Bulletin of Environment, Pharmacology and Life Sciences
Bull. Env. Pharmacol. Life Sci., Vol 10 [12]November 2021 : 134-145
©2021 Academy for Environment and Life Sciences, India
Online ISSN 2277-1808
Journal's URL:http://www.bepls.com
CODEN: BEPLAD

ORIGINAL ARTICLE **OPEN ACCESS**

Reversal of deltamethrin-induced oxidative stress in hepatic and pancreatic tissues by *Allium sativum*

Pragnesh Patel[1, 2], Ketaki Desai[2], Hyacinth Highland[1], Nihar Nimbark*[1]
1 - Department of Zoology, BMT and HG, School of Sciences, Gujarat University, Ahmedabad.
2 –President Science College, Affiliated to Gujarat University, Ghatlodia, Ahmedabad
*E-mail address: niharnimbark21@gmail.com

ABSTRACT
Deltamethrin (DLM) is a well-known synthetic pyrethroid insecticide which is widely used for veterinary, farming purposes and home pest control due to restriction on the sale of organophosphate. Although its exposure to humans and other animals leads to toxicity in the hepatic and other vital tissues, the present study was designed to examine the protective effects of Allium sativum (AS) against hepatic and pancreatic sub-chronictoxicity in female mice. Forty-two female mice were divided into seven experimental groups: Group A served as the control group. Group B served as vehicle control (peanut oil) and Group C administered with 200 mg/kg b.wt. AS only. Group D and Group E animals were given 3 mg/kg b.wt and 6 mg/kg b.wt of DLM respectively while Group F and Group G mice were given 3 mg/kg b.wt. of DLM+200 mg/kg b.wt AS and 6 mg/kg b.wt of DLM+200 mg/kg b.wt AS respectively for a period of 21 days to female Swiss albino mice. Results showed that DLM intoxication altogether increased hepatic and pancreatic lipid peroxidation and critically restrained antioxidative biomarkers including SOD, CAT, GSH, GPx, GRx and GST. Further, co-administration of AS along with DLM brought the parameters near to the control. Hence, it can be concluded that AS has beneficial influences and could be able to antagonize DLM caused oxidative stress. Thus, AS supplementation could overcome DLM-induced toxicity to liver and pancreas by abolishing oxidative tissue injuries and detrimental effects of DLM.
KEYWORDS: Deltamethrin,Hepatic,Pancreatic,Oxidative stress, *Allium sativum*.

Received 29.08.2021 Revised 20.10.2021 Accepted 18.11.2021

INTRODUCTION

Deltamethrin (DLM) is a broad-spectrum synthetic pyrethroid insecticide widely used for to protect agricultural crops, fruits and vegetables against pests like weevils, mites and beetles[1]. DLM is globally used as an insecticide in most of the countries because of its rapid metabolism, strong effect on many pests and low toxicity to humans and other non-target animals[2]. However, recent reports suggest several side effects of DLM which includes hepatotoxicity, nephrotoxicity, neurotoxicity, immunosuppression, allergy, hypertension and decreased testosterone levels[3].

Major target organ of such pesticides is liver [2,4]. Another important digestive gland is pancreas which plays an important role in the digestion of proteins and lipids along with the liver. Toxic effects of the pesticides can be associated with the formation of reactive oxygen species (ROS) in the hepatic tissue [4] as well as in the pancreatic tissue. The reactive oxygen species causes the oxidative deterioration of cellular macromolecules such as lipids, proteins and DNA which may ultimately results into several pathologies [5]. Cells contains enzymatic oxidants like catalase, SOD, GPx, GRx, GST as well as non-enzymatic antioxidants composed of endogenous substances like GSH to scavenge free radicals generated by reactive oxygen species. Moreover, this endogenous antioxidant mechanism can be reinforced by certain exogenous substances supplemented in the diet. Such type of exogenous antioxidants can be derived from *Allium sativum*. Itcontains organosulfur compounds which are capable to scavenge free radicals and protect the liver and pancreatic tissue against oxidative damage.

*Allium sativum*is widely used as a dietary supplement throughout the world. Due to its antioxidant properties and biological activities, it can be used as one of nature's wonderful productwhich has potential to prevent and curevarious diseases [6].*A. sativum* and its preparations such as powder, distilled garlic, oil of garlic and aged garlic extract have been declared as more repeatedly used dietary component compared to others by the third National Health and Nutrition Examination Survey [7,8].*A. sativum* is comprising of around 200 compounds including organic compounds like thiosulfinates, sulfur

compounds, flavonoids, sapogenins and saponins which provides various biological activities to it. These components of *A. sativum* are categorized based on the chemistry of the compounds into thiosulfinates, organosulfur volatiles, vinyldithiins, ajoene and water-solubleorganosulfur compounds [9].Further, these compounds also enhance the levels of antioxidant enzymes of cell such superoxide dismutase (SOD), catalase and glutathione peroxidase [10,11], and increasing glutathione in the cells [12]. Recent investigations are being carried out to evaluate the potency of *Allium sativum* and its extracts or products to prevent and treat chronic diseases [13,14].

Hence, the aim of the present investigation was to determine the toxic effects of deltamethrin on hepatic and pancreatic biomarkers of oxidative damage in mice and to evaluate the potential benefits of *Allium sativum* DLM induced toxicity.

MATERIAL AND METHODS

Housing and care of animals

In the present study, adult, pathogen free, healthy, colony bred female albino mice (*Mus musculus*)of Swiss strain weighing between 30-40 gms were obtained from Cadila Health Care and Pharmaceutical, Ahmedabad, Gujarat, India were used. The experimental protocol was approved by the local animal ethics committee meeting under registration (167/GO/ReBi/S/99/CPCSEA) from the Ministry of Social Justice and Empowerment, Government of India and Committee for the Purpose of Control and Supervision of Experiments on Animals, Chennai, India.

Animals of different experimental groups were caged separately and housed in an air- conditioned animal house at a temperature of 26 ± 3 °C and exposed to 10-12 hours ofday light and relative humidity of 30-60%. Maximum of five animals per cage were maintained on a standard animal food obtained from Pranav Agro Industries, containing wheat - 70%, gram - 20%, fish meat - 5% and yeast powder - 5% and distilled water was given *ad libitum*. All the animals were acclimatized seven days prior to the commencement of the treatment. The treatment was given daily before feeding so as to avoid interference with food intake.

Rationale for Selection of Doses

Technical grade Deltamethrin of 98.99% purity was generously gifted from Meghmani Organics Ltd., Ahmedabad, India. Deltamethrin was dissolved in peanut oil and administered via oral gavage at two dose concentrations: Low Dose (3 mg/kg body weight) and High Dose (6 mg/kg body weight). The dose was determined on the basis of LD_{50} of deltamethrin in peanut oil i.e. 30 mg/kg body weight [15].

