Acta Veterinaria (Beograd), Vol. 61, No. 2-3, 227-237, 2011.

DOI: 10.2298/AVB1103227K UDK 619:636.5.085.19

EFFECTS OF FEEDING WHEAT NATURALLY CONTAMINATED WITH *FUSARIUM* MYCOTOXINS ON BLOOD BIOCHEMISTRY AND THE EFFECTIVENESS OF DIETARY LIGNIN TREATMENT TO ALLEVIATE MYCOTOXIN ADVERSE EFFECTS IN BROILER CHICKENS

KLAPÁČOVÁ KATARÍNA*, FAIXOVÁ ZITA*, GREŠÁKOVÁ L'UBA**, FAIX Š**, MIKLÓSOVÁ LUCIA* and LENG L'**

*University of Veterinary Medicine and Pharmacy in Košice, Slovak Republic
**Institute of Animal Physiology Slovak Academy of Sciences, Košice, Slovak Republic

(Received 26th April 2010)

A study was conducted to investigate the effects of feeding wheat naturally contaminated with Fusarium mycotoxins on some biochemical parameters and the efficacy of lignin to alleviate adverse effects of fusariotoxins in broiler chickens.

Eighty, 1-d-old ROSS 308 broiler chicks of both sexes were used in the experiment. All birds received the control diet for two weeks and then they were fed experimental diets for two more weeks. The 4 diets included the following: 1.) negative control diet (0.1 mg DON/kg diet; 0.005 mg ZEA/kg diet), 2.) positive control diet (0.1 mg DON/kg diet, 0.005 mg ZEA/kg diet + 0.5% lignin), 3.) mycotoxin-contaminated diet (2.95 mg DON/kg diet, 1.59 mg ZEA/kg diet) and 4.) mycotoxin-contaminated diet with the addition of lignin at 0.5% of the diet (2.95 mg DON/kg diet, 1.59 mg ZEA/kg diet + 0.5% lignin). The feeding of contaminated wheat did not significantly affect parameters of mineral metabolism, including calcium, chlorides and phosphorus. Decreased levels of total protein, albumin and potassium were observed. However, dietary supplementation with lignin prevented this effect. Plasma triglycerides and free glycerol levels were not affected by dietary treatments. There was a significant increase in aspartate aminotransferase and alkaline phosphatase activities and magnesium and cholesterol levels in plasma from birds fed contaminated wheat. Inclusion of lignin in the diet reversed elevated alkaline phosphatase activity in chicks induced by mycotoxin-contaminated diet. Results indicated that consumption of grain naturally contaminated with Fusarium mycotoxins can adversely affect chickens' metabolism. As a food additive, lignin was not effective in the prevention of Fusarium mycotoxins effects in chickens.

Key words: chicken, deoxynivalenol, Fusarium mycotoxin, lignin, plasma chemistry, zearalenone

INTRODUCTION

There is increasing evidence that global supplies of cereal grains for animal feedstuffs are commonly contaminated with *Fusarium* mycotoxins. Trichothecenes are the most studied group of *Fusarium* mycotoxins and have been implicated in many cases of mycotoxicoses in animals and humans (Placinta *et al.,* 1999; Dönmez and Keskin, 2008). Trichothecenes are of great importance because they may occur in toxicologically relevant concentrations in grains which can affect the health and productivity of animals (Doll and Dänicke, 2004; Nešič *et al.,* 2008). The trichothecenes are recognized as having inhibitory effects on cells, including inhibition of protein, DNA and RNA synthesis, interference with cell-membrane integrity, and induction of apoptosis (Balogh *et al.,* 2007; Weber *et al.,* 2010). The rapidly proliferating cells and tissues with high rates of protein turnover, including the immune system, liver, and small intestine, are primarily effected by deoxynivalenol (Eriksen and Pettersson, 2004). There are many reports on the effects of feeding *Fusarium* mycotoxins on the health and performance of broiler chickens (Faixová *et al.,* 2006; Yegani *et al.,* 2006).

In order to avoid mycotoxicosis, several strategies have been investigated.

