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Extra-pulmonary oxidative stress investigations of an over-the-counter pyrethroid insecticide product in rats

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Extra-pulmonary oxidative stress investigations of exposure to aerosol, of an over-the-counter pyrethroid insecticide product in Nigeria in Wistar rats were carried out. Four groups of five animals each were used in this study, and were exposed to different concentrations of the insecticide aerosol. Malondialdehyde (MDA) and reduced glutathione (GSH) concentrations were determined in the liver, kidney and testes, while the activities of catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx) and glutathione S-transferase (GST) were determined in liver only. Liver, kidney and testes showed no significant difference in their weights and relative weights when compared with control, except groups II and III where significant increase in liver weight was recorded. There was no significant increase in liver, kidney and testis MDA concentration when compared with the control. There was significant decrease in testis GSH for all groups, while significant increase was seen in groups III and IV for kidney GSH and group II only for liver GSH. The activities of liver CAT, SOD, GST and GPx were reduced, but showed no significant difference when compared with the control. Our investigations therefore reveal that the compositions of the pyrethroid insecticide product may not play any role in extra-pulmonary tissue oxidative damage.

Key words: Insecticide aerosol, pyrethroid, oxidative stress, lipid peroxidation, rats.

INTRODUCTION

Eliminating mosquito vectors of the malaria parasite is one of the most effective measures against malaria. Pyrethroids are human-made forms of pyrethrins. There are two types that differ in chemical structure and symptoms of exposure. Type I pyrethroids include allethrin, tetramethrin, resmethrin, d-phenothrin, bioresmethrin and permethrin (Klaassen et al., 1996; Ray, 1991). Some examples of type II pyrethroids are cypermethrin, cyfluthrin, deltamethrin, cyphenothrin, fenvalerate, and fluvalinate (Klaassen et al., 1996; Ray, 1991). They act primarily on the nervous system by prolonging the open state of voltage-sensitive sodium channels (Soderlund et al., 2002). Since the nervous system of all the animals is quite similar, these

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compounds have the potential for non-target species (Soderlund et al., 2002).

Pyrethroid insecticide products are commonly used in the control of mosquitoes in Nigeria. The active insecticidal ingredients of pyrethroid insecticide product used in this study include cyfluthrin, imiprothrin and prallethrin. β-Cyfluthrin is a newer pyrethroid insecticide used all over the world. It is reported to be neurotoxic (Satpathy et al., 1997), hepatotoxic (Omotuyi et al., 2006) and teratogenic (Soni et al., 2011). β-Cyfluthrin toxicity is exhibited by its metabolites which in turn generate free radicals (El-Demerdash, 2007). *In vitro* study on human erythrocytes has demonstrated that cyfluthrin generates the reactive oxygen species (ROS) in cells (Sadowaska-Woda et al., 2010).

A study of imiprothrin in rats has indicated that repeated non-contiguous inhalation of an insecticide that contains imiprothrin can have immunotoxic effects in sites distal to the lungs (Emara and Draz, 2007). In human volunteers who were exposed regularly to prallethrin containing mosquito repellent, alterations in the biochemical composition of erythrocyte membranes, erythrocytic osmotic haemolysis, and plasma levels of nitrite and nitrate were found (Narendra et al., 2007).

In addition to the active insecticidal ingredients, insecticide aerosols also contain ‘‘inert’’ ingredients which are referred to as “trade secrets” by their manufacturers. Inert ingredients may not be inert in the usual sense of the word; often they are not chemically, biologically or toxicologically inert. Occupational exposure to these solvents has been associated with an increased rate of leukemia and a range of lymphoma types (McMichael, 1988; Cocco et al., 2010).

Pesticides induce oxidative stress as well as alter the defense mechanisms of detoxification and scavenging enzymes (Rasoul et al., 2012; Mossa et al., 2012; Mansour and Mossa, 2010a, 2011; Marzouk et al., 2011). Overproduction of ROS can exacerbate oxidative stress and ROS generation has been linked to a number of disease processes, such as heart disease (Giordano, 2005), diabetes (Rolo and Palmeira, 2006), liver injury (Mansour and Mossa, 2010a, 2011; Mossa, 2004; Jaeschke, 2000), cancer (Klaunig and Kamendulis, 2004) and aging (Bokov et al., 2004). It is therefore necessary to maintain the balance between ROS generation and antioxidant enzymes. This balance has been suggested to have an important role in preventing pesticides toxicity (Mansour and Mossa, 2010a, 2011; Mossa, 2004; Jaeschke, 2000).

There is a widespread misuse, incessant and inadvertent exposure to pyrethroid insecticide products in Nigeria, and there is still a dearth of information on the toxicological consequences of frequent exposure. In this study, we therefore assessed the risk and the hazard associated with exposure to aerosols of a commonly used over-the-counter pyrethroid insecticide product, using rats as an experimental animal model. We investigated if exposure to the insecticide could lead to extra-pulmonary oxidative damage by monitoring the activities of liver catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx) and glutathione S-transferase (GST), as well as concentrations of reduced glutathione (GSH) and malondialdehyde (MDA) in liver, kidney and testes of rats.