Crude extract of *Allium sativum* (garlic) of the single clove variety was prepared from bulbs purchased in bulk from the market. The cloves were sliced into pieces, ground into a fine paste. Working solution was then prepared by dissolving 5 gm of this paste in 100 ml of deionized water, where 1 ml of the extract contained 50 mg of crude *Allium sativum*. Freshly prepared *Allium sativum* extract was then administered to mice at a dose level of 200 mg/kg body weight accordingly. Dose of the *Allium sativum* was based on previous studies [16].

Experimental design

Studies on the effect of deltamethrin at two dose concentrations were carried out and compared with control (untreated) and vehicle treated (peanut oil only) mice as per the experimental protocol. *Allium sativum* and curcumin were administered along with the low dose and high dose of deltamethrin to investigate mitigative potency of the same. The animals were treated orally using a gavage. Control animals were provided only distilled water throughout the study and the vehicle control animals were given 0.2 ml peanut oil. The duration of the treatment was 21 days(Table 1).

Table 1: Experimental Protocol

GROUPS	TREATMENT AND DOSE	DURATION (DAYS)	DAY OF NECROPSY
A	Control (Untreated)	-	Sacrificed along with treated animals
B	Vehicle Control (Peanut oil)	21	22nd Post treatment
C	Control + *Allium sativum* (AS) (200 mg/kg B.wt.)	21	22nd Post treatment
D	Deltamethrin (DM) Low Dose (LD) (3 mg/kg B.wt.)	21	22nd Post treatment
E	Deltamethrin (DM) High Dose (HD) (6 mg/kg B.wt.)	21	22nd Post treatment
F	DM (LD) + AS	21	22nd Post treatment
G	DM (HD) + AS	21	22nd Post treatment

Number of animals in each group (n) = 6.

Hypothesis

1. **Null hypothesis**: *Allium sativum* shows no ameliorative effects on Deltamethrin-induced sub-chronic toxicity in hepatic and pancreatic tissues of Swiss albino female mice.
2. **Alternative hypothesis**: *Allium sativum* shows ameliorative effects on Deltamethrin-induced sub-chronic toxicity in hepatic and pancreatic tissues of Swiss albino female mice.

Parameters studied

Protein estimation

Protein levels in the liver and pancreas of control and all treated groups of animals were estimated by the method of Lowry et al., (1951) [17]. Protein containing preparation when treated with phenol reagent of Folin-Ciocalteau, a deep blue colouration develops, which is measured colorimetrically at 540 nm.

Lipid Peroxidation (LPO) / Thiobarbutiric Acid Reactive Species (TBARS)

The thiobarbutiric acid reactive species (TBARS) levels in liver and pancreas of control and all treated animals were determined by the method of Ohkawa et al.,(1979) [18]. The method is based on the formation of a red chromophore that absorbs at 532 nm following the reaction of thiobarbituric acid with MDA and other breakdown products of peroxidized lipids collectively called as TBARS.

Superoxide Dismutase (SOD)

The activity of superoxide dismutase (SOD) in liver and pancreas of control and all treated animals was assayed by the modified spectrophotometric method of Kakkar et al. (1984) [19]. In this method, the formazan formed at the end of the reaction indicates the presence of the enzyme. One unit of enzyme activity is defined as the enzyme concentration required to inhibit 50% of the optical density of chromogen formed in 1 minute at 560 nm under the assay condition.

Catalase (CAT)

Catalase activity in liver and pancreas of control and treated mice was assayed by the modified method of Sinha (1972) [20].

Glutathione (GSH)

The concentration of glutathione (GSH) in liver and pancreas of control and all treated groups of mice was assayed by the method of Ellman [21]. Glutathione present in the tissue oxidizes 5,5$'$-dithio-bis-(2-nitrobenzoic acid) to form yellow-coloured complex that can be read at 412 nm. The absorbance is proportional to the amount of GSH.

Glutathione Peroxidase (GPx)

Activity of glutathione peroxidase (GPx) was estimated in liver and pancreas of control and treated animals by the method of Rotruck et al. (1973) [22]. Glutathione peroxidase acts on hydrogen peroxide (H_2O_2) and splits it into 2 water molecules. While doing so it consumes 2 hydrogen atoms from 2 molecules of GSH. As a by-product, 1 molecule of reduced glutathione (GS-SG) is obtained. Thus, the amount of GSH consumed per 10 minutes is related to activity of GPx.

Glutathione Reductase (GRx)

The estimation of glutathione reductase (GRx) in hepatic and pancreatic tissue was done by the method of Carlberg and Mannervik (1985) [23]. Glutathione reductase can be measured by measuring the rate of nicotinamide adenine dinucleotide phosphate (NADPH) oxidation. The oxidation of NADPH to $NADP^+$is accompanied by a decrease in absorbance at 340 nm. Since GRx is present at rate limiting concentrations, the rate of decrease is directly proportional to the GRx activity in the sample.

Glutathione-S-Transferase (GST)

Glutathione-S-transferase (GST) activity was measured in liver and pancreas of control and treated group animals by modified method of Habig *et al.* [24]. Glutathione-S-transferase catalyse the conjugation of GS-SGvia a sulfhydryl groupto electrophilic centres on a wide variety of substrates. Hence, the quantification of GSH–CDNB (1-Chloro-2,4-dinitrobenzene) conjugate formed by reaction of CDNB and GSH in presence of enzyme source may be used to measure the activity of GST.

Statistical analysis

For each parameter, minimum of 6 replicates were done and the results were expressed as Mean ± Standard Error (S.E.). The data was then statistically analysed by Analysis of Variance (One way - ANOVA) taking significance at $p<0.05$ level by Graphpad Prism 7.0 software. Sidak's post hoc test was used for comparison among different treatment groups ($p<0.05$).

RESULTS

Total Protein

Protein content of the hepatic tissue was significantly depleted after 21 days ($p<0.002$) in DLM low dose treatment (Group D) compared to control mice (Group A). When high dose of DLM was given (Group E),

hepatic protein content resulted in a highly significant reduction after 21 days (p<0.001) treatment compared to control animals (Group A). Further, treatment of *Allium sativum* along with low dose DLM (Group F), significant recovery was registered after 21 days (p<0.002) of treatment when compared to low dose DLM treatment (Group D). Administration of *Allium sativum* along with high dose DLM (Group G) significant recovery after 21 days (p<0.002) of treatment (Group E) (Table 2).
Protein content in pancreas showed significant reduction in both the deltamethrin treatment groups, in Group-D in order of (p<0.033) and Group-E in order of p<0.001 when compared to control Group- A (Table-2). Co-treatment of *Allium sativum* with DLM showed non-significant restoration in protein content in pancreas in both Group F and Group-G when compared with Group-D and E (Table-2).

