Protective Effect of Modified Glucomannans against Changes in Antioxidant Systems of Quail Egg and Embryo due to Aurofusarin Consumption

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ABSTRACT : The aim of this study was to evaluate effects of modified glucomannan (Mycosorb[TM]) on the antioxidant profile of egg yolk and tissues of newly hatched quail after aurofusarin inclusion in the maternal diet. Fifty-four 45 day-old Japanese quail were divided into three groups and were fed a corn-soya diet balanced in all nutrients *ad libitum*. The diet of the experimental quail was supplemented with aurofusarin at the level of 26.4 mg/kg feed in the form of *Fusarium graminearum* culture enriched with aurofusarin or with aurofusarin plus Mycosorb[TM] at 1 g/kg feed. Eggs obtained after 8 weeks of feeding were analysed and incubated in standard conditions of 37.5°C/55% RH. Samples of quail tissues were collected from newly hatched quail. The main carotenoids, retinol, retinyl esters and malondialdehyde were analysed by HPLC-based methods. Inclusion of aurofusarin in the maternal diet was associated with decreased carotenoid and vitamin A concentrations in egg yolk and liver of newly-hatched quail. Furthermore, lipid peroxidation in quail tissues was enhanced. Inclusion of modified glucomannan (Mycosorb[TM]) in the toxin-contaminated diet provided a significant protective effect against changes in antioxidant composition in the egg yolk and liver. It is suggested that a combination of mycotoxin adsorbents and natural antioxidants could be the next step in counteracting mycotoxins in animal feed. *(Asian-Aust. J. Anim. Sci. 2004. Vol 17, No. 3 : 434-440)*

Key Words : Aurofusarin, Quail, Mycosorb, Carotenoids, Vitamin A, Lipid Peroxidation

INTRODUCTION

Mycotoxin contamination of feed and food ingredients is a worldwide problem. The number of mycotoxins that have been shown to induce signs of toxicity in mammalian and avian species exceeds 300 and is steadily increasing (Leeson et al., 1995; Fink-Gremmels, 1999). It has been estimated that 25% of the world's cereal grains are contaminated with mycotoxins (Fink-Gremmels, 1999).

Acute mycotoxicosis outbreaks are rare events in modern poultry production. However, low mycotoxin doses that may go undetected are often responsible for reduced efficiency and greater susceptibility to infectious disease. The problem is made even more difficult by the fact that many mechanisms of mycotoxin action at the molecular level have not been fully elucidated. Biochemical changes in mycotoxicosis vary greatly but oxidative stress in combination with apoptosis is regarded as an important mechemnism of mycotoxin action (Surai, 2002). In fact, aflatoxins, ochratoxins, T-2 toxin, vomitoxin, fumonisins and zearalenone are involved in promotion of lipid peroxidation.

A recently discovered mycotoxin called aurofusarin (a pink pigment produced by *Fusarium graminearum*; Kotyk, 1999) has been shown to compromise antioxidant system activity and stimulate lipid peroxidation (Dvorska et al., 2001; 2002). Furthermore, aurofusarin was shown to alter

egg yolk colour with no clinical signs of toxicosis (Kotyk et al., 1990; 1995; Dvorska, 2001). On the other hand, aurofusarin compromised immune function of laying chickens (Sakhatsky, 1999) and quail (Dvorska et al., 2001) and decreased fertility and hatchability.

Our previous study (Dvorska and Surai, 2002) showed a protective effect of modified glucomannan against T-2 toxin toxicosis in quail. In particular, protective effects were associated with prevention of antioxidant depletion in quail tissues. Similarly, modified glucomannans prevented changes in fatty acid composition and vitamin E level in quail egg and tissues due to aurofusarin consumption (Dvorska et al., 2003).

The aim of the present work was to further study protective effects of modified glucomannan (Mycosorb[TM]) on the antioxidant systems in quail egg yolk and tissues of newly hatched quail after aurofusarin inclusion into the maternal diet.