MATERIALS AND METHODS

Animals, test materials and reagents

Twenty albino Wistar rats with an average weight of 150 g used for this study were obtained from the animal house of the College of Veterinary Medicine, Federal University of Agriculture, Abeokuta, Nigeria. Baygon^{TM} multi-purpose pyrethroid insecticide product of Johnson Wax Nigeria Limited, Lagos (composition Cyfluthrin, 0.015%; Imiprothrin, 0.05%; Prallethrin, 0.05%; and undisclosed inert ingredients, 99.885%) was purchased from a local supermarket near the university. All other reagents and chemicals were of analytical grade, products of Sigma Chemical Co., Saint Louis, MO, USA or BDH Chemical Ltd, Poole, England.

Animal handling and insecticide aerosol exposure chamber design

The experimental animals were handled and used in accordance with the international guide for the care and use of laboratory animals (National Research Council, 1996). They were allowed to acclimatize, and kept in standard laboratory conditions under natural light-dark cycle. The experimental animals had access to diet and water *ad libitum* throughout the duration of the experiment. Four identical wooden-glass exposure chambers each with internal volume of 0.167 m^3 (0.405 x 0.800 x 0.515 m) were used. Into each chamber (containing five rats) was sprayed 0, 1, 2, or 3 ml respective volume of the pyrethroid product multi-purpose insect killer. This is equivalent to 0, 6.0, 12.0 and 18.0 mL m^{-3} of the exposure chamber, respectively. The chambers were closed for 1 h immediately after spraying. A glass top enabled observations to be made on reactions of the animals to the aerosols. These procedures were carried out once a day for 6 weeks. Effective light usage of the insecticide in Nigerian homes ranges from about 3.5 - 6.0 mL m^{-3}.

Three different doses, multiples of this light usage, were experimented within different groups of rats exposed to the insecticide aerosols thereby giving allowance for heavy aerosols spray and high level of exposure to the insecticide. The volume of the insecticide sprayed as aerosols usually depend on the users and the perceived population of insects in the house.

Experimental animals and treatments

The rats were divided randomly into four groups (I to IV) of five animals per group as follows: group I (control), not exposed to insecticide aerosols; group II, exposed to 6.0 mL of the aerosols per 1 m^3 treatment chamber volume (1 mL of the insecticide product in 0.167 m^3 exposure chamber); group III, exposed to the aerosols at 12.0 mL m^{-3}; group IV, exposed to the aerosols at 18.0 mL m^{-3}.

Sample preparation

At the end of the experimental period, the animals were sacrificed

Table 1. Liver, kidney and testis weights of control and experimental rats exposed to pyrethroid insecticide aerosols.

Group	Liver weight (g)	Kidney weight (g)	Testis weight (g)	Relative liver weight (%)	Relative kidney weight (%)	Relative testis weight (%)
I	6.51 ± 0.67	1.09 ± 0.07	2.36 ± 0.18	3.05 ± 0.24	0.51 ± 0.02	1.11 ± 0.09
II	5.15 ± 0.33[a]	1.00 ± 0.03	2.56 ± 0.08	2.67 ± 0.14	0.52 ± 0.01	1.33 ± 0.06
III	5.17 ± 0.22[a]	0.99 ± 0.05	2.23 ± 0.15	2.73 ± 0.21	0.52 ± 0.03	1.19 ± 0.10
IV	5.58 ± 0.38	1.06 ± 0.06	2.51 ± 0.06	2.77 ± 0.04	0.53 ± 0.02	1.26 ± 0.06

Each value is a mean of 5 rats ± SEM. [a]Significantly different from negative control group ($p<0.05$). Relative organ (liver/kidney/testis) weight = (organ weight/final body weight) x 100. I, Group of rats not exposed to aerosols. II, Rats in this group were exposed to 1 mL of the insecticide product aerosols in 0.167 m^3 treatment chamber (equivalent to 6.0 mL m^{-3} treatment chamber volume). III, Animals in this group were exposed to 2 mL of the aerosols (equivalent to 12.0 mL m^{-3} treatment chamber volume). IV, Rats in this group were exposed to 3 mL of the aerosols (equivalent to 18.0 mL m^{-3} treatment chamber volume).

and liver, kidney and testis were harvested. They were washed in ice-cold saline (0.9% w/v) solution, and were blotted dry before suspension in 0.1 M phosphate buffer (pH 7.4) and homogenized (1:4 w/v), followed by centrifugation at 5000 rpm for 10 min. The homogenate was kept frozen until used.

Assay for biochemical parameters

CAT activity was determined by the method of Sinha (1972), and expressed in µmole/min/mg protein. SOD was determined by the method of Misra and Fridovich (1972), and expressed in unit/mg protein, GPx was determined by the method of Paglia and Valentine (1967), expressed in µg/ml/mgprotein, and GST was determined by the method of Habig et al. (1974), expressed in µmole/min/mgprotein. MDA levels, a marker of lipid peroxidation was determined by the method of Beuge and Aust (1978) and expressed in nmol/mg, while GSH levels was determined by the method of Moron et al. (1979), and expressed in µg/ml. Protein concentration was done according to the method of Gornall et al. (1949).