Table 2: Total protein level (mg/100 mg tissue weight) in Liver and Pancreas of control and treated animals after 21 days treatment

GROUPS	TREATMENT AND DOSE	LIVER	PANCREAS
A	Control (Untreated)	20.27 ± 0.207	14.86±0.063
B	Vehicle Control (Peanut oil)	20.01 ± 0.1479NS	14.88±0.117NS
C	Control + *Allium sativum* (AS) (200 mg/kg B.wt.)	20.38 ± 0.2541NS	14.85±0.097NS
D	Deltamethrin (DM) Low Dose (LD) (3 mg/kg B.wt.)	18.94 ± 0.1819**	14.11±0.189*
E	Deltamethrin (DM) High Dose (HD) (6 mg/kg B.wt.)	18.19 ± 0.102***	13.98±0.085**
F	DM (LD) + AS	20.28 ± 0.355##	14.67±0.2197ns
G	DM (HD) + AS	19.53 ± 0.341##	14.39±0.108ns

Values are represented as Mean ± S.E., Analysis of variance at P<0.05 level
Comparison of Group A with Group B, C, D and E; *p<0.033, **p<0.002, ***p<0.001, NS – non significant
Comparison of Group D and E with F and G respectively; #p<0.033, ##p<0.002, ###p<0.001, ns – non significant

Lipid peroxidation (LPO) (TBARS)
LPO levels in liver of treated mice registered significant increase after 21 days (p<0.002) treatment with low dose DLM (Group D) compared to control animals (Group A). Animals administered with high dose of DLM (Group E) showed highly significant elevation (p<0.001) in liver LPO levels after 21 days post treatment compared to control animals (Group A).Administration of *Allium sativum* along with low dose of DLM (Group F) showed non-significant alteration in LPO level after 21 days compared to DLM low dose treated group (Group D). When experimental animals were supplemented with *Allium sativum* along with high dose of DLM (Group G), significant reduction in LPO levels was observed after 21 days (p<0.002) compared to high dose treated group (Group E) (Table 3).

Table 3: LPO level (nanomoles of MDA/100 mg tissue weight) in Liver and Pancreas of control and treated animals after 21 days treatment

GROUPS	TREATMENT AND DOSE	LIVER	PANCREAS
A	Control (Untreated)	124.9 ± 0.29	44.55±0.961
B	Vehicle Control (Peanut oil)	125.3 ± 0.40NS	44.13±0.866NS
C	Control + *Allium sativum* (AS) (200 mg/kg B.wt.)	125.0 ± 0.80NS	43.98±0.356NS
D	Deltamethrin (DM) Low Dose (LD) (3 mg/kg B.wt.)	134.6 ± 0.48**	47.72±0.649NS
E	Deltamethrin (DM) High Dose (HD) (6 mg/kg B.wt.)	145.2 ± 1.21***	49.23±1.543*
F	DM (LD) + AS	127.9 ± 2.34ns	45.14±0.856ns
G	DM (HD) + AS	135.7 ± 2.52##	49.74±0.863ns

Values are represented as Mean ± S.E., Analysis of variance at P<0.05 level
Comparison of Group A with Group B, C, D and E; *p<0.033, **p<0.002, ***p<0.001, NS – non significant
Comparison of Group D and E with F and G respectively; #p<0.033, ##p<0.002, ###p<0.001, ns – non significant

LPO in pancreas showed non-significant increase in low-dose deltamethrin treatment group in Group-D and significant in Group-E in order of ($p<0.033$) when compared to control Group- A (Table-2). Co-treatment of *Allium sativum* with DLM showed non-significant decrease in LPO in pancreas in both Group F and Group-G when compared with Group-D and E (Table-3).

Superoxide dismutase (SOD)

Activity of SOD in liver of experimental mice with low dose of DLM treatment (Group D)showed significant decline after 21 days ($p<0.033$) compared to control (Group A) while treatment with high dose of DLM (Group E), SOD activity in liver was found to deplete significantly after 21 days ($p<0.001$) compared to control (Group A).*Allium sativum* along with low dose of DLM (Group F) revealed non-significant elevation in SOD activity after 21 days post treatment compared to low dose treated group (Group D). Further, *Allium sativum* along with DLM high dose (Group G) showed significant increase in SOD activity after 21 days ($p<0.002$) compared to high dose DLM treated group (Group E) (Table 4).

SOD activity in pancreas showed non-significant decrease in low dose treated deltamethrin group Group-D but showed significant reduction in SOD activity in Group-E in order of ($p<0.033$) when compared to control Group- A (Table-4). Co-treatment of *Allium sativum* with DLM showed non-significant increase in SOD activity in pancreas in both Group F and Group-G when compared with Group-D and E (Table-4).

Catalase (CAT)

Activity of catalase in liver of treated female mice registered significant decline after 21 days ($p<0.033$) treatment with low dose of DLM (Group D) compared to control. When experimental animals were administered with DLM high dose (Group E), compared to control (Group A) catalase activity was found to fall significantly in liver after 21 days ($p<0.001$) post treatment (Group E).*Allium sativum* administration along with the low dose of DLM (Group F) showed non-significant rise in catalase activity after 21 days compared to DLM low dose treated group (Group D). Supplementation of *Allium sativum* along with DLM high dose (Group G) recorded significant increase in catalase activity after 21 days ($p<0.002$) duration compared to DLM high dose treated group (Group E) (Table 5).

Catalase activity in pancreas showed non-significant reduction in low dose treated deltamethrin group-Group-D but showed significant reduction in catalase activity in Group-E in order of ($p<0.001$) when compared to control Group- A (Table-5). Co-treatment of *Allium sativum* with DLM showed non-significant increase in catalase activity in pancreas in Group F when compared to Group-D, whereasSupplementation of *Allium sativum* along with DLM high dose (Group G) recorded significant increase in catalase activity after 21 days ($p<0.033$) duration compared to DLM high dose treated group (Group E) (Table 5).

Glutathione (GSH)

GSH level in liver of DLM low dose treated animals (Group D) revealed a duration dependent significant decrease after 21 days ($p<0.001$) compared to control (Group A). Highly significant depletion ($p<0.001$) in GSH level was registered after 21 days in liver of high dose DLM treated mice (Group E) compared to control. Administration of *Allium sativum* along with DLM low dose (Group F) revealed significantly increased GSH level after 21 days ($p<0.002$) post treatment compared to DLM low dose treated group (Group D). When animals treated with *Allium sativum* and DLM high dose (Group G), significant increase ($p<0.001$) in GSH level was observed after 21 days compared to high dose DLM treated group (Group E) (Table 6).

GSH content in pancreas showed non-significant reduction in both the deltamethrin treatment groups, in Group-D and Group-E when compared to control Group- A (Table-6). Co-treatment of *Allium sativum* with DLM showed non-significant restoration in protein content in pancreas in both Group F and Group-G when compared with Group-D and E (Table-6).

