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Effect of Different Levels of Green Tea on Hepatotoxicity and Nephrotoxicity Induced by Malathion in Rats

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Abstract:

The present study was designed to determine the modulating effect of Green tea at different doses against adverse effects of malathion. Rats were divided into seven groups (5 rats /group). Group one was used as a control group control(2,3) and given malathion (50 mg/kg/day and 200mg/kg/day, respectively, for four weeks), and group four was given malathion (50 mg/kg/day and Green tea 200mg four weeks), Group five were given malathion (50 mg/kg/day and Green tea 400mg four weeks), Group six were given malathion (200 mg/kg/day and Green tea 200mg four weeks), and Group seven were given malathion (200 mg/kg/day and Green tea 400mg four weeks respectively. The experiment continued for four weeks, then rats fasted, and blood samples were collected for biochemical analysis. The results showed that the malathion-treated group had significantly higher serum Alkaline Phosphatase (ALP), Alanine aminotransferase (ALT), Aspartate aminotransferase (AST), Alkaline phosphatase (ALP), urea, creatinine, uric acid, low-density lipoprotein (LDL), Very low-density lipoprotein (VLDL), Triglycerides (TG) and Cholesterol (CHOL) levels than the control group and the malathion-treated group had significantly lower Body Weight Gain (BWG), feed intake(FI) and feed efficiency ratio (FER), High-density lipoprotein (HDL) levels than the control group. In conclusion, green tea can ameliorate the liver and kidney deterioration caused by malathion among experimental rats.

Keywords: Functional Food, Blood Lipids, ALT, AST, Kidney Functions

Introduction

Extensively using of organophosphorus pesticides in different fields such as agriculture, medicine and industry can cause many disturbances in human and wildlife. These organophosphorus (OP) compounds are immediately degraded in the environment. Their concept was introduced following the ban on organochlorines which can bioaccumulate and biomagnifies, which results in ecotoxicological effects [1].

Particularly, malathion [O,O-dimethyl S-(1,2-dicarcethoxyethyl) phosphorodithioate] is an OP pesticide habitually used to eradicate ectoparasites, household insects, to conserve stored grain and to eliminate disease-inducing arthropods [2]. On the negative side, it is one of OPs agents that exerts diverse toxic effects throught the inactivation of serine esterases, mostly acetylcholinesterase (AChE) and butyrylcholinesterase which leads to an overstimulation of the cholinergic pathways [3].

The OPs can achieve all the tissues leading eventually to several pathological difficulties, this is due to their lipophilic naturem and their simple and rapid intestinal assimilation, including a insufficiency of the immune system pancreatitis, liver disease hematological pathosis disorder, kidney injurydecrease fertility and reproduction capability [4].

Many studies have reported toxic effects of this Ops in both humans and animals. Being the main actors of xenobiotic biotransformation, regulation of hepatic gene expression may play a central role in the adaptive response to altered metabolism by changing the capacity of enzymes in relevant metabolic pathways [5].

Liver is the principal metabolizing site for mediating biotransformation of thin organophosphates and with kidney contributing to the elimination of toxic products. These tissues are considered among the main targets of malathion toxicity which is mediated through oxidative stress generated by reactive oxygen species (ROS) [2].

ROS such as superoxide anion, peroxy radicals, hydroperoxy radical, hydrogen peroxide are produced from the molecular oxygen as a consequence of normal cellular metabolism. At low or moderate concentrations, ROS are considered as part of normal oxidative metabolism, but at elevated concentrations, they cause tissue injuries, including lipids, proteins oxidation, DNA damage, and enzyme inactivation. They are also implicated in many pathological conditions such as cancer, diabetes, cardiovascular, pulmonary and autoimmune diseases, neurological disorders and aging, among others [6].

Tea is the most consumed drink in the world after water. Green tea is a 'non-fermented' tea, and contains more catechins, than black tea or oolong tea. Catechins are in vitro and in vivo strong antioxidants. In addition, its content of certain minerals and vitamins increases the antioxidant potential of this type of tea. Since ancient times, green tea has been considered by the traditional Chinese medicine as a healthful beverage [7].