Data were analyzed by one-way analysis of variance (ANOVA), followed by least significant difference (LSD) to test for significant differences among the groups of rats using Statistical Package for Social Sciences program version 17.0. Data were expressed as mean ± standard error of mean. P values less than 0.05 were considered statistically significant.

RESULTS

Liver, kidney, testis weights and relative weights of control and experimental rats exposed to pyrethroid insecticide aerosols are shown in Table 1. Exposure of rats to the insecticide aerosol did not produce any significant effect ($p > 0.05$) on liver, kidney and testis relative weights as compared to the control. Apart from 6.0 and 12.0 mL m^{-3} of the insecticide aerosol that produced significant difference ($p < 0.05$) in liver weights, there was no significant difference ($p > 0.05$) in kidney and testis weights as compared to the control.

The effects of pyrethroid insecticide aerosol on liver, kidney and testis MDA concentrations are shown in Figure 1. The obtained results showed no significant increase ($p > 0.05$) in the liver and testis MDA concentrations as compared to control, while 18.0 mL m^{-3} of the insecticide aerosol significantly increased ($p < 0.05$) the kidney MDA concentration as compared to the control.

Effects of the insecticide aerosol on liver, kidney and testis levels of GSH are represented also in Figure 2. All the concentrations of insecticide aerosol assessed (Groups II to IV) significantly reduced ($p < 0.05$) the levels of GSH in testis as compared to the control. A significant increase ($p < 0.05$) in kidney levels of GSH was recorded by 12.0 and 18.0 mL m^{-3} of the insecticide, while only 6.0 mL m^{-3} of the aerosol significantly raised ($p < 0.05$) the liver GSH level as compared to the control.

Lastly, exposure to the insecticide aerosol did not give significant reductions ($p > 0.05$) in liver GST, SOD, GPx and CAT activities as compared to the control.

DISCUSSION

Misuse of pyrethroids insecticides could cause toxicity in non-target species (Cantalamessa, 1993). Insecticides exposure constitutes a source of potent hazard especially in children and animals (Eisler, 1989; Nebeker et al., 1992; Menegaux et al., 2006). The multipurpose insect killer used, a pyrethroid insecticide product, is a popular brand for the eradication of mosquito in households in Nigeria and in some cases, it is used daily in homes. This increases the exposure of people, especially children sleeping under the aerosol.

Our investigation on the pyrethroid insecticide aerosol revealed no significant effects on liver, kidney and testes weights (Table 1), as well as their relative weights (Table 2). Measurement of MDA is mostly used to monitor lipid peroxidation and indirectly, oxidative stress *in- vitro* and *vivo* (Beltowski et al., 2000). The lipid oxidation causes disruption of the bilayer and cell integrity accompanied by leakage of cellular content from the damaged organ into the blood stream (Ologundudu et al., 2010). Our investigations in the present study revealed that the insecticide aerosol caused no significant increase ($p >$