Glutathione Peroxidase (GPx)

Activity of GPx in liver of low dose DLM treated mice registered significant decline after 21 days ($p<0.002$) treatment (Group D) compared to control group (Group A).Administration of higher dose of DLM in experimental animals recorded significant decrease in GPx activity of liver compared to control animals after 21 days ($p<0.001$) post treatment (Group E).Supplementation of *Allium sativum* along with the low dose of DLM (Group F) showed significant increase ($p<0.033$) after 21 days in GPx activity compared to low dose DLM treated group (Group D). *Allium sativum* when co-supplemented with high dose of DLM (Group G), non-significant increase in GPx activity was registered after 21 days post treatment compared to high dose of DLM (Group E) (Table 7).

GPx activity in pancreas showed non-significant decrease in both the groups treated with DLM Group-D and Group-E when compared to control Group- A (Table-7). Co-treatment of *Allium sativum* with DLM showed non-significant increase in GSH content in pancreas in both Group F and Group-G when compared with Group-D and E (Table-7).

Glutathione Reductase (GRx)

Mice treated with low dose of DLM (Group D) revealed significantly reduced GRx activity in liver after a period of 21 days ($p<0.002$) compared to control group (Group A). When high dose of DLM was administered in experimental mice (Group E), highly significant reduction in GRx activity was noticed in hepatic tissue after 21 days ($p<0.001$) post treatment.When *Allium sativum* was administered along with the DLM low dose (Group F) in experimental female mice, significant rise was noted after 21 days ($p<0.033$) compared to low dose DLM treated group (Group D). Supplementation of *Allium sativum* along with the DLM high dose (Group G) in experimental mice exhibited significant elevation in GRx activity after 21 days ($p<0.033$) compared to DLM high dose treated group (Group E) (Table 8).

GRx activity in pancreas showed non-significant reduction in low dose treated deltamethrin group- Group-D but showed significant reduction in GRx activity in Group-E in order of ($p<0.001$) when compared to control Group- A (Table-5). Co-treatment of *Allium sativum* with DLM showed non-significant increase in GRxin pancreas in Group F when compared to Group-D, whereas Supplementation of *Allium sativum* along with DLM high dose (Group G) recorded significant increase in GRx activity after 21 days ($p<0.001$) duration compared to DLM high dose treated group (Group E) (Table 8).

Table 4: SOD activity (units/mg protein) in Liver and Pancreas of control and treated animals after 21 days treatment

GROUPS	TREATMENT AND DOSE	LIVER	PANCREAS
A	Control (Untreated)	0.612 ± 0.009	0.400±0.014
B	Vehicle Control (Peanut oil)	0.623 ± 0.009NS	0.403±0.010NS
C	Control + *Allium sativum* (AS) (200 mg/kg B.wt.)	0.628 ± 0.009NS	0.4117±0.0188NS
D	Deltamethrin (DM) Low Dose (LD) (3 mg/kg B.wt.)	0.545 ± 0.016*	0.386±0.006NS
E	Deltamethrin (DM) High Dose (HD) (6 mg/kg B.wt.)	0.497 ± 0.018***	0.340±0.008*
F	DM (LD) + AS	0.597 ± 0.008ns	0.393±0.008ns
G	DM (HD) + AS	0.580 ± 0.017##	0.376±0.004##

Values are represented as Mean ± S.E., Analysis of variance at P<0.05 level
Comparison of Group A with Group B, C, D and E; *p<0.033, **p<0.002, ***p<0.001, NS – non significant
Comparison of Group D and E with F and G respectively; #p<0.033, ##p<0.002, ###p<0.001, ns – non significant

Table 5: Catalase activity (µmoles H_2O_2 consumed/mg protein) in Liver and Pancreas of control and treated animals after 21 days treatment

GROUPS	TREATMENT AND DOSE	LIVER	PANCREAS
A	Control (Untreated)	27.28 ± 0.64	20.22±0.116
B	Vehicle Control (Peanut oil)	26.35 ± 0.58NS	20.37±0.365NS
C	Control + *Allium sativum* (AS) (200 mg/kg B.wt.)	27.16 ± 1.04NS	20.61±0.152NS
D	Deltamethrin (DM) Low Dose (LD) (3 mg/kg B.wt.)	23.12 ± 0.97*	19.69±0.807NS
E	Deltamethrin (DM) High Dose (HD) (6 mg/kg B.wt.)	19.85 ± 0.88***	17.90±0.202***
F	DM (LD) + AS	26.29 ± 1.05ns	20.05±0.255ns
G	DM (HD) + AS	25.07 ± 1.22##	19.60±0.130#

Values are represented as Mean ± S.E., Analysis of variance at P<0.05 level
Comparison of Group A with Group B, C, D and E; *p<0.033, **p<0.002, ***p<0.001, NS – non significant
Comparison of Group D and E with F and G respectively; #p<0.033, ##p<0.002, ###p<0.001, ns – non significant

Table 6: GSH level (µg/100mg tissue weight) in Liver and Pancreas of control and treated animals after 21 days treatment

GROUPS	TREATMENT AND DOSE	LIVER	PANCREAS
A	Control (Untreated)	66.75 ± 0.76	36.66±1.461
B	Vehicle Control (Peanut oil)	66.33 ± 0.47NS	36.84±1.091NS
C	Control + *Allium sativum* (AS) (200 mg/kg B.wt.)	66.29 ± 0.45NS	36.90±0.825NS
D	Deltamethrin (DM) Low Dose (LD) (3 mg/kg B.wt.)	58.89 ± 1.69***	34.03±0.338NS
E	Deltamethrin (DM) High Dose (HD) (6 mg/kg B.wt.)	54.17 ± 1.54***	33.22±0.771NS
F	DM (LD) + AS	64.96 ± 1.58##	36.34±0.971ns
G	DM (HD) + AS	61.93 ± 1.70###	34.41±0.0763ns

Values are represented as Mean ± S.E., Analysis of variance at P<0.05 level
Comparison of Group A with Group B, C, D and E; *p<0.033, **p<0.002, ***p<0.001, NS – non significant
Comparison of Group D and E with F and G respectively; #p<0.033, ##p<0.002, ###p<0.001, ns – non significant

Table 7: GPx activity (GSH consumed / mg protein) in Liver and Pancreas of control and treated animals after 21 days treatment

GROUPS	TREATMENT AND DOSE	LIVER	PANCREAS
A	Control (Untreated)	13.61 ± 0.04	6.33±0.22
B	Vehicle Control (Peanut oil)	13.51 ± 0.03NS	6.40±0.11NS
C	Control + *Allium sativum* (AS) (200 mg/kg B.wt.)	13.61 ± 0.02NS	6.38±0.01NS
D	Deltamethrin (DM) Low Dose (LD) (3 mg/kg B.wt.)	12.52 ± 0.28**	6.10±0.22NS
E	Deltamethrin (DM) High Dose (HD) (6 mg/kg B.wt.)	12.32 ± 0.32***	5.53±0.13**
F	DM (LD) + AS	13.42 ± 0.25#	6.00±0.04ns
G	DM (HD) + AS	13.12 ± 0.07ns	5.96±0.136ns

Values are represented as Mean ± S.E., Analysis of variance at P<0.05 level
Comparison of Group A with Group B, C, D and E; *p<0.033, **p<0.002, ***p<0.001, NS – non significant
Comparison of Group D and E with F and G respectively; #p<0.033, ##p<0.002, ###p<0.001, ns – non significant

Glutathione-S-Transferase (GST)

GST activity in liver of experimental animals registered duration dependent significant decline after 21 days (p<0.002) treatment with low dose of DLM (Group D) compared to control animals. Treatment of experimental female mice with high dose of DLM exhibited significantly reduced GST activity after 21 days (p<0.001) in liver (Group E) compared to control animals (Group A).Treatment of *Allium sativum* along with DLM low dose (Group F) showed significant increase (p<0.033) in GST activity after all the experimental duration of 21 days compared to DLM low dose treated group (Group D). Results of liver GST activity in experimental mice treated with *Allium sativum* and DLM high dose (Group G) registered significant increase after 21 days (p<0.033) compared to DLM high dose treated group (Group E) (Table 9).

