Journal of Animal and Veterinary Advances 7 (5): 539-544, 2008
ISSN: 1680-5593


# The Effects of Yeast Glucomannan (Mycosorb) on Lipid Peroxidation and Non-Enzymatic Antioxidative Status in Experimentally Induced Aflatoxicosis in Broilers

[1]Miyase Çinar, [2]Ebru Yildirim, [3]İlkay Yalçinkaya and [4]Gökhan Eraslan
[1]Department of Biochemistry, [2]Department of Pharmacology and Toxicology,
[3]Department of Animal Nutrition and Nutritional Diseases, Faculty of Veterinary Medicine,
University of Kırıkkale, Yahşihan, 71451 Kırıkkale, Turkey
[4]Department of Pharmacology and Toxicology, Faculty of Veterinary Medicine,
University of Erciyes, Sümer Mah. Barış Manço Cad. 38090 Kayseri, Turkey

**Abstract:** The aim of this study was to determine the effect of yeast glucomannan (Mycosorb), an aflatoxin binder, on lipid peroxidation and non-enzymatic antioxidative status in experimentally induced aflatoxicosis in broilers. Forty, 1-day-old male broiler (Ross 308) chickens were randomly divided into 4 groups of 10 birds each. The control group was fed a basal diet and the remaining groups received 0.75 g $kg^{-1}$ Mycosorb, 2 ppm Aflatoxin (AF) and 0.75 g $kg^{-1}$ Mycosorb +2 ppm Aflatoxin (AF) for 21 days. At the end of day 21, blood samples were collected and plasma Malondialdehyde (MDA), Ceruloplasmin (CP), albumin (alb), uric acid, vitamin A (vit A), β-carotene, vitamin C (vit C) and vitamin E (vit E) levels were determined. Plasma MDA levels increased insignificantly and alb levels decreased significantly in AF and AF+Mycosorb groups when compared to control and mycosorb groups. Plasma CP, vit A and β-carotene levels decreased significantly in AF groups when compared to control. In AF and AF+Mycosorb group, significant decreases were determined in vit E levels when compared to control and in vit C levels when compared to mycosorb group. Plasma uric acid levels were not affected by these treatments. It is concluded that subacute dietary intake of AF altered nonenzymatic components (CP, alb, vit A, β carotene, Vit E) of antioxidant defense systems and 0,75 g $kg^{-1}$ yeast glucomannan was not sufficient to ameliorate the oxidative damage caused by AF in broilers.

**Key words:** Aflatoxicosis, broiler, lipid peroxidation, mycosorb, non-enzymatic antioxidant

## INTRODUCTION

Two hundred and fifty, 300 different species of fungi are known to produce mycotoxin till now and 20-25 mycotoxin group are found as a natural pollutant on feed and feedstuffs Aflatoxins, produced by *Aspergillus flavus* and *Aspergillus parasiticus,* are the most frequently encountered mycotoxins in feed and feedstuffs. They lead considerable economic losses in poultry sector due to naturally contaminated feedstuffs (Kaya, 2002).

There are many toxic effects of AF, one of these is lipid peroxidation (Rastogi *et al*., 2001; Eraslan *et al*., 2004, 2005a,b; Preetha *et al*., 2006; Umarani *et al*., 2008). The main manifestation of oxidative damage is lipid peroxidation. It plays an important role in toxicity (Rastogi *et al*., 2001; Umarani *et al*., 2008). From the studies conducted on poultry it has been found that the most affected organ is liver following the administration of the toxin (Ortatatli and Oguz, 2001; Karaman *et al*., 2005). Therefore, aflatoxins are hepatotoxic (Preetha *et al*., 2006; Ortatatli and orguz, 2001; Karaman *et al*., 2005) and hepatocarcinogenic agents. They cause free radical producing during hepatic cells damage (Preetha *et al*., 2006). These radicals initiate lipid peroxidation, since all cell membranes contain the fatty acids which are substrates for such a reaction (Rastogi *et al*., 2001). This is thought to be the mechanism underlying the hepatotoxicity of aflatoxins (Preetha *et al*., 2006). Aflatoxins are not directly toxic by themselves, they become effective when transform into AFB1-8,9 epoxide by cytochrome P-450 in the liver (Coulombe, 1993). Probably an increase in AFB1-8, 9-epoxide causes increases in hepatic lipid peroxide level (Toskulkao and Glinsukon, 1988).