Recent human studies suggest that green tea may contribute to a reduction in the risk of cardiovascular disease and some forms of cancer, as well as to the promotion of oral health and other physiological functions such as anti-hypertensive effect, body weight control, antibacterial and antivirasic activity, solar ultraviolet protection, bone mineral density increase, anti-fibrotic properties, and neuroprotective power [2].

Material and Methods

Materials

1.1 Green tea from agriculture research center. Giza, Egypt.

1.2 Malathion from agriculture society, Damietta.

2- Animals:

A total of 35 Sprague-Dawley rats (aged 12 weeks; weighing 200 – 220 g) were collected from the Animal House Colony of The agriculture research, Giza, Egypt.

3- Method

3.1 Acclimatization of experimental rats

The rats were housed in plastic cages at a controlled temperature of $22 \pm 3^{\circ}\text{C}$ with a 12 h light/dark cycle for 2 weeks in the Faculty of Home Economics, Helwan University, Egypt. Animals were fed with standard chow diet, composed of crude protein 20.0%, crude fat 4.0%, crude fiber 3.5%, ash 6.0%, salt 0.5%, calcium 1.0%, phosphorus 0.6%, vitamin A 20.0 IU/g, vitamin D 2.2 IU/g, vitamin E 70.0 IU/kg, energy 2,850 ME kcal/kg, and trace minerals such as cobalt, copper, iodine, iron, manganese, selenium, zinc **AIN-93M**.

Ethical approval: The research related to animal use has been complied with all the relevant national regulations and institutional policies for the care and use of animals.

3.2 Experimental Design

- 1- Added of Malathion 50 mg to 1,000 ml of corn oil.
- 2- Added of malathion 200 mg to 1,000 ml of corn oil
- 3- The rhizomes of green tea were dried at room temperature and were crushed to powder. 400mg of the powder were macerated in 1000 ml of distilled water for 12 h at room temperature and were then filtered through a 5 μcm strainer to obtain the final aqueous extract.
- 4- The rhizomes of green tea were dried at room temperature and were crushed to powder. 800mg of the powder were macerated in 1000 ml of distilled water for 12 h at room temperature and were then filtered through a 5 μcm strainer to obtain the final aqueous extract.

Group one: "control negative" was normal rats (not infected) and was fed based diet only.

Group two: "control positive1" was fed standard diet with Malathion 50 mg to 1,000 ml of corn oil via oral.

Group three: "control positive2" was fed standard diet with Malathion 200 mg to 1,000 ml of corn oil via oral.

Group four: was fed based diet with Malathion 50 mg to 1,000 ml of corn oil and green tea 200mg of the powder were macerated in 1000 ml via oral.

Group five: was fed standard diet with Malathion 50 mg to 1,000 ml of corn oil and green tea 400mg of the powder were macerated in 1000 ml via oral.

Group six: was fed standard diet with Malathion 200 mg to 1,000 ml of corn oil and green tea 200mg of the powder were macerated in 1000 ml via oral.

Group seven: was fed standard diet with Malathion 200 mg to 1,000 ml of corn oil and green tea 400mg of the powder were macerated in 1000 ml via oral.

All groups received experimental diet for 4 consecutive weeks. The animals were weighed twice weekly. Feed consumption and body weight gain were calculated at the end experiment.

By the end of the experiment, all rats were sacrificed under diethyl ether anaesthetic and blood samples were collected by the retro-orbital plexus. Serum was separated and kept in plastic vial at -20° until analysis.

3.3 Biological evaluation:

Food intake: The body weight gain “(BWG %), food efficiency ratio (FER), and also organ/body weight % were determinate according to the following equation **chapman et al., (1959)**.

$$\text{BWG\%} = \frac{(\text{final weight} - \text{initial weight}) \times 100}{\text{initial weight}}$$

$$\text{FER} = \frac{\text{grams body weight gain}}{\text{grams feed consumed}}$$

3.4 Biochemical analysis:

3.4.1 Liver functions:

Aspartate Aminotransferase (AST/GOT) and Alanine Aminotransferase (ALT/GPT) were determined according to the methods described in Huang et al., [8]

Alkaline phosphates (ALP):_was determined by immunosorbent assay according to the method described by Richard et al., [9].

