Efficacy of Dietary Selenium to Counteract Toxicity of Deoxynivalenol in Growing Broiler Chickens

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Abstract

Faixová Z, Š. Faix, R. Bořutová, Ľ. Leng: Efficacy of Dietary Selenium to Counteract Toxicity of Deoxynivalenol in Growing Broiler Chickens. Acta Vet. Brno 2007, 76: 349-356.

The aim of this study was to evaluate the effect of deoxynivalenol on plasma indicators and efficacy of dietary selenium to counteract toxicity of deoxynivalenol in growing broiler chicks. Three groups of broilers were formed with 14 birds in each group. Three diets included control (0.2 ppm deoxynivalenol, 0.4 mg selenium/kg diet), deoxynivalenol-contaminated (3 ppm deoxynivalenol, 0.4 mg selenium/kg diet) and deoxynivalenol-contaminated (3 ppm deoxynivalenol) plus selenium-enriched yeast (1.4 mg selenium/kg diet). After 6 weeks of feeding all birds were sacrificed and blood samples for chemical analyses were collected. Plasma calcium, chloride and alanine aminotransferase activity were significantly elevated and magnesium, total proteins, triglycerides and free glycerol were decreased in chicks fed deoxynivalenol-contaminated diet compared with those fed the control diet. Supplementation of selenium-enriched yeast to the diet reversed plasma levels of calcium, magnesium and alanine aminotransferase activity in chicks induced by dietary deoxynivalenol. Phosphorus, albumin and cholesterol levels and alkaline phosphatase, aspartate aminotransferase and lactate dehydrogenase activities were not affected by diets. The inclusion of selenium to DON-contaminated diet, however, did not completely alleviate toxic effect on protein and lipid metabolism by the liver. Supplementation of selenium-enriched yeast product counteracted most of the plasma indicator alterations caused by deoxynivalenol-contaminated diet in chicks.

Deoxynivalenol, chicken, plasma indices, selenium-enriched yeast

Fusarium species occur widely on plants. They are found in a variety of agricultural products mainly on corn, wheat and other cereal grains for human and animal consumption.

Although more than 100 *Fusarium* mycotoxins are known, those fusariotoxins of most concern based on toxicity and occurrence on a worldwide basis are trichothecenes, zearalenone, fumonisins and moniliformin.

Deoxynivalenol (DON) is the most prevalent trichothecene in crops used for food and feed production. Toxic effects of DON on animals have been well documented and concern mainly the immune system and the gastrointestinal tract (Rotter et al. 1996). The toxicity of DON is thought to be due to inhibition of protein synthesis and cytotoxicity was reported in a variety of cells (Parent-Massin 2004).

Trichothecenes are also known to interfere with the metabolism of membrane phospholipids and to increase liver lipid peroxides *in vivo* (Mezes et al. 1999).

The sensitivity to DON varies considerably between species. Poultry are more sensitive to DON than ruminants but less sensitive than pigs.

In order to avoid mycotoxicosis, several strategies have been investigated. The most frequently applied method for protecting animals against mycotoxicosis is the utilization of adsorbants mixed with the feed which are supposed to bind mycotoxins efficiently in the gastrointestinal tract. Many compounds have been tested for adsorptive effects on mycotoxins, but only few have proven successful (Swamy et al. 2002; Dvorska and Surai 2004; Diaz et al. 2005).

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Since some mycotoxins are known to produce membrane damage through increased lipid peroxidation, the protective properties of antioxidant substances have been extensively used.

Selenium, some vitamins (A, C and E) and their precursors act as superoxide anion scavangers. For these reasons, these substances have been used as protecting agents against toxic effects of mycotoxins. The effect of selenium as protecting agent against toxic effects of mycotoxins has been reported by several researchers.

In a controlled study, Lin et al. (1994) observed that selenium is able to reduce *in vitro* toxic effects of T-2 toxin on cultured chicken embryonic chondrocytes. These findings are in agreement with the report of Shi et al. (1994). They demonstrated that selenium inhibits aflatoxin B_1 –DNA binding. The same authors (Shi et al. 1995) found in an *in vitro* study on cultured hamster ovary cells that sodium selenite and selenium enriched yeast extract protect cells from aflatoxin B_1 cytotoxicity but not from mutagenicity.