Lignin is a natural component of plant cell walls, and in its intact form, it represents a barrier to digestion of feedstuffs. A purified form of lignin is a by-product of paper manufacture, composed of low-molecular weight polyphenolic fragments (Lora *et al.,* 1993). It is non-digestible for both ruminants and monogastric animals. There are several reports of the effects of purified lignin on the growth performance of animals. Phillip *et al.* (2000) reported veal calves growth performance improvement and the growth *Escherichia coli* inhibition in *in vitro* experiments. In studies with chickens, the dietary inclusion of a purified form of lignin, has been shown to improve weight gain and feed efficiency and to reduce the concentration of volatile fatty acids in the ceca and large intestine (Ricke *et al.,* 1982). Nelson *et al.* (1994) reported that a purified form of lignin reduced intestinal translocation of pathogenic bacteria following burn injury in rats and inhibited *in vitro* growth of *E. coli, Staphylococcus aureus*, and *Pseudomonas.* Purified lignin has the potential to improve poultry performance by altering the microbial ecology of the hindgut (Baurhoo *et al.,* 2007).

Dietary fiber has been demonstrated to protect against toxicoses resulting from numerous xenobiotic compounds. There are reports that unrefined plant fibers can overcome the toxicity of food dyes when fed to rats (Carson and Smith, 1983). Such effects have been attributed to the physical properties of fibers (Takeda and Kiryama, 1979), as well as their ability to alter intestinal transit time (Takeda and Emoto, 1982).

There are only limited literature reports of the ability of dietary lignin treatment to alleviate adverse effects of *Fusarium* mycotoxins of broiler chickens.

Therefore, the objectives of this study were to determine the effects of feeding a diet contaminated with *Fusarium* mycotoxins on metabolic parameters and the efficacy of dietary addition of purified lignin to broiler chicken diet to reverse toxic effects of fusariotoxins.

MATERIAL AND METHODS

Animals and diets

Eighty, 1-d-old ROSS 308 broiler chickens of both sexes were obtained from a commercial local hatchery (Párovské háje, the Slovak Republic) and grown over a 28-d period. The chicks were raised in pens covered with wood shaving litter. Throughout the study, the birds were brooded following standards temperature regimens, which gradually decreased from 32 to 28ºC, and under a 20L:4D cycle. All birds had free access to water and feed.

The experimental procedures were in accordance with European guidelines for care and use of animals for research purpose. The protocol was approved by the local ethic committee and scientific authorithies.

Birds were randomly assigned to 4 treatments. The 4 experimental diets included the following: 1.) negative control diet (0.1 mg DON/kg diet, 0.005 mg ZEA/kg diet), 2.) positive control diet (0.1 mg DON/kg diet, 0.005 mg ZEA/kg diet + 0.5 % lignin), 3.) mycotoxin – contaminated diet (2.95 mg DON/kg diet, 1.59 mg ZEA/kg diet) and 4.) mycotoxin-contaminated diet with the addition of lignin at 0.5% of the diet (2.95 mg DON/kg diet, 1.59 mg ZEA/kg diet + 0.5 % lignin).

All birds were fed the same control diet for two weeks. To provide stable dietary contents of mycotoxins during the experiment, the chickens were fed the same diet (HYD-01). The composition of the diet is given in Table 1.

Table 1 Composition and nutrient content of diet HYD-01 fed to broiler chickens four weeks during the entire experiment

Component	Contents
Wheat ground, 11% of crude protein (CP) [g.kg^{-1}]	204.2
Maize (ground 8.3% CP) [g.kg^{-1}]	400.0
Oil-rape seed [g.kg^{-1}]	10.0
Soybean extracted ground meal (46% CP) [g.kg^{-1}]	330.0
Fish meal (62% CP) [g.kg^{-1}]	20.0
Monocalcium-phosphate [g.kg^{-1}]	7.0
Limestone [g.kg^{-1}]	16.0
Feed salt [g.kg^{-1}]	2.8
Premix HYD 01 ARO [g.kg^{-1}]	10.0
Dry matter [g.kg^{-1}]	883.0
Metabolizable energy [MJ.kg^{-1}]	12.75
Crude protein (analysed) [g.kg^{-1}]	210.0

Thereon the broilers of experimental groups started being fed diets contaminated with mycotoxins and supplemented by lignin. The control group of birds continued to be fed the same control diet. The experimental diets were obtained by mixing the basal diet (BD, the part of complete diet before addition of 40% portion of control or contaminated wheat) supplied by Agrokonzult s.r.o.,

Nové Zámky, the Slovak Republic with control or contaminated wheat batches. The contents of deoxynivalenol (DON), zearalenone (ZEA), total aflatoxins, ochratoxin A and ergosterol in control wheat, batches of contaminated wheat and in basal diet are summarized in Table 2.