3.4.2 Kidney functions:

Serum creatinine:_was determined. By immunosorbent assay according to Glod et al. [10].

Urea: was determined. By immunosorbent assay according to the method described by Wadehra, [11].

Uric acid: was determined. By immunosorbent assay according to the method described by Kageyama [12].

Cholesterol (CHOL): was determined by immunosorbent assay according to the method described by Allain, et al. [13].

High-density lipoprotein (HDL): was determined by immunosorbent assay according to the method described by van't Hof, et al. [14].

Triglycerides (TG): was determined. By immunosorbent assay according to the method described by Fossati and Prencipe, [15].

Very low-density lipoprotein (VLDL): was determined by immunosorbent assay according to the method described by Lee and Nieman [16].

low-density lipoprotein (LDL): was determined by immunosorbent assay according to the method described by Lee and Nieman [16].

3.5 Statistical analysis:

The data were presented as mean value \pm SD and were statistically compared for the determination of levels of significance between the experimental rat groups by One-Way ANOVA followed by LSD *post hoc* analysis using SPSS (version 22, Chicago, IL, USA). The $p < 0.05$ was considered statistically significant.

Results and Discussion

Data presented in table (1) show the feed intake (FI), feed efficiency and body weight gain (BWG) after exposure to malathion toxicity and treatment with green tea: malathion administration induce a decrease in feed intake but this reduction wasn't enough to produce significant decrease in FER.

Over and above, analysis of variance (ANOVA) test revealed that the difference between groups for BWG% was statistically significant ($p < 0.001$).

It is clear that due to malathion, the FI (g/d) was reduced from 22.40 ± 0.24 to control (-), 19.60 ± 0.24 and 17.80 ± 0.20 , respectively for control+ (malathion 50g) and control+ (malathion 200g) rats. this was a sufficient indication of the effect of malathion on weight loss in rats. Also It is clear that due to malathion, the FER% was decrease from 0.017 ± 0.003 in control (-), 0.016 ± 0.004 and 0.015 ± 0.002 respectively for control (-), control+ (malathion 50g) and control+ (malathion 200g) rats. So the BWG% was reduced from 43.70 ± 4.07 for control (-) rats to, 40.70 ± 4.07 and 32.45 ± 3.35 , respectively for control (-), control+ (malathion 50g) and control (malathion 200g) rats. this was a sufficient indication of the effect of malathion on weight loss in rats.

The results of Daly [17] disagreed with our results and reported that decreased body weight gain was in male and female Fischer-344 rats administered 359 or 415 mg/kg/day, respectively, of malathion (97.1% pure) in the diet for 2 years no significant effects were observed at 35 mg/kg/day. In the other study, food intake was not reduced by administration of malathion. Male and female mice also National Toxicology Program [18] showed a reduction in body weight gain after administration of approximately 1,490 mg/kg/day of malathion (95% pure) for 80 weeks.

A similar finding was reported by a Slauter [19] in male mice after dietary administration of 1,476 mg/kg/day malathion (96.4% pure) for 18 months; in this

Table (1): Effect of green tea by different doses on body weight, food intake and food efficiency ratio of rats toxicity by malathion and treatment with green tea:

Groups	BWG(%)	FI(g/d)	FER
1	$43.70 \pm 4.07c$	$22.40 \pm 0.24a$	$0.017 \pm 0.003b$
2	$40.35 \pm 5.59d$	$19.60 \pm 0.24b$	$0.016 \pm 0.004b$
3	$32.45 \pm 3.35e$	$17.80 \pm 0.20a$	$0.015 \pm 0.002b$
4	$47.85 \pm 4.44a$	$21.60 \pm 0.24a$	$0.020 \pm 0.002a$
5	$37.70 \pm 2.95d$	$21.60 \pm 0.24a$	$0.017 \pm 0.001b$
6	$38.72 \pm 4.65d$	$21.60 \pm 0.24a$	$0.018 \pm 0.002b$
7	$42.58 \pm 1.93b$	$21.60 \pm 0.24a$	$0.020 \pm 0.001a$