**Corresponding Author:** Miyase Çinar, Department of Biochemistry, Faculty of Veterinary Medicine, University of Kırıkkale, Yahşihan, 71451 Kırıkkale, Turkey

Antioxidants are substances that protect cells against the effects of free radicals. Antioxidants inhibit lipid peroxidation by breaking the chain reactions and scavenging initiating free radicals (Dundar and Aslan, 1999). Antioxidants are classified as enzymatic and non-enzymatic antioxidants. Non enzymatic antioxidants are α- tocopherol (Vitamin E), β-carotene, vitamin A, ubiquinols, ascorbate, glutathione, melatonin, cysteine, ceruloplasmin, haemoglobin, bilirubin, albumin and other compounds. Enzymatic antioxidants are superoxide dismutase, catalase, glutathione S transferase, glutathione peroxidase (Akkuş, 1995).

It is important to prevent the feed and feedstuffs from AF contamination. One of the promising and practical way is to use adsorbents (Huwig *et al*., 2001). Adsorbents bind to AF in the gastrointestinal system and decrease the rate of AF absorbed (Diaz *et al*., 2002).

Yeast glucomannan is a natural toxin binder. Yeast glucomannan has been found to counteract the toxic effect of aflatoxin in feed of growing broiler chicken (Ortatatli and Oguz, 2001; Karaman *et al*., 2005; Raju and Devegowda, 2000; Basmacioglu *et al*., 2005; Safameher and Shivazad, 2007). Karaman *et al*. (2005) showed that Yeast glucomannan (Mycosorb) alleviated the adverse effects of aflatoxin on the pathological changes and that the higher concentration of yeast glucomannan (1 g $kg^{-1}$) was more effective than the lower concentration (0.5 g $kg^{-1}$) and mycosorb itself had no adverse effect.

On different species, there have been many reports indicating the detrimental effects of AF (Eraslan *et al*., 2004, 2005b, Ortatatli and Orguz, 2001; Çam *et al*., 2008) and the protective effect of adsorbent from AF toxication (Eraslan *et al*., 2004, Ortatatli and Orguz, 2001; Karaman *et al*., 2005; Raju and Devegowda, 2000; Basmacioğlu *et al*., 2005; Safameher and Shivazad, 2007; Sehu *et al*., 2005; Jansen *et al*., 2006). However, there is limited number of studies about oxidative stress of aflatoxicated broilers (Eraslan *et al*., 2004, 2005).

Therefore, the objective of this study was determine the efficacy of aflatoxin in feed on lipid peroxidation and some non-enzymatic components of antioxidant defense system. In addition, to evaluate the role of the efficacy of Mycosorb at 0.75 g $kg^{-1}$ to prevent the adverse effects of AF in contaminated feed in broiler chicken.

## MATERIALS AND METHODS

Aflatoxin was produced, by the method described by Shotwell *et al*. (1966) modified by Demet *et al*. (1995), from the strain of Aspergillus parasiticus NRLL 2999 (National Center for Agricultural Utilization Research, Peoria, IL, USA) on rice. Immunoaffinity columns (Vicam AflaTest-Affinity Column) were used to analyse the aflatoxin content of the culture material and quantified via high performance liquid chromatography (HPLC) (Agillent 1100 Series) The AFs $B_1$, $B_2$, $G_1$ and $G_2$ standards were purchased from Sigma-Aldrich (Taufkirchen, Germany). The stock solutions, working standards and the calibration curve were prepared as described by Stroka *et al*. (2000). The total amount of AFs (AF $B_1$, $B_2$, $G_1$ and $G_2$) were 90.35 mg $kg^{-1}$ in rice powder and consisted of 83.33% AF $B_1$, 10.42% AF $G_1$, 4.17% AF $B_2$ and 2.08% AF $G_2$, based on the total AF in the rice powder.