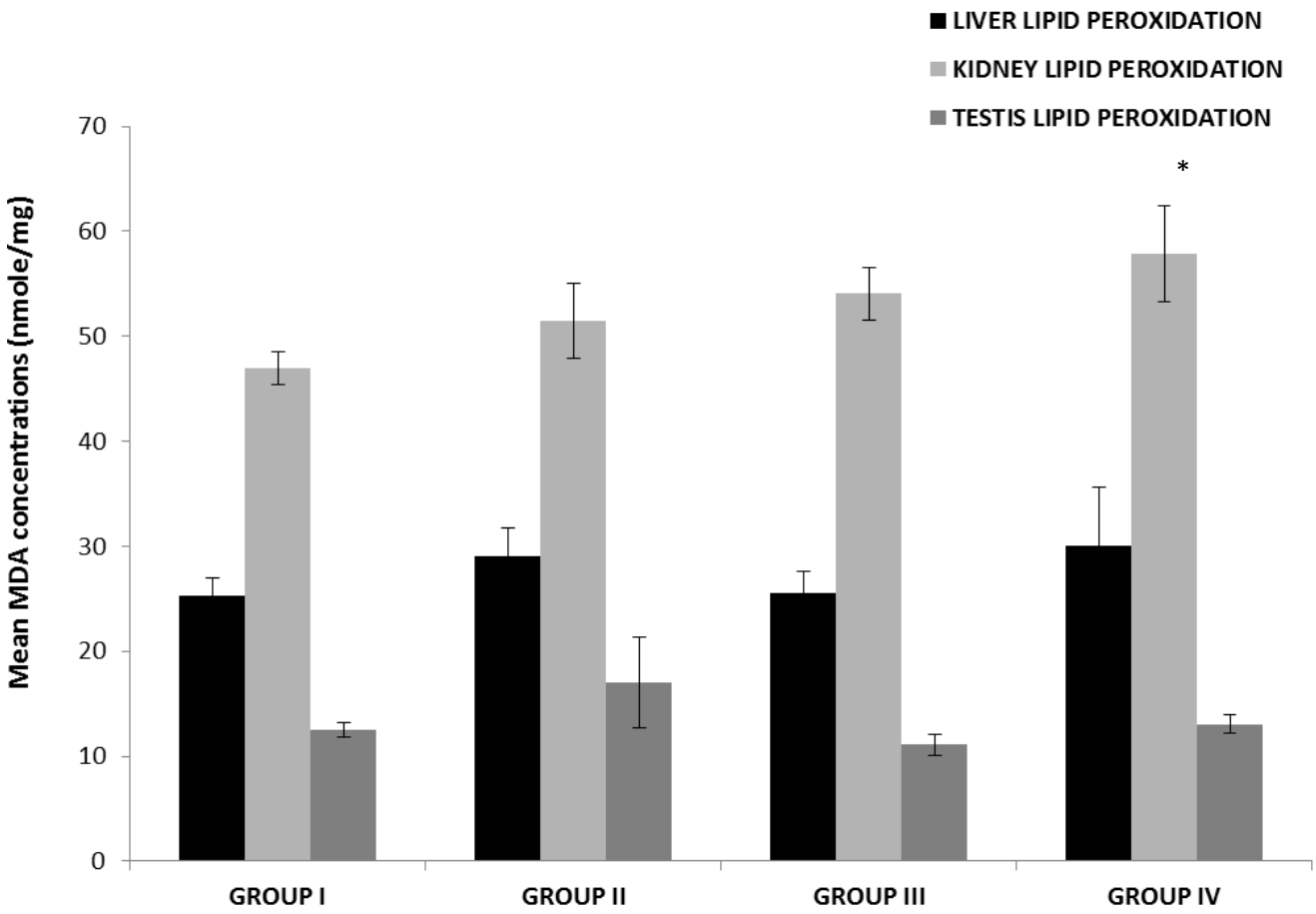

Figure 1. Effects of pyrethroid insecticide aerosols on liver, kidney and testes MDA concentrations. I, Group of rats not exposed to aerosols. II, Rats in this group were exposed to 1 mL of the insecticide product aerosols in 0.167 m^3 treatment chamber (equivalent to 6.0 mL m^{-3} treatment chamber volume). III, Animals in this group were exposed to 2 mL of the aerosols (equivalent to 12.0 mL m^{-3} treatment chamber volume). IV, Rats in this group were exposed to 3 mL of the aerosols (equivalent to 18.0 mL m^{-3} treatment chamber volume). Values are mean ± SEM; n = 5. *Significantly different from control group I ($p < 0.05$).

Table 2. Relative liver, kidney, and testis weights of control and experimental rats exposed to pyrethroid insecticide aerosols.

Group	Relative liver weight (%)	Relative kidney weight (%)	Relative testis weight (%)
I (Control)	3.05 ± 0.24	0.51 ± 0.02	1.11 ± 0.09
II (6.0 mL m^{-3} of insecticide as aerosols)	2.67 ± 0.14	0.52 ± 0.01	1.33 ± 0.06
III (12.0 mL m^{-3} of insecticide as aerosols)	2.73 ± 0.21	0.52 ± 0.03	1.19 ± 0.10
IV (18.0 mL m^{-3} of insecticide as aerosols)	2.77 ± 0.04	0.53 ± 0.02	1.26 ± 0.06

No significant difference from negative control group ($p<0.05$). Relative organ (liver/kidney/testis) weight = (organ weight/final body weight) x 100.
I, Group of rats not exposed to aerosols. II, Rats in this group were exposed to 1 mL of the insecticide product aerosols in 0.167 m^3 treatment chamber (equivalent to 6.0 mL m^{-3} treatment chamber volume). III, Animals in this group were exposed to 2 mL of the aerosols (equivalent to 12.0 mL m^{-3} treatment chamber volume). IV, Rats in this group were exposed to 3 mL of the aerosols (equivalent to 18.0 mL m^{-3} treatment chamber volume).

0.05) in the concentrations of liver and testis MDA (Figure 1), except for kidney MDA concentration in Group IV. Deltamethrin, a pyrethroid insecticide was reported to significantly increase the MDA level in testis, liver and kidney of rats (Sharma et al., 2014; Manna et al., 2005; Rehman et al., 2006). Cyfluthrin administered to rats has been reported to increase the concentration of MDA in liver and kidney of rats (Yilmaz et al., 2015).

