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Antioxidant activity of Selenium in Cherry Valley Ducks fed Aflatoxin B1

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Abstract: This study was conducted to determine the ameliorative effects of selenium in cherry valley ducks. For this purpose a total of 180 one-day-old cherry valley ducks were randomly divided into three groups. Group I was used as a blank control. Ducks in group II and group III on day 7 were fed a 50μg/kg of AFB1–contaminated diet for 2 weeks then received normal saline (group I and group II) or sodium selenite (group III) intragastrically administered for 6 days. Ducks were sacrificed at days 2, 4 and 6 after treatment of the three groups. The activity of serum glutamic-pyruvic transaminase (GPT), glutamic oxaloacetic transaminase enzymes (GOT) and glutathione S-transferase (GST) were increased significantly in the group II, but the activity of superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px) were significantly decreased. Furthermore, the concentration of malondialdehyde (MDA) significantly increased in group II, compared with group I. The level of malondialdehyde was decreased, but the activity of superoxide dismutase and glutathione peroxidase in group III was increased as compared with group II. In addition, AFB1 treatment induced significant hepatocyte apoptosis in group II, and sodium selenite treatment ameliorated these changes in group III. These results indicate that selenite may have effects against the toxicity induced by AFB1 that contains antioxidant and anti-hepatocyte apoptosis properties.

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1. Introduction

Aflatoxins are a group of fetal toxic metabolites that are harmful to the health of certain animals, especially sensitive species such as turkey and duck (Hussein et al., 2001). AflatoxinB1 (AFB1) is the main toxic metabolite to cause low level of feed intake, growth rate, feed utilization and induce liver injury or immunosuppression on poultry (Gómez, 2009; A.P. Magnoli et al., 2011; Xin-Yan Han et al., 2008). AFB1 is thought to be the main residue in liver and eggs after AFs exposure in poultry. Liver is the main target organ for AFs; it may promote liver fibrosis or liver cancer occurrence when there is chronic exposure to low levels AFB1 in foodstuffs (Denli et al., 2009). Furthermore, the liver cell injury of AFB1 was demonstrated through changes in serum biochemical parameters, such as increased activity of glutamic oxaloacetic transaminase enzymes,serum glutamic-pyruvic transaminase and glutathione S-transferase (Basmacioglu et al.,2005). The International Agency for Research on Cancer (IARC) classify AFB1 as a group I carcinogen, because aflatoxinB1 may induce prolonged genetic damage in mice exposed to aflatoxin (Roongtiwa Wattanawaraporn et al., 2012).

Negative effects of AFB1 include cell damage, release of free radicals and lipid peroxidation. Since lipid peroxidation plays a major role in the toxicity of AF, a protective effect of antioxidants is possible (Nisarani et al.,2009;Choi,K. C et al., 2010; Aziza A. El-Nekeetya et al.,2011). There is direct evidence that some antioxidant would reduce aflatoxicosis and genotoxicity of AFB1 (FatimeGeyikoglu et al.,2006). Glutathione peroxidase can reduce reactive products by reducing H2O2 and lipid hydroxides. Selenium is essential to guarantee activity, so the antioxidant capacity of ducks intoxicated with AFB1 increased by selenium is shown in a recent study (Hoekstra W C, 1975;Da-you Shi et al.,2012). Selenium can reduce oxidative stress, DNA damage, and apoptosis in hepatocytes (Aiguo Wang et al.,2004; Jie Yu et al., 2009). We therefore deduce there may be relationship between oxidative stress and hepatocyte apoptosis in duck.

Our study was undertaken to investigate oxidative stress and hepatocyte apoptosis induced by AFB1 and also estimated the effect of selenium on antioxidant capacity and anti-hepatocyte apoptosis in Cherry Valley Ducks administered with AFB1.

