

Effects of T-2 Toxin, Zeolite and Mycosorb on Antioxidant Systems of Growing Quail

J. E. Dvorska¹ and P. F. Surai*

Avian Science Research Centre, Scottish Agricultural College, Auchincruive, Ayr, KA6 5HW, Scotland, UK

ABSTRACT : The present study was conducted to assess the dietary effect of T-2 toxin on the antioxidant systems of the liver in growing quail and to comparatively evaluate the protective properties of two different mycotoxin-adsorbent additives, Mycosorb and zeolite, in preventing inhibition of the antioxidant system. Four groups of 4 day old quail were formed with 20 birds in each group. The birds were maintained on the floor for the course of the study. The three treatment diets consisted of the basal diet with T-2 toxin added in the form of *Fusarium sporotrichioides* culture (8.1 mg/kg feed), T-2 toxin (8.1 mg/kg) plus zeolite (30 g/kg feed), and T-2 toxin (8.1 mg/kg) plus Mycosorb (1 g/kg feed). After 30 days of feeding (34 days old) all birds were sacrificed and liver samples for biochemical analyses were collected from five quail in each of the four groups. Antioxidant concentrations were evaluated by HPLC-based methods. Inclusion of T-2 toxin in the quail diet was associated with a significant ($p < 0.05$) decrease in concentrations of all forms of antioxidants studied, including α - and γ -tocopherols, ascorbic acid, retinol and retinyl esters. At the same time, liver susceptibility to lipid peroxidation significantly ($p < 0.05$) increased. Inclusion of zeolite in the quail diet at the level of 3% was ineffective in preventing antioxidant depletion in the liver by mycotoxicosis. In contrast, Mycosorb in the diet at a 0.1% level was able to significantly inhibit liver antioxidant depletion and as a result decreased lipid peroxidation in the liver. Concentrations of all forms of antioxidants studied were significantly higher in the livers of the quails fed the basal and T-2 toxin/Mycosorb combination in comparison to birds fed the basal with T-2 toxin alone. (*Asian-Aust. J. Anim. Sci.* 2001. Vol 14, No. 12 : 1752-1757)

Key Words : T-2 Toxin, Antioxidants, Vitamin A, Mycosorb, Zeolite

INTRODUCTION

Mycotoxin contamination of various feed and food commodities is a global problem. More than 300 mycotoxins have been characterised (Fink-Gremmels, 1999); this number is growing quickly. The trichothecene group of mycotoxins accounts for over one hundred fungal metabolites, of which T-2 toxin, produced by the *Fusarium fungus*, was the first to be studied (Leeson et al., 1995; Bondy and Pestka, 2000). The adverse effects of trichothecene toxin on animal health is expressed in a diverse range of symptoms including skin lesions, immunosuppression, hepatotoxicity, nephrotoxicity, neurotoxicity, genotoxicity and even death (Hollinger and Ekperigin, 1999). The damage caused by mycotoxins results primarily from the toxin's interruption of cell division activity in bone marrow, immunocompetent organs and intestinal mucosa (Bondy and Pestka, 2000), resulting in a serious immunosuppressive effect (Bondy and Pestka, 2000; Pitt, 2000). T-2 toxin also has a strong inhibitory effect on protein synthesis, which in turn results in the inhibition of DNA and RNA synthesis (Leeson et al., 1995).

The mechanisms of T-2 toxicity include effects on other metabolic functions of the body. For example, the chemical

structure of T-2 toxin makes it fat soluble, which allows it to be incorporated into cell membranes, potentially changing membrane structural and functional properties (Coulombe, 1993). Lipid peroxidation by T-2 toxin in the liver has also been identified as an important underlying mechanism of T-2 toxin-induced cell injury (Leeson et al., 1995; Hoehler and Marquardt, 1996) and DNA damage (Atroshi et al., 1997). While a variety of different strategies to combat mycotoxicosis have been developed, the most promising strategies are based on the addition of adsorbents to contaminated feed. The adsorbent material selectively binds mycotoxins during digestion, preventing the absorption from the gastrointestinal tract, thereby decreasing toxic effects (Ledoux and Rottingham, 1999; Devegowda et al., 1998). Of several different adsorbent additives studied, an esterified glucan (Mycosorb, Alltech, Inc. USA) has been shown to be the most effective (Devegowda et al., 1998).

