



Protective effect of *Funalia trogii* crude extract on deltamethrin-induced oxidative stress in rats

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ABSTRACT

In this study the protective effects of cold buffer extract of *Funalia trogii* ATCC 200800 (FtE) and vitamin E (VitE) on oxidative stress induced with deltamethrin using oral administration in rats were investigated. Deltamethrin treatment caused an increase in liver enzyme activities of aspartate transaminase (AST), alanine transaminase (ALT), alkaline phosphatase (ALP) ($p < 0.05$); however, it caused a decrease in activities of catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx), and glutathione reductase (GRd) when compared to control group ($p < 0.05$). Activities of AST, ALT, ALP enzymes and level of thiobarbituric acid reactive substances (TBARS) decreased significantly after VitE administration ($p < 0.05$). Both enzyme activities and TBARS levels were found similar in VitE and FtE treated rats shortly after pesticide administration ($p < 0.05$). In conclusion, it appears that FtE prepared in cold buffer has capability to prevent the liver damage like VitE against the toxic effect of deltamethrin.

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1. Introduction

Deltamethrin, a synthetic pyrethroid type II, is globally used in crop protection and control of malaria and other Vector-borne diseases. The main sources of general population exposure to this pesticide are contaminated food and water, deltamethrin is readily absorbed by the oral route (Barlow, Sullivan, & Lines, 2001). Several studies have demonstrated the genotoxic and immunotoxic effects of deltamethrin in mammalian species (Lukowicz-Ratajczak & Krechniak, 1992). Induction of oxidative stress is one of the main mechanisms of deltamethrin toxicity (Tuzmen, Candan, Kaya, & Demiryas, 2008; Yousef, Awad, & Mohamed, 2006).

Reactive oxygen species (ROS) are constantly formed as by-product of normal metabolic reactions and their formation is accelerated by accidental exposure to occupational chemicals like pesticides. In healthy individuals, the generation of reactive oxygen species is well balanced by the counterbalancing act of antioxidant defences. Hence an imbalance between ROS generation and antioxidant status has been described as oxidative stress (Marubayashi, Dohi, Ochi, & Kawasaki, 1985). The cellular antioxidant pool com-

prises of integral antioxidants like glutathione and other thiols and antioxidant free radical scavenging enzymes like catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx) and glutathione reductase (GRd). Long-term oxidative stress is also expressed as changes in extent of lipid peroxidation (Yu, 1994).

Vitamin E is a naturally occurring antioxidant nutrient that plays an important role in animal health by inactivating harmful free radicals produced through normal cellular activity and various stressors. Also vitamin E inhibits free radical formation for the protection of cells against oxidative damage due to pesticides toxicity (El-Demerdash, Yousef, Kedwany, & Baghdadi, 2004). Yousef et al. (2006) found that deltamethrin (1.28 mg/kg BW) exposure of rats resulted in free radical-mediated liver damage, as indicated and hepatic lipid peroxidation, which was prevented by vitamin E (100 mg/kg BW).

Mushrooms have long been appreciated for their flavour and texture as vegetables and also they provide a wealth of protein (~22%), fibre (~63%), fat (~5%), vitamins (thiamin, riboflavin, niacin, and biotin), and minerals (~10%) (Mattila, Sounpa, & Piironen, 2000). Moreover, mushrooms could accumulate a variety of secondary metabolites including phenolics, steroids, terpenes, poly-peptides. The antioxidative and free radical scavenging properties of phenolics of mushroom extracts have been reported (Mau, Chang, Huang, & Chen, 2004). Hot water extract of *Lentinus edodes*

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and basidiomycetes-X was also found to have high antioxidant activity against lipid peroxidation (Cheung & Cheung 2005). *Funalia trogii* ATCC 200800 has been reported to degrade xenobiotic compounds such as azo, heterocyclic, and reactive dyes (Ozsoy, Unayyar, & Mazmancı, 2005). Antitumor activity of *F. trogii* extract was also showed on mammalian cells (Unayyar et al., 2006).