Table 2. Deoxynivalenol, zearalenone, total aflatoxins, ochratoxin A and ergosterol levels in batches of wheat and in the basal diet for chickens

Component of diet	Concentration ($mg.kg^{-1}$ feed)				
	DON	ZEA	AFLA total	OTA	Ergosterol
Wheat Control group	0.06	0.0	0.0	0.0006	14.25
Wheat Contaminated group	7.18	3.95	0.0	0.0005	242.76
Basal diet	0.13	0.009	0.0	0.0004	11.07

DON = deoxynivalenol; ZEA = zearalenone; AFLA total = total aflatoxins; OTA = ochratoxin A

Contaminated batches of wheat were obtained by cultivation with *Fusarium graminearum* at the Slovak Agriculture University in Nitra according to the method of Labuda *et al.* (2003). Ready made contaminated batches of wheat were used in the experiment. The final concentration of mycotoxins and ergosterol in control and the experimental diets are shown in Table 3.

Table 3 Total content of deoxynivalenol, zearalenone, total aflatoxins, ochratoxin A and ergosterol in control and experimental diets

	Concentration ($mg.kg^{-1}$ feed)				
	DON	ZEA	AFLA total	OTA	Ergosterol
Negative control group	0.1	0.005	0.0	0.0005	12.3
Positive control group	0.1	0.005	0.0	0.0005	12.3
Contaminated group	2.95	1.59	0.0	0.0005	103.8
Contaminated group + 0.5% lignin	2.59	1.59	0.0	0.0005	103.8

DON = deoxynivalenol; ZEA = zearalenone; AFLA total = total aflatoxins; OTA = ochratoxin A

Experimental diets were prepared by mixing the basal diet containing 200.4 g wheat with 400 g contaminated or non-contaminated wheat/kg basal diet.

Sample collection

At the age of four weeks, eight chickens from each group were anaesthetized by intraperitoneal injection of xylazine (Rometar 2%, SPOFA, Czech Republic) and ketamine (Narkamon 5%, Spofa, Czech Republic) at 0.6 and 0.7 mL/kg body weight, respectively. After laparothomy, blood was collected into heparinised tubes by intracardial punction. Plasma was separated by centrifugation at 1 180 g for 15 min and stored at -65° C until analysis.

Sample analysis

Mycotoxins in all wheat batches used and in the basal diet were detected using the commercial competitive enzyme-linked immunosorbent assay (VERATOX 5/5 kit, Neogen, Lansing, MI, USA). Concentrations of ergosterol in wheat batches and the basal diet were analyzed by a fluorodensitometric method (Bailly *et al.,* 1999).

The plasma levels of albumin, potassium, cholesterol and activities of alanine aminotransferase (ALT, EC 2.6.1.2), aspartate aminotransferase (AST, EC 2.6.1.1) and alkaline phosphatase (ALP, EC 3.1.3.1) were determined using a Reflotron spectrophotometer auto analyzer (Boehring Manheim, Germany).

Ready made modified lignin, molecular weight 1 900 and 20.8 % OCH_3 and 0.015 % ash was kindly provided by Biochemical Institute of Slovak Academy of Sciences in Bratislava, the Slovak Republic.

The level of total protein was measured by the method of Doumas *et al.* (1981), the concentration of chloride by the method of Kuffer *et al.* (1975) and calcium concentration was determined according to Ray Sarikar and Chauchan (1967) using commercial kits (Pliva-Lachema, Brno, Czeck Republic). Phosphorus concentration was measured by the method of Daly and Ertingshausen (1972), the concentration of magnesium by the method of Mann and Yoe (1957), triglycerides and free glycerol concentrations were measured using the method of Koditchek and Umbreit (1969) with commercial kits (RANDOX, Ardmore, UK).

Statistical analysis

The results are expressed as means ± S.E.M. Statistical analysis was done by a one-way analysis of variance (ANOVA) with *post hoc* Tukey multiple comparison test (Graph Pad Software, La Jolla, USA).

RESULTS

Table 4 shows that the diet contaminated with *Fusarium* mycotoxins reduced plasma levels of total protein, albumin and potassium as compared to control and dietary supplementation with lignin partially prevented this effect.