All values represented as Mean \pm SD

Means with different subscript in the column are significantly different ($P < 0.05$)

Data presented in table (2) showed the effect of exposure to malathion toxicity and treatment with green tea on serum liver enzyme (GPT, GOT and ALP) in rats. It was found that the levels of serum glutamic pyruvic transaminases (GPT) enzyme (u/l) increased from 21.33 ± 1.20 , 26.67 ± 2.73 and 27.34 ± 3.28 respectively for control (-) and control (malathion 50g) and control (malathion 200g) rats. As act of leading to possibly liver inflammation. Also It was reported that the levels of serum glutamic oxaloacetic transaminases (GOT) enzyme increased from 32.33 ± 4.40 , 45.33 ± 1.85 and 47.67 ± 3.17 respectively for control (-), control (malathion 50g) and control (malathion 200g) rats. As well it was found that the levels of serum alkaline phosphates (ALP mg/l) enzyme increased from 133.33 ± 19.28 , 190.67 ± 0.86 , and 203.33 ± 0.79 and respectively for control (-), control (malathion 50g) and control (malathion 200g) rats.

Sakr et al., [20] reported that Treating animals with water extract of green tea and Adriamycin led to an improvement in the histological changes induced by Adriamycin together with significant decrease in ALT and AST activity.

These finding are in agreement with previous studies [21] who reported that the transaminases such ALT and AST are major cytolysis markers in the liver and their activities increasing in the plasma of male mice resulted from the impairment and necrosis of the function of tissues with subsequent liberation of enzymes into the circulation from the damaged tissues.

Kalender et al. [1] findings agreed with the present study revealed that malathion treatment caused a significant increase in the activities of ALT, AST, ALP and LDH in serum of male albino rats in comparison with control group.

Table (2): Effect of exposure to malathion toxicity and treatment with green tea on GPT, GOT and ALP:

GROUPS	GPT	GOT U/L	ALP
1	21.33±1.20b	32.33±4.40d	133.33±19.28d
2	26.67±2.73a	45.33±1.85a	190.67±0.88a
3	27.34±3.28a	47.67±3.17a	203.33±0.79a
4	25.33±1.45a	45.33±0.89a	150.33±2.02c
5	22.66±1.20b	40.66±0.34b	167.67±1.45b
6	26.67±0.88a	46.32±0.33a	197.67±25.43a
7	23.00±0.57b	41.66±0.65b	170.67±0.33b

All values represented as Mean±SE

Means with different subscript in the column are significantly different(P<0.05)

The result of table (3) stated the effect of exposure to malathion toxicity and treatment with green tea on serum urea, uric acid and creatinine in rats (mg/dl):

It is evident that due to oral administration of malathion for 1 week without treatment, blood urea (mg/dL) was by 40.33±2.03 in control (-) but raised to, 45.66±1.20 and 47.67±6.17 respectively for control (-) and control+1 (malathion 50g) rats and control+2 (malathion 200g) respectively. Also urea (mg/dL) increased from 2.23±0.03 to 3.37±0.14 and 3.73±0.57 respectively for control (-) and control (malathion 50g) rats and control (malathion 200g) respectively. As well serum creatinine (mg/dl) increased from 1.37±0.03 to 1.80±0.11 and 1.90±0.05 respectively for control (-), control (malathion 50g) and control (malathion 200g) rats.

But after the therapeutic intervention using green tea some groups began to improve, and the results were the best and the closest to the control (-) group was compared to control. These finding are in agreement with previous studies carried out by Dive et al, [22], who reported that in a subject who invested approximately 514 mg/kg of malathion, protein was found in the urine, and mild renal insufficiency (measured by creatinine clearance) was observed. Crowley and Johns [23] in another case, found that after ingestion of

approximately 600 mg/kg of malathion, protein, sugar, and white blood cells were found in the urine. Healy [24] found increased secretion of ketone bodies and glucose in the urine in an 18-month-old boy who ingested malathion.