The aim of the present work was two-fold. The first was to study the dietary effect of T-2 toxin on the antioxidant systems of the liver in growing quail. The second was to comparatively evaluate the protective properties of two different mycotoxin-adsorbent additives, Mycosorb and zeolite, in preventing inhibition of the antioxidant system.

MATERIALS AND METHODS

Quail

Four groups of 4 day old quail were formed with 20 birds in each group. The birds were maintained on the floor

* Address reprint request to P. F. Surai. Tel: +44-1292-525-160, fax: +44-1292-525177. E-mail: P.Surai@au.sac.ac.uk

¹ Department of Veterinary Medicine, Sumy State Agrarian University, Sumy, Ukraine.

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during the course of the study. The quail received a maize-based basal diet (240 g/kg crude protein, 12.1 MJ ME/kg; and (g/kg) yellow maize 435, barley 44, soyabean meal 341, sunflower seed meal 55, fish meal 51, vegetable oil 50, limestone flour 11, sodium chloride 5, mineral premix 3.5, vitamin premix 1.0, methionine 1.0) formulated to meet the NRC (1994) requirements for all nutrients. The three treatment diets consisted of the basal diet with T-2 toxin added in the form of *Fusarium sporotrichioides* culture (8.1 mg/kg feed), T-2 toxin (8.1 mg/kg) plus zeolite (30 g/kg feed), and T-2 toxin (8.1 mg/kg) plus Mycosorb (Mycosorb is a trademark of Alltech, Inc. that has been registered with the U.S. Trademark Office, 1 g/kg feed). Temperature and all other environmental conditions were maintained in accordance with existing norms for the facility. After 30 days of feeding (34 days old) all birds were sacrificed and samples for biochemical analyses were collected from five quail in each of the four groups. The livers were collected and immediately frozen in liquid nitrogen.

Analytical procedures

Liver vitamin E (α - and γ -tocopherols) levels were analysed by saponifying liver tissue with ethanolic KOH in the presence of pyrogallol as previously described in Surai et al. (1996).

Total carotenoids were determined in the liver as described by Surai and Speake (1998). Briefly, 2 ml of tissue homogenate (20% in 0.01 M phosphate buffer, pH 7.4) were mixed with 2 ml of ethanol. Hexane (5 ml) was then added and the mixture was shaken vigorously for 5 minutes. The hexane phase, containing the carotenoids, was separated by centrifugation and collected. The extraction was repeated two additional times each using 5 ml of hexane. The hexane extracts were then combined and the total aggregated carotenoids were determined spectrophotometrically at 446 nm using lutein as the calibration standard.

Retinyl esters were determined by HPLC-based methods as described by Furr et al. (1986) with minor modifications (Surai et al., 2000). In this modified technique, tissues (0.2-0.5 g) were homogenised in 2 ml of 1:1 (v/v) mixture of 5% NaCl solution and ethanol. After adding 3 ml hexane the mixture was further homogenised for 3 minutes. After centrifugation, the hexane extract was collected. This extraction process was repeated twice. The hexane extracts were then combined and evaporated under nitrogen gas. The extract residue was dissolved in 1 ml of acetonitrile:methanol:dichloromethane (0.5:0.25:0.25, v/v/v), centrifuged, and the retinol, retinyl ester and carotenoid levels determined from the supernatant by injecting it into the HPLC system (autosampler; Shimadzu, Japan). This HPLC system included an isocratic pump (Spectra System P100, Spectra-Physics Analytical) fitted with a Spherclone