However, much less information is available from studies on other mycotoxins, such as ochratoxin, zearalenone, deoxynivalenol, citrinin, moniliformin and fusaric acid.

The objective of this study was to evaluate the protective effect of selenium enriched product to counteract toxicity of deoxynivalenol in growing broiler chicks.

Materials and Methods

Animals, diets and treatments

Fourty-two chickens of Ross 308 hybrid broilers were randomly divided on the day of hatching into 3 groups (n = 14). The birds were kept on the floor during the study. Chickens were reared under lighting regimen of 23L:1D. The initial room temperature of 32-23 °C was reduced every week by 3 °C to a final temperature of 23 °C. All birds had free access to feed and water.

Birds were fed diets for 6 weeks which differed only in DON contamination and/or amount of selenium supplemented. Three diets included control (0.2 ppm deoxynivalenol, 0.4 mg selenium/kg diet), deoxynivalenol-contaminated (3 ppm deoxynivalenol, 0.4 mg selenium/kg diet) and deoxynivalenol-contaminated (3 ppm deoxynivalenol) plus Selplex® (Alltech Inc. USA), (1.4 mg selenium/kg diet).

To provide stable dietary contents of mycotoxins throughout the experiment, the chickens were fed only one type of diet HYD-02. The composition of this diet is given in Table 1.

Table 1. The composition of diet HYD-02

Component	g·kg^{-1}
Wheat ground, 10.5% of crude protein (CP)	260
Soya bean oil	14
Maize ground (8.3% CP)	400
Soya bean extracted ground meal (45% CP, 1.5% fat)	273.5
Fish meal (72% CP)	12.5
Premix HYD-02 (vitamins and minerals)	40

1 kg of diet contained: vitamin A 12 450 IU; vitamin D3 3800 IU; vitamin K 2.85 mg; vitamin E 61.2 mg; thiamine 5.6 mg; riboflavin 7.67 mg; pyridoxine 8.01 mg; cyanocobalamin 22.79 µg; niacin 69 mg; panthotenic acid 20.01 mg; biotin 0.34 mg; folic acid 1.81 mg; lysine 11.9 g; methionine 5.0 g; Zn 70.97 mg; I 0.81 mg; Co 0.06 mg; Mn 87.08 mg; Cu 13.98 mg; Fe 120.0 mg; Se 0.4 mg.

The final diets were obtained by mixing the basal diet supplied by Agrokonzult, s.r.o., the Slovak Republic (the part of HYD-02 diet before addition of 40% portion of maize) with control or contaminated maize. Maize used for the diets of control contained DON background level 0.5 mg·kg^{-1} while zearalenone and 15-acetyldeoxynivalenol were below detection limits. Diets for groups 2 and 3 contained DON-contaminated maize at a concentration of 7.5 mg·kg^{-1}. Zearalenone and 15-acetyldeoxynivalenol levels in the contaminated maize were 0.3 and 0.6 mg·kg^{-1}, respectively. Concentrations of T-2 toxin, iso T-2 toxin, T-2 triol, T-2 tetraol, HT-2 toxin, fusarenon-X, 3-acetyldeoxynivalenol, DAS, scirpentriol, nivalenol, 15-acetylscirpentriol, neosolaniol, zearalenol, aflatoxins and fumonisins were below detection limits in both control and contaminated maize.

The mycotoxin contents in the basal diet (the part of HYD-02 diet before addition of 40% portion of control or

contaminated maize) were found to be 0.05 and 0.0026 mg·kg^{-1} for zearalenone and total aflatoxins, respectively. DON, T-2 toxin and total fumonisins were below the detection limit of the method used.

All experimental procedures with animals were in accordance with European Guidelines for care and use of animals for research purposes and they were approved by the local Ethic Committee.

Sample analysis

All birds were sacrificed and blood samples for chemical analyses were collected. Plasma was separated by centrifugation at 1 600 g for 10 min and stored at -20 °C until analysis.

Alkaline phosphatase, alanine aminotransferase and aspartate aminotransferase activities and concentration of calcium, magnesium, chlorides, phosphorus, total proteins, albumin, cholesterol, triglycerides and free glycerol were determined by the colorimetric methods using spectrophotometric kits.