Feeding mycotoxin-contaminated diet resulted in a significant increase in aspartate aminotransferase and alkaline phosphatase activities and magnesium and cholesterol plasma levels as compared to control. Inclusion of lignin in the diet reversed alkaline phosphatase activity in chicks induced by mycotoxin-contaminated diet.

The feeding of contaminated wheat did not significantly affect parameters of mineral metabolism, including calcium, chlorides and phosphorus. Plasma triglycerides and free glycerol levels were not affected by dietary treatment.

Table 4 Effects of feeding diets contaminated with mycotoxins (2.95 mg DON.kg^{-1} feed and 1.59 mg ZEA.kg^{-1} feed) and supplemented with 0.5 % lignin on blood biochemistry of broiler chickens

Parameter	Negative control group	Positive control group (0.5% lignin)	Mycotoxin-contaminated group	Mycotoxin-contaminated group + 0.5 % lignin
Total protein (g.l^{-1})	43.75±0.53^{ab}	42.10±2.24^{cd}	23.89±0.80^{ace}	34.84±0.88^{bde}
Albumin (g.l^{-1})	21.49±0.99^{abc}	15.18±0.22^{a}	12.24±0.12^{b}	14.08±0.09^{c}
Aspartate aminotransferase (μkat.l^{-1})	2.63±0.10^{ab}	2.54±0.05^{cd}	3.31±0.01^{ac}	3.01±0.28^{bd}
Alkaline phosphatase (μkat.l^{-1})	7.98±0.10^{a}	9.64±0.29^{b}	14.99±0.10^{abc}	8.44±0.21^{c}
Potassium (mmol.l^{-1})	6.27±0.14^{ab}	6.03±0.4^{c}	4.37±0.30^{acd}	5.12±0.42^{bd}
Calcium (mmol.l^{-1})	1.87±0.08	2.06±0.03	1.61±0.01	1.90±0.07
Magnesium (mmol.l^{-1})	1.0±0.02^{abc}	1.42±0.03^{ad}	1.61±0.05^{be}	1.90±0.07^{cde}
Chlorides (mmol.l^{-1})	129.9±5.11^{ab}	128.3±8.6^{c}	137.9±2.73^{a}	105.6±3.9^{bc}
Phosphorus (mmol.l^{-1})	2.12±0.04	2.65±0.32	2.02±0.19	1.92±0.10
Cholesterol (mmol.l^{-1})	2.14±0.07^{abc}	2.72±0.11^{a}	2.85±0.17^{b}	2.90±0.13^{c}
Triglycerides (mmol.l^{-1})	1.31±0.01	1.17±0.01	1.22±0.02	1.28±0.02
Free glycerol (mmol.l^{-1})	1.20±0.02	1.05±0.02	1.10±0.01	1.16±0.01

Significant differences within a row are indicated by the same superscript letters at $p<0.05$ level, mean ± S.E.M., n = 8

DISCUSSION

In our study, the plasma total protein and albumin levels of chicken fed the contaminated diet was decreased as compared to control.

DON is a well-known inhibitor of protein synthesis (Goyarts *et al.,* 2006). Its effect is explained by binding to the ribosomal peptidyl-transferase site, causing protein and DNA synthesis inhibition. Bergsjo *et al.* (1993) reported a significant decrease in serum protein and albumin in growing pigs fed a diet containing DON

at 3.5 mg/kg of feed. These effects may be explained by reduced feed intake, and/or uptake, but the inhibition of protein synthesis may play a role. In another study, plasma level of total protein was found to be significantly decreased in chicks fed DON at 3 mg/kg of feed for 42 days (Faixová *et al.,* 2006).

In the current study, plasma potassium was decreased in birds fed mycotoxin contaminated diet. This finding might be supported by the fact that the rapidly proliferating cells and tissues with high rates of protein turnover, including the small intestine, are primarily affected by deoxynivalenol (Eriksen and Pattersson, 2004). Moreover, Hunder *et al.* (1991) reported reduced intestinal absorption of minerals due to excessive necrosis in the gastrointestinal tract caused by deoxynivalenol. Similar results were reported by Rotter *et al.* (1996).