Type ODS (2), 3 μ C18 reverse phase HPLC column, 15 cm \times 4.6 mm (Phenomex, UK), a Security Guard cartridge system (Phenomex, UK), Programme wavelength detector (Spectra-Physics, San Jose, California) and a Shimadzu integrator. Chromatography was performed using a mobile phase of acetonitrile/dichloromethane (80:20) at a flow rate of 1.5 ml/min. UV detection of retinol and retinyl esters was performed at 325 nm. Standard solutions of retinyl palmitate, retinyl oleate, retinyl stearate and retinyl linoleate in mobile phase were used for instrument calibration.

Using the same extract, free retinol was determined with the HPLC system (Shimadzu Liquid Chromatograph, LC-10AD, Japan Spectroscopic Co. LTD; JASCO Intelligent Spectrofluorometer 821-FP, Spherisorb, type S30DS2, 3 μ C₁₈ reverse phase HPLC column, 15 cm \times 4.6 mm; Phase Separations Limited, UK). Chromatography was performed using a mobile phase of methanol:water (97:3, v/v) at a flow rate of 1.05 ml/min. Fluorescence detection of retinol involved excitation and emission wavelengths of 330 and 480 nm, respectively (Surai et al., 2000). A standard solution of retinol in mobile phase was used for instrument calibration.

Ascorbic acid was determined by HPLC-based method described by Mori et al. (1998). Tissue was homogenised in 3% metaphosphoric acid - 8% acetic acid mixture (1:1) and precipitated proteins were removed by centrifugation. Ascorbic acid was determined from the supernatant by HPLC after oxidation by 2,6-dichlorophenolindophenol and derivatisation of dehydro-ascorbic acid with 4,5-dimethyl-o-phenylenediamine. The HPLC's fluorimetric detection used an excitation wavelength of 360 nm and an emission wavelength of 440 nm. The column was the same as used for vitamin E, while using a mobile phase of methanol-0.1% methaphosphoric acid (7:3 by volume).

Tissue susceptibility to lipid peroxidation was determined measuring thiobarbituric acid reactive substances (TBARS) accumulation as a result of Fe-stimulated lipid peroxidation, as previously described in Surai and Sparks (2000). Results were expressed as μ g malondialdehyde (MDA) equivalents per g of fresh tissue using 1, 1, 3, 3-tetramethoxypropane as the standard for calibration.

Statistical analysis

Results are presented as mean of measurements on tissue samples from five quail per treatment. Statistical analysis was performed by one-way ANOVA and means were separated using Student's t-test.

RESULTS

The antioxidant profile for the quail liver is shown in table 1. Inclusion of T-2 toxin in the quail diet significantly

Table 1. Antioxidants in the quail liver, $\mu\text{g/g}$ ($n=5$)

Antioxidant	Control	T-2 toxin	T-2+Zeolite	T-2+Mycosorb
α -tocopherol	18.4 \pm 1.33 ^a	10.2 \pm 0.96 ^b	11.2 \pm 1.03 ^b	14.6 \pm 0.99 ^c
γ -tocopherol	1.9 \pm 0.17 ^a	1.0 \pm 0.09 ^b	1.2 \pm 0.15 ^{bc}	1.5 \pm 0.07 ^{ac}
Carotenoids	4.3 \pm 0.13 ^a	2.7 \pm 0.18 ^b	3.1 \pm 0.30 ^{cb}	3.6 \pm 0.16 ^{cd}
Ascorbic acid	166.4 \pm 8.6 ^a	101.1 \pm 5.1 ^b	111.1 \pm 4.8 ^b	150.6 \pm 8.0 ^a

Values are mean \pm SE of measurements from the tissues from 5 quails.