In an earlier study it was demonstrated the protective role of *F. trogii* on lipid peroxidation generated by deltamethrin in liver, by monitoring the chances of MDA concentration and by electron microscopy observation (Balli et al., 2009). The aim of this study was to investigate the protective effect of cold buffer extract of *F. trogii* ATCC 200800, grown on wheat bran and soybean flour, on deltamethrin-induced oxidative stress in rats. The protective effect of the extract was also compared with that of a well known antioxidant, vitamin E.

2. Materials and methods

2.1. Chemicals

A commercial formulation of the pyrethroid insecticide deltamethrin, [IUPAC name: (S)-alpha-cyano-3-phenoxybenzyl (1R, 3R)-3-(2,2-dibromovinyl)-2,2-dimethylcyclopropan carboxylate and CA name: 1R-[1(S),3]-3-(2,2-dibromoethenyl)-2,2-dimethylcyclopropane carboxylic acid, cyano(3-phenoxyphenyl)methyl ester] DECIS 2.5 EC (deltamethrin 25 g L⁻¹-Bayer) was used. Vitamin E (Dietvit® E, 53% α-tocopherol acetate) was purchased from Merck (Germany). Other chemicals and reagents were purchased from Sigma-Aldrich Chemical Co. and Merck. Wheat bran and soybean flour were supplied from a farm land.

2.2. Preparation of *F. trogii* extracts (FtE)

F. trogii culture was obtained from Environmental Biotechnology Laboratory, Environmental Engineering Department, University of Mersin, Turkey. The fungus was maintained on Potato Dextrose Agar (PDA; Merck) slants and incubated at 30 °C for 5 days and stored at 4 °C.

The solid-substrate fermentation (SSF) medium used for producing the fungal biomass consisted of wheat bran and soybean flour (90:10). The substrate was humidified with a 0.1 M, pH 6.0 sodium phosphate buffer (added at 60% v/w). The humidified medium was placed in 1-L Erlenmeyer flasks and autoclaved at 120 °C for 60 min. The autoclaved medium was inoculated with the fungal stock cultures that had been grown on PDA. The flasks were incubated for 10 days at 30 °C. Then, the contents of the flasks were dried (Sanyo MIR 152 incubator) for 24 h at 40 °C. The dried material was ground in a coffee grinder for 2 min. The ground biomass powder (1 g) was suspended in potassium phosphate buffer (10 mL, 0.1 M, pH 6.0) for 15 min. Solids were removed by centrifugation at 12,000g (Hettich Micro 22R) for 15 min (Unayyar et al., 2006). The supernatant (extract) was sterilized using a 0.22 μm filter and diluted with aforementioned phosphate buffer to desired concentrations. Hot water or alcohols were not used for extraction in any step of this work.

2.3. Specification of FtE

Cold buffer extract of FtE used in this research was analysed and summarised in Table 1. Laccase and peroxidase enzyme activity was determined using ABTS as a substrate. One unit of enzyme activity (U) was defined as the amount of enzyme that formed 1 μmol ABTS per min. Protein concentration (expressed in mg mL⁻¹ FtE) was determined by a modified Lowry method (1951) with bovine serum albumin as a standard protein. Specific

activities (SA) of enzymes were calculated by dividing total enzyme activity to total protein (Unit/mg protein). The yield of extract was found 128 ± 0.08 mg g⁻¹ of dry SSF media.

2.4. Preparation of deltamethrin and vitamin E (VitE)

The tested dose of deltamethrin was adjusted at 1.28 mg/kg BW (1/100 LD₅₀) in corn oil (Worthing, 1983). The dose 100 mg/kg BW of Vitamin E prepared with corn oil was used because previous studies showed that this dose was effective against the toxicity of deltamethrin (Yousef et al., 2006).