In the present study, feeding the diet contaminated with *Fusarium* mycotoxins caused an elevation in aspartate aminotransferase activity in plasma, indicating liver damage and leakage of the enzymes into the blood. The findings of the current study are in agreement with previous studies of Faixová *et al*. (2007), who have also observed an increase in liver enzyme activities – serum ALT from broiler chickens fed diets containing 3 mg of deoxynivalenol . kg^{-1} of feed for 6 weeks. Recently, it has been shown that feeding a diet naturally contaminated with *Fusarium* mycotoxins deoxynivalenol and zearalenone each in 3.4 mg/kg feed has toxic effects on liver function of broiler chickens (Faixová *et al.,* 2010). An increase of plasma alkaline phosphatase activity in the current study indicates hepatic disorders and biliary obstruction. The findings of the current study are in agreement with Leung *et al.* (2007), who also observed an increase in serum ALP from dog fed-cereal-based *Fusarium* mycotoxins (2.7 mg DON/kg feed, 0.2 mg ZEA/kg feed and 8.4 mg fusaric acid/kg feed for 14 days).

Inclusion of lignin in the mycotoxin- contaminated diet provided insufficient protective effects against changes in hepatocyte integrity.

Other biochemical parameters, including calcium, chlorides, phosphorus, triglycerides and free glycerol levels were not affected by dietary treatments.

The concentration of lignin in one of the contaminated diets was based on the recommendations of the manufacturer, as well as on reports regarding other species (Baurhoo *et al.,* 2007). Higher levels of lignin can adversely affect performance of animals. Inclusion of high lignin concentration in feed caused diarrhea in rats. Lignin has been described as an inhibitor of digestion and this may have contributed to the growth depression when high levels of lignin were fed (Davis *et al.,* 1980).

There are several literature reports of the protective mechanism of lignin against mycotoxin toxicity.

Stangroom and Smith (1984) reported that the fiber fraction of alfalfa could protect against 250 mg zearalenone/kg feed in rat and this effect was not likely mediated by hepatic 3-alpa-hydroxysteroid dehydrogenase, the enzyme believed to metabolize zearalenone.

Carson and Smith (1983) studied the effect of dietary fibers on T-2 toxicosis in rats. Lignin was found to overcome feed refusal and growth depression in animals fed T-2 toxin (3 mg/kg feed) for 2 weeks in rats. Alfalfa is a fibrous feedstuff relatively rich in lignin. Inclusion of 20% alfalfa to T-2 toxin-contaminated diet in

rats was able to completely overcome the toxic effect of T-2 toxin. However, there was no effect of diet on activity of hepatic esterase, the enzyme believed to catabolize T-2 toxin. Alfalfa is believed to reduce T-2 toxicosis in rats by binding to toxins in the intestinal lumen, thereby promoting fecal excretion. Inclusion of 20% alfalfa corresponds to feeding 3.5% lignin, since the alfalfa was determined to contain 16.7% lignin.

The lignin in concentrations included in the contaminated diet in the present study appeared to be insufficient to provide protective effects in chickens, particularly in liver damage manifested by an increase in AST activity. Other researchers have reported, however, that lignin could be effective in the protection of animals against a single mycotoxin in diet in rats. Its protective effect is explained mostly by binding to toxins in the intestinal lumen and by increasing intestinal motility, thereby reducing intestinal absorption and promoting fecal excretion.

The reason for this discrepancy might be attributed to differences in the source of contamination (natural and purified), using a single source of contaminated grain compared with a blend of contaminated grains, and the level and duration of exposure. These studies have also been conducted under different experimental conditions, with different experimental animals (chickens and rats), which may influence the effect of feeding contaminated grains on metabolism.

CONCLUSION

In the current study, broiler chickens were fed a diet naturally contaminated *Fusarium* mycotoxins. Deoxynivalenol at 2.95 mg/kg diet and zearalenone at 1.59 mg/kg diet were the major mycotoxins contained in the contaminated diets.

Poultry is relatively resistant to the effects of DON in comparison to pigs and pet animals and no effect was found in chicks fed DON at 5 mg/kg feed (Eriksen and Pettersson, 2004).

In this experiment, however, several biochemical parameters were found to be altered by toxic effects of mycotoxins.

It should be noted that contaminations of mycotoxins as found naturally in mouldy feeds, caused greater effects than the feed containing a single mycotoxin (Pál *et al.,* 2009). This is probably due to the presence of unidentified mycotoxins and precursors in the contaminated grains and to resulting in synergistic effects among mycotoxins (Smith *et al.*, 1997).