Values within a row that do not share a common letter are significantly different ($p<0.05$).

decreased the concentrations of all forms of antioxidants studied ($p<0.05$). It appeared from data that α -tocopherol and γ -tocopherol concentrations were decreased by almost 50%, the carotenoid level was reduced by 37% and ascorbic acid concentration decreased by 39%.

Inclusion of zeolite in the quail diet (Group 3) at the level of 3% was ineffective in preventing antioxidant depletion in the liver by mycotoxicosis (table 1). In contrast, Mycosorb in the diet at a 0.1% level was able to significantly inhibit liver antioxidant depletion. Concentrations of all forms of antioxidants studied were significantly higher in the livers of the quails fed the basal and T-2 toxin/Mycosorb combination (Group 4) in comparison to birds fed the basal with T-2 toxin alone (Group 2). While Mycosorb was not able to completely restore the concentrations of all forms of liver antioxidants, in terms of concentrations of ascorbic acid and γ -tocopherol concentrations in the liver, the Mycosorb treatment group did not differ ($p>0.05$) from the basal diet control group (Group 1).

T-2 toxin had a profound effect on vitamin A accumulation in the liver. As a result of including T-2 toxin in the growing quail's diet, concentrations of six different forms of vitamin A were significantly decreased, retinol (31%, $p<0.05$), retinyl stearate (48%, $p<0.001$), palmitate (44%, $p<0.01$), oleate (36%, $p<0.01$), linoleate (66%, $p<0.001$) and total vitamin A (45%, $p<0.001$). Zeolite was minimally effective in preventing liver vitamin A depletion as a result of T-2 toxicosis. Only the level of retinyl linoleate was significantly ($p<0.05$) higher in the liver of the quail fed the T-2 toxin/zeolite diet combination (Group 3) compared to the quail fed T-2 toxin alone (Group 2).

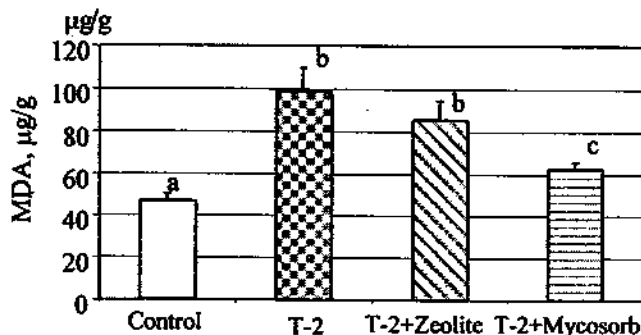
In contrast, Mycosorb supplementation completely restored concentrations of the retinol, retinyl palmitate, retinyl oleate and retinyl linoleate forms of vitamin A (table 2). Only concentrations of retinyl stearate ($p<0.01$) and total vitamin A ($p<0.05$) in the liver of quail from the T2 toxin/Mycosorb treatment group (Group 4) were lower than those of the control; however, liver concentrations of the measured forms of vitamin A in the Mycosorb treatment were significantly higher than those found in the livers of quail fed the basal plus T-2 toxin diet.

As a result of antioxidant depletion in the liver, susceptibility to lipid peroxidation increased more than two-

fold (figure 1). Inclusion of zeolites in the diet did not prevent antioxidant depletion; and as a result the susceptibility to lipid peroxidation in the liver was increased, showing no significant difference from the group fed the T-2 toxin treatment without an adsorbent additive. On the other hand, the inclusion of Mycosorb in the T-2 adulterated diet significantly ($p<0.05$) decreased tissue susceptibility to lipid peroxidation in comparison to diets containing toxin only, although the inclusion of the Mycosorb adsorbent material was unable to completely mitigate the powerful stimulating effect of T-2 toxin on lipid peroxidation. Therefore, MDA accumulation in the livers of the quail in the Mycosorb group (4) was still significantly ($p<0.05$) higher than MDA levels in the control group.