Results obtained by Eraslan et al., [25] are in good agreement with current study, they observed that uric acid increase may be related to either increase in protein degradation, which is involved in uric acid formation, or the toxic effect of malathion on the kidneys during Malathion exposure that also induced an increase in creatinine and urea levels, indicating a kidney dysfunction. These findings corroborated with previous investigation in adult rats and their suckling pups treated with dimethoate [26] and in adult rats treated with chlorfenvinfos [27] or with phosphonodithioate [28].

Table (3): Effect of exposure to malathion toxicity and treatment with green tea on serum urea, serum uric acid and serum creatinine:

GROUPS	Urea	Uric Acid mg/dL	Creatinine
1	40.33±2.03c	2.23±0.03c	1.37±0.03c
2	45.66±1.20b	3.37±0.14a	1.80±0.11a
3	47.67±6.17a	3.73±0.57a	1.90±0.05a
4	47.67±4.84a	2.83±0.03b	1.73±0.12a
5	44.33±3.38b	2.20±0.07c	1.90±0.06a
6	47.66±3.28a	2.63±0.08b	1.66±0.06b
7	46.34±1.45b	2.50±0.05b	1.80±0.11a

All values represented as Mean±SE

Means with different subscript in the column are significantly different(P<0.05)

Data presented in table (4) showed the effect of exposure to malathion toxicity and treatment with green tea on serum (HDL, VLDL, LDL, TG and CHOL mg/dl) in rats:

It is clear that due to malathion, high Density Lipoproteins (HDL mg/dl) decreased dramatically from 60.00±0.58, 52.00±0.58 and 50.00±0.57 respectively for control (-), control (malathion 50g) and control (malathion200g) rats. While serum very low-density lipoproteins (VLDL) increased dramatically from 18.47±0.64 to 22.13±2.21 and 23.20±1.51 respectively for control (-), control (malathion 50g) and control (malathion200g) rats. Also serum Low Density Lipoproteins (LDL) increased dramatically from 0.19±3.51 to 22.60±4.70 and 15.8±8.14 respectively for control (-), control (malathion 50g) and control (malathion200g) rats. Also serum triglycerides (TG.mg/dl) increased dramatically from 89.33±2.85 to 117.00±7.94 and 147.67±36.03 respectively for control (-), control (malathion 50g) and control (malathion200g) rats. As well serum cholesterol (CHOL. mg/dl) increased dramatically from 79.33±5.84 to 98.00±5.51 and 103.00±13.07 respectively for control (-), control (malathion 50g) and control (malathion200g) rats.

This result is agreement with the findings of Selmi et al., [29], who found that malathion in acute exposure leads to a disruption of lipid metabolism with an enhancement in LDL and triglyceride contents and may play an important role in the development of atherosclerosis and cardiovascular disease.

Lasram et al. [5] reported that disruption in plasma lipid profile may lead to a kind of insulin resistance which results in hyperglycemia. Moreover, Błasiak [30] reported these results that the presence of cholesterol may be of importance in the interaction of organophosphorus insecticide with biological membrane. Cholesterol action may be a result of either competition for similar or the same interaction sites or change in the structural organization of phospholipids

Bhandari et al., [31] reported the green tea was found to have hypocholesterolemic effects and cause decrease in body weight, glucose in blood, serum total cholesterol and serum alkaline phosphatase in adult male rats.