**Experimental design and management:** In this study, 41 days old Ross 308 broiler chicks were distributed by similar body weight to four groups. The control group was fed a basal diet and the remaining groups received 0.75 g $kg^{-1}$ Mycosorb, 2 ppm AF and 2 ppm AF+0.75 g $kg^{-1}$ Mycosorb for 21 days. The chicks were housed in electrically heated batteries under fluorescent lighting and feed and water was supplied *ad libitum*. This study was approved by Ethics Committee of University of Kırıkkale, Faculty of Veterinary Medicine (Approval number, 17. 03. 2006-06/03).

**Biochemical assays:** At the end of the 21 day treatment period, blood samples were collected into heparinized test tubes from wein vena to determine lipid peroxidation and some non-antioxidant enzyme components. Plasma samples were separated by centrifugation at 3000 rpm for 10 min and stored -30°C until the analysis. Lipid peroxidation levels were measured with Thiobarbituric Acid Reactions (TBARs) in plasma by the method of Moreno *et al*. (2003). The values of TBARs material were expressed in terms of MDA. Plasma albumin (TECO, USA) and uric acid (DDS, Germany) levels were determined by a Shimadzu UV 1700 spectrophotometer using commercial kits. Plasma CP, vit A, vit C and vit E levels were determined by the methods of Colombo and Richeric (1964), Suzuki and Katoh (1990), Haag (1985) and Martinek (1964), respectively.

**Statistical analysis:** Statistical analysis of data were performed using the by SPSS 13. 0 version for Windows. One-way Analysis of Variance (ANOVA) was used for the differences between groups. When the F values were significant, Duncan's Multiple Range Test was performed. All data were expressed as means±SEM.

## RESULTS AND DISCUSSION

Aflatoxins were reported to produce free radicals. The free radicals generated by $AFB_1$, can be important mediators of damage to cell structures, nucleic acids,

Table 1: Plasma MDA and some non-enzymatic antioxidant levels of control and treatment groups in experimentally induced aflatoxicosis in broilers (n=10)

| | Group | | | | |
|---|---|---|---|---|---|
| Parameters | Control | Mycosorb | AF | AF+Mycosorb | p |
| MDA (μmol $L^{-1}$) | 1.95±0.36 | 1.80±0.45 | 2.44±0.63 | 2.23±0.52 | - |
| CP (mg $dL^{-1}$) | 2.76±0.46$^{a}$ | 2.13±017$^{ab}$ | 1.56±0.29$^{b}$ | 2.35±0.13$^{ab}$ | * |
| Uric acid (mg $dL^{-1}$) | 6.23±0.45 | 5.66±0.61 | 4.98±0.68 | 5.38±0.61 | - |
| Alb (g $dL^{-1}$) | 0.83±0.03$^{a}$ | 0.96±0.03$^{a}$ | 0.43±0.05$^{b}$ | 0.46±0.06$^{b}$ | *** |
| Vit A (μg $dL^{-1}$) | 137.02±16.23$^{a}$ | 111.92±17.78$^{ab}$ | 82.60±12.98$^{b}$ | 87.14±7.64$^{b}$ | * |
| β-carotene (μg $dL^{-1}$) | 187.54±11.31$^{a}$ | 166.20±9.35$^{ab}$ | 121.97±9.12$^{c}$ | 137.67±12.50$^{bc}$ | *** |
| Vit C (μg $mL^{-1}$) | 15.87±0.51$^{ab}$ | 18.61±0.86$^{a}$ | 14.75±1.32$^{b}$ | 14.81±0.97$^{b}$ | * |
| Vit E (mg $dL^{-1}$) | 3.64±0.20$^{a}$ | 3.52±0.15$^{ab}$ | 2.81±0.21$^{b}$ | 2.91±0.34$^{b}$ | * |

$^{a-c}$: Values within each row with different superscripts differ significantly, -:not significant (p>0.05), *: p<0. 05, **: p<0. 01, ***: p<0. 001

lipids and proteins and then expected to induce lipid peroxidation (Rastogi *et al*., 2001; Umarani *et al*., 2008). Antioxidants prevent Reactive Oxygen Species (ROS) concentrations from reaching a high-enough level within a cell that damage may occur (Seifried *et al*., 2007).