It could be concluded that consumption of grains naturally contaminated with 2.95 mg deoxynivalenol/kg feed and 1.95 mg zearalenone/kg feed for 2 weeks can adversely affect chickens' protein and mineral metabolism and liver function. As a food additive, lignin was not effective in the prevention of *Fusarium* mycotoxins in chickens.

ACKNOWLEDGEMENTS:
The study was supported by the Grant Agency for Science (VEGA) of the Slovak Republic, Grants No. 1/0420/08, No. 2/0010/10 and No. 2/006/9 and Grant LPP No. 0213-06.
Our thanks belong to Dr. Christine Lang from University of Veterinary Medicine, Vienna for analysis of mycotoxins and Dr. Jean-Denis Bailly from National Veterinary School in Toulouse for analysis of ergosterol levels in experimental diets. We are grateful to Professor Ing. Božena Košíková, DSc. from Biochemical Institute of Slovak Academy of Sciences in Bratislava, the Slovak Republic for providing a modified lignin.

Address for correspondence:
Associate Professor Zita Faixová, D.V.M., PhD.
Institute of Pathological Physiology
The University of Veterinary Medicine and Pharmacy in Košice
Komenského 73
041 83 Košice, Slovak Republic
E-mail: faixova@uvm.sk

REFERENCES

1. *Bailly JD, Le Bars P, Pietri A, Benard G, le Bars J*, 1990, Evaluation of a fluorodensitometric method for analysis of ergosterol as a fungal marker in compound feeds. *J Food Protect,* 62, 686-90.
2. *Balogh K, Hausenblasz J, Weber M, Erdélyi M, Fodor J, Mézes M*, 2007, Effects of ochratoxin A on some production traits, lipid peroxide and glutathione redox status of weaned piglets, *Acta Vet Hung*, 54, 463-70.
3. *Baurhoo B, Phillip L, Rui-Feria CA*, 2007, Effects of purified lignin and mannan oligosaccharides on intestinal integrity and microbial populations in the ceca and litter of broiler chickens, *Poult Sci,* 86, 1070-8.
4. *Bergsjo B, Langseth W, Nafstad I, Jansen JH, Larsen HJS*, 1993, The effects of naturally deoxynivalenol-contaminated oats in the clinical conditions, blood parameters, performance, and carcass composition of growing pigs, *Vet Res Commun,* 17, 283-94.
5. *Carson MS, Smith TK*, 1983, Effects of feeding alphalpha and refined plant fibers on the toxicity and metabolism of T-2 toxin in rats, *J Nutr,* 113, 304-13.
6. *Daly JA, Ertingshausen G*, 1972, Direct method for determinated inorganic phosphate in serum with "CentrifiChem", *Clin Chem,* 18, 263-5.
7. *Davis ND, Dickens JW, Freie RL et al*., 1980, Protocols for surveys, sampling, post-collection handling, and analysis of grain samples involved in mycotoxin problems, *J AOAC Int*, 36, 95-102.
8. *Doll S, Dänicke S*, 2004, *In vivo* detoxification of *Fusarium* toxins, *Arch Anim Nutr,* 58, 419-41.
9. *Doumas BT, Bayse DD, Carter RJ, Peters Tjr, Schaffer R*, 1981, A candidate reference method for determination of total protein in serum. I. Development and validation, *Clin Chem,* 27, 1642-50.
10. *Dönmez N, Keskin E*, 2008, The effects of aflatoxin and glucomannan on some antioxidants and biochemical parameters in rabbits, *Acta Vet (Beograd),* 58, 307-13.
11. *Eriksen GS, Patterson H*, 2004, Toxicological evaluation of trichothecenes in animal feed, *Anim Feed Sci Technol*, 114, 205-39.
12. *Faixová Z, Faix Š, Leng L', Váczi P, Szabóová R, Maková Z*, 2006, Effects of feeding diet contaminated with deoxynivalenol on plasma chemistry in growing broiler chickens and the efficacy of glucomannan mycotoxin adsorbent, *Acta Vet (Beograd),* 56, 479-87.
13. Faixová Z, Faix Š, Borutová R, Leng L', 2007, Efficacy of dietary selenium to counteract toxicity of deoxynivalenol in growing broiler chickens, *Acta Vet Brno*, 76, 349-56.
14. Faixová Z, Faix Š, Borutová R, Leng L', 2010, Effects of feeding diets contaminated with *Fusarium* mycotoxins on blood biochemistry of broiler chickens, *Acta Vet Hung,* 58, 275-85.
15. *Goyarts T, Grove N, Danicke S*, 2006, Effects of the *Fusarium* toxin deoxynivalenol from naturally contaminated wheat given subchronically or as one single dose on the *in vivo* protein synthesis

of peripheral blood lymphocytes and plasma protein in the pig, *Food Chem Toxicol,* 44, 1953-65.

