Biol Trace Elem Res (2009) 128:184–190
DOI 10.1007/s12011-008-8264-y

Differential Effects of Sodium Selenite and Nano-Se on Growth Performance, Tissue Se Distribution, and Glutathione Peroxidase Activity of Avian Broiler

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Received: 21 June 2008 / Accepted: 13 October 2008 /
Published online: 30 October 2008

Abstract The present research evaluated differential effects of sodium selenite and nano-Se on growth performance, tissue Se distribution, and glutathione peroxidase (GSH-Px) activity of avian broiler. Broilers were randomly segregated into 12 groups so that three replicates were available for each of the three treatments (T-1, T-2, and T-3) and control groups. The control groups were fed basal diets without Se addition. T-1, T-2, and T-3 were fed with diets containing 0.2 mg kg^{-1} sodium selenite, 0.2 mg kg^{-1} nano-Se, and 0.5 mg kg^{-1} nano-Se, respectively. Compared with the control, Se supplementation remarkably improved daily weight gain and survival rate and decreased feed conversion ratio ($P<0.05$). However, no significant difference was observed between T-1, T-2, and T-3. The tissue Se content was significantly higher ($P<0.05$) in Se-supplemented groups than the control, and T-3 showed the highest. Furthermore, higher Se content was observed in liver, and there was a significant difference ($P<0.05$) compared with that in muscle. As for serum and hepatic GSH-Px activities, Se supplementation remarkably improved GSH-Px activity ($P<0.05$), and there was no significant difference ($P>0.05$) between treatments (T-1, T-2, and T-3).

Keywords Selenium · Sodium selenite · Nano-Se · Growth performance · Glutathione peroxidase · Avian broiler

Introduction

Selenium (Se) is an essential trace element which is important for both human and animal health [1]. The first known biological function of Se, as a component of glutathione peroxidase (GSH-Px), was reported in 1973 [2]. GSH-Px catalyzes the reduction of

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hydrogen peroxide and a variety of organic hydroperoxides using glutathione (GSH) as the hydrogen donor to water and corresponding alcohols to protect cells and membranes from oxidative damage [3]. The activity level of this enzyme in liver or plasma is indicative of selenium supply to the organism. The Se status of avian species has been demonstrated to affect bird's resistance to different diseases, antioxidant protection level, hatchability, and survival [4–7].

It is known that gray and black elemental selenium are biologically inert. Therefore, the supplementation of feed with selenium is usually limited to sodium selenite and other selenium-containing organic compound. However, the recently developed red elemental selenium has promising uses in the environmental protection from the pollution of the excessive selenium [8–11]. Zhang et al. [8] synthesized nano red elemental selenium (nano-Se) of size 5–100 nm and observed that nano-Se had a similar bioavailability in rat and much less acute toxicity in mice compared with selenite. Recently, an animal trial performed by Wang et al. [12] showed that nano-Se (20–60 nm) possesses equal efficacy in increasing the activities of GSH-Px in plasma and liver from male Kunming mice compared with selenomethionine. Therefore, this study attempted to investigate differential effects of sodium selenite and nano-Se on growth performance, tissue Se distribution, and GSH-Px activity of avian broiler.

Materials and Methods

Experimental Design

Healthy avian (n=600) were used in this study and reared in an environmentally controlled isolation facility for 42 days. All broiler chickens had similar initial weights (44.8±3.2 g). Broilers were randomly segregated into 12 groups so that three replicates were available for each of the three treatments (T-1, T-2, and T-3) and control groups. The basal diet used in this experiment was Se-deficient, which contained approximately 0.05 mg kg^{-1} total Se as determined by atomic absorption spectrophotometer (AA6501, Shimadzu Ltd., Japan). The ingredient and chemical composition were according to the National Research Council, except Se [13]. The control groups were fed basal diets without Se addition during the entire trial period. T-1, T-2, and T-3 were fed with diets containing 0.2 mg kg^{-1} sodium selenite, 0.2 mg kg^{-1} nano-Se, and 0.5 mg kg^{-1} nano-Se, respectively. In this research, nano-Se (30–80 nm) and sodium selenite were provided by Nano-Biology Lab of Zhejiang University, and the actual concentration of selenium in diet was shown in Table 1.