In this study, 2 ppm AF increased the level of plasma MDA slightly as an indicator of lipid peroxidation and this increase was statistically insignificant (p>0.05) in comparison to the control and 0.75 g $kg^{-1}$ mycosorb given groups (Table 1). Eraslan *et al*. (2005a) showed that erythrocyte MDA levels in broilers increased slightly in the groups administered 0.5 and 1 ppm AF on days 15 and 30. But these increases were statistically insignificant, whereas, these increases were found to be statistically significant on day 45. Eraslan *et al*. (2005a) suggested that the increase of MDA level has been observed at high doses and known to depend on the exposure period.

In this study, the levels of CP decreased significantly (p<0.05) in AF treatment group when compared to control and alb levels also decreased significantly (p<0.001) in AF and AF+ Mycosorb groups when compared to control and mycosorb groups. However, uric acid levels were not affected by these treatments (Table 1). The significant decreases in the level of alb of aflatoxicated group than to control are in agreement with the studies on broiler chickens (Basmacioğlu *et al*., 2005; Safameher and Shivazad, 2007). Ceruloplasmin and alb are proteins synthesized in the liver (Johnson *et al*., 1999). Aflatoxin can cause damage to the liver (Preetha *et al*., 2006; Ortatatli and Oguz, 2001; Coulombe, 1993). Thus levels of these protein synthesized in the liver may be decreased. The decrease of alb level may be due to the hepatic protein inhibition, a characteristic indicator of hepatotoxic effect of AF (Fernandez *et al*., 1994). Ceruloplasmin has antioxidative properties in biological systems. It can protect polyunsaturated fatty acids in cellular membranes from ROS (Kim *et al*., 1998). Ceruloplasmin and alb have the ability to bind copper ion in organisms. Binded copper ions could not catalyze free radical reactions. The antioxidants such as CP and alb show their activity by inhibiting the encountering of free radicals, which find in interstitial fluids, to a catalyzer metal ion like copper (Aslan and Dundar, 2000). Also decreasing of CP and alb in the plasma may cause an increase in free copper in organism and therefore, lead to deficiency of the defense system against lipid peroxidation (Kohen and Nyska, 2002; Agli *et al*., 1995). Copper ion does not initiate the lipid peroxidation, in fact it catalysts the chain reaction of lipid peroxidation and degration of the synthesized lipid hydroperoxides, so the free radicals become more harmful (Akkuş, 1995).

The levels of Vit A decreased (p<0.05) significantly in AF treatment group when compared to control in this study (Table 1). There have been many reports indicated that AF administration effected the levels of serum Vit A and β-carotene in broilers (Salmanoğlu, 2002; Altıntaş *et al*., 2003). Salmanoğlu (2002) reported that AF decreased the levels of serum Vitamin A and β-carotene on 21 days of broilers. Our results agree with the findings of Salmanoğlu (2002). In some studies (Ergun *et al*., 2006; Sehu *et al*., 2007) it was indicated that there is a decrease in the number of ribosomes in hepatocytes and this lead to a decrease in protein synthesis. Thus, a decrease may be seen in the retinol binding protein, a protein that carries Vit A inside and outside of the cell (Salmanoğlu, 2002). $AFB_1$ can reduce the level of Vit A *in vivo* and this effect on Vit A, is correlative with induction of hepatic microsome cytochrome P 450 (Liu and Zhou, 1989).

Significantly decreases (p<0.05) were obtained in the vit C levels in AF and AF+ Mycosorb group than to mycosorb group and the level of β-carotene also decreased significantly (p<0.001) in AF treatment group when compared to both control and Mycosorb treatment group in this study (Table 1). Vitamin C is the most important antioxidant found in extracellular fluids. It can protect biomembranes against lipid peroxidation damage by eliminating peroxyl radicals before the latter can initiate peroxidation (Mcdowell, 2002). Vitamin C shows antioxidant activity by enhancing the immunity via maintaining the functional and structural integrity of important immune cells (Mcdowell, 2002). Aflatoxins inhibit immune function (Oguz *et al*., 2003). This may be

the reason why Vit C and β-carotene levels were decreased in all aflatoxin given groups. Yeast glucomannan was found to have beneficial effects on immune response in broilers exposed to AF (Stanley *et al*., 1993). Beta carotenes are known to stimulate immune function (Kalaycioglu *et al*., 2000). This can explain the reason why the levels of Vit C and β- carotene was high in Mycosorb given group.