2. Materials and Methods

2.1 Drugs and Chemicals

Detection kits which include glutamic-pyruvic transaminase (GPT, Cat NO.C009-2), glutamic oxaloacetic transaminase enzymes (GOT, Cat NO.C010-2), glutathione S-transferase (GST, CatNO. A004), superoxide dismutase (SOD, Cat NO.A001-3), glutathione peroxidase (GSH-Px, Cat NO.A005), and malondialdehyde (MDA, Cat NO. A003-4) were purchased from Nanjing Jiancheng Bioengineering Institute. Standards of AFB1 purchased from Sigma (Sigma-Aldrich, St. Louis, MO). Annexin V-FITC Kit (Cat NO. LHK601-100) were purchased from Becton, Dickinson and Company.

2.2 AFB1 synthesis

AFB1 was produced via fermentation of rice according to the method of shotwell et al. (1966), Aspergillusflavus (CICC2219) was purchased from the China center of industrial culture collection. The rice culture medium was placed in a conical flask, and inoculated with 2 mL of aqueous suspension of spores. Cultures were cultured for 7 days at 28°C in a water jacket thermostatic constant incubator. On the seventh day, the conical flasks were autoclaved, and the rice was dried for 48 h at 40°C in a forced-air oven and then ground to a fine powder. The AFB1 contents in the rice powder were measured by HPLC as described in the data section (Jun-Ho Hwang et al., 2006). The rice contaminated with AFB1 was added to the basal diet at a concentration is 50μg of AFB1/kg of feed. Aflatoxin B1 content was 5 and 6μg /kg in the growing and finishing basal diets.

2.3 Animal and Experimental Design

A total of 180 one-day-old cherry valley ducks from a large hatch company were used in the study. Ducks were weighed (59±1g) and divided into three groups, which were fed at the department of pharmacy animal research center of the Sichuan Agriculture University. Ducks in group Ⅰ were used as the control and fed basal diet; Ducks in group Ⅱ were used as the AFB1 treated group; and Ducks in groupⅢ were the selenium treated group. Ducks in group Ⅱ and group Ⅲ were fed AFB1 (50 μ g/kg) contaminated diet from day 8 to 21. Then ducks in groupⅢwere administered sodium selenite water solution intragasterically (1.53 mg/kg body weight), Ducks in group Ⅰ and group Ⅱ were administered an equivalent saline from day 22 to 27 intragasterically. Diets were compounded according to the NRC (1994) recommendations, and met the nutrient requirements of ducks from day 1 to 21 and 22 to 27. Ingredient formulation and nutrients are presented in Table 1. The three groups of ducks were raised in separate animal rooms, each with a pool,. Sufficient food and clean drinking water was provided throughout the experiment.

Table 1. Composition and nutrient levels of the diets

Item	Grower	Finisher
Ingredients (%)		
Corn	58.40	66.00
Soybean meal	31.80	25.00
Wheat middling	5.00	5.00
Vegetable oil	0.70	0.60
Unite bran	0.46	
CaHPO4	1.65	1.53
Calcium powder	1.11	1.08
Methionine	0.17	0.14
Lysine	0.06	
Choline chloride(50%)	0.10	0.10
Salt	0.30	0.30
Vitamin premix[1]	0.10	0.10
Mineralpremix[2]	0.15	0.15
Composition[3]		
ME, Mcal/kg	2900	2950
CP, %	19.74	17.53
Ca, %	0.95	0.87
P, %	0.65	0.61
AFB1(μg/kg)	50	6

[1] Vitamin premix supplied per kilogram: Growers vitamin A, 3000 IU; vitamin B1,3 mg; vitamin B6, 3 mg; biotin, 0.1 mg; choline, 1650 mg; folic acid,1 mg; vitamin B12, 0.02 mg; vitamin D3, 600 IU; vitamin E, 8 IU; vitamin K, 2 mg; riboflavin, 5 mg; D-pantothenic acid, 11 mg; nicotinic acid,60 mg; Finisher: vitamin A, 2500 IU; vitamin B1, 3 mg; vitamin B12, 0.02 mg; vitaminD3, 500 IU; vitamin E, 8 IU; vitamin K, 2 mg; riboflavin, 5 mg; D-pantothenic acid, 11 mg; nicotinic acid, 55 mg; vitamin B6, 3 mg; biotin, 0.1 mg; choline, 1400 mg; folic acid, 1 mg;

[2]Mineral premix supplied per kilogram: Growers:Cu,8 mg; Se, 0.15 mg; I,0.45 mg Fe, 96 mg; Mn, 30 mg; Zn, 60 mg;. Finisher: Cu, 8 mg; Se,0.15 mg; I,0.45 mg Fe, 96 mg; Mn, 80 mg; Zn, 60 mg;.