Table (4): Effect of exposure to malathion toxicity and treatment with green tea on HDL, VLDL, LDL, TG and CHOL:

GROUPS	HDL	VLDL	LDL mg/dL	TG	Cholesterol
1	60.67±1.45a	18.47±0.64b	0.19±3.51e	89.33±2.85c	79.33±5.84e
2	52.00±0.58d	23.4±2.21a	23.60±5.03a	117.00±7.94b	98.00±5.51b
3	50.00±0.57e	29.53±1.51a	23.47±8.14a	147.67±36.03a	103.00±13.07a
4	52.33±1.76d	16.53±1.33d	15.8±5.19c	82.67±1.45c	84.66±2.60d
5	57.00±2.65b	17.4±0.38b	13.93±6.96b	87.00±0.57c	88.33±14.83c
6	55.00±0.57c	17.2±0.98c	13.93±4.35c	86.00±1.15c	88.66±6.17c
7	54.67±0.88c	17.6±0.55b	18.73±4.70b	88.00±1.15c	91.00±8.66c

All values represented as Mean±SE

Means with different subscript in the column are significantly different(P<0.05)

Conclusion

Taken together, it can be concluded that exposure to organophosphorus malathion caused a decrease in feed intake, hepatotoxicity, and nephrotoxicity, which caused a decrease in feed enhancement and a change in the tissue composition of both kidneys and liver. Decreased feed efficiency and altered tissue composition of the kidneys and liver can alter body weights, liver, and kidneys and alter biochemical markers such as ALT, AST, hepatic ALP, renal urea, creatinine, and uric acid. In addition to depleting the antioxidant enzyme system, malathion also caused molecular alterations. Attenuation of toxicity induced by malathion was observed by adding green tea during exposure to pesticides.

References

- [1] Kalender S, Uzun FG, Durak D, Demir F, Kalender Y. Malathion-induced hepatotoxicity in rats: the effects of vitamins C and E. *Food Chem Toxicol.* 2010 Feb;48(2):633-8. doi: 10.1016/j.fct.2009.11.044.
- [2] Suresh Babu N, Malik JK, Rao GS, Aggarwal M, Ranganathan V. Effects of subchronic malathion exposure on the pharmacokinetic disposition of pefloxacin. *Environ Toxicol Pharmacol.* 2006 Sep;22(2):167-71. doi: 10.1016/j.etap.2006.03.001.
- [3] Bartling A, Worek F, Szinicz L, Thiermann H. Enzyme-kinetic investigation of different sarin analogues reacting with human acetylcholinesterase and butyrylcholinesterase. *Toxicology.* 2007 Apr 20;233(1-3):166-72. doi: 10.1016/j.tox.2006.07.003.