MATERIALS AND METHODS

Quail

Fifty-four 45 day-old Japanese quail (*Coturnix Japonica*) were divided into three groups (each group consisted of three quail families with 5 females and one male in each of them); control and two experimental groups. Each quail family was kept in separate sections and was fed a corn-soya diet balanced in all nutrients *ad libitum* (Table 1). The diet of the experimental quail was supplemented with aurofusarin at 26.4 mg/kg feed in the form of *Fusarium graminearum* culture enriched with aurofusarin

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Table 1. Quail diet composition

Ingredient	g/kg	Ingredient	g/kg
Yellow corn	528	Sodium chloride	4
Corn gluten (60% CP)	50	Choline chloride	2
Soyben meal (50% CP)	300	Limestone	60
Trace mineral mix[1]	5	Dicalcium phosphate	20
Vitamin mix[2]	10	DL-methionine	1
Corn oil	20		
Calculated analysis			
ME, MJ/kg	12.13	Methionine	5.2
Protein	223	Cystine	3.5
Lysine	11.3	Calcium	28.5

[1] Provided (in milligrams per kilogram of diet): $FeSO_4 \cdot 7H_2O$, 330; $MnSO_4 \cdot H_2O$, 330; KI, 2.6; $CuSO_4 \cdot 5H_2O$, 16.7; $ZnCO_3$, 115; $Na_2SeO_3 \cdot 5H_2O$, 0.66.

[2] Provided (in milligrams per killogram of diet): niacin, 50; Ca pantotenate, 20; pyridoxine HCl, 4.5; folic acid, 4; menadione, 1.5; biotin, 0.2; thiamine, 11; riboflavin, 11; cyanocobalamin, 0.02; retinyl acetate, 3.44; cholecalciferol, 0.0375; tocopheryl acetate, 35.

(a gift from Dr. Kotyk, Poultry Research Institute, Borky, Ukraine; Kotyk and Trufanova, 1998) or with aurofusarin plus Mycosorb™ (trademark of Alltech, Inc. USA, 1 g/kg feed). It was confirmed that the culture contained exclusively aurofusarin. In particular, spectral analyses of extracted aurofusarin revealed three major peaks at 248, 265 and 345 nm, which are characteristic for pure aurofusarin (Kotyk, 1999). The aurofusarin dose was the same as in our previous experiments (Dvorska et al., 2001; 2002). Our previous observations indicated that Mycosorb alone in the quail diet at the same rate of inclusion (1 g/kg) did not affect lipid or antioxidant composition of eggs or embryonic tissues (Dvorska et al., unpublished). Eggs obtained after 8 weeks of feeding were analysed and incubated in standard conditions of 37.5°C/55% RH. Tissue samples were harvested from newly hatched quail.

Analytical procedures

Vitamin and carotenoid extraction from egg yolk and tissues was performed as previously described (Surai et al., 2000a). In brief, egg yolk (0.2-0.3 g) or tissues (0.2-0.5 g) were homogenised in 2 ml of 1:1 (v/v) mixture of 5% NaCl solution and ethanol following by addition of 3 ml hexane and further homogenisation for 3 min. After centrifugation, the hexane layer was collected and the extraction was repeated twice. Hexane extracts were combined and evaporated under N_2 and the residue was dissolved in 1 ml of methanol:dichloromethane (1:1, v/v), centrifuged and the supernatant was used for vitamin A and carotenoid determination.

Retinol and retinyl esters were determined by HPLC-based methods as described by Furr et al. (1986) with minor modifications (Surai et al., 2000a). The extract was injected into the HPLC system: autosampler (Shimadzu, Japan), isocratic pump (Spectra System P100, Spectra-Physics Analytical), fitted with a Sphereclone Type ODS (2), 3 μ 18

reverse phase HPLC column, 15 cm×4.6 mm (Phenomex, Cheshire, UK) with Security Guard cartridge system (Phenomex, UK). Programme wavelength detector (Spectra-Physics, San Jose, California) and 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 retinol, retinyl palmitate, retinyl oleate, retinyl stearate and retinyl linoleate in mobile phase were used for instrument calibration.