Mycotoxins in maize were detected using GC-MS method (Raymond et al. 2003). The mycotoxin contents in the basal diet (the part of HYD-02 diet before addition of 40% portion of control or contaminated maize) were analyzed using NOACK kits for ELISA with spectrophotometric evaluation. The concentration of selenium in diets was measured using the fluorometric method of Rodriguez et al. (1994).

The results are expressed as mean ± S.E.M. Statistical significance was evaluated by one-way ANOVA test.

Results

Dietary suplementation of DON increased plasma alanine aminotransferase activity and decreased total proteins concentration (ALT 0.25 ± 0.01 to 0.47 ± 0.01 µkat·l^{-1}, $P < 0.01$, total protein 35.54 ± 1.53 to 18.59 ± 1.92 g·l^{-1}, $P < 0.01$, Fig. 1). Supplementation of selenium- enriched yeast to the contaminated diet reversed plasma alanine aminotransferase activity (0.47 ± 0.01 to 0.25 ± 0.02 µkat·l^{-1}, $P < 0.01$). Alkaline phosphatase and aspartate aminotransferase activities were not affected by diets (ALP 8.13 ± 0.91, 6.72 ± 1.26, 8.54 ± 0.62 µkat·l^{-1}, NS, AST 1.84 ± 0.08, 2.12 ± 0.09, 1.40 ± 0.08 µkat·l^{-1}, NS).

Plasma triglycerides and free glycerol levels were decreased in animals fed the diet containing 3 ppm DON (triglycerides 0.59 ± 0.04 to 0.34 ± 0.02 mmol·l^{-1}, $P < 0.01$, free glycerol 0.48 ± 0.04 to 0.23 ± 0.02 mmol·l^{-1}, $P < 0.01$, Fig. 2). Plasma cholesterol concentration and lactate dehydrogenase activity, however, were not affected by diets (cholesterol 3.29 ± 0.12, 3.80 ± 0.34, 2.81 ± 0.12 mmol·l^{-1}, NS, lactate dehydrogenase 2.18 ± 0.16, 2.88 ± 0.43, 3.83 ± 0.26 µkat·l^{-1}, NS).

There were no differences between groups in plasma albumin level (12.3 ± 0.17, 15.8 ± 0.32, 16.8 ± 0.52 g·l^{-1}, NS).

Plasma calcium and chloride levels were significantly higher and magnesium concentration was significantly lower in birds fed the diet containing 3 ppm (Table 2). Supplementation of selenium-enriched yeast to DON-contaminated diet reversed plasma levels of calcium and magnesium.
Inclusion of selenium-enriched yeast to DON-contaminated diet, however, did not completely alleviate the toxic effect on protein and lipid metabolism by the liver (triglycerides 0.34 ± 0.02 to 0.50 ± 0.20 mmol·l^{-1}, NS, free glycerol 0.23 ± 0.02 to 0.39 ± 0.01 mmol·l^{-1}, NS).

Discussion

The deoxynivalenol treatment significantly decreased plasma level of total protein of chicks. Our results are consistent with those of Kubena et al. (1988) who found decreased total protein level in broiler chicks exposed to a DON (16 mg/kg) contaminated diet from 1 to 3 weeks of age.

Bergsjø et al. (1993) reported a significant decrease in serum protein in growing pigs fed a diet containing 3.5 mg/kg DON. They considered that these effects may be secondary to the reduced feed uptake but the inhibition of protein synthesis may play some role, too. One of the toxicities of DON was thought to be derived from the inhibition of protein