2.5. Animals treatment schedule

Twenty healthy adult male Swiss albino Wistar rats (6–8 weeks of age and average body weight 180–200 g) were used in this study. Rats were obtained from the Experimental Animal Centre, University of Mersin, Turkey. The study was approved by the research and ethical committee of the Mersin University. The rats were housed in polycarbonate boxes with steel wire tops and rice husk bedding. They were maintained in a controlled atmosphere of 12 h dark/light cycle, 22 ± 2 °C temperature, and 50–70% humidity, with free access to pelleted feed and fresh tap water. After 2 weeks of acclimation, animals were randomly divided into four groups of six animals each. All groups were fed with standard pellets. Group I (control group) was orally given corn oil. Group II (deltamethrin group) was treated with deltamethrin alone. Group III (deltamethrin + VitE group) was treated with deltamethrin, after 30 min Vit E was given; Group IV (deltamethrin + *F. trogii* group) was treated with deltamethrin after 30 min FtE (50 mg/kg BW) was given. Rats were orally treated at 48 h intervals with repeated doses of deltamethrin, VitE and FtE, for 30 days. At the end of the experiments all rats were sacrificed by decapitation under ketamine (KETALAR-Eczacibasi) anaesthesia. Trunk blood samples were collected from the sacrificed animals placed immediately on ice. Heparin was used as an anticoagulant and plasma samples were obtained by centrifugation at 860g for 20 min. The liver was quickly excised, rinsed in ice-cold 0.175 M KCl/25 mM Tris-HCl (pH 7.4) and homogenised in glass-Teflon homogenizer (Heidolph S01 10R2R0). The liver homogenates were centrifuged at 10,000g for 15 min (Hettich U32) and the supernatants were used for antioxidant enzyme assays and lipid peroxidation determination in spectrophotometer (Perkin-Elmer Lambda EZ210).

2.6. Biochemical analysis

Plasma aspartate transaminase (AST; EC 2.6.1.1), alanine transaminase (ALT 2.6.1.2) and alkaline phosphatase (ALP 3.1.3.1) were assayed using kits approved by the IFCC. In liver SOD (EC 1.15.1.1) activity was measured at 505 nm and 37 °C and was calculated according to the inhibition percentage of formazon formation (McCord & Fridovich, 1969). The catalase (CAT; EC 1.11.1.6) assay, consisting of the spectrophotometric measurement of 10 mM hydrogen peroxide (H₂O₂) breakdown at 240 nm was assayed following the method of Beers and Sizer (1952). GPx catalyses the oxidation of GSH to GSSG by H₂O₂. The rate of GSSG formation was then measured by following a decrease in absorbance of the reaction mixture containing NADPH and glutathione reductase at 37 °C and 340 nm as NADPH is converted to NADP. t-Butyl hydroperoxide was used as a substrate. GRd (EC 1.8.1.7) activity was assayed by the oxidation of NADPH by GSSG at 37 °C and 340 nm (Beutler, 1984). GPx activity (EC 1.11.1.9) was determined according to the method of Beutler (1984). TBARS, as a marker for LPO, were measured at 532 nm by using 2-thiobarbituric acid (2,6-dihydroxypyrimidine-2-thiol; TBA) (Ohkawa, Ohishi, & Tagi, 1979).

Table 1

Analysis results of cold buffer extract of fungus grown on SSF process.

	<i>Funalia trogii</i> extract	Methods
Laccase	58 SA	Majcherczyk, Johannes, and Huttermann (1999)
Peroxidase	71 SA	Keesey (1987)
Protein	2.53 mg	Lowry, Rosenbrough, Farr, and Randall (1951)
Phenol	26.30 µg	Quettier-Deleu et al. (2000)

SA: specific activity of enzymes in FtE (Unit/mg protein).

Specific activity of enzymes is defined as units of activity per mg of protein (Unit/mg protein).

2.7. Statistical analysis

Statistical analysis was performed using SPSS 12.0 for Windows (SPSS, Chicago, IL). Data (mean \pm SE) were compared using one-way analysis of variance (ANOVA). Data significant at $p < 0.05$ in ANOVA were further analysed by Least Significant Differences (LSD) multiple comparison test to determine statistical difference between groups ($p < 0.05$).

All results were statistically compared with group I and group II separately. In the statistical analysis symbol “*” gives the difference between group I (control group) and the other groups. The symbol “a” gives the difference between group II (deltamethrin) and the other groups (group III and group IV).