Carotenoids were determined by HPLC as described previously (Surai et al., 2001) using a Spherisorb type S3ODS2, 5-μ C18, reverse-phase column, 25 cm×4.6 mm (Phase Separation, Clwyd, UK) with a mobile phase of mobile phase of acetonitrile-methanol (85:15) and acetonitrile-dichloromethane-methanol (70:20:10) in gradient elution using detection by absorbance at 445 nm. Peaks were identified by comparison with the retention times of a range of carotenoid standards (variously obtained from Sigma, Poole. UK; Fluka, Gillingham. UK; Apin, Abingdon. UK; and Hoffman-La Roche, Basel, Switzerland) as well as using co-elution of individual carotenoids with known standards.

Yolk and tissue susceptibility to lipid peroxidation was determined as in Surai et al. (1996). Yolk or tissue homogenates (10% w/v), in sodium phosphate buffer, 10 mM, pH 7.4, containing 1.15% (w/v) KCl, were prepared and incubated at 37°C for 180 min. under air with gentle shaking. At the end of the incubation, butylated hydroxytoluene was added (0.01% v/v). The accumulation of malondialdehyde was determined by HPLC as previously described (Surai, 2000). In brief, after incubation of tissue homogenate (1 ml) at 37°C, 0.2 ml of sodium dodecyl sulphate (8%) was added and samples were vortexed. Then, 1.5 ml of 20% CH_3COOH at pH-3.5 (adjusted by KOH) and 1.5 ml of thiobarbituric acid (TBA, 0.8% in water) were added, samples were vortexed and incubated at 95°C for 60 min. After cooling, an aliquot was mixed with methanol and centrifuged at 5,000 ppm for 10 minutes. The clear supernatant was transferred to a vial and 5 μl of the solution was injected onto a 3 μ C_{18} reverse phase HPLC column (Phenomenex. Sphereclone ODS2, 100×3.2 mm, Cheshire. England). Separation of thiobarbituric acid-malondialdehyde adduct from other chromogens was achieved using a mobile phase of 50 mM phosphate buffer (pH 7.0) mixed with methanol in proportion 65:35 by volume and fluorescent detection with excitation at 515 and emission at 553 nm, respectively. Results were expressed as μg malondialdehyde /g wet yolk. 1,1,3,3-tetramethoxypropane was used as a standard for malondialdehyde determination.

Statistical analysis

Results are presented as mean (±SE) of measurements

Table 2. Effect of aurofusarin (Au) and Mycosorb™ (Msb) on carotenoids and vitamin A in quail egg yolk, µg/g

Measurements	Control	Control+Au	Control+Au+Msb
Total carotenoids	18.33±1.8[a]	12.29±1.46[b]	16.34±1.43[a]
Lutein	12.21±0.97[a]	7.11±0.93[b]	11.09±1.05[a]
Zeaxanthin	4.12±0.43[a]	2.44±0.25[b]	3.08±0.22[c]
Retinol	5.75±0.51[a]	4.08±0.46[b]	5.40±0.54[a]

Values are means±SEM (n=10). Values within a row that do not share a common letter are significantly different (p<0.05).

on 10 egg yolks or 6 tissues from each group. Statistical analysis was performed by Student's t-test.

RESULTS

Quail egg yolk carotenoids were represented mainly by lutein and zeaxanthin together comprising about 89% of total carotenoids. Aurofusarin consumption was associated with a significant reduction in concentration of total carotenoids (by 33%, p<0.05), lutein (by 42%, p<0.01), zeaxanthin (by 33 %, p<0.05) and retinol (by 29%, p<0.05; Table 2). Dietary inclusion of Mycosorb™ showed a protective effect against antioxidant depletion. In fact, total carotenoids, lutein and retinol levels returned to the control level. Zeaxanthin concentration in the egg yolk was also improved, however, it remained significantly lower than that in the control group (Table 2).