Alkaline phosphatase

Alanine aminotransferase

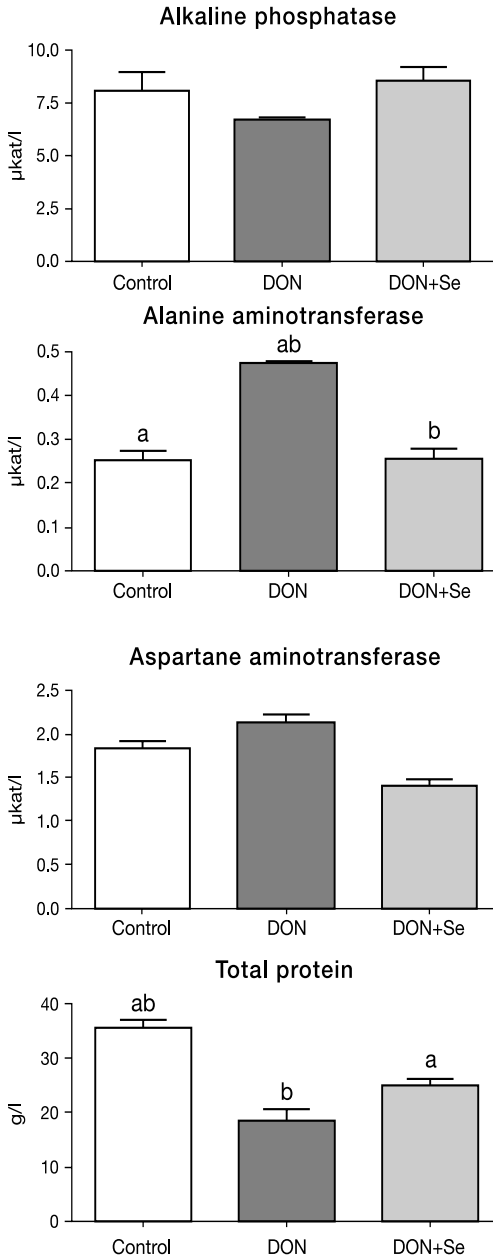## Aspartane aminotransferase

Total protein

Fig. 1. Plasma alkaline phosphatase, alanine aminotransferase and aspartate aminotransferase activities and total protein concentration after DON and selenium-enriched yeast intake in chickens. Values are mean ± S.E.M. (n = 14). Significant differences between columns are indicated by using the same superscript letter, $P < 0.01$.

synthesis (Rotter et al. 1996). These data were confirmed by Mikami et al. (2004).

We failed to demonstrate a protective effect of organic selenium against changes in protein metabolism in the liver induced by DON. Burguera et al. (1983), however, reported that selenium supplementation was effective in reducing the adverse effects of aflatoxin in turkey poults expressed by higher values of total protein.

The toxicity of DON was expressed through decreased plasma triglycerides and free glycerol in broiler chicks. These findings are in agreement with the previous reports of Kubena et al. (1987). DON has been reported to increase liver triglycerides and total liver lipid in White Leghorn hens fed a diet containing 0.25 or 0.70 ppm DON for 86 or 135 days (Farnworth et al. 1983).

Our results showed that selenium in the diet was ineffective in reducing the adverse effects of DON on lipid metabolism.

Dietary inclusion of 3 ppm DON resulted in increased plasma alanine aminotransferase activity, indicating liver damage. DON has also been reported to increase activities of aspartate aminotransferase, lactate dehydrogenase and gamma glutamyltransferase in broiler chicks fed DON at 15 mg/kg, indicating possible tissue damage and leakage of the enzymes into the blood (Kubena et al. 1997). Similar results were observed in horses (Raymond et al. 2003) and piglets (Doll et al. 2005) fed *Fusarium* culture material.

Supplementation of organic selenium to the contaminated diet decreased plasma alanine aminotransferase activity. Similar results were observed by Atroshi et al. (1999) who reported that pretreatment with Co Q_{10} (30 mg Co Q_{10}/kg diet) together with carnitine (2.8 mg carnitine/kg diet), alpha-tocopherol (30 IU vitamin E/kg diet) and selenium (1 mg selenium as sodium selenite/kg diet) decreased DNA damage and the activities of AST and ALT in the liver induced by fumonisin B_1 in rats.

Table 2. Effect of dietary inclusion of deoxynivalenol and selenium-enriched yeast on plasma indices in growing broiler chickens

Indicator (mmol·l[-1])	Control	DON	DON+Se
Calcium	1.73 ± 0.13[a]	3.36 ± 0.35[ab]	1.69 ± 0.09[b]
Magnesium	1.02 ± 0.07[a]	0.35 ± 0.02[ab]	0.89 ± 0.05[b]
Chloride	81.9 ± 5.37[ab]	114.4 ± 4.37[a]	107.9 ± 1.28[b]
Phosphorus	3.01 ± 0.23	2.16 ± 0.25	3.01 ± 0.73

Values are mean ± S.E.M., n = 14. Significant differences within a row are indicated by using the same superscript letter, $P < 0.01$.