16. *Hunder G, Schuhann K, Strugala G, Gropp J, Fichtl B, Forth W*, 1991, Influence of subtoxic exposure to low dietary deoxynivalenol, a trichothecene mycotoxin, on intestinal absorption of nutrients in mice, *Food Chem Toxicol*, 29, 809-14.
17. *Koditchek IK, Umbreit WW*, 1969, α-Glycerophosphate oxidase in *Streptococcus faecium* F24, *J Bacteriol*, 98, 1063-8.
18. *Kuffer H, Richterich R, Kraft R, Peheim E, Colombo JAP*, 1975, The determination of chloride in plasma and serum (mercury III)- thiocyanate method) with Greiner Electronic selective Analyzer GSA II. *Z Klin Chem Klin Biochem,* 5, 203-11.
19. *Labuda R, Trančinová D, Hudec K*, 2003, Identification and enumeration of *Fusarium* species in poultry feed mixtures from Slovakia, *Ann Agric Environ Med*, 10, 661-6.
20. *Leung MC, Smith TK, Karrow NA, Boermans HJ*, 2007, Effects of foodborne *Fusarium* mycotoxins with and without a polymeric glucomannan mycotoxin adsorbent on food intake and nutrient digestibility, body weight, and physical and clinicopathologic variables of mature dogs, *Am J Vet Res*, 68, 1122-29.
21. *Lora JH, Creamer AW, Wu LCF, Goyal GC*, 1993, Industrial scale of organo solo lignin: Characteristics and application, In: Kennedy JF, Phillips GO, Williams PA (editors.), Eillulosics: Chemical, biochemical and Material aspects, Ellis Horwoods LTD, West Sussex, UK, 252-6.
22. *Mann CK, Yoe JH*, 1957, Spectrophotometric determination of magnesium with sodium1-azo-2-hydroxy-3-(2,4-dimethycarboxanilidonaphtalene-1'-(2hydroxybenzene), *Anal Clin Act,* 16, 155-60.
23. *Nelson JL, Alexander JW, Gianotti L, Chalk CL, Pyles T,* 1994, Influence of dietary fiber on microbial growth *in vitro* and bacterial translocation after burn injury in mice, *Nutrition,* 10, 32-6.
24. *Nešić K, Resanović R, Nešić V, Sinovec Z*, 2008, Efficacy of mineral and organic adsorbent in alleviating harmful effects of zearalenone on pigs performance and health, *Acta Vet (Beograd),* 58, 211-9.
25. *Pál L, Dublecz K, Weber M, Krisztián B, Erdélyi M, Szigeti G, Mézes M*, 2009, Effects of combined treatment with aflatoxin B1 and T-2 toxin and metabolites on some production traits and lipid peroxide status parameters of broiler chickens, *Acta Vet Hung,* 57, 75-84.
26. *Phillip LE, Idziak ES, Kubov S*, 2000, The potential use of lignin in animal nutrition, and in modifying microbial ecology of the gut, pages 1-9 in East Nutr Conf Anim Nutr Assoc of Canada, Montreal, Quebec, Canada.
27. *Placinta CM, D´Mello JPF, Mac Donald AMC*, 1999, A review of worldwide contamination of cereal grains and animal feed with *Fusarium* mycotoxins, *Anim Feed Sci Technol*, 78, 21-37.
28. *Ray Sarkar BC, Chauchan USP*, 1967, A new method for the determination of microquantities of calcium in biological material, *Anal Biochem,* 20, 155-60.
29. *Ricke SC, van der Aar PJ, Fahey GC, Berger L,* 1982, Influence of dietary fiber on performance and fermentation characteristics of gut contents from growing chicks, *Poult Sci,* 61, 1335-43.
30. *Rotter BA, Prelusky DB, Pestka JJ,* 1996, Toxicology of deoxynivalenol (vomitoxin), *J Toxicol Environ Health,* 48, 1-34.
31. *Smith TK, Mc Millan EG, Castillo JB*, 1997, Effects of feeding blend of *Fusarium* mycotoxin-contaminated grains containing deoxynivalenol and fusaric acid on growth and feed consumption of immature swine, *J Anim Sci*, 75, 2174-91.
32. *Stangroom KE, Smith TK*, 1984, Effects of whole and fractionated dietary alfalfa meal on zearalenone toxicosis and metabolism in rats and swine, *Can J Physiol Pharmacol*, 62, 1219-24.
33. *Takeda H, Kiryama S*, 1979, Correlation between the physical properties of dietary fibers and their protective activity against amaranth toxicity in rats, *J Nutr,* 109, 388-96.
34. *Takeda H, Emoto T,* 1982, Nutritional significance of dietary fiber in counteracting the amaranth-toxicity in rats: A possible explanation of the mechanism, *Nutr Rep Int,* 25, 169-87.