This feeding trial process was acceptable to the commercial producer. The experimental broilers were provided continuous lighting from incandescent lamps in the ceilings of each

Table 1 The Actual Concentration of Selenium (mg kg^{-1}) in Diet

Group/Treatment	Control	T-1	T-2	T-3
Selenium sources	–	Sodium selenite	Nano-Se	Nano-Se
Additive concentration (mg kg^{-1})	0.0	0.2	0.2	0.5
Actual concentration (mg kg^{-1})	0.055±0.007	0.253±0.008	0.254±0.011	0.548±0.012

Nano-Se nano red elemental selenium

room, but each brood-grow battery provided an area of subdued light for sleeping and resting. Fresh water was provided on a daily basis during the first week of the experiment to all the broilers and then every other day thereafter. Remaining water from the previous day was discarded before adding fresh water, and the water intake was not measured. The feeding trial was conducted under the supervision of the Animal Care and Use Committee of the university. Body weight, feed intake, feed conversion ratio (FCR), and mortality rate were recorded and analyzed.

Sampling and Analytical Methods

Weight of all collected broilers from each room determined at 2 and 42 days were treated as initial weight and final weight, respectively. The daily weight gain (DWG, g day^{-1}) was calculated as (mean final weight − mean initial weight)/42 (g day^{-1}). The FCR used the following formula: total feed consumption/(total final weight − total initial weight + total mortality weight).

For the Se concentration analysis of broilers tissues, six randomly selected broilers were collected from each group at the end of the experiment and slaughtered by severing the jugular vein. The Se concentration of tissues was determined according to the method described by Tinggi [14] by hydride generation atomic absorption spectrophotometer (AA6501, Shimadzu Ltd.).

At the end of the 6-week dietary treatment, blood was collected into tubes and allowed to coagulate at 4°C for 2 h and then centrifuged (4°C, 3,000 rpm×10 min). The supernatants were collected and used for the serum GSH-Px assays. The liver samples were also collected and homogenized using a handheld glass homogenizer, respectively, to measure the GSH-Px activity. The enzyme activity of GSH-Px was determined and expressed as specific activity (U mg^{-1}) in liver and as unit per milliliter in serum [15, 16]. All enzymatic assays were conducted within 24 h after extraction.

Statistic Analysis

Analysis of variance was used to determine the significant ($P<0.05$) difference between the tested groups. All statistics were performed using SPSS for Windows version 11.5 (SPSS, Chicago, USA).

Results

Growth Performance

DWG, FCR, and survival rate of avian broilers treated with (T-1, T-2, and T-3) or without (control) Se were shown in Table 1. Compared with the control, Se supplementation remarkably improved DWG ($P<0.05$). However, analysis of data revealed no difference in DWG between treatments at 42 days, although the highest value was observed in T-3. FCR was better ($P<0.05$) in Se-added groups (T-1, 1.98±0.03, T-2, 1.97±0.09, and T-3, 2.01±0.08, respectively) than the control (2.29±0.11), and there were also no significant differences between T-1, T-2, and T-3. As for the survival rate, the lowest value ($P<0.05$) was observed in the control than the other treatments. No statistical differences ($P>0.05$) were observed in survival rate between T-1 (97.33±1.15), T-2 (98.00±2.00), and T-3 (96.67±1.15; Table 2).

Table 2 Growth Performance and Feed Utilization of Broiler Chickens Fed with (T-1, T-2, and T-3) or without (Control) Selenium

Group/Treatment	Control	T-1	T-2	T-3
DWG (g day^{-1})	31.54±2.42 a	42.58±1.27 b	41.72±1.52 b	43.24±0.74 b
FCR	2.29±0.11 a	1.98±0.03 b	1.97±0.09 b	2.01±0.08 b
Survival rate (%)	68.67±3.06 a	97.33±1.15 b	98.00±2.00 b	96.67±1.15 b

T-1, T-2, and T-3 fed with diets containing 0.2 mg kg^{-1} sodium selenite, 0.2 mg kg^{-1} nano-Se, and 0.5 mg kg^{-1} nano-Se, respectively. Results were presented as means ± SE of triplicate observations. Means in the same row with different letter were significantly different ($P<0.05$)

DWG daily weight gain, *FCR* feed conversion ratio

Tissue Concentration of Selenium

The tissue distribution concentration of Se was significantly higher ($P<0.05$) in Se-supplemented groups as compared to control groups (Table 3). The level of Se in muscle and liver from avian broiler increased significantly ($P<0.05$) in T-3-supplemented with 0.5 mg kg^{-1} nano-Se than the others after 42 days of feeding. However, no remarkable difference was observed between T-1 added with 0.2 mg kg^{-1} sodium selenite and T-2 added with 0.2 mg kg^{-1} nano-Se. Furthermore, higher Se content was observed in liver and there was significantly different ($P<0.05$) compared with that in muscle.