3. Results

Cold buffer extract of FtE used in this research was analysed and specific activity of laccase, peroxidase and protein, phenol contents were summarised in Table 1.

Table 2 shows ALT, AST and ALP enzyme activities of liver which increased significantly ($p < 0.05$) after deltamethrin treatment in group II. Following treatment with VitE in group III and FtE in group IV, enzyme activities also increased ($p < 0.05$) when com-

Table 2

Changes of liver enzymes in control and experimental groups.

Group	AST (UI^{-1})	ALT (UI^{-1})	ALP (UI^{-1})
Group I	83.2 \pm 4.37	50.9 \pm 0.2	119 \pm 13.0
Group II	154 \pm 14.0*	65.2 \pm 2.03*	193 \pm 18.3*
Group III	107 \pm 3.74**	56.7 \pm 0.7**	167 \pm 8.2**
Group IV	104 \pm 1.01***	58.2 \pm 0.3***	154 \pm 3.4***

Values are mean \pm SE from six rats from each group.

Group I, control; group II, deltamethrin alone; group III, deltamethrin + VitE; and group IV, deltamethrin + FtE.

AST: aspartate transaminase; ALT: alanine transaminase; and ALP: alkaline phosphatase.

* Significantly different from group I ($p < 0.05$).** Significantly different from group II ($p < 0.05$).**Table 3**

Antioxidant enzyme activities in liver of experimental groups.

Group	CAT (Unit/mg prot)	SOD (Unit/mg prot)	GRd (Unit/mg prot)	GPx (Unit/mg prot)
Group I	1.94 \pm 0.02	0.6 \pm 0.06	0.74 \pm 0.03	1.71 \pm 0.22
Group II	1.42 \pm 0.05*	0.10 \pm 0.00*	0.37 \pm 0.00*	0.85 \pm 0.08*
Group III	1.64 \pm 0.04**	0.16 \pm 0.00*	0.49 \pm 0.01**	1.07 \pm 0.03*
Group IV	1.64 \pm 0.04**	0.15 \pm 0.01*	0.51 \pm 0.02**	1.09 \pm 0.03*

Values are mean \pm SE from six rats from each group.

Group I, control; group II, deltamethrin alone; group III, deltamethrin + VitE; and group IV, deltamethrin + FtE.

CAT: catalase; SOD: superoxide dismutase; GPx: glutathione peroxidase; and GRd: glutathione reductase.

* Significantly different from Group I ($p < 0.05$).** Significantly different from Group II ($p < 0.05$).

pared to group I, but these activities were found significantly ($p < 0.05$) lower than group II. No such significant difference between group III and IV enzyme activities was observed, however significant difference between group II and group III, IV was observed. According to our results it can be said that VitE and *F. trogii* decreased the effects of deltamethrin on liver enzymes.

Table 3 shows CAT, SOD and GSH-dependent antioxidant enzymes, GRd and GPx, in the liver of the control and experimental rats. CAT, SOD, GRd and GPx enzyme activities significantly ($p < 0.05$) decreased in all experimental groups as compared with control group (group I). However in the group III and group IV the antioxidant enzyme activities were found higher than group II. Following treatment with VitE and FtE after 30 min of deltamethrin administration, CAT and GRd enzyme activity significantly ($p < 0.05$) increased in group III and IV as compared with group II. However, there were no significant differences between SOD and GPx enzyme activities in group III and IV.

The extent of lipid peroxidation of control, deltamethrin and deltamethrin + VitE was investigated (Table 4). Lipid peroxidation was significantly ($p < 0.05$) increased on deltamethrin group when compared to group I ($p < 0.05$). VitE and FtE treatment after deltamethrin significantly decreased the TBARS concentrations when compared to group II ($p < 0.05$). It can be said that VitE and *F. trogii* decreased the deleterious effects of deltamethrin in lipid peroxidation.