Vitamin E is one of the major antioxidants. It can limit lipid peroxidation by terminating chain reactions initiate in the membrane lipids (McDowell, 2000). Significant decreases ($p<0.05$) were determined in vit E in AF and AF+Mycosorb group than to control in this study (Table 1). It might be due to the excessive utilization of vitamin E for scavenging free radicals produced under these conditions (Umarani *et al*., 2008). We could not find any study about the level of plasma Vitamin E and C in aflatoxicated animals. However, decrease of some antioxidant vitamin levels of aflatoxin treatment group may be related to increased utilization for scavenging of ROS (2008). Wilson *et al*. (1975) reported that damage caused by aflatoxin to hepatic cells, had decreased the synthesis of bile salts, which would affect the absorption of the fat-soluble vitamins. Also AF is known to cause damage in the gastrointestinal system. This may impair the intestinal adsorption and increase excretion. Thus, AF may decrease vitamin absorption and reduce their levels in body and hence weaken the antioxidant defense mechanism (Eraslan *et al*., 2005).

## CONCLUSION

The results of this study showed that subacute toxication of 2 ppm AF in feed did not cause lipid peroxidation significantly, however decreased some non-enzymatic antioxidants and 0.75 g $kg^{-1}$ Mycosorb was not sufficient to prevent the adverse effects of AF in contaminated feed in broilers.

## REFERENCES


Agil, A., C.J. Fuller and I. Jialal, 1995. Susceptibility of plasma to ferrous iron/hydrogen peroxide-mediated oxidation: demonstration of a possible Fenton reaction. Clin. Chem., 41: 220-225.

Akkuş, 1995. Serbest radikaller ve fizyopatolojik etkileri. Mimoza Basım, Yayım ve Dağıtım A.Ş. Konya, pp: 1-133.

Altnta, A., A. Bilgili, T. Konta and G. Eraslan, 2003. Diyetle aflatoksin alan broylerlerde karaci er ve serum vitamin A-karotin düzeyleri. Ankara Üniv. Vet. Fak. Derg., 50: 1-6.

Aslan, R. and Y. Dündar, 2000. Kan Vizkositesi ve Oksidan Stres. In: Hekimlikte Oksidatif Stres ve Antioksidanlar, Dündar, Y. and R. Aslan (Eds.). Afyon Kocatepe Yayınları, Afyon, pp: 47-58.

Basmacıoğlu, H., H. Oğuz, M. Ergul, R. Col and Y.O. Bırdane, 2005. Effect of dietary esterified glucomannan on performance, serum biochemistry and haematology in broilers exposed to aflatoxin. Czech. J. Anim. Sci., 50: 31-39.

Buyukokuroglu, M.E., M. Cemek, Y. Yurumez, Y. Yavuz and A. Aslan, 2008. Antioxidative role of melatonin in organophosphate toxicity in rats. Cell Biol. Toxicol., 24: 151-158.

Çam, Y., G. Eraslan, A. Atasever, M. Eren, B.C. Liman and N. Şeybek, 2008. Efficacy of N-acetylcysteine on aflatoxicosis in rabbits. Polish J. Environ. Stud., 17: 189-197.

Colombo, J.P. and R. Richerich, 1964. Zur bestimmung des ceruloplazmin in plazma. Schweiz. Med. Wschr., 94: 715-720.

Coulombe, R.A., 1993. Biological action of mycotoxins. J. Dairy Sci., 76: 880-891.

Demet, O., H. Oğuz, I. Celik and F. Nizamlıoğlu, 1995. Pirinçte aflatoksin üretilmesi. Vet. Bil. Derg. 11: 19-23.

Diaz, D.E., W.M. Jr. Hagler, B.A. Hopkins and L.W. Whitlow, 2002. Aflatoxin binders I: *in vitro* binding assay for aflatoxin B1 by several potential sequestering agents. Mycopathology, 156: 223-226.

Dündar, Y. and R. Aslan, 1999. Oksidan-antioksidan denge ve korunmas nda vitaminlerin rolü. Hay. Ara. Derg., 9: 32-39.