The antioxidant system of newly hatched quail was also compromised as a result of decreased carotenoid and vitamin A concentrations in the egg yolk (Table 3). For example, lutein and zeaxanthin concentrations in the liver of newly hatched quail decreased by 47% (p<0.01) and

48% (p<0.01), respectively. Dietary Mycosorb™ inclusion prevented not only changes in yolk carotenoid concentration, but also was protective in terms of maintaining carotenoids in tissues of the newly hatched quail. In fact, liver lutein concentrations of group number 3 (fed aurofusarin and Mycosorb™) were not different from those of control birds. Again, as in the case of egg yolk, liver zeaxanthin concentration of newly hatched quail increased (p<0.05) as a result of Mycosorb™ inclusion, but did not reach the level of a control group.

As can be seen from Table 3, vitamin A concentration in the liver of newly hatched quail significantly decreased due to aurofusarin inclusion in the maternal diet. In fact, all 5 major forms of vitamin A in the liver were significantly decreased. Mycosorb™ was effective in preventing vitamin A decrease in the liver, returning levels of retinol, retinyl palmitate, retinyl linoleate and retinyl oleate to the initial level. Only concentration of retinyl stearate was still reduced in comparison to controls.

Compromising the antioxidant system of developing quail due to aurofusarin in the maternal diet increased tissue susceptibility to lipid peroxidation significantly (p<0.05) (Table 4). This was obvious for all tissues studied except muscle, where the increase did not reach statistical significance. Brain tissue had the highest (4.5-fold) increase in lipid peroxidation. Dietary Mycosorb™ added to the maternal diet contaminated with aurofusarin largely prevented increase in the tissue susceptibility to peroxidation. After Mycosorb addition to the contaminated maternal diet, only brain tissue contained elevated lipid peroxidation in comparison to the control group (Table 4).

Table 3. Effect of aurofusarin (Au) and Mycosorb™ (Msb) on carotenoids and vitamin A in liver of newly hatched quail, µg/g

Measurements	Control	Control+Au	Control+Au+Msb
Lutein	15.31±1.00[a]	8.12±1.33[b]	13.67±1.30[a]
Zeaxanthin	6.23±0.42[a]	3.15±0.62[b]	4.9±0.25[c]
Retinyl linoleate	3.63±0.40[a]	2.10±0.30[b]	3.43±0.23[a]
Retinyl oleate	7.75±0.94[a]	4.22±0.71[b]	7.20±1.05[a]
Retinyl palmitate	14.22±1.25[a]	9.07±0.59[b]	14.13±0.79[a]
Retinyl stearate	6.42±0.47[a]	3.83±0.84[b]	4.78±0.39[b]
Retinol	1.26±0.17[a]	0.68±0.19[b]	1.43±0.27[a]
Total vitamin A	33.30±2.91[a]	19.90±3.00[b]	30.93±0.49[a]

Values are means±SEM (n= 6). Values within a row that do not share a common letter are significantly different (p<0.05).

Table 4. Effect of aurofusarin (Au) and Mycosorb™ (Msb) on lipid peroxidation in tissues of newly hatched quail, µg malondialdehyde/g

Groups	Control	Control+Au	Control+Au+Msb
Initial yolk	4.63±0.91[a]	8.9±0.68[b]	5.86±0.90[ab]
Liver	33.16±2.67[a]	44.28±3.01[b]	36.83±2.56[a]
Kidney	35.63±3.81[a]	47.18±1.48[b]	35.92±2.42[a]
Lung	34.05±2.71[a]	62.18±5.88[b]	41.12±2.98[c]
Brain	91.63±10.29[a]	411.7±81.60[b]	122.4±6.91[c]
Pectoral muscle	32.07±2.82[a]	36.15±2.62[a]	32.95±4.52[a]
Femoral muscle	36.63±5.15[a]	42.30±3.37[a]	35.23±7.46[a]

Values are means±SEM (n=6). Values within a row that do not share a common letter are significantly different (p<0.05).