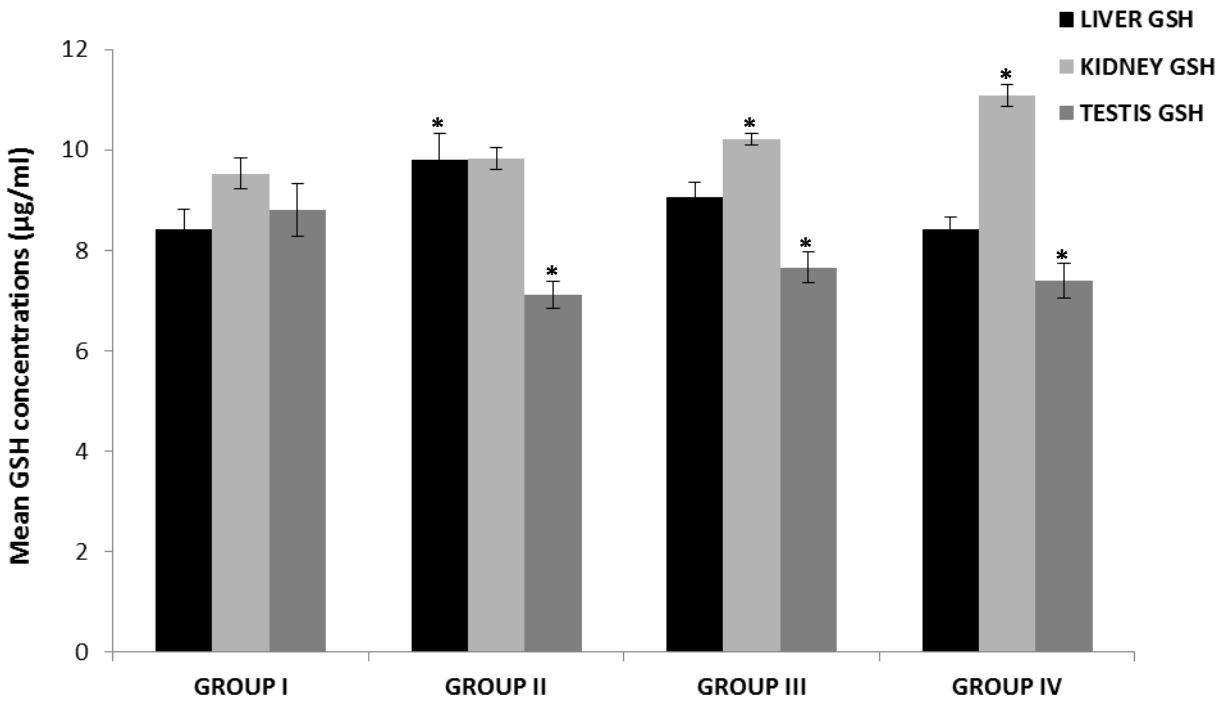

Figure 2. Effects of pyrethroid insecticide aerosols on liver, kidney and testes GSH levels. I, Group of rats not exposed to aerosols. II, Rats in this group were exposed to 1 mL of the insecticide product aerosols in 0.167 m^3 treatment chamber (equivalent to 6.0 mL m^{-3} treatment chamber volume). III, Animals in this group were exposed to 2 mL of the aerosols (equivalent to 12.0 mL m^{-3} treatment chamber volume). IV, Rats in this group were exposed to 3 mL of the aerosols (equivalent to 18.0 mL m^{-3} treatment chamber volume). Values are mean ± SEM; n = 5. *Significantly different from control group I ($p < 0.05$).

Also, in the present study, our investigations revealed that the insecticide aerosol increased significantly ($p < 0.05$) the levels of GSH in the kidney (Groups 3 and 4) of rats (Figure 2), while it significantly ($p < 0.05$) decreased the levels of GSH in testis in all the groups. The elimination of H_2O_2 is either affected by catalase or glutathione peroxidase, with the latter predominating (using reduced glutathione as substrate) in the case of testes (Zini and Schlegel, 1996; Peltola et al., 1992). This could be responsible for the decrease in testes GSH levels seen in our investigations. Incubation of testis homogenate with different concentrations of insecticide mixture for different time intervals significantly decreased the activity of antioxidant enzymes, like GST, SOD and CAT, and the level of GSH (El-Demerdash et al., 2013). Cypermethrin-induced toxicity has been reported to decrease the level of GSH in testis of rats (Huq et al., 2014). GSH participate in the elimination of ROS, acting both as non-enzymatic oxygen radical scavenger and as a substrate for various enzymes such as GPx (Ashar and Muthu, 2012). The liver is the primary site for the detoxification of xenobiotics, and it is rich in drug metabolizing enzymes. All these factors may protect the liver from the overwhelming toxic effects of the insecticide. Again, from our investigations, the non-significant decrease ($p > 0.05$) in liver GST, GPx, CAT and SOD activities (Figure 3) following exposure to the insecticide aerosol could be also due to the prompt and balanced response of these tissues to the increased and continuous generation of ROS particularly superoxide radical ($O_2^{\cdot-}$), which can be dismutated to H_2O_2 by SOD, and then to H_2O and O_2 by CAT, GPx and GST. It has been reported that 14 days exposure to beta-cyfluthrin caused a significant decrease in CAT and SOD activity as compared to the control in a dose dependent manner (Verma et al., 2013). Cyfluthrin has been reported to increase the generation of free radical and decrease SOD and CAT activity in mice (Omotuyi et al., 2006; Eraslan et al., 2007) and in cultured human erythrocytes (Sadowaska-Woda et al., 2010). Cyfluthrin administration was reported to lower the activities of CAT and GPX in liver and kidney of rats (Yilmaz et al., 2015). It was recently reported that the activities of kidney SOD and GPx, as well as activities of liver SOD, GST and CAT were significantly decreased by prallethrin administration in rats (Rafaie et al., 2014; Mossa et al., 2013). Cypermethrin-induced toxicity has been reported to decrease the activities of CAT, SOD, GST and GPx in testis of rats (Huq et al., 2014). Deltamethrin administration has been reported to significantly reduce the activities of SOD, CAT, GST and GPx, as well as levels of GSH in liver, testis and kidney of rats exposed to

