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Full Length Research Article

EFFECT OF AFLATOXIN AND TOXIN BINDERS ON THE SERUM GLUCOSE LEVEL OF BREEDING JAPANESE QUAILS, COTURNIX COTURNIX JAPONICA

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ABSTRACT

Mycotoxins play a global role in human and animal health. Molds in poultry feed are a source of significant economic loss to poultry producers. Molds adversely influence performance of animals by altering the nutrient composition of feed ingredients, decreasing the efficiency of nutrient utilization, and by producing toxic secondary metabolites. This work was carried out to study the role of locally available herbal plant extracts such as *Azadirachta indica, Cynodon dactylon* and *Curcuma longa* in counteracting the effects of experimental aflatoxicosis in laying Japanese quail by feeding them with diets containing aflatoxin at 1.5 and 3ppm levels for a period of six weeks.

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INTRODUCATION

Poultry performance and efficient disease prevention are obvious goals for the poultry industry worldwide. Most disease problems in poultry today are usually caused by interaction of many factors where immunosuppression plays a key role. There are varieties of agents that can impair the normal resistance mechanism of birds. The presence of such agents in modern poultry operation is of great importance as they can directly affect performance of birds and incidence of diseases. One of the most common immunosuppressive agents in poultry is mycotoxin produced by fungi. Fungi produce an array of toxic chemical byproducts. Penicillin is an example of a secondary metabolite produced by fungi that has medicinal applications. Unfortunately, not all secondary metabolites are as useful as penicillin.

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Mycotoxins are one such secondary metabolites of fungal origin that are toxic to animals and humans. Mycotoxins play a global role in human and animal health some of their effects are well documented while others are not. Examples of the known biological activity of the major Fusarium mycotoxins include the fumonisins (leukoencephalomalacia and promoters of carcinogenesis), T-2 toxin (hemorrhagic), deoxynivalenol (feed refusal), zearalenone (estrogenism) and fusarochromanone (bone deformation).

MATERIALS AND METHODS

Laying Japanese quail, *Coturnix coturnix japonica* was selected for the present investigation. The birds, about 13 weeks of age were obtained from Poultry Research station, nandanam, Chennai. *Aspergillus parasiticus* strain NRRL 2999 was used to produce aflatoxin. Inoculum was prepared by inoculating the tubes of potato-dextrose agar slant with spores of *Aspergillus parasiticus* NRRL 2999. *Aspergillus parasiticus* were scraped with a sterilized inoculating wire and the spores were spread on the slant of the agar medium. The inoculated test tubes were placed undisturbed for about 7-11 days. On the 11th day, a velvety growth of green spores of Aspergillus parasiticus was seen. Aflatoxin was produced by inoculating the cultured Aspergillus parasiticus spores on rice culture (Shotwell et al., 1966). The aflatoxin was extracted and estimated Pons et al. (1966). Leaves of Azadirachta indica and Cynodon dactylon and rhizome of Curcuma longa were collected from locally available plants. They were sun dried and ground into a fine powder. Aminovit was obtained from Intervet India Pvt. Ltd, Pune. The present study dealt with the effect of dietary aflatoxin (1.5 and 3 ppm) on laying Japanese quails and the role of herbal extracts such a Azadirachta indica, Cynodon dactylon and Curcuma longa in counteracting the negative effects of dietary aflatoxin. The experimental groups of birds were divided into 12 groups. Animals within each treatment groups were treated daily with dietary aflatoxin for a period of six weeks. The different dietary treatments were as follows:

Group1: Control- without any dietary aflatoxin feed.

Group2: Fed with estimated amount of aflatoxin containing diet (1.5 ppm).

Group3: Fed with estimated amount of aflatoxin (1.5 ppm) containing diet along with powdered extract of *Azadirachta indica*.

Group4: Fed with estimated amount of aflatoxin (1.5 ppm) containing diet along with powdered extract of *Cynodon dactylon*.

Group5: Fed with 1.5ppm aflatoxin containing diet along with powdered extract of *Curcuma longa*.

Group6: Fed with 1.5ppm aflatoxin containing diet with powdered extract mixture of all the above three mentioned herbals.

Group7: Fed with estimated amount of aflatoxin (3 ppm).

Group8: Fed with estimated amount of aflatoxin (3 ppm) containing diet along with aminoacid supplementation.