4. Discussion

Lipid peroxidation associated with reduction of antioxidants is a characteristic observation in the tissues and malondialdehyde is used as an indicator of tissue damage (Ohkawa et al., 1979). On the other hand, not only reduction of antioxidants but also increase of radicals, such as the trichloromethyl radical generated by biochemical reactions, could enhance the malondialdehyde level and these radicals may react with oxygen to indicate lipid peroxidation (Jayakumar, Sakthivel, Thomas, & Geraldine, 2008). Earlier study has reported the effect of *F. trogii* extract on lipid peroxidation generated by deltamethrin in liver (Balli et al., 2009). They found significant decrease in MDA concentration in extract treated rats shortly after treating with deltamethrin. In this study, similar results were found and significant increase was observed in TBARS level of group II (only deltamethrin). The level of TBARS significantly decreased shortly after VitE treatment when compared to the control group.

Formation of free radicals is prevented by antioxidants which block some of destructive reactions in the cell. Some antioxidant rich extracts have been shown to protect against hepatotoxicity, as indicated by a decrease in the level of MDA (Lee et al., 2004). The extracts of mushroom *Ganoderma lucidum* and *Pleurotus ostreatus* have been demonstrated to reduce level of MDA in CCl₄ treated rats (Jayakumar et al., 2008; Lin & Lin, 2006). The observed reduction in the TBARS level in the liver of rats following treatment

Table 4

TBARS concentrations in control and experimental groups.

Group	TBARS (nmol/mg prot)
Group I	0.88 ± 0.04
Group II	1.92 ± 0.14 ^{*,**}
Group III	1.19 ± 0.06 ^{*,**}
Group IV	1.30 ± 0.06 ^{*,**}

Values are mean ± SE from six rats from each group. Group I, control; group II, deltamethrin alone; group III, deltamethrin + VitE; and group IV, deltamethrin + FtE. TBARS, thiobarbituric acid reactive substances.

* Significantly different from group I ($p < 0.05$).

** Significantly different from group II ($p < 0.05$).

with cold buffer extract suggests the protective potential of *F. trogii* grown in SSF media.

Reduced activities of antioxidant enzymes (CAT, SOD, GRd and GPx) after treatment of pesticides are important indicators for the toxicity of these chemicals (Jayakumar, Ramesh, & Geraldine, 2006). In the present study, significantly changes of these enzymes were observed in all groups when compared to control group. The lowered enzyme levels may reflect to increased oxidative damage (Srinivasan, Rukkumani, Ram Sudheer, & Menon 2005). Vitamin E is a well known, non-enzymatic scavenger of free radicals that protects cell membranes from peroxidative damage (Macdonald-Wicks & Garg, 2003). However, some antioxidant-rich extracts such as those of *Platycodi radix* (Lee & Jeong, 2002) and *P. ostreatus* (Jayakumar et al., 2008) increased the GSH levels. Thus, significant increase in antioxidant enzyme levels in group III (deltamethrin + VitE) and IV (deltamethrin + FtE) when compared to group II (only deltamethrin) suggests protective potential of FtE relatively similar to Vit E.

Pesticide-induced increase in plasma ALT, AST and ALP activities can be attributed to the effect of deltamethrin on liver cell membrane and thus can cause liver dysfunction (El-Demerdash et al., 2004; Yousef et al., 2006). An earlier study showed a correlation between enzyme leakage and cell damage (Awad, Abdel-Rahman, & Hassan, 1998). The present results show that the activities of ALT, AST and ALP significantly decreased in group III and IV when compared to group II. The results indicate antioxidant or free radical removing action of FtE, as well as VitE, which played an important role in reducing the toxicity and preserving liver membrane integrity in rats treated with deltamethrin.

In conclusion, the oral administration of deltamethrin to rats was found to significantly increase the TBARS, ALT, AST and ALP levels, but significantly decreased CAT, SOD, GRd and GPx enzyme activities. The post-treatment with vitamin E after deltamethrin administration prevented liver injury when compared to group II. Our results demonstrate that treatment with cold water extract of *F. trogii* shortly after deltamethrin treatment leads to a significant revival of the oxidative status, like VitE, which confirms the protective effect of FtE. Further molecular studies should be focused to find the individual components present in the extract.

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