fenitrothion and chlorpyrifos). This elevation in liver enzymes in the plasma or serum may be due to tissuedamage, particularly in the liver .histopathological examination confirm these findings where congestion of central veins and hepatic sinusoids as well as necrosis of hepatocytes may be the cause of permeability alteration and leakage of lysosomal enzymes (Choudhary *et al*., 2003; Ksheerasagar *et al.*, 2011). Slight reduction in renal biomarkers urea and creatinine was recorded in plasma of chronic intoxicated animals with different doses of TZ. Congestion of glomerular tuft ,intertubular blood capillaries and atrophy of some glomerular tuft were recorded in histopathological examination . These findings may be explained by reduction output of these metabolite by liver or congestion of kidney tissues for clearance of pesticide metabolite as recorded by (Ashade and Joseph; 2014). Moreover, significant elevation in plasma tiiodothyronine (T3) at $p<0.05$ and significant reduction in plasma (T4) was recorded in both intoxicated groups. Thyroid is vulnerable to some pesticides; endocrine disrupting chemicals (Nicolle-Mir, 2010); this effect may be considered as hypothyroidism due to most of the T4 is converted into T3 in the liver. Approximately sixty percent of the T4 is converted into T3, twenty percent is converted into an inactive form of thyroid hormone known as reverse T3 (irreversible), Reverse T3 can be problematic; even though it is inactive, it will still bind to T3 receptors and block T3 from binding and working its magic on metabolism. Too much or too little cortisol that is produced by the adrenal glands will increase circulating levels of reverse T3. Stress due to toxic metals including mercury, cadmium and lead will also increase reverse T3 production. This mechanism is due to suppressed liver detoxification and clearance of reverse T3 from excess cortisol production. Stress can not only cause signs of hypothyroidism but it will also impair the liver's ability to detoxify (Lidia and Bruno, 2011; Walter *et al.*, 2012).

In conclusion; subchronic intoxication withlow doses oftriazophoseorganophosphorous base pesticide induced adverse health effect on different organs like liver, kidneys and thyroid due induction of oxidative stressconcurrent with its mechanism of action on central nervous system.

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