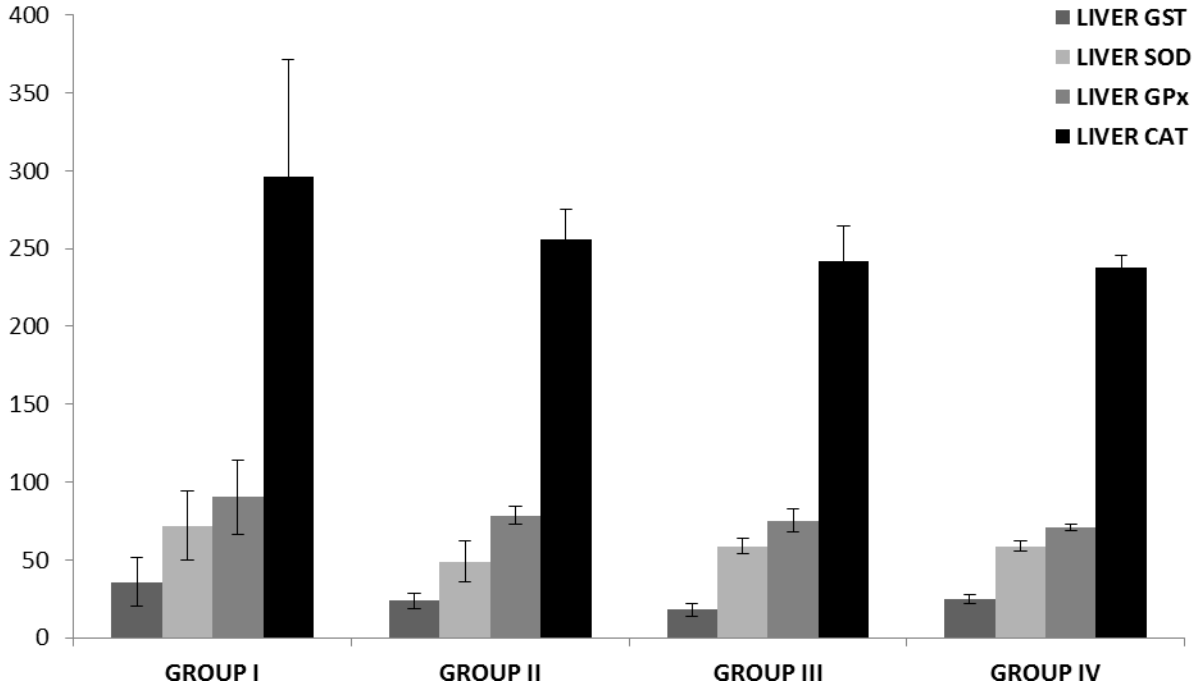

Figure 3. Effects of pyrethroid insecticide aerosols on liver GST, SOD, GPx and CAT activities. I, Group of rats not exposed to aerosols. II, Rats in this group were exposed to 1 mL of the insecticide product aerosols in 0.167 m^3 treatment chamber (equivalent to 6.0 mL m^{-3} treatment chamber volume). III, Animals in this group were exposed to 2 mL of the aerosols (equivalent to 12.0 mL m^{-3} treatment chamber volume). IV, Rats in this group were exposed to 3 mL of the aerosols (equivalent to 18.0 mL m^{-3} treatment chamber volume). Values are mean ± SEM; n = 5GST, SOD, GPx, and CAT activities are expressed as µmole/min/mgprotein, unit/mgprotein, µg/ml/mgprotein, and µmole/min/mgprotein, respectively.

it (Sharma et al., 2014; Manna et al., 2005; Rehman et al., 2006).

In conclusion, results from these investigations reveals that the concentrations of the active pyrethroid ingredients in the aerosol of this commonly used over-the-counter pyrethroid insecticide product at the dose assessed may not be associated or implicated in extra-pulmonary oxidative tissue damage.

Conflict of interests

The authors did not declare any conflict of interest.

REFERENCES

Ashar WMP, Muthu MHS (2012). Fenvalerate induced hepatotoxicity and its amelioration by quercetin. Int. J. Pharm. Tech. Res. 4(4):1391-1400.