[3] Measured values.

2.4 Sampling

On 2th, 4th, and 6th days after treatment, 10 ducks were randomly selected from each group and fasted 12h before sampling. The blood samples were collected by severing the jugular vein. Then the blood samples were centrifuged for 10 min at 4000g, and serum was transferred to Eppendorf tubes from each group. All of the serum samples were frozen at -20°C for analysis.

2.5 Assessment of Serum Biochemistry and Antioxidant Capacity

The activities of GPT, GOT, GST, SOD and GSH-Px were detected according to direction that the kit company had recommended. The content of MDA was also detected.

2.6 Assessment of Hepatocyte Apoptosis

The liver tissue samples were removed for hepatocyte apoptosis analysis from the different groups. Liver tissue was cleaned with phosphate buffer and transferred to Eppendorf tubes (10ml) for apoptosis analysis. Annexin-PI apoptotic assay was performed using Annexin V-FITC Kit (Becton,Dickinson and Company. FAC scan Becton-Dickinson (BD) flow-cytometer was used for apoptosis testing and cell quest software was used for data statistics.

2.7 Statistical Analysis

Data were presented as the mean ± S.D. and the difference was assessed by a one-way analysis of variance. The significance statistical significance was $P<0.05$.

3. Results

3.1 Serum Biochemistry

As shown in Table 2, GOT activity was significantly increased on 2th, 4th and 6th days after the ducks were fed the AFB1-containing diet. The activity of GOT was increased by 129.21%, 87.51%, and 96.05% respectively, compared to the control group ($p<0.01$).

Table 2 The activity of serum GOT (U/L)

groups	Three detected times after treatment (days)		
	2	4	6
I	6.47±0.74	7.61±0.78	7.85±0.42
II	14.83±1.22**	14.27±1.59**	15.39±1.93**
III	11.14±1.69	13.04±1.20	14.63±1.11

*P<0.05, ** P<0.01, AFB1 treated group (group II) compared with control group (group I); AFB1 and selenite treated group (group III) compared with AFB1 treated group (group II).

However, concerning the ducks that were fed AFB1 contaminated diet plus selenium, GOT activity was not significant difference, compared to the AFB1intoxed group.

According to the information in Table 3, the GPT activity significantly increased on 2nd, 4th and 6th after the ducks were fed AFB1 diet. The GPT activity increased by 114.11%, 109.36%, and 94.60% respectively, compared to the control group ($p<0.01$).

Table 3 The activity of serum GPT(U/L)

groups	Three detected times after treatment (days)		
	2	4	6
I	15.38±0.65	16.88±0.34	16.31±0.59
II	32.93±1.72**	35.34±1.97**	31.74±1.69**
III	31.21±1.14	29.47±1.73	22.27±1.35*

*P<0.05, ** P<0.01, AFB1 treated group (group II) compared with control group (group I); AFB1 and selenite treated group (group III) compared with AFB1 treated group (group II).

However, concerning the ducks that were fed AFB1 contaminated diet plus selenium, the GPT activity was not significantly different on the 2nd and 4th days but decreased by 29.84% ($p<0.05$) on 6th after the sodium selenite intragastrically administered treatment, compared to the control group.

Table 4 shows that GST activity was significantly affected on 2nd, 4th and 6th days after the ducks were fed the AFB1 diet. The activity of GST increased by 107.69%, 114.60%, and 79.86% respectively, compared to control group ($p<0.01$). In group III ducks fed the AFB1 diet plus selenium, the activity of GST was not of significant difference, compared to the group II.