GSH-Px Activity

Specific enzyme activities for GSH-Px in serum and liver across all treatments were presented in Figs. 1 and 2. GSH-Px activities ($P<0.05$) both in serum and liver were improved remarkably with the Se supplementation during the experimental period. The groups that received the sodium selenite with a concentration 0.2 mg kg^{-1} (T-1) showed an increasing trend in GSH-Px activity of serum and liver. However, there were no significant differences ($P>0.05$) between treatments (T-1, T-2, and T-3).

Discussion

The research of different Se source for their potential use as additives in poultry was still increasing [17–19]. It was clear from our studies that the administration of Se via the basal

Table 3 Selenium Concentration of Muscle and Liver from Broiler Chickens Fed with (T-1, T-2, and T-3) or without (Control) Selenium

Group/Treatment	Selenium concentration (μg g^{-1})			
	Control	T-1	T-2	T-3
Muscle	0.11±0.04 a*	0.26±0.05 b*	0.30±0.04 b*	0.43±0.05 c*
Liver	0.24±0.04 a	0.51±0.06 b	0.55±0.06 b	0.69±0.06 c

T-1, T-2, and T-3 fed with diets containing 0.2 mg kg^{-1} sodium selenite, 0.2 mg kg^{-1} nano-Se, and 0.5 mg kg^{-1} nano-Se, respectively. Results were presented as means ± SE of triplicate observations. Means in the same row with different lowercase and in the same column with asterisk were significantly different ($P<0.05$)

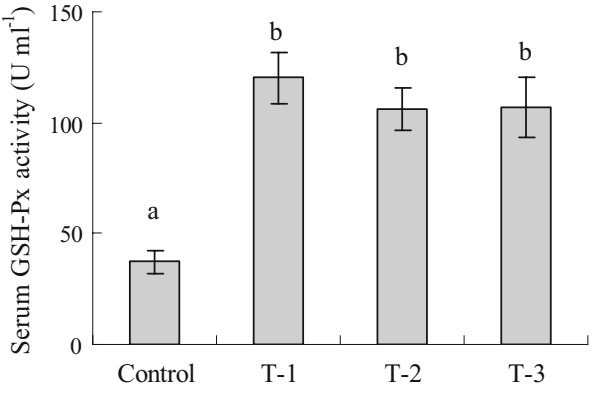

Fig. 1 Glutathione peroxidase (*GSH-Px*) activity of serum from broiler chickens fed a basal diet (control) and three diets containing 0.2 mg kg^{-1} sodium selenite (T-1), 0.2 mg kg^{-1} nano-Se (T-2), and 0.5 mg kg^{-1} nano-Se (T-3) at the end of 42 days. Means with different letters were significantly different ($P<0.05$)

diet had beneficial effect on avian broiler performance. In the present research, FCR was significantly reduced in groups of Se treatment compared with that of the control. Similar results were observed by Mahmoud and Edens [20] who demonstrated that the FCR of broiler chickens (*Gallus gallus*) is affected by dietary Se level. Similar improvements in growth performance had been reported for poultry receiving Se [21]. However, there was no significant difference among the treatment groups (T-1, T-2, and T-3) with different source and concentrations of Se. This indicated that the forms and quantity of Se was only one of the factors improving the DWG and FCR of avian broilers.