Beltowski J, Wójcicka G, Górny D, Marciniak A (2000). The effect of dietary-induced obesity on lipid peroxidation, antioxidant enzymes and total plasma antioxidant capacity. J. Physiol. Pharmacol. 51:883-896.

Bokov A, Chaudhuri A, Richardson A (2004). The role of oxidative damage and stress in aging. Mech. Ag. Dev. 125(10-11):811-826.

Buege JA, Aust SD (1978). Microsomal lipid peroxidation. Methods Enzymol. 52:302-310.

Cantalamessa F (1993). Acute toxicity of two pyrethroids, permethrin, and cypermethrin in neonatal and adult rats. Arch. Toxicol. 67(7):510-513.

Cocco P, T'Mannetje A, Fadda D, Melis M, Becker N, de Sanjose S, Foretova L, et al (2010). Occupational exposure to solvents and risk of lymphoma subtypes: results from the Epilymph case-control study. Occup. Environ. Med. 67(5): 341-347.

Eisler R (1989). Pentachlorophenol hazards to fish, wildlife and invertebrates: A synoptic review. US fish and wildlife service. Biol. Rep. 85:72.

El-Demerdash FM (2007). Lambdacyhalothrin-induced changes in oxidative stress biomarkers in rabbit erythrocytes and alleviation effects of some antioxidants. Toxicol. In-Vitro. 21: 392-397.

El-Demerdash FM, Jebur AB, Nasr HM (2013). Oxidative stress and biochemical perturbations induced by insecticides mixture in rat testes. J. Environ. Sci. Health B. 48(7): 593-599.

Emara AM, Draz EI (2007). Immunotoxicological study of one of the most common over the counter pyrethroid insecticide products in Egypt. Inhal. Toxicol. 19(12): 997-1009.

Eraslan G, Saygi S, Essiz D (2007). Evaluation of aspect of some oxidative stress parameters using vitamin E, proanthocynidin and N-acetylcysteine against exposure to cyfluthrin in mice. Pest. Biochem. Physiol. 88:43-49.

Giordano FJ (2005). Oxygen, oxidative stress, hypoxia, and heart failure. J. Clin. Invest. 115(3): 500-508.

Gornall AG, Bardawill CJ, David MM (1949). Determination of serum protein by biuret method. J. Biol. Chem. 117: 751-766.

Habig WH, Pabst MJ, Jakoby WB (1974). Glutathione S-transferases: The first enzymatic step in mercapturic acid formation. J. Biol. Chem. 249:7130-7139.