GST activity in pancreas showed non-significant reduction in low dose treated deltamethrin group- Group-D but showed significant reduction in GST activity in Group-E in order of (p<0.033) when compared to control Group- A (Table-5). Co-treatment of *Allium sativum* with DLM showed non-significant increase in GST in pancreas in Group F when compared to Group-D, whereas Supplementation of *Allium sativum* along with DLM high dose (Group G) recorded significant increase in GST activity after 21 days (p<0.001) duration compared to DLM high dose treated group (Group E) (Table 8).

Table 8: GRx activity (moles NADPH oxidized/ min/mg protein) in Liver and Pancreas of control and treated animals after 21 days treatment

GROUPS	TREATMENT AND DOSE	LIVER	PANCREAS
A	Control (Untreated)	1.53 ± 0.022	1.235±0.009
B	Vehicle Control (Peanut oil)	1.543 ± 0.019NS	1.225±0.022NS
C	Control + *Allium sativum* (AS) (200 mg/kg B.wt.)	1.532 ± 0.025NS	1.237±0.020NS
D	Deltamethrin (DM) Low Dose (LD) (3 mg/kg B.wt.)	1.432 ± 0.024**	1.178±0.007NS
E	Deltamethrin (DM) High Dose (HD) (6 mg/kg B.wt.)	1.405 ± 0.013***	1.113±0.020***
F	DM (LD) + AS	1.512 ± 0.020#	1.233±0.020ns
G	DM (HD) + AS	1.497 ± 0.010#	1.218±0.010###

Values are represented as Mean ± S.E., Analysis of variance at P<0.05 level
Comparison of Group A with Group B, C, D and E; *p<0.033, **p<0.002, ***p<0.001, NS – non significant
Comparison of Group D and E with F and G respectively; #p<0.033, ##p<0.002, ###p<0.001, ns – non significant

Table 9: GST activity (units/mg protein) in Liver and Pancreas of control and treated animals after 21 days treatment

GROUPS	TREATMENT AND DOSE	LIVER	PANCREAS
A	Control (Untreated)	0.418 ± 0.015	0.260±0.012
B	Vehicle Control (Peanut oil)	0.415 ± 0.015NS	0.276±0.011NS
C	Control + *Allium sativum* (AS) (200 mg/kg B.wt.)	0.418 ± 0.017NS	0.266±0.007NS
D	Deltamethrin (DM) Low Dose (LD) (3 mg/kg B.wt.)	0.335 ± 0.015**	0.236±0.011NS
E	Deltamethrin (DM) High Dose (HD) (6 mg/kg B.wt.)	0.320 ± 0.014***	0.206±0.010*
F	DM (LD) + AS	0.400 ± 0.020#	0.256±0.008ns
G	DM (HD) + AS	0.387 ± 0.019#	0.249±0.007##

Values are represented as Mean ± S.E., Analysis of variance at P<0.05 level
Comparison of Group A with Group B, C, D and E; *p<0.033, **p<0.002, ***p<0.001, NS – non significant
Comparison of Group D and E with F and G respectively; #p<0.033, ##p<0.002, ###p<0.001, ns – non significant

DISCUSSION

Accelerated population growth and consequent, constant increase in the demand for food has led to evaluate and monitor the agricultural activities and mass production of agricultural products at a global level. Hence, nowadays agrochemicals and pesticides are extensively used to produce more plant products by controlling the various vectors of plant pathogens [25]. Deltamethrin is one such second generation pesticide, widely used to control ectoparasites like ticks, mites, flies, fleas and to kill agricultural pests for enhancing crop production [26]. Contrary to this DLM has also been reported as environmental and industrial pollutant which can cause toxic manifestations either directly or indirectly into fish, birds and mammals including humans [27].Liver is considered as the major organ for deltamethrin accumulation as it is the principal site for metabolism [28]. Recent studies have also reported deltamethrin induced hepatotoxicity and renotoxicity in rats [29,30], reproductive toxicity [31] and neurotoxicity [32,33].

Various natural as well as anthropogenic xenobiotics induce enhanced production of ROS [34]. Neutralization of these ROS inside organisms occurs by various endogenous antioxidants [35]. This defense system keeps a balance between free radicals, free radical generation and antioxidative profile.In the present study, experimental female mice revealed significant elevation in LPO and concurrent reduction in SOD, catalase, GPx, GRx and GST activity as well as depletion in GSH level in liver and pancreas.In agreement with this study, Kadry et al. (2012) [36] also reported that excess of ROS generation is due to various pollutants such as pesticides which ultimately lead to induced oxidative stress and altered antioxidant levels.

Among the oxidative stress parameters lipid peroxidation (LPO) is considered as a marker of oxidative damage which is responsible for inducing insecticide mediated toxicity in human and other non-target organisms [37,38]. In the present study, dose and time dependent elevation in LPO level was found after deltamethrin treatment in liver of experimental female mice.Further, this has been also supported by disrupted integrity of cellular membranes, noticed due to elevated LPO in Wistar rats [39]. Increased level of malondialdehyde (MDA) in hepatic and pancreatic tissue in the present study might be due excess free radical generation especially hydroxyl radicals which further disturb oxidant and antioxidant equilibrium. This fact is in accordance with the previous work which has demonstrated pyrethroid induced hepatotoxicity [40,41].

In the present study, superoxide dismutase (SOD) activity has been evaluated which is a potent oxidant to scavenge the superoxide anions by catalyzing dismutation of superoxide radicals and converting it into H_2O_2 and oxygen. Further, hydrogen peroxide is converted into water and oxygen [42,43] by catalase and GPx. Exposure of female mice to deltamethrin in this study stimulates overproduction of ROS that suppresses antioxidative potency of SOD, catalase and GPx. This is by either direct damage and denaturation of enzyme [44] or affecting synthesis of enzyme due to deltamethrin [45]. Similar results were also obtained in male mice with deltamethrin treatment [46]. Further, the inhibition or alteration in activity of SOD and mainly catalase may lead to accumulation of oxyradicals and concurrent increase in oxidative stress. Here it was also noted that antioxidant profile in female mice was more affected compared to male mice after deltamethrin treatment. Further, recent work of Abdelkhalek et al. (2015) [47] has also revealed reduction in SOD, catalase and GPx levels in liver, kidney and gills of tilapia after deltamethrin exposure.