Table 4 The activity of serum GST(U/mL)

groups	Three detected times after treatment (days)		
	2	4	6
I	11.58±0.23	12.60±0.12	13.21±0.22
II	24.05±1.97**	27.04±1.73**	23.76±2.14**
III	24.07±1.34	27.25±2.07	24.40±1.57

*P<0.05, ** P<0.01, AFB1 treated group (group II) compared with control group (group I); AFB1 and selenite treated group (group III) compared with AFB1 treated group (group II).

Antioxidant Capacity

Table 5 shows that SOD activity was significantly affected on 2nd, 4th and 6th days after the ducks were fed the AFB1 diet. The SOD activity decreased by 11.35%, 11.12%, and 9.80% respectively, compared to the control group (p<0.05).

Table 5 The activity of serum SOD(U/mL)

groups	Three detected times after treatment (days)		
	2	4	6
I	34.88±1.15	35.89±2.55	33.38±3.04
II	30.92±2.55*	31.90±3.39*	30.11±3.00*
III	41.31±3.18**	39.61±3.86**	37.91±2.44**

*P<0.05, ** P<0.01, AFB1 treated group (group II) compared with control group (group I); AFB1 and selenite treated group (group III) compared with AFB1 treated group (group II).

However, the ducks that were fed the AFB1 diet plus selenium, displayed SOD activity which was increased by 25.15%, 20.89%, and 20.58% respectively, compared to the control group (P<0.01).

Table 6 demonstrates that GSH-PX activity was significantly affected on 2nd, 4th and 6th days after ducks were fed the AFB1 diet. GSH-PX activity decreased by 23.74%, 19.27%, and 32.53% respectively, compared to the control group (p<0.05).

Table 6 The activity of serum GSH-Px (U/mL)

groups	Three detected times after treatment (days)		
	2	4	6
I	125.14±11.63	124.57±16.35	125.14±11.42
II	95.43±14.18*	100.52±17.37*	84.43±14.31*
III	193.14±13.78**	313.14±18.90**	307.43±19.23**

* P<0.05, ** P<0.01, AFB1 treated group (group II) compared with control group (group I); AFB1 and selenite treated group (group III) compared with AFB1 treated group (group II).

Ducks that were fed AFB1 diet plus selenium, showed the GSH-Px activity significantly increased by 102.39%, 211.52%, and 264.12% respectively, compared to the group II (P<0.01).

According to the data in Table 7, the concentration of MDA was significantly affected on 2nd, 4th and 6th days after ducks were fed the AFB1 diet. The concentration of MDA increased by 114.35%, 126.79% and 124.56% respectively, compared to the control group (p<0.01).

Table 7 The content of serum MDA(nmol/mL)

groups	Three detected times after treatment (days)		
	2	4	6
I	6.27±0.06	5.60±0.17	6.27±0.13
II	13.44±0.77**	12.70±0.56**	14.08±0.17**
III	7.62±0.69**	6.03±0.40**	7.62±0.12**

* P<0.05, ** P<0.01, AFB1 treated group (group II) compared with control group (group I); AFB1 and selenite treated group (group III) compared with AFB1 treated group (group II).

Ducks that were fed the AFB1 diet plus selenium, showed that the concentration of MDA decreased by 43.30%, 52.52%, and 45.88% respectively, compared to the group II (P<0.01).

3.2 Percentage of Hepatocyte Apoptosis

Data in Table 8 and figure 1 show the percentage of hepatocyte apoptosis was significantly affected on 2nd, 4th and 6th days after ducks were fed the AFB1 diet. The percentage of hepatocyte apoptosis increased by 77.01%, 58.81% and 63.44% respectively, compared to the control group (p<0.05).

Table 8 The percentage of hepatocyte apoptosis

groups	Three detected times after treatment (days)		
	2	4	6
I	5.48±0.16	6.75±0.06	6.51±0.24
II	9.70±0.75*	10.72±0.56*	10.64±0.66*
III	6.86±0.84*	9.33±0.65*	7.66±0.23*

* P<0.05, ** P<0.01, AFB1 treated group (group II) compared with control group (group I); AFB1 and selenite treated group (group III) compared with AFB1 treated group (group II).