- [4] Gokalp O, Buyukvanlı B, Cicek E, Ozer MK, Koyu A, Altuntas I, Koylu H. The effects of diazinon on pancreatic damage and ameliorating role of vitamin E and vitamin C. *Pesticide biochemistry and physiology*. 2005 Feb 1;81(2):123-8.
- [5] Lasram MM, Annabi AB, El Elj N, Selmi S, Kamoun A, El-Fazaa S, Gharbi N. Metabolic disorders of acute exposure to malathion in adult Wistar rats. *J Hazard Mater*. 2009 Apr 30;163(2-3):1052-5. doi: 10.1016/j.jhazmat.2008.07.059.
- [6] Dal-Pizzol F, Klamt F, Dalmolin RJ, Bernard EA, Moreira JC. Mitogenic signaling mediated by oxidants in retinol treated Sertoli cells. *Free Radic Res*. 2001 Dec;35(6):749-55. doi: 10.1080/10715760100301251.
- [7] Cabrera C, Artacho R, Giménez R. Beneficial effects of green tea--a review. *J Am Coll Nutr*. 2006 Apr;25(2):79-99. doi: 10.1080/07315724.2006.10719518.
- [8] Huang XJ, Choi YK, Im HS, Yarimaga O, Yoon E, Kim HS. Aspartate Aminotransferase (AST/GOT) and Alanine Aminotransferase (ALT/GPT) Detection Techniques. *Sensors (Basel)*. 2006;6(7):756-782.
- [9] Richard J, Henry MD, Donald C. "Clinical chemistry: principles and techniques. 2nd Ed., Harper and Publishers, New York. Philadelphia, 1974. (1974): 1468-1471.
- [10] Glod BK, Wantusiak PM, Piszcz P, Lewczuk E, Zarzycki PK. Application of micro-TLC to the total antioxidant potential (TAP) measurement. *Food Chem*. 2015 Apr 15;173:749-54. doi: 10.1016/j.foodchem.2014.10.058.
- [11] Wadehra NR. A colorimetric method for the estimation of cholesterol from high density lipoprotein and its subclasses. *Indian Journal of Clinical Biochemistry*. 1990 Jul;5(2):131-4. doi.org/10.1007/BF02873500.
- [12] Kageyama N. A direct colorimetric determination of uric acid in serum and urine with uricase-catalase system. *Clinica Chimica Acta*. 1971 Feb 1;31(2):421-6. doi: 10.1016/0009-8981(71)90413-x.
- [13] Allain CC, Poon LS, Chan CS, Richmond W, Fu PC. Enzymatic determination of total serum cholesterol. *Clin Chem*. 1974 Apr;20(4):470-5. doi:10.1093/clinchem/20.4.470.
- [14] van't Hof RJ, Armour KJ, Smith LM, Armour KE, Wei XQ, Liew FY, et al. Requirement of the inducible nitric oxide synthase pathway for IL-1-induced osteoclastic bone resorption. *Proc Natl Acad Sci U S A*. 2000 Jul 5;97(14):7993-8. doi: 10.1073/pnas.130511497.
- [15] Fossati P, Prencipe L. Serum triglycerides determined colorimetrically with an enzyme that produces hydrogen peroxide. *Clinical chemistry*. 1982 Oct 1;28(10):2077-80.
- [16] Lee, JR, Nieman, D. *Nutritional Assessment*. 2nd Ed., Mosby, Missouri, USA. 1996. (33-60)
- [17] Daly IW. A 24-month oral toxicity/oncogenicity study of malathion in the rat via dietary administration. Unpublished study performed by Huntingdon Life Sciences, East Millstone, NJ, Study. 1996 (90-3641).
- [18] National Toxicology Program. Bioassay of malathion for possible carcinogenicity. *Natl Cancer Inst Carcinog Tech Rep Ser*. 1978;24:1-102.
- [19] Slauter R. 18-Month oral (dietary) oncogenicity study in mice: Malathion. Lab project No. 668- 001. Unpublished study prepared by International Research and Development Corp. MRID 43407201. (As cited in EPA 2000a, 2000b).

- [20] Sakr SA, Mahran HA, Lamfon HA. Protective effect of ginger (*Zingiber officinale*) on adriamycin-induced hepatotoxicity in albino rats. *J Med Plant Res*. 2011 Jan 4;5(1):133-40.
- [21] Videira RA, Antunes-Madeira MC, Lopes VI, Madeira VM. Changes induced by malathion, methylparathion and parathion on membrane lipid physicochemical properties correlate with their toxicity. *Biochim Biophys Acta*. 2001 Apr 2;1511(2):360-8. doi: 10.1016/s0005-2736(01)00295-4.
- [22] Dive A, Mahieu P, Van Binst R, Hassoun A, Lison D, De Bisschop H, Nemery B, Lauwerys R. Unusual manifestations after malathion poisoning. *Hum Exp Toxicol*. 1994 Apr;13(4):271-4. doi: 10.1177/096032719401300409.
- [23] Crowley WJ Jr, Johns TR. Accidental malathion poisoning. *Arch Neurol*. 1966 Jun;14(6):611-6. doi: 10.1001/archneur.1966.00470120043007.
- [24] Healy JK. Ascending paralysis following malathion intoxication: a case report. *Med J Aust*. 1959 Jun 6;1(23):765-7. doi: 10.5694/j.1326-5377.1959.tb88846.x.
- [25] Eraslan G, Kanbur M, Silici S. Evaluation of propolis effects on some biochemical parameters in rats treated with sodium fluoride. *Pesticide Biochemistry and Physiology*. 2007 Jul 1;88(3):273-83.
- [26] Mahjoubi-Samet A, Fetoui H, Zeghal N. Nephrotoxicity induced by dimethoate in adult rats and their suckling pups. *Pesticide biochemistry and physiology*. 2008 Jun 1;91(2):96-103.
- [27] Jabłońska J, Brzeziński J. The acute toxic effects of chlorfenvinphos (CHF) on rat kidney. *Toxicology Letters*. 1996(88):32.
- [28] Dheranetra W, Keadtisuke S, Swatek FE, Fukuto TR. Renal effects in the delayed toxicity of O, S, S-trimethyl phosphorodithioate. *Pesticide Biochemistry and Physiology*. 1988 Jan 1;30(1):95-100.
- [29] Selmi S, Rtibi K, Grami D, Sebai H, Marzouki L. Malathion, an organophosphate insecticide, provokes metabolic, histopathologic and molecular disorders in liver and kidney in prepubertal male mice. *Toxicol Rep*. 2018 Jan 9;5:189-195. doi: 10.1016/j.toxrep.2017.12.021.
- [30] Błasiak J. Protective action of cholesterol against changes in membrane fluidity induced by methylparathion. *Acta Biochimica Polonica*. 1993 Mar 31;40(1):35-8.
- [31] Bhandari U, Kanojia R, Pillai KK. Effect of ethanolic extract of *Zingiber officinale* on dyslipidaemia in diabetic rats. *J Ethnopharmacol*. 2005 Feb 28;97(2):227-30. doi: 10.1016/j.jep.2004.11.011.