35. *Weber M, Balogh K, Fodor J, Erdélyi M, Ancsin Z, Mézes M*, 2010, Effect of T-2 and HT-2 toxin during the growing period on body weight, lipid peroxide and glutathione redox status of broiler chickens, *Acta Vet Brno*, 79, 27-31.
36. *Yegani M, Smith TK, Leeson S, Boermans HJ*, 2006, Effects of feeding grains naturally contaminated with *Fusarium* mycotoxins on performance and metabolism of broiler breeders, *Poult Sci*, 85, 1541-9.

EFEKTI PŠENICE PRIRODNO KONTAMINIRANE FUZARIJUM MIKOTOKSINIMA NA BIOHEMIJSKE PARAMETRE U KRVI I EFIKASNOST DIJETETSKOG TRETMANA LIGNINOM RADI UBLAŽAVANJA ŠTETNIH EFEKATA MIKOTOKSINA NA BROJLERE

KLAPÁČOVÁ KATARÍNA, FAIXOVÁ ZITA, GREŠÁKOVÁ L'UBA, FAIX Š,
MIKLÓSOVÁ LUCIA i LENG L'

SADRŽAJ

Ovo istraživanje je izvršeno radi utvrđivanja efekata pšenice, prirodno kontaminirane fuzarijum mikotoksinima, na vrednosti pojedinih biohemijskih parametara u krvi i efikasnosti lignina u ublažavanju štetnih efekata ovih toksina kod brojlera.

U ogledu je korišćeno 89 jednodnevnih ROSS 308 brojlerskih pilića oba pola. Svi pilići su bili hranjeni standardnom smešom prve dve nedelje, a zatim su hranjeni eksperimentalnim smešama tokom naredne dve nedelje. Smeše su bile formulisane na sledeći način 1. negativna kontrolna smeša (0.1 mg DON/kg i 0.005 mg ZEA/kg smeše 2. pozitivna kontrolna smeša (0.1 mg DON/kg i 0.005 mg ZEA/kg smeše + 0.5 % lignina), 3. mikotoksinska smeša (2.95 mg DON/kg i 1.59 mg ZEA/kg smeše) i 4.) mikotoksinska smeša uz dodavanje lignina (2.95 mg DON/kg i 1.59 mg ZEA/kg dijeta + 0.5 % lignina).

Ishrana kontaminiranom pšenicom nije značajno uticala na vrednosti parametara mineralnog metabolizma, uključujući kalcijum, hloride i fosfor. Uočeno je smanjenje nivoa ukupnih proteina, albumina i kalijuma ali je smeša sa ligninom neutralisala ove efekte. Koncentracije triglicerida i slobodnog glicerola u plazmi nisu bile izmenjene dijetetskim tretmanima. Dokazano je značajno povećanje aktivnosti aspartat aminotransferaze i alkalne fosfataze, kao i nivoa magnezijuma i holesterola u plazmi pilića hranjenih kontaminiranom pšenicom. Uključivanje lignina u dijetu smanjivalo je samo aktivnost alkalne fosfataze kod pilića hranjenih smešama sa mikotoksinima. Dobijeni rezultati ukazuju da pšenica sa fuzarijum mikotoksinima štetno utiče na metabolizam pilića a da, kao aditiv u hrani, lignin nije u potpunosti bio efikasan u prevenciji njihovih efekata.