Poultry diets deficient in selenium result in poor growth and development, increased mortality, reduced egg production, decreased hatchability, pancreatic fibrosis, and muscle myopathies [22–24]. The present research result proved this point, and the control groups fed with basal diet unsupplemented with any forms of Se showed the symptoms of selenium deficiency such as lower survival rate, DWG, and higher FCR. The minimum level of supplemental selenium to sustain growth and performance in broiler chickens was 0.1 mg kg^{-1} according to the National Research Council. However, the Se content of basal diet was only 0.055±0.007 mg kg^{-1} and lower than the standard. In contrast, no significant survival increases were detected, and the survival rates of all the groups supplemented with Se (T-1, T-2, and T-3) were 97.33%, 98.00%, and 96.67%, respectively, after 42 days feeding. It indicated that the nano-Se had the same biological functions as sodium selenite in avian broilers. Moreover, no remarkable significance was observed between T-2 and T-3

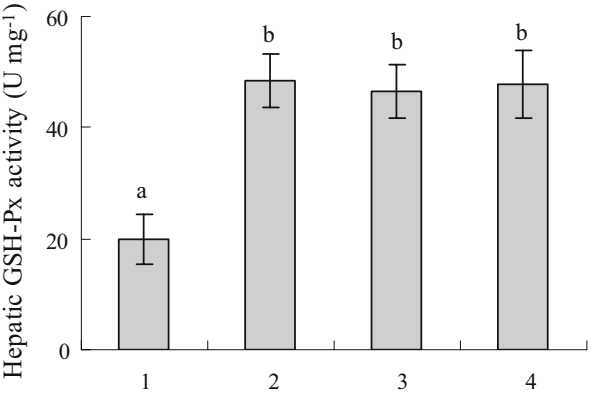

Fig. 2 Hepatic glutathione peroxidase (*GSH-Px*) activity of broiler chickens fed a basal diet (control) and three diets containing 0.2 mg kg^{-1} sodium selenite (T-1), 0.2 mg kg^{-1} nano-Se (T-2), and 0.5 mg kg^{-1} nano-Se (T-3) at the end of 42 days. Means with different letters were significantly different ($P<0.05$)

in the present study, and it suggested from the opposite side that the addition of 0.5 mg kg^{-1} nano-Se was acceptable in avian feeding.

It was obvious that the tissues with Se content were markedly increased as the dietary Se level increased. Similar results were observed in Rohman laying hens by Pan et al. [25] who reported that breast muscles and whole body, liver, kidney, spleen selenium concentrations were higher in the groups given selenium compared with that of the control. Animal studies have demonstrated that the liver is the major target organ of selenium accumulation [26]. In the present study, higher Se content was observed in liver than in muscle across all treatments.

A substantial research has also defined an important role for Se in antioxidant defense. Se is important for the control of oxidative stress, and therefore the redox state of the cell, due to its incorporation as selenocysteine into GSH-Px [27] and thioredoxin reductase [28]. In this study, broilers fed a diet deficient in Se showed decreased GSH-Px activity in serum and liver. By Supplementation of the diet with Se, both sodium selenite and nano-Se, increased the GSH-Px activity. However, GSH-Px activity was not linearly related to the concentration of the dietary nano-Se. This was not in agreement with the previous studies which showed that the GSH-Px activity increased as a logarithmic function of the dietary selenium (sodium selenite and selenomethionine) level [29]. However, it was difficult to directly assess different studies using Se because the efficacy of a Se application depended on many factors such as species composition and viability, administration level, application method, frequency of application, overall diet, bird age, overall farm hygiene, and environmental stress factors. Essentially, there was no difference in GSH-Px activities both in serum and liver from broilers fed equal gram-atoms of selenium as sodium selenite and as nano-Se in the present research. This indicated that the form of Se was only one of the factors promoting the GSH-Px activity of avian broilers.

Based on the findings of our study, nano-Se could serve as another Se form and successfully improved DWG, FCR, survival rate, tissue Se content, and the GSH-Px activity of avian broilers compared with the control. Furthermore, different tissue Se contents were observed in the groups fed with different concentration nano-Se. However, no significant differences were found in DWG, FCR, survival rate, and GSH-Px activity of serum and liver across all treatments fed with 0.2 mg kg^{-1} sodium selenite (T-1), 0.2 mg kg^{-1} nano-Se (T-2), and 0.5 mg kg^{-1} nano-Se (T-3), respectively. The addition, however, of a different form of Se, especially selenomethionine, the predominant chemical form of organic selenium in feedstuffs, and nano-Se to avian broilers in general requires further research to compare the bioavailability and clearly understand the functional mechanism between the Se and animals. Moreover, modern molecular techniques should be applied to study whether there are other metabolic pathways of nano-Se which differed from sodium selenite and/or selenomethionine.

Acknowledgments This study was supported by the Doctoral Natural Science Foundation of University (No. 1110XJ-030628). We also acknowledge valuable help provided by all involved workers.

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