DISCUSSION

There is little information available on aurofusarin contamination of various feedstuffs. Recently, it has been suggested that aurofusarin be considered a new crop pollutant (Dvorska et al., 2000). Grains contaminated with aurofusarin have a pinkish colour; and this pigment was detected in 11 samples of wheat growing in Ukraine in 1988-1990 (Kotyk and Trufanova, 1990). For many years this Fusarium pigment has not been considered a potential toxicant. However, information accumulated over the last decade has clearly shown adverse effects of this compound on poultry. The primary effect of aurofusarin on poultry is a change in egg yolk colour from yellow-orange to dark-brown with a greenish tinge (Kotyk et al., 1990; Kotyk et al., 1995; Kotyk, 1999; Dvorska, 2001). Furthermore, aurofusarin consumption is associated with detrimental changes in fatty acid and antioxidant composition of egg yolk and tissues of quail (Dvorska et al., 2001; 2002; 2003), deterioration of chicken meat quality (Dvorska, 2000; 2000a,b), compromised immunity in laying hens (Sakhatsky, 1999) and quail (Dvorska, 2001a; Dvorska et al., 2001) and decreased fertility and hatchability in chickens (Sakhatsky, 1999) and quail (Dvorska, 2001a; Dvorska et al., 2001). These data indicated that aurofusarin should be considered a mycotoxin.

This study confirmed our previous observations of the protective effect of glucomannans against pro-oxidant properties of aurofusarin. Indeed, Mycosorb™ effectively prevented depletion of vitamin E in the egg yolk and liver of day old quail caused by aurofusarin (Dvorska et al., 2003). Furthermore, fatty acid profile changes in egg yolk were also prevented. The results of this study extended those data showing that protective effect of glucomannans is physiologically broader and also related to prevention in carotenoid and vitamin A depletion in the egg yolk and tissues of newly hatched quail. More importantly, a stabilised antioxidant system due to dietary Mycosorb™ inclusion was associated with a significant decrease in lipid peroxidation of quail tissues.

Our previous observations indicated that the antioxidant system in the developing chicken embryo is an important determinant of chick hatchability and, more importantly, chick viability in early postnatal development (Surai and Sparks, 2001). It seems likely that increased quail embryonic mortality due to aurofusarin consumption is also associated with a compromised antioxidant system (Dvorska et al., 2002). Therefore, dietary inclusion of glucomannans could be considered as a preventive measure against detrimental effects of mycotoxins. Indeed, a glucomannan derived from yeast cell walls (Mycosorb™) has been shown effective against a wide range of mycotoxins (Devegovda et al., 1998; Raju and Devegowda, 2000; Swamy et al., 2002; 2002a). For example, when broiler chicks were fed diets containing blends of corn and wheat naturally contaminated with Fusarium mycotoxins, biliary IgA was significantly increased and glucomannans prevented this change (Smith et al., 2003). Similarly, dietary glucomannan prevented detrimental changes in the immune system of ducklings due to consumption of Fusarium contaminated grains. Furthermore, changes in IgM and IgA in pig serum due to mycotoxin contamination were also prevented by glucomannans (Smith et al., 2003). Modified glucomannan alleviated the growth depression in broilers caused by mycotoxin contaminated diets (Aravind et al., 2003). In laying hens fed T-2 toxin (1-2 mg/kg diet) egg production and ovary weight were decreased and modified glucomannan prevented those changes (Manoj and Devegowda, 2000). Furthemore, glucomannan added to the mycotoxin-contaminated (ochratoxin, T-2 toxin and aflatoxin) diet of broilers increased body weight, food intake and prevented detrimental biochemical changes in blood (Raju and Devegowda, 2000). Supplementation of glucomannans to an ochratoxin A (0.5 ppm) contaminated diet improved body weight gain (3.6%), total proteins (11%), hematocrit values (4.7%) and reduced mortality of broilers (Devegowda and Aravind, 2003). Recently, chemically modified 1.3-β-D-glucan derived from yeast has shown binding of zearalenone and T-2 toxin (Freimund et al., 2003).