Eraslan, G., M. Akdogan, E. Yarsan, D.Essiz, F. Sahindokuyucu, S.E. Hismiogullari and L. Altintas, 2004. Effects of aflatoxin and sodium bentonite administered in feed alone or combined on lipid peroxidation in the liver and kidneys of broilers. Bull. Vet. Inst. Pulawy., 48: 301-304.

Eraslan, G., M. Akdogan, E.Yarsan, F. Sahindokuyucu, D. Essiz and L. Altintas, 2005a. The effects of aflatoxins on oxidative stress in broiler chickens. Turk. J. Vet. Anim. Sci., 29: 701-707.

Eraslan, G., Y. Çam, M. Eren, B.C. Liman, Ö. Atalay and N. Seybek, 2005b. Aspects of using N-Acetylcysteine in aflatoxicosis and its evaluation regarding some lipid peroxidation parameters in rabbits. Bull. Vet. Inst. Pulawy., 49: 243-247.

Ergün, E., L. Ergün and D. Eşsiz, 2006. Light and electron microscopic studies on liver histology in chicks fed aflatoxin. Dtsch Tierarztl Wochenschr., 113: 363-8.

Fernandez, A., M. Verde, M. Gascon, J. Ramos, J. Gomez, D.F. Luco and G. Chavez, 1994. Variations of clinical, biochemical parameters of laying hens and broiler chickens fed aflatoxin-containing feed. Avian Pathol., 23: 37-47.

Haag, W., 1985. Zur methodik und praktischen Bedeutung der C vitamin-bestimmung beim rind in vergangenheit und gegenwart, Inaugural Dissertation, Justus Liebig Universitaet, Giessen.

Huwig, A., S. Friemud, O. Kappeli and H. Dutler, 2001. Mycotoxin detoxication of animal feed by different adsorbents. Toxicol. Lett., 122: 179-188.

Jansen Van Rensburg, C., C.E.J. Van Rensburg, J.B.J Van Ryssen, N.H. Casey and G.E. Rottinghaus, 2006. *In vitro* and *in vitro* assessment of humic acid as an aflatoxin binder in broiler chickens. Poult. Sci., 85: 1576-1583.

Johnson, A.M., E.M. Rohlfs and L.M. Silverman, 1999. Proteins. In: Tietz Textbook of Clinical Chemistry, 3nd Edn., Eds: Burtis, C. A. and E.R. Ashwood, W.B. Saunders Company, pp: 477-540.

Kalaycıoglu, L., B. Serpek, M. Nizamlioglu, N. Başpınar and A.M. Tiftik, 2000. Vitaminler. In: Biyokimya. Nobel. Ankara, pp: 332-381.

Karaman, M., H. Basmacioglu, M. Ortatatli and H.Oguz, 2005. Evaluation of the detoxifying effect of yeast glucomannan on aflatoxicosis in broilers as assessed by gross examination and histopathology. Br. Poult. Sci., 46: 394-400.

Kaya, S., 2002. Mikotoksinler. In: Veteriner Hekimli Inde Toksikoloji (Eds.). Kaya S, Pirinçci İ., Bilgili, A 2. Bask Medisan Ankara, pp: 537-573.

Kim, I.G., S.Y. Park, K.C. Kim and J.J. Yum, 1998. Thiol-linked peroxidase activity of human ceruloplasmin. FEBS Lett., 431: 473-475.

Kohen, R. and A. Nyska, 2002. Oxidation of biological systems: oxidative stress phenomena, antioxidants, redox reactions and methods for their quantification. Toxicol. Pathol., 30: 620-650.

Liu, X.L and Y.Z. Zhou, 1989. The effect of aflatoxin B1 on vitamin A status and on microsomal mixed function oxidase in male mouse. Zhonghua Yu Fang Yi Xue Za Zhi., 23: 218-221 (Abstract).

Martinek, R.G., 1964. Method for determination of vitamin E (total tocopherols) in serum. Clin. Chemist., 10: 1078-1086.

McDowell, L.R., 2000. Vitamins in Animal and Human Nutrition. 2nd Edn. Iowa State University Press Inc. San Diego New York, pp: 1-810.

Moreno, I.M., A. Mate, G. Repetto, C.M. Vazquez and A.M. Camean, 2003. Influence of Microcystin-LR on the activity of membrane enzymes in rat intestinal mucosa. J. Physiol. Biochem., 59: 293-300.