In the present study, the administration of 3 ppm DON to diet altered plasma calcium. Previous data of Bergsjø et al. (1993) reported a significant decrease in serum calcium and phosphorus in growing pigs fed a diet containing 3.5 mg DON/kg diet. DON has also been reported to induce a weak hypocalcaemia in rats fed 1 mg/kg DON diet for 6 months, suggesting that calcium metabolism disorders during chronic action of mycotoxin could be partially associated with secondary vitamin D deficiency (Sergeev et al. 1990).

However, recently Gouze et al. (2006) reported that electrolytes in plasma appeared to be insensitive to a 4-week exposure to low DON in mice.

The discrepancy between these results and our data could be due to a number of factors, including sensitivity to DON between species, DON-concentration, DON source, animal genetics, sex and nutritional status.

Inclusion of organic selenium in DON-enriched diet of growing broiler chicks provided a significant protective effect against changes in calcium metabolism.

On the basis of many reports about antimycotoxin action of antioxidant compounds and our findings it may be concluded that nutritional approaches for protection against mycotoxins should include increased levels of methionine, selenium and vitamin supplementation of the affected diet. Mycotoxins, upon being absorbed, are detoxified in the liver, utilizing the glutathione system, which contains cystine (derivative of methionine). Hence the metabolic level of methionine is depleted, leading to poor growth and feed efficiency.

Higher levels of methionine, selenium and vitamin supplementation in feed, therefore, have been found to be beneficial.

The role of dietary antioxidants such as vitamin C, E and selenium in preventing mycotoxin toxicity has attracted increasing attention at present and numerous trials are currently in progress to ascertain the benefits of these compounds in the diet.

Peng and Yang (2003) observed that sodium selenite is able to reduce *in vitro* the toxic effect of DON on cultured cardiomyocytes. Studies of Jakhar and Sadana (2004) showed that supplementation of selenium (5 ppm sodium selenite) had some protective effect against the toxic effect of 1 ppm aflatoxin B$_1$ in Japanese quail.

However, McLeod et al. (1997) reported that rats fed a selenium-deficient diet were resistant to aflatoxin B$_1$ than those fed a selenium-sufficient diet. According to the authors, the protection conferred by selenium deficiency against aflatoxin B$_1$ is associated with the hepatic expression of an aldo-keto reductase and a glutathione S-transferase subunit that efficiently metabolizes the mycotoxin.

Based on a study on rats Atroshi et al. (1995) concluded that selenium, vitamin E and vitamin C act as an antioxidant system and free radical scavenger that protects spleen and brain against membrane damage caused by T-2 toxin and DON. The same authors, Atroshi et al. (2000) reported a decrease of the GSH activity after a two-week-treatment with ochratoxin in mice. Treatment of mice with the combined antioxidants could enhance the hepatic oxidant/detoxification system, as indicated by an increase in hepatic-reduced glutathione level. The authors suggest that use of the combined antioxidants may be of
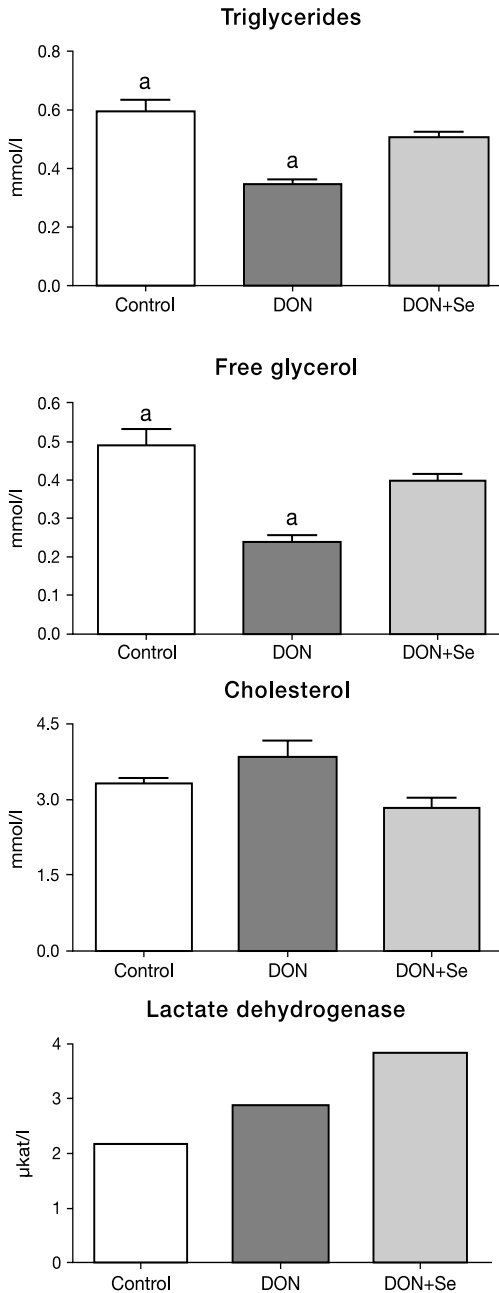
Triglycerides