DISCUSSION

Oxidative damage caused by T-2 toxin may be one of the underlying mechanisms for T-2 toxin-induced cell injury and DNA damage, which eventually leads to tumourigenesis. Our data clearly indicate increased lipid peroxidation in quail liver as a result of T-2 toxin consumption. These data are in agreement with other observations on the stimulating effect of trichothecenes on lipid peroxidation in avian tissues. The most sensitive species were geese, followed by ducks and chickens, while



Values are means of measurement from 5 livers.

Values that do not share a common letter are significantly different ($p<0.05$).

Figure 1. Lipid peroxidation in the quail liver, μg MDA/g fresh tissue

Table 2. Vitamin A in the quail liver, $\mu\text{g/g}$ (n=5)

Antioxidant	Control	T-2 toxin	T-2+Zeolite	T-2+Mycosorb
Retinol	3.02± 0.21 ^a	2.07± 0.27 ^b	2.34± 0.26 ^b	3.01± 0.27 ^a
Retinol-stearate	13.45± 1.09 ^a	6.92± 0.69 ^b	6.27± 0.54 ^b	9.61± 0.29 ^c
Retinol-palmitate	27.81± 2.36 ^a	15.48± 1.17 ^b	18.59± 1.74 ^{bc}	21.80± 2.22 ^{ac}
Retinol-oleate	4.81± 0.42 ^a	3.04± 0.28 ^b	3.13± 0.43 ^b	5.04± 0.31 ^a
Retinol-linolate	3.72± 0.43 ^a	1.26± 0.21 ^b	2.15± 0.20 ^c	3.01± 0.34 ^a
Total A	52.81± 3.11 ^a	28.77± 1.49 ^b	32.47± 2.38 ^b	42.07± 2.12 ^c

Values are mean±SE of measurements from the tissues from 5 quails.

Values within a row that do not share a common letter are significantly different ($p < 0.05$).

the most sensitive tissues were the liver, blood plasma, and red blood cells (Mezes et al., 1999). Leal et al. (1999) found that after 7 days of toxin treatment (1.5 mg T-2 toxin/kg body weight/day) hepatic MDA concentration in male broiler chicks increased 128%, while glutathione concentration decreased. However, Hoehler and Marquardt (1996) found that T-2 toxin was not always effective in stimulating lipid peroxidation in chickens. This finding could be attributed to differences in dietary composition, levels of antioxidants and mycotoxins in the diet, or other unidentified factors.

At present, it is not clear whether T-2 toxin stimulates lipid peroxidation directly by enhancing free radical production, or the increased susceptibility of tissues to peroxidation is a reflection of a compromised antioxidant system. For the quail in this experiment, the levels of the primary liver antioxidants (α -tocopherol, γ -tocopherol, carotenoids and ascorbic acid) were significantly decreased as a result of T-2 toxin consumption. Other studies showed similar findings. Hoehler and Marquardt (1996) found that the presence of T-2 toxin in the diet decreased the concentration of α -tocopherol in chicken liver, while Coffin and Combs (1981) showed that T-2 toxin consistently depressed concentrations of vitamin E in chicken plasma. However, the addition of micelle-promoting compounds (taurocholic, monoolein, and oleic acids) alleviated the reduction of plasma vitamin E, indicating that T-2 toxin interferes with micelle formation during vitamin E absorption. Therefore, it is possible that the decreased level of vitamin A in the liver is a reflection of the decreased intestinal absorption of fat-soluble nutrients due to T-2 toxin in the feed. Decreased antioxidant absorption from the diet is just one of the possible mechanisms of antioxidant depletion from the tissues.