GSH acts as a cofactor for other enzymes associated with it like glutathione peroxidase (GPx), glutathione reductase (GRx), and glutathione-S-transferase (GST) to scavenge free radicals and cause detoxification of peroxides generated by lipid peroxidation[48]. Hence, depletion in GSH level may result into altered activity of enzymatic action of GPx, GRx and GST.Reduced GSH levels of liver in this study corroborates with previous reports [47,49]. Further, Jayasree et al. (2003) [50] also reported significant reduction in GSH of deltamethrin treated broiler chicks. In the present study, GPx was noticed to be reduced significantly in hepatic and pancreatic tissues after deltamethrin treatment. Declined GPx observed in this study might be due to increased use of GPx to convert GSH to GSSG [51].Furthermore, reduction in GRx activity of all the tissues has been noticed in the present study which might be due to toxic effects induced by excess of oxidizing compounds. Similar results of declined activity of GRx were also demonstrated by other workers in various tissues of rodents [46,52].In the present investigation significant dose dependent changes in Glutathione-S-transferase (GST) activity was observed after deltamethrin treatment. Decreased GST activity in hepatic and pancreatic tissues of experimental animals might be due to excess of GSH utilization to curb deltamethrin induced toxicity. Other researchers have also found deltamethrin induced depletion in GST activity in experimental animals [53,54]. Contrary results of significantly increased GST activity have been recorded in vital tissues of deltamethrin exposed *Channa punctatus* and *Cyprinus carpio*[55,56].

Allium sativum is one of such medicinal plant which has been well documented for its dietary and medicinal qualities throughout the world since many years [57]. Further, *Allium sativum* stimulated reduction in lipid peroxidation (LPO) might be due to different chemical mechanisms such as free radical quenching, electron transfer, radical addition, or radical recombination [58]. *Allium sativum* also revealed significant restoration in non-enzymatic parameters like GSH and enzymatic parameters i.e., LPO, SOD, catalase, GPx, GRx and GST in both the test tissues of the present work. These results are in accordance with the recent work of Ncir et al. (2018) [59] who have also recorded similar trend of reduction in LPO level and re-established enzymatic activity of SOD, catalase and GPx and concomitant recovery in damaged histoarchitecture of nervous and renal tissues of rats treated with deltamethrin. Al-Snafi [60] has also documented anti- oxidative potential of *Allium sativum* by increasing the level of endogenous antioxidant GSH and regulating the enzymatic functions of SOD, catalase, GPx and GRx.Our results also corroborate with studies of previous workers who have observed free radical scavenging properties of *Allium sativum* that further helps in re-establishment of damaged histomorphology of renal tissue in albino rats exposed to cisplatin [61]. Thus, *Allium sativum* has been found to be potent antioxidant to mitigate deltamethrin raised hepatotoxicity and pancreatic toxicity.

CONCLUSION

Based on the aforementioned data, results of the present investigation directly demonstratethe toxic manifestations of deltamethrin on hepatic and pancreatic tissues. Hence, rampant use of suchpyrethroids should be regulated and monitored strictly especially when it is used for domestic purposes. Furthermore, sensitive sub-groups of population like pregnant women and children should avoid any

direct or indirect exposure as even low concentration of these insecticides can interfere with the normal physiology and overall well-being of the exposed organism. Injudicious and indiscriminate usage should be curbed and suitable alternatives should be employed wherever feasible. Moreover, the results of the present investigation justify the mitigating action of *Allium sativum* to attenuate deltamethrin induced hepatotoxicity and pancreatic toxicity due to its anti-peroxidative and antioxidant effects against free radical induced oxidative stress and toxicity. Since *Allium sativum* has come out as excellent remedy to attenuate deleterious effects of deltamethrin, it is therefore recommended as a dietary supplement to the individuals exposed to such toxicant.

CONFLICT OF INTEREST

The authors have no conflicts of interest regarding this investigation.