Free glycerol

Cholesterol

Lactate dehydrogenase

Fig. 2. Plasma triglycerides, free glycerol and cholesterol concentrations and lactate dehydrogenase activity after DON and selenium- enriched yeast intake in chickens. Values are mean ± S.E.M.(n = 14). Significant differences between columns are indicated by using the same superscript letter, $P < 0.01$.

interest in conditions when certain toxin-mediated forms of cell death/apoptosis contribute significantly to toxicity.

A curious fact is that in some cases the source of potential protective agents are also a potential way to the assumption of mycotoxins, especially AFB_1 and OTA (Halt 1998). Moreover, caution should be used in promoting antimycotoxin action of discussed substances, since some may have toxic properties (selenium).

Our results show that dietary inclusion of deoxynivalenol resulted in changes of plasma indices in growing broiler chicks. Supplementation of organic selenium counteracted most of the plasma indicator alterations caused by deoxynivalenol in broiler chicks.

Efektívnosť selénu v diéte pôsobiť proti toxicite deoxynivalenolu u rastúcich kurčiat

Cieľom práce bolo vyhodnotiť vplyv deoxynivalenolu na ukazovatele v krvnej plazme rastúcich broilerových kurčiat a stanoviť efektívnosť pôsobenia selénu proti toxicite deoxynivalenolu. V pokuse boli použité tri skupiny kurčiat; počet kurčiat v každej skupine bol 14. Kontrolná diéta obsahovala 0,2 ppm deoxynivalenolu a 0,4 mg selénu/kg diéty, diéta kontaminovaná deoxynivalenolom obsahovala 3 ppm deoxynivalenolu a 0,4 mg selénu/kg diéty a tretia diéta obsahovala 3 ppm deoxynivalenolu a selénom obohatené kvasnice v dávke 1,4 mg/kg diéty. Zvieratá boli kŕmené diétami 6 týždňov, potom boli usmrtené a vzorky krvi boli odobraté na chemické vyšetrenia. Koncentrácia vápnika, chloridov a aktivita alanínaminotransferázy boli signifikantne zvýšená a koncentrácia horčíka, celkových bielkovín, triglyceridov a voľného glycerolu v plazme poklesla u kurčiat kŕmených diétou kontaminovanou deoxynivalenolom v porovnaní s kurčatami kŕmenými kontrolnou diétou. Pridanie prípravku obsahujúcom selénom obohatené kvasnice ku diéte eliminovalo zmeny v koncentrácií vápnika, horčíka a aktivity alanínaminotransferázy v plazme kurčiat

vyvolané deoxynivalenolom. Koncentrácia fosforu, albumínu, cholesterolu a aktivita alkalickej fosfatázy, aspartátaminotransferázy a laktátdehydrogenázy nebola ovplyvnená diétou. Prídavok selénu ku diéte kontaminovanej deoxynivalenolom však úplne nezmiernil toxický účinok deoxynivalenolu na metabolizmus tukov a bielkovín v pečeni. Prídavok prípravku, ktorý obsahoval selenizované kvasnice zmiernil väčšinu zmien v plazme vyvolaných deoxynivalenolom u kurčiat.

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