Ducks that were fed the AFB1 diet plus selenium, showed the percentage of hepatocyte apoptosis decreased by 29.28%, 12.97%, and 28.01% individually, compared to the group II (P<0.05).

Figure

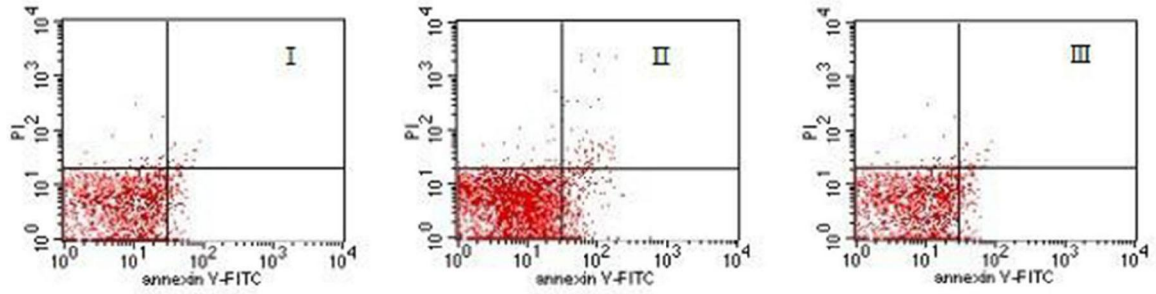

Figure 1: Flow cytometer shows Percentage of Hepatocyte Apoptosis at 2day after sodium selenite treatment

4. Discussion

AflatoxinB1 (AFB1) is produced by certain species of Aspergillus and AFB1-contaminated diets are thought to be toxigenic, carcinogenic, mutagenic and teratogen to animals. The liver is the target organ for AFB1 and duck administered aflatoxin-contaminated feed increased the activity of liver-specific enzymes (M. Denli et al.,2009; Thalita et al.,2010). Current research has found that oxidative stress is the principle manifestation of AFB1–induced toxicity. When chicks were fed the AFB1 diet, the lipid peroxide level was increased and the antioxidant enzyme was decreased (N. K. S.Gowda et al.,2008; Koohi et al.,2010). In the present study, the liver

enzymes (GOT, GPT and GST) in the AFB1 -treated group were significantly higher than in the control group. These finding confirm that the liver injury in ducks were induced by a low level of AFB1 diet which concurs with other research. AFB1 can cause cell injury and induce the release of free radicals, which activate lipid peroxidation. The decreased antioxidant enzymes (SOD and GSH-PX) and increased lipid peroxidative product (MDA) have shown that AFB1-containing diets put the ducks into a high oxidative stress situation. The excessive lipid peroxidation causes cell membrane damage, and the products of lipid peroxidation can easily diffuse through the cell membranes and modulate DNA, leading to cell apoptosis (Machalinska et al.,2001; S. M. Rustemeyer et al., 2011). Therefore in our study the percentage of hepatocyte apoptosis increased, compared with the control group.

Evidence has accumulated showing that AFB1-induced hepatotoxicity and hepatocarcinogenesis via oxidative stress could be inhibited by non-natural and natural antioxidants (Aiguo Wang et al.,2004; Rekha Gupta et al.,2011). Many scholarshave found that selenium can increase the antioxidant capacity, which is decreased by AFB1 (Dayou Shi et al.,2012; Handan Uysal and Güleray Agar, 2005). In a recent study, we found that selenium has no effect on aminotransferase, which relates to morbidity.. However selenium can increase the activity of antioxidative enzymes and decrease the concentration of MDA. When the antioxidative capability was improved, the percentage of hepatocyte apoptosis was decreased at the same time. María Teresa Ronco also discussedthe relationship between Apoptosis and Proliferation (María Teresa Ronco et al.,2002).

The present data suggest that the sodium selenite significantly improved the negative effect of AFB1-containing diets on oxidative stress and hepatocyte apoptosis, but have little effect on serum biochemistry enzymes in ducks. It is indicated that sodium selenite ameliorated the aflatoxicosis, which is reliant on its antioxidant capability.

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