Oguz, H., H.H. Hadimli, V. Kurtoglu and O. Erganiş, 2003. Evaluation of humoral immunity of broilers during chronic aflatoxin (50 and 100 ppb) and clinoptilolite exposure. Rev. Med. Vet., 154: 483-486.

Ortatatli, M. and H. Oguz, 2001. Ameliorative effects of dietary clinoptilolite on pathological changes in broiler chickens during aflatoxicosis. Res. Vet. Sci., 71: 59-66.

Preetha, S.P, M. Kanniappan, E. Selvakumar, M. Nagaraj and P. Varalakshmi, 2006. Lupeol ameliorates aflatoxin B1-induced peroxidative hepatic damage in rats. Comp. Biochem. Physiol, Part C., 143: 333-339.

Raju, M.V.L.N. and G. Devegowda 2000. Influence of esterified-glukomannan on performance and organ morphology, serum biochemistry and haematology in broiler exposed to individual and combined mycotoxicosis (aflatoxin, ochratoxin and T-2 toxin). Br. Poult. Sci., 41: 640-665.

Rastogi, R., A.K. Srivastava and A.K. Rastogi, 2001. Long term effect of aflatoxin B1 on lipid peroxidation in rat liver and kidney: Effect of picroliv and silymarine. Phytother Res., 15: 307-310.

Safameher, A. and M. Shivazad, 2007. The effects of saccharomyces cerevisiae on performance and biochemical parameters in broiler chicks during aflatoxicosis. J. Anim. Vet. Adv., 6: 917-919.

Salmanoğlu, B., 2002. Broyler civcivlerde aflatoksin B1'in karaciğer vitamin A depolamas üzerine etkisi. Turk. J. Vet. Anim. Sci., 26: 1341-1344.

Sehu, A., S. Cakir, O. Cengiz and D. Eşsiz, 2005. MYCOTOX and aflatoxicosis in quails. Br. Poult. Sci., 46: 520-4.

Sehu, A., L. Ergün, S. Cakir, E. Ergün, Z. Cantekin, T. Sahin, D. Essiz, B. Sareyyüpo lu, Y. Gürel and Y. Yiğit, 2007. Hydrated sodium calcium aluminosilicate for reduction of aflatoxin in quails (Coturnix coturnix japonica). Dtsch Tierarztl Wochenschr., 114: 252-259.

Seifried, H.E., D.E. Anderson, E.I. Fisher and J.A. Milner, 2007. A review of the interaction among dietary antioxidants and reactive oxygen species. J. Nutr. Biochem., 18: 567-579.

Shatwell, D.L., C.W. Hesseltine, R.D. Stubblefield and W.G. Sorensor, 1966. Production of aflatoxin on rice. Applied Microbiol., 14: 425-428.

Stanley, V.G., R. Ojo, S. Woldensenbent, D.H. Hutchinson and L. F. Kubena, 1993. The use of Saccharomyces cerevisiae to suppress the effects of aflatoxicosis in broiler chicks. Poult. Sci., 72: 1867-1872.

Stroka, J., E. Anklam, U. Jorıssen and J. Gılbert, 2000. Immunoaffinity column cleanup with liquid chromatography using post-column bromination for determination of AF in peanut butter, pistachio paste, fig paste and paprika powder: collaborative study. J. AOAC Int., 83: 320-340.

Suzuki, J. and N. Katoh, 1990. A simple and cheap methods for measuring serum vitamin A in cattle using only a spectrophotometer. Jpn. J. Vet. Sci., 52: 1281-1283.

Toskulkao, C and T. Glinsukon, 1988. Hepatic lipid peroxidation and intracellular calcium accumulation in ethanol potentiated aflatoxin B1 toxicity. J. Pharmacobio. Dyn., 11: 191-197.

Umarani, M., P. Shanthi and P. Sachdanandarm, 2008. Protective effect of Kalpaomruthaa in competing the oxidative stress posed by aflatoxin B1-induced hepatocellular carcinoma with special reference to flavonoid structure-activity relationship. Liver Int., pp: 200-213.

Wilson, H.R., C.R. Douglas, R.A. Harms and G.T. Edds. 1975. Reduction of aflatoxin effects on quail. Poult. Sci., 54: 923-925.