From data presented above it is clear that inclusion of glucomannans into the chicken/animal diet could be a valuable solution for mycotoxin-contaminated feeds. However, when choosing among commercial adsorbents it is important to take into account whether an absorbent is effective against a variety of mycotoxins or just a single mycotoxin. Most are mainly effective against aflatoxins only (Huwig et al., 2001). For example, recently two experiments with male broilers examined excretion kinetics of zearalenone and its metabolites and their occurrence in blood plasma and bile fluid after a single oral dose of zearalenone. Mycofix-Plus supplementation had only minor or no effects on the parameters examined (Danicke et al., 2001). Similarly, feeding maize highly contaminated with Fusarium mycotoxins adversely influenced performance of hens and modulated immune response, however inclusion of Micofix-Plus was not able to overcome the problem (Danicke et al., 2002). Furthermore, supplementation of the diets with Mycofix Plus decreased performance in a manner independent of mycotoxin concentration and some clinical-chemical serum parameters were significantly altered due to Mycofix Plus (Danicke et al., 2003). Hydrated sodium calcium aluminosilicate did not alter impaired productivity (Santin et al., 2002), the reduced humoral immune response

against Newcastle disease vaccination or the number of mitotic cells in the bursa of broilers (Santin et al., 2002a) due to ochratoxicosis. Zeolites alone were not effective in preventing effects of T-2 toxin (Dvorska and Surai, 2001). In contrast, the inclusion of yeast glucomannan in quail diets containing T-2 toxin significantly slowed the depletion of natural antioxidants and vitamin A in the liver. The protective effects of glucomannans probably reflect its ability to prevent the absorption of T-2 toxin into systemic circulation (Reddy and Devegowda, 2003). Our data also show that the inclusion of the yeast glucomannan in a diet contaminated with T-2 toxin significantly decreased tissue susceptibility to lipid peroxidation. However, the adsorbent could not completely mitigate the powerful stimulating effect of T-2 toxin on lipid peroxidation. Similar data are reported here in relation to aurofusarin-induced lipid peroxidation in quail tissues, where glucomananns were also shown to have a protective effect.

Considering the wide variety of mycotoxins contaminating typical feed ingredients, technical difficulties in controlling mycotoxin presence and detrimental effects of mycotoxins on productive and reproductive performance of farm animals and poultry, it is critical to develop strategies for combating mycotoxicosis. For example, immunosuppresive action of mycotoxins could be responsible for low efficiency of vaccinations, as well as for poor animal growth and development. It is also necessary to take into account that a combination of various mycotoxins (Huff and Doerr, 1981; Bata et al., 1983) even in low concentrations can often cause more problems than individual mycotoxins (Kubena et al., 1989; Raju and Devegowda, 2000; Swamy et al., 2002; 2002a). Therefore, a toxicological synergism between various mycotoxins is an important issue and needs more attention (Smith and Seddon, 1998).

CONCLUSION

These data, together with above mentioned other findings in relation to Mycosorb™ efficacy against various mycotoxicoses clearly suggest that glucomannans could be considered as an effective means to deal with mycotoxin-contaminated feeds. Indeed, it is likely that inclusion of Mycosorb into standard feeds for various farm animals and poultry could be used as a preventive measure for producers to decrease or even prevent detrimental effects of mycotoxins. It seems likely that a combination of glucomannans with additional antioxidants (e.g. organic selenium, vitamin E. etc.) could further improve protection against mycotoxicoses. Clearly, more work should be done in this area to further develop the idea of preventive animal nutrition.

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