تأثير مستويات مختلفة من الشاي الأخضر على سمية الكبد والكلى المستحثة بالملاثيون في

الفئران

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الملخص العربي:

صممت هذه الدراسة لتقييم تأثير الشاي الأخضر بجرعات مختلفة على التأثيرات الضارة للملاثيون. تم تقسيم الفئران إلى سبع مجموعات (5 فئران / مجموعة). تم استخدام المجموعة الأولى كعنصر-تحكم، المجموعة الثانية والثالثة أعطيت الملثيون (50 ملجم / كجم / يوم و 200 ملجم / كجم / يوم لمدة أربعة أسابيع)، المجموعة الرابعة أعطيت الملثيون (50 ملجم / كجم / يوم والشاي الأخضر- 200 ملجم لأربعة أسابيع)، المجموعة الخامسة أعطيت الملثيون (50 ملجم / كجم / يوم والشاي الأخضر- 400 ملجم لأربعة أسابيع)، المجموعة السادسة أعطيت الملثيون (200 ملجم / كجم / يوم والشاي الأخضر- 200 ملجم لمدة أربعة أسابيع)، المجموعة السابعة أعطيت الملثيون (200 ملجم / كجم / يوم والشاي الأخضر- 400 ملجم لمدة أربعة أسابيع) على التوالي. في نهاية الأسبوع الرابع تم صيام الفئران وجمع عينات الدم وأجراء التحاليل الكيموحيوية. أظهرت النتائج ان المجموعة المعالجة بالملثيون (الضابطة+) تثير أعلى بكثير من الفوسفاتيز القلوي والالانين ترانزامينيز واسبارتات ترانزامينيز واليوريا والكرياتينين وحمض البوليك والليبوبروتينات منخفضة الكثافة و الليبوبروتينات منخفضة جدا في الكثافة والدهون الثلاثية والكوليسترول. وكما لوحظ ان المجموعة المعالجة بالملثيون اكتسبت وزن أقل بشكل ملحوظ وتناول كمية أقل من الطعام ونسبة كفاءة الغذاء وقل مستوي الليبوبروتينات مرتفعة الكثافة مقارنة بمجموعة الضابطة السالبة. الخلاصة انه يمكن ان يساعد الشاي الأخضر- في تحسين التدهور الحادث في الكبد والكلى الناجم عن الملثيون بين فئران التجارب

الكلمات المفتاحية: الغذاء الوظيفي، دهون الدم، الالانين ترانزامينيز، اسبارتات ترانزامينيز، وظائف الكلى