Huq AU, Sharma P, Singh R (2014). Cypermethrin-induced reproductive

toxicity in the rat is prevented by resveratrol. J. Hum. Reprod. Sci. 7(2):99-106.
Jaeschke H (2000). Reactive oxygen and mechanisms of inflammatory liver injury. J. Gastroenterol. Hepatol. 15(7):718-724.
Klaassen CD, Amdur MO, Doull J Eds (1996). Casarett & Doull's Toxicology. The Basic Science of Poisons (5 ed.). Toronto: th McGraw-Hill Companies, Inc.
Klaunig JE, Kamendulis LM (2004). The Role of oxidative stress in carcinogenesis. Ann. Rev. Pharmacol. Toxicol. 44: 239-267.
Manna S, Bhattacharyya D, Mandal TK, Das S (2005). Repeated dose toxicity of deltamethrin in rats. Ind. J. Pharmacol. 37(3): 160-164.
Mansour SA, Mossa ATH (2010a). Oxidative damage, biochemical and histopathological alterations in rats exposed to chlorpyrifos and the antioxidant role of zinc. Pest. Biochem. Physiol. 96(1):14-23.
Mansour SA, Mossa ATH (2011). Adverse effects of exposure to low doses of chlorpyrifos in lactating rats. Toxicol. Ind. Health. 27(3):213-224.
Marzouk MA, Abbassy MA, Mansour SA, Mossa AH, Elsayed SR (2011). Effect of dimethoate, dicofol and voltaren on oxidant/antioxidant status in male rats: role of selenium. J. Agric. Environ. Sci. 10(2): 40-60.
McMichael AJ (1988). Carcinogenicity of benzene, toluene and xylene: Epidemiological and experimental evidence. IARC Scientific Publications. 85:3-18.
Menegaux F, Baruchel A, Bertrand Y, Lescoeur B, Leverger G, Nelken B, et al (2006). Household exposure to pesticides and risk of childhood acute leukaemia. Occup. Environ. Med. 63(2):131-134.
Misra HP, Fridovich I (1972). The role of superoxide anion in the auto-oxidation of epinephrine and a simple assay for superoxide dismutase. J. Biol. Chem. 247:3170-3175.
Moron MS, Depierre JW, Mannervik B (1979). Levels of glutathione. Glutathione reductase and glutathione S-transferase activities in rat lung and liver. Biochem. Biophys. Acta. 582:67-78.
Mossa AH (2004). Genotoxicity of pesticides [Ph.D. thesis], Pesticide Chemistry and Toxicology, Faculty of Agriculture, Damanhour, Alexandria University.
Mossa AH, Refaie AA, Ramadan A, Bouajila J (2013). Amelioration of prallethrin-induced oxidative stress and hepatotoxicity in rat by the administration of *Origanum majorana* essential oil. BioMed Research International, doi.org/10.1155/2013/8590851-11.
Mossa AT, Heikal TM, Omara EA (2012). Physiological and histopathological changes in the liver of male rats exposed to paracetamol and diazinon. Asian Pac. J. Trop. Biomed. 2:S1683-S1690.
Narendra M, Bhatracharyulu NC, Padmavathi P, Varadacharyulu NC (2007). Prallethrin induced biochemical changes in erythrocyte membrane and red cell osmotic haemolysis in human volunteers. Chemosphere 67(6):1065-1071.
Nebeker AV, Griffis WL, Stutzman TW, Schuytema GS, Carey LA, Scherer SM (1992). Effects of aqueous and dietary exposure of dieldrin on survival, growth and bioconcentration in mallard ducklings. Environ. Toxicol. Chem. 11(5):687-699.
Ologundudu A, Ologundudu AO, Oluba OM, Omotuyi IO, Obi FO (2010). Effect of Hibiscus sabdariffa anthocyanins on 2,4-dinitrophenyl hydrazine-induced tissue damage in rabbits. J. Toxicol. Environ. Health Sci. 2(1):1-6.
Omotuyi IO, Oluyemi KA, Omofoma CO, Josiah SJ, Adesanya OA, Saalu LC (2006). Cyfluthrin- induced hepatotoxicity in rats. Afr. J. Biotech. 5(20):1909-1912.
Omotuyi IO, Oluyemi KA, Omofoma CO, Josiah SJ, Adesanya OA, Saalu LC (2006). Cyfluthrin- induced hepatotoxicity in rats. Afr. J. Biotech. 5(20):1909-1912.
Paglia DE, Valentine WN (1967). Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. J. Lab. Clin. Met. 70:158-169.
Peltola V, Huhtaniemi I, Ahotupa M (1992). Antioxidant enzyme activity in the maturing rats testis. J. Androl. 13:450-455.
Rasoul MA, Marei GI, Abdelgaleil SA (2012). Evaluation of antibacterial properties and biochemical effects of monoterpenes on plant pathogenic bacteria. Afr. J. Microbiol. Res. 6(15):3667-3672.
Ray DE (1991). Pesticides derived from plants and other organisms. In: Hayes WJ, Jr. & Laws ER (Eds.), Handbook of Pesticide Toxicology, 2:585-593. Toronto: Academic Press.
Rehman H, Ali M, Atif F, Kaur M, Bhatia K, Raisuddin S (2006). The modulatory effect of deltamethrin on antioxidants in mice. Clin. Chim. Acta. 369(1):61-65.
Rolo AP, Palmeira CM (2006). Diabetes and mitochondrial function: role of hyperglycemia and oxidative stress. Toxicol. Appl. Pharmacol. 212(2):167-178.
Sadowaska-Woda I, Wojcik N, Agata K, Bieszczad-bedrejczuk E (2010). Effect of selected antioxidants in cyfluthrin-induced oxidative stress in human erythrocytes in vitro. Toxicol. In-Vitro. 24:879-884.
Sadowaska-Woda I, Wojcik N, Agata K, Bieszczad-bedrejczuk E (2010). Effect of selected antioxidants in cyfluthrin-induced oxidative stress in human erythrocytes in vitro. Toxicol. In-vitro. 24: 879-884.
Satpathy SK, Tyagi PK, Das BS, Srivastava P, Yadav RS (1997). Evaluation of possible toxic effects of cyfluthrin during short-term, relevant community exposue. Environ. Contam. Toxico. 59: 681-687.
Sharma P, Singh R, Jan M (2014). Dose-dependent effect of deltamethrin in testis, liver, and kidney of wistar rats. Toxicol. Int. 21:131-139.
Sinha AK (1972). Colorimetric Assay of Catalase. Anal. Biochem. 47:389-394.
Soderlund DM, Clark JM, Sheets LP, Murlin LS, Piccirillo VJ, Weiner ML (2002). Mechanisms of pyrethroid neurotoxicity: Implications for cumulative risk assessment. Toxicol. 171:3-59.
Soni I, Syed F, Bhatnagar P, Mathur R (2011). Perinatal toxicity of cyfluthrin in mice: developmental and behavioural effects. Hum. Exp. Toxicol. 30(8):1096-1105.
Verma R, Awasthi KK, Soni I, John PJ (2013). Evaluation of Cytogenetic Effects of -Cyfluthrin in Swiss Albino Mice. Int. J. Curr. Microbiol. Appl. Sci. 2(6):30-40.
Yilmaz M, Rencuzogullari E, Canli M (2015). The effects of cyfluthrin on some biomarkers in the liver and kidney of wistar rats. Environ. Sci. Poll. Res. 22(6):4747-4752.
Zini A, Schlegel PN (1996). Catalase mRNA expression in the male rat reproductive tract. J. Androl. 17:473-480.