Another possible cause of antioxidant depletion stems from an overproduction of free radicals in the liver as it has been observed in other animal species fed T-2 toxin. For example, in rat liver T-2 toxin has been shown to be involved in the generation of free radicals, which increased lipid peroxidation (Suneja et al., 1989). Similarly, Rizzo et al. (1994) demonstrated that T-2 toxin stimulated lipid peroxidation and increased MDA accumulation in rats by

268%, with a consequent decrease of GSH content. Treatment of fasted mice with a single dose of T-2 toxin (1.8 or 2.8 mg/kg body weight) by oral gavage led to 76% hepatic DNA fragmentation and a marked decrease in hepatic glutathione levels (Atroshi et al., 1997). Ahmed and Ram (1986) administered T-2 mycotoxin to rats (1.25 mg/kg) orally for five days causing an increase in lipid peroxidation in hepatic nuclei (ascorbate-induced, as well as NADPH-dependent), while the activity of liver glutathione-S-transferase (EC. 2.5.1.18) was decreased. Segal et al. (1983) found that, like T-2 toxin, deoxynivalenol stimulated lipid peroxidation in rat liver. Yeast studies yielded data demonstrating that T-2 toxin stimulated lipid peroxidation in a biological system due to increased generation of hydroxyl radicals (Hoehler et al., 1998). Male rats treated with a sublethal dose (2 mg/kg body weight) of the trichothecene roridin E showed a significant increase in lipid peroxidation in the liver (Omar et al., 1997).

Lipid peroxidation is associated with other forms of mycotoxicoses, including ochratoxicosis (Hoehler and Marquardt, 1996; Hasinoff et al., 1990), fumonisin B1 toxicosis (Abel and Gelderblom, 1998; Abado-Becognee et al., 1998), aflatoxin B1 toxicosis (Shen et al., 1994; Harvey et al., 1994), aurofusarintoxicosis (Dvorska et al., 2001; Surai and Dvorska, 2001) and citrinin toxicosis (Ribeiro et al., 1997).

Since lipid peroxidation plays an important role in mycotoxin toxicity, it is expected that antioxidants in the diet will provide a protective effect. The results from a number of experiments in a variety of animal species provide support for this argument. For example, vitamin E supplementation ameliorated the prooxidative effects of ochratoxin A in chickens (Hoehler and Marquardt, 1996) and mice (Grosse et al., 1997). The protective effect of vitamin E was also found in T-2 toxicoses in rats (Rizzo et al., 1994), in mice (Atroshi et al., 1997) and in a cell-line *in vitro* system (Shokri et al., 2000). Vitamin E can also protect against both fumonisin B1 (Mobio et al., 2000; Abel and Gelderblom, 1998) and zearalenone (Grosse et al., 1997).

Our data indicate that zeolites were not effective in prevention of toxic effects of T-2 toxin. These data are in

agreement with observations of Kubena et al. (1990, 1998) indicating absence of protective effect of aluminosilicate sorbents against T-2 toxicoses. Superactivated charcoal (Edrington et al., 1997) and organic sorbents (Bailey et al., 1998) were also ineffective against T-2 toxicosis. In contrast, inclusion of esterified glucomannans (Mycosorb) in the diet of quail containing T-2 toxin significantly slowed the depletion of natural antioxidants in the liver, while also significantly decreasing lipid peroxidation. Mycosorb's protective effects are attributed to the high adsorbent capability that esterified glucomannans have for T-2 (and other) mycotoxins (Dawson, 2001). However, inclusion of Mycosorb in the quail diet was unable to completely prevent the adverse effects of T-2 toxin on the antioxidant systems of the liver of the growing quail. Results of this study are consistent with Dawson's (2001) findings and conclusions that Mycosorb's T-2 toxin adsorbent capacity is about 33.4%.

CONCLUSION

The principal finding from this research is that a yeast cell wall-derived glucomannan product, Mycosorb, has a significant protective effect against T-2 toxicosis by shielding the antioxidant systems of the quail liver from toxin damage. Additional research into the relationship between different forms of mycotoxicosis, antioxidant status in the liver, and nutritional supplementation with various forms of dietary antioxidants will provide greater insight into this fascinating and important area of animal health and nutrition.

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