REFERENCES

1. Mehlhorn, H., Schumacher, B., Jatzlau, A., Abdel-Ghaffar, F., Al-Rasheid, K.A., Klimpel, S. & Pohle, H. (2011). Efficacy of deltamethrin (Butox® 7.5 pour on) against nymphs and adults of ticks (Ixodes ricinus, Rhipicephalus sanguineus) in treated hair of cattle and sheep. Parasitology research, 108(4): 963-971.
2. Chargui, I., Grissa, I., Bensassi, F., Hrira, M.Y., Haouem, S., Haouas, Z. & Bencheikh, H.(2012). Oxidative stress, biochemical and histopathological alterations in the liver and kidney of female rats exposed to low doses of deltamethrin (DM): a molecular assessment. Biomedical and Environmental Sciences, 25(6): 672-683.
3. Abdel-Daim, M.M., Abuzead, S.M. & Halawa, S.M. (2013). Protective role of Spirulina platensis against acute deltamethrin-induced toxicity in rats. Plos one, 8(9): e72991.
4. Saoudi, M., Ncir, M., Ben Ali, M., Grati, M., Jamoussi, K., Allouche, N. & El Feki, A. (2017). Chemical components, antioxidant potential and hepatoprotective effects of Artemisia campestris essential oil against deltamethrin-induced genotoxicity and oxidative damage in rats. Gen. Physiol. Biophys, 36: 331-342.
5. Ncir, M., Ben Salah, G., Kamoun, H., Makni Ayadi, F., Khabir, A., El Feki, A. & Saoudi, M. (2016). Histopathological, oxidative damage, biochemical, and genotoxicity alterations in hepatic rats exposed to deltamethrin: modulatory effects of garlic (*Allium sativum*). Canadian journal of physiology and pharmacology, 94(6): 571-578.
6. Ncir, M., Ali, M.B., Sellami, H., Allagui, M.S., Lahyani, A., Ayadi, F.M., Boudawara, T., Allouche, N., El Feki, A. & Saoudi, M. (2020). Protective effects of *Allium sativum* essential oil rich in disulfides against deltamethrin induced oxidative stress and hepatotoxicity in rats. Journal of Food Measurement and Characterization, 14(5):2667-2675.
7. Radimer, K.L., Subar, A.F. & Thompson, F.E. (2000). Nonvitamin, nonmineral dietary supplements: issues and findings from NHANES III. Journal of the American Dietetic Association, 100(4): 447-454.
8. Suleria, H.A.R., Butt, M.S., Khalid, N., Sultan, S., Raza, A., Aleem, M. & Abbas, M. (2015). Garlic (*Allium sativum*): diet based therapy of 21st century–a review. Asian pacific journal of tropical disease, 5(4): 271-278.
9. Ramirez, D.A., Locatelli, D.A., González, R.E., Cavagnaro, P.F. & Camargo, A.B. (2017). Analytical methods for bioactive sulfur compounds in *Allium*: An integrated review and future directions. Journal of food composition and analysis, 61: 4-19.
10. Lobo, V., Patil, A., Phatak, A. & Chandra, N. (2010). Free radicals, antioxidants and functional foods: Impact on human health. Pharmacognosy reviews, 4(8): 118-126.
11. Desai, K.R., Moid, N., Patel, P.B. & Highland, H.N. (2015). Protective efficacy of *Allium sativum* on deltamethrin induced toxicity in reproductive tissues of male mice. International journal of pharmaceutical sciences and research, 6(4): 1711-1720.
12. Mukthamba, P. & Srinivasan, K. (2016). Hypolipidemic and antioxidant effects of dietary fenugreek (*Trigonella foenum-graecum*) seeds and garlic (*Allium sativum*) in high-fat fed rats. Food bioscience, 14: 1-9.
13. Lawson, L.D. & Gardner, C.D. (2005). Composition, stability, and bioavailability of garlic products used in a clinical trial. Journal of agricultural and food chemistry, 53(16): 6254- 6261.
14. Zeng, Y., Li, Y., Yang, J., Pu, X., Du, J., Yang, X., Yang, T. & Yang, S. (2017). Therapeutic role of functional components in *Allium* for preventive chronic disease in human being. Evidence-based complementary and alternative medicine, 2017: 1-14.
15. The European Agency for the Evaluation of Medicinal Plants (EMEA). (2001). Veterinary medicine and information technology, EMEA/MRL/779/01-FINAL.
16. El-Kott, A.F. (2012). Amelioration of Nitrate induced Hepatotoxicity. J Med Sci, 12: 85-91.
17. Lowry, O.H., Rosebrough, N.J., Farr, A.L. & Randall, R.J. (1951). Protein measurement with the Folin phenol reagent. Journal of biological chemistry, 193(1): 265-275.
18. Ohkawa, H., Ohishi, N. & Yagi, K. (1979). Assay for lipid peroxides in animal tissues by thiobarbituric acid reaction. Analytical biochemistry, 95(2): 351-358.
19. Kakkar, P., Das, B. & Viswanathan, P.N. (1984). A modified spectrophotometric assay of superoxide dismutase. Ind J Biochem Biophysics, 21:130-132.
20. Sinha, A.K. (1972). Colorimetric assay of catalase. Analytical biochemistry, 47(2): 389-394.
21. Ellman, G.L. (1959). Tissue sulfhydryl groups. Archives of biochemistry and biophysics, 82(1): 70-77.
22. Rotruck, J.T., Pope, A.L., Ganther, H.E., Swanson, A.B., Hafeman, D.G. & Hoekstra, W. (1973). Selenium: biochemical role as a component of glutathione peroxidase. Science, 179(4073): 588-590.

23. Carlberg, I. & Mannervik, B. (1985). Glutathione reductase. Methods in enzymology, 113: 484- 490.
24. Habig, W.H., Pabst, M.J. & Jakoby, W.B. (1974). Glutathione S-transferases the first enzymatic step in mercapturic acid formation. Journal of biological Chemistry, 249(22): 7130-7139.
25. Refaie, A.A.E.R., Ramadan, A. & Mossa, A.T.H. (2014). Oxidative damage and nephrotoxicity induced by prallethrin in rat and the protective effect of *Origanum majorana* essential oil. Asian pacific journal of tropical medicine, 7(S1): S506-S513.
26. Nieradko-Iwanicka, B. & Borzęcki, A. (2015). Subacute poisoning of mice with deltamethrin produces memory impairment, reduced locomotor activity, liver damage and changes in blood morphology in the mechanism of oxidative stress. Pharmacological reports, 67(3): 535-541.
27. Ensibi, C., Hernández-Moreno, D., Míguez Santiyán, M.P., Daly Yahya, M.N., Rodríguez, F.S. & Pérez-López, M. (2014). Effects of carbofuran and deltamethrin on acetylcholinesterase activity in brain and muscle of the common carp. Environmental toxicology, 29(4): 386-393.
28. Rehman, H., Ali, M., Atif, F., Kaur, M., Bhatia, K. & Raisuddin, S. (2006). The modulatory effect of deltamethrin on antioxidants in mice. Clinica chimica acta, 369(1): 61-65.
29. Maalej, A., Mahmoudi, A., Bouallagui, Z., Fki, I., Marrekchi, R. & Sayadi, S. (2017). Olive phenolic compounds attenuate deltamethrin-induced liver and kidney toxicity through regulating oxidative stress, inflammation and apoptosis. Food and chemical toxicology, 106(Part A): 455- 465.
30. Khalatbary, A.R., Ghabaee, D.N.Z., Ahmadvand, H., Amiri, F.T. & Lehi, S.T. (2017). Deltamethrin- Induced Hepatotoxicity and Virgin Olive Oil Consumption: An Experimental Study. Iranian journal of medical sciences, 42(6): 586-592.
31. Sharma, P., Khan, I.A. & Singh, R. (2018). Curcumin and quercetin ameliorated cypermethrin and deltamethrin-induced reproductive system impairment in male Wistar rats by upregulating the activity of pituitary-gonadal hormones and steroidogenic enzymes. International journal of fertility and sterility, 12(1): 72-80.
32. Mani, V.M., Asha, S. & Sadiq, A.M.M. (2014). Pyrethroid deltamethrin-induced developmental neurodegenerative cerebral injury and ameliorating effect of dietary glycoside naringin in male Wister rats. Biomedicine and aging pathology, 4(1): 1-8.
33. Ali, M., Gomaa, M. & Mohammed, Z. (2017). Study of chronic toxic effect of deltamethrin and dimethoate on brain of adult male albino rats. Zagazig Journal of Forensic Medicine, 16(1): 29-46.
34. Livingstone, D.R. (2001). Contaminant-stimulated reactive oxygen species production and oxidative damage in aquatic organisms. Marine pollution bulletin, 42(8): 656-666.
35. Ibrahim, A.E. & Abdel-Daim, M.M. (2015). Modulating effects of *Spirulina platensis* against tilmicosin-induced cardiotoxicity in mice. Cell journal (Yakhteh), 17(1): 137-144.
36. Kadry, S.M., Marzouk, M.S, Amer, A.F., Hanna, M.I., Azmy, A.H. & Hamed, H.S. (2012). Vitamin E as antioxidant in female african catfish (*Clarias gariepinus*) exposed to chronic toxicity of atrazine. Egyptian journal of aquatic biology and fisheries, 16(2): 83-98.
37. Saxena, R., Garg, P. & Jain, D.K. (2011). *In vitro* anti-oxidant effect of vitamin E on oxidative stress induced due to pesticides in rat erythrocytes. Toxicology international, 18(1): 73-76.
38. Mossa, A.T.H. & Abbassy, M.A. (2012). Adverse Haematological and Biochemical Effects of Certain. Research journal of environmental toxicology, 6(4): 160-168.
39. Sharma, P., Singh, R. & Jan, M. (2014). Dose-dependent effect of deltamethrin in testis, liver, and kidney of Wistar rats. Toxicology international, 21(2): 131-139.
40. Abbassy, M. & Mossa, A.H. (2012). Haemato-biochemical effects of formulated and technical cypermethrin and deltamethrin insecticides in male rats.Journal of Pharmacology and Toxicology, 7(7): 312-321.
41. Abbassy, M.A., Marzouk, M.A., Mansour, S.A., Shaldam, H.A. & Mossa, A.H. (2014). Impact of oxidative stress and lipid peroxidation induced by lambdacyhalothrin on p450 in male rats: the ameliorating effect of zinc. Journal of Environmental and Analytical Toxicology, 4(4): 1-5.
42. Salvi, M., Battaglia, V., Brunati, A.M., La Rocca, N., Tibaldi, E., Pietrangeli, P., Marcocci, L., Mondovì, B., Rossi, C.A. & Toninello, A. (2007). Catalase takes part in rat liver mitochondria oxidative stress defense. Journal of biological chemistry, 282(33): 24407-24415.
43. Safhi, M.M. (2018). Nephroprotective effect of Zingerone against CCl4-induced renal toxicity in Swiss albino mice: molecular mechanism. Oxidative medicine and cellular longevity, 2018: 1-7.
44. Dubey, N., Khan, A.M. & Raina, R. (2013). Sub-acute deltamethrin and fluoride toxicity induced hepatic oxidative stress and biochemical alterations in rats. Bulletin of environmental contamination and toxicology, 91(3): 334-338.
45. Yonar, M.E., Yonar, S.M., Pala, A., Silici, S. & Saglam, N. (2015). Trichlorfon-induced haematological and biochemical changes in *Cyprinus carpi*o: ameliorative effect of propolis. Diseases of aquatic organisms, 114(3): 209-216.
46. Desai, K.R., Moid, N., Patel, P.B. & Highland, H.N. (2015). Protective efficacy of *Allium sativum* on deltamethrin induced toxicity in reproductive tissues of male mice. International journal of pharmaceutical sciences and research, 6(4): 1711-1720.
47. Abdelkhalek, N.K., Ghazy, E.W. & Abdel-Daim, M.M. (2015). Pharmacodynamic interaction of *Spirulina platensis* and deltamethrin in freshwater fish *Nile tilapia*, *Oreochromis niloticus*: impact on lipid peroxidation and oxidative stress. Environmental Science and Pollution Research, 22(4): 3023-3031.
48. Zhang, H. & Forman, H.J. (2009). Redox regulation of γ-glutamyl transpeptidase. American journal of respiratory cell and molecular biology, 41(5): 509-515.