Group9: Fed with estimated amount of aflatoxin (3 ppm) containing diet along with powdered extract of *Azadirachta indica*.

Group10: Fed with 3ppm aflatoxin containing diet along with powdered extract of *Cynodon dactylon*.

Group11: Fed with 3ppm aflatoxin containing diet along with powdered extract of *Curcuma longa*.

Group12: Fed with 3ppm aflatoxin containing diet along with powdered extract mixture of all the above three mentioned herbals.

At the end of the sixth week experimental period the birds were sacrificed and the samples of blood collected in test tubes were allowed to clot and centrifuged at 1500 rpm for 20 min to separate the sera. Serum samples were analyzed individually for total protein, albumin, globulin, glucose, cholesterol, serum glutamate oxaloacetate transaminase (SGOT), serum glutamatepyruvate transaminase (SGPT), gamma- glutamyl transferase (GGT) and lactate dehydrogenase (LDH) using colorimeter or BTS 320 Semi-auto analyzer.

RESULTS AND DISCUSSION

The mean $(\pm S.E)$ of serum glucose levels (mg/dl) in laying Japanese quail treated with dietary aflatoxin is shown in Table 1 A highly significant (P < 0.01) difference was observed between the control and treated groups at the end of 6 weeks. The serum glucose level showed a gradual decrease with increasing concentration of the aflatoxin. The groups fed with diet containing aflatoxin and toxin binders showed higher levels of blood sugar as compared to the quails fed with aflatoxin alone. Aminovit fed quails also showed a significant depression in the serum glucose levels. At the end of the sixth weeks, the experiment showed, a significant decrease of 4.64% and 7.87% in the mean serum glucose levels in the 1.5 ppm and 3 ppm aflatoxin fed quails respectively. Panda et al. (1987) also reported a gradual reduction in the blood glucose levels of quail consequent to feeding of aflatoxin. The blood glucose level varies in poultry in the case of fatty liver which is a major outcome of aflatoxicosis. For this reason, the glucose level is taken into consideration in aflatoxicosis.

 Table 1. Effect of aflatoxin and toxin binders on mean serum glucose (mg/dl) in Breeding Japanese quail

Treatments	Blood sugar $(Mean \pm S.E.)^*$	
T1	$174.93^{a} \pm 0.46$	
T2	$166.80^{\circ} \pm 0.53$	
Т3	$170.49^{cd} \pm 0.58$	
T4	$171.00^{cd} \pm 0.53$	
Τ5	$170.97^{cd} \pm 0.83$	
T6	$174.52^{ab} \pm 0.47$	
Τ7	$161.16^{\rm f} \pm 0.78$	
Т8	$159.75^{\rm f} \pm 0.59$	
Т9	$169.69^{cd} \pm 0.58$	
T10	$170.55^{cd} \pm 0.72$	
T11	$169.04^{de} \pm 1.44$	
T12	$171.97^{bc} \pm 0.83$	

^{*}Means carrying atleast one common superscripts do not differ significantly $(P \ge 0.01)$.

ANOVA FOR SERUM GLUCOSE

Source	DF	SS	MS	F
Treatments	11	2864.08	26.3713	
Error	132	879.35	6.6617	39.08**
Total	143	3743.43		

[∗]Highly Significant (P≤0.01)

The aflatoxin induced liver injury probably induces glycogen synthesis in the liver by inhibiting phosphorylase or by stimulating glycogen synthetase resulting in low levels of glucose in the aflatoxin fed quail. Supportively Kumar *et al.* (1993) fed broiler chicks aged 15 days with aflatoxin for 30 days and found that there was a marked depression in the levels of serum glucose. However, Johri *et al.* (1990) reported that blood glucose was not influenced by various dietary treatments of aflatoxin. Contrarily, Chang and Hamilton (1982) reported that feeding different levels of aflatoxin/g to Japanese quail from 0-4 weeks did not alter serum lipids and glucose concentration.

However, the various kinds of herbal toxin binders fed in combination with aflatoxin tend to bind with the aflatoxin in the digestive tract of quail, but the binding was not at the rate of 100%. Thus it could be explained that the unbound part of aflatoxin might be responsible for the adverse effects in the group of quail fed with aflatoxin coupled with toxin binders. However, the toxin binder not only inhibit the absorption of aflatoxin from the digestive tract but it might also play a role in reversing the liver enzyme inhibitions brought about by aflatoxin leading to normalizing the levels of glucose.

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