49. Gündüz, E., Ülger, B.V., İbiloğlu, İ., Ekinci, A., Dursun, R., Zengin, Y., İçer, M., Uslukaya, Ö., Ekinci, C. & Güloğlu, C. (2015). Glutamine provides effective protection against deltamethrin-induced acute hepatotoxicity in rats but not against nephrotoxicity. Medical science monitor: International medical journal of experimental and clinical research, 21: 1107-1114.
50. Jayasree, U., Reddy, A.G., Reddy, K.S., Anjaneyulu, Y. & Kalakumar, B. (2003). Evaluation of Vitamin E against Deltamethrin Toxicity in Broiler Chicks. Indian journal of physiology and pharmacology, 47(4): 447-452.
51. Dubey, N., Raina, R. & Khan, A.M. (2012). Toxic effects of deltamethrin and fluoride on antioxidant parameters in rats. Fluoride, 45(3): 242-246.
52. Sharma, P., Khan, I.A. & Singh, R. (2018). Curcumin and quercetin ameliorated cypermethrin and deltamethrin-induced reproductive system impairment in male Wistar rats by upregulating the activity of pituitary-gonadal hormones and steroidogenic enzymes. International journal of fertility and sterility, 12(1): 72-80.
53. Ayaz, N.O. (2017). Modulating impacts of coustus *Sassura lappa* extract against oxidative stress and genotoxicity induced by deltamethrin toxicity in rat kidneys. International journal of pharmaceutical research and allied sciences, 6(2): 49-60.
54. Mekircha, F., Chebab, S., Gabbianelli, R. & Leghouchi, E. (2018). The possible ameliorative effect of *Olea europaea* L. oil against deltamethrin-induced oxidative stress and alterations of serum concentrations of thyroid and reproductive hormones in adult female rats. Ecotoxicology and environmental safety, 161: 374-382.
55. Atif, F., Parvez, S., Pandey, S., Ali, M., Kaur, M., Rehman, H., Khan, H.A. & Raisuddin, S. (2005). Modulatory effect of cadmium exposure on deltamethrin-induced oxidative stress in *Channa punctata* Bloch. Archives of environmental contamination and toxicology, 49(3): 371-377.
56. Ensibi, C., Perez-Lopez, M., Rodríguez, F.S., Miguez-Santiyan, M.P., Yahya, M.D. & Hernández- Moreno, D. (2013). Effects of deltamethrin on biometric parameters and liver biomarkers in common carp (*Cyprinus carpio* L.). Environmental toxicology and pharmacology, 36(2): 384- 391.
57. Lawal, A., Dangoggo, S.M. & Umar, K.J. (2010). Phytochemical and antibacterial screening of garlic (*Allium sativum*). Katsina journal of pure and applied sciences, 2: 101-104.
58. Borek, C. (2001). Antioxidant health effects of aged garlic extract. The Journal of nutrition, 131(3): 1010S-1015S.
59. Ncir, M., Saoudi, M., Sellami, H., Rahmouni, F., Lahyani, A., Makni Ayadi, F., El Feki, A. & Allagui, M.S. (2018). *In vitro* and *In vivo* studies of *Allium sativum* extract against deltamethrin- induced oxidative stress in rats brain and kidney. Archives of physiology and biochemistry, 124(3): 207-217.
60. Al-Snafi, A.E. (2015). Therapeutic properties of medicinal plants: a review of plants with antioxidant activity (part 1). International Journal of Pharmacology and Toxicology, 6(3): 159-182.
61. El-Din, M.M.M., Mostafa, A.M. & Abd-Elkader, A. (2014). Experimental studies on the effect of (Lambda-Cyhalothrin) insecticide on lungs and the ameliorating effect of plant extracts (Ginseng (*Panax Ginseng*) and garlic (*Allium sativum* L.) on asthma development in albino rats. BMC research notes, 7(1): 243.

CITATION OF THIS ARTICLE

P Patel, K Desai, H Highland, N Nimbark. Reversal of deltamethrin-induced oxidative stress in hepatic and pancreatic tissues by *Allium sativum*.. Bull. Env. Pharmacol. Life Sci., Vol 10[12] November 2021 : 134-145.