

Influence of Dietary Selenomethionine Supplementation on Performance and Selenium Status of Broiler Breeders and Their Subsequent Progeny

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Abstract The study was conducted to investigate the effects of dietary maternal selenomethionine or sodium selenite supplementation on performance and selenium status of broiler breeders and their next generation. Two hundred and forty 39-week-old Lingnan yellow broiler breeders were allocated randomly into two treatments, each of which included three replicates of 40 birds. Pretreatment period was 2 weeks, and the experiment lasted 8 weeks. The groups were fed the same basal diet supplemented with 0.30 mg selenium/kg of sodium selenite or selenomethionine. After incubation, 180 chicks from the same parental treatment group were randomly divided into three replicates, with 60 birds per replicate. All the offspring were fed the same diet containing 0.04 mg selenium/kg, and the experiment also lasted 8 weeks. Birth rate was greater ($p < 0.05$) in hens fed with selenomethionine than that in hens fed with sodium selenite. The selenium concentration in serum, liver, kidney, and breast muscle of broiler breeders, selenium deposition in the yolk, and albumen and tissues' (liver, kidney, breast muscle) selenium concentrations of 1-day-old chicks were significantly ($p < 0.01$) increased by maternal selenomethionine supplementation compared with maternal sodium selenite supplementation. The antioxidant status of 1-day-old chicks was greatly improved by maternal selenomethionine intake in comparison with maternal sodium selenite intake and was evidenced by the increased glutathione peroxidase activity in breast muscle ($p < 0.05$), superoxide dismutase activity in breast muscle and kidney ($p < 0.05$), glutathione concentration in kidney ($p < 0.01$), total antioxidant capability in breast muscle and liver ($p < 0.05$), and decreased malondialdehyde concentration in liver and pancreas ($p < 0.05$) of 1-day-old chicks. Feed utilization was better ($p < 0.05$), and mortality was lower ($p < 0.05$) in the progeny from hens fed with selenomethionine throughout the 8-week growing period compared with those from hens fed with sodium selenite. In summary, we concluded that maternal selenomethionine supplementation increased birth rate and Se deposition in serum and tissues of broiler

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breeders as well as in egg yolk and egg albumen more than maternal sodium selenite supplementation. Furthermore, maternal selenomethionine intake was also superior to maternal sodium selenite intake in improving the tissues Se deposition and antioxidant status of 1-day-old chicks and increasing the performance of the progeny during 8 weeks of post-hatch life.

Keywords Selenomethionine · Broiler breeder · Antioxidant status · Selenium deposition · Mortality

Introduction

Selenium (Se) has been recognized as an essential dietary nutrient that plays important roles in animal health and productivity. There are two main sources of Se in the diet: organic Se such as Se-enriched yeast (SY) and inorganic Se such as sodium selenite (SS). Usually, organic Se has higher bioavailability and antioxidant properties and rates of tissue accumulation as well as lower toxicities as compared to inorganic Se [1–3].

The impact of maternal nutrition on the nutritional status or antioxidant system of the offspring has attracted more and more researchers' attention in recent years. Selenium is an essential component of the antioxidant enzyme glutathione peroxidase (GSH-Px) [4]. The GSH-Px family of enzymes is a crucial player in the integrated antioxidant system, neutralizing potential threats to the integrity of cellular macromolecules by eliminating hydrogen peroxide and detoxifying lipid hydroperoxides [5]. Selenium derived from the diet of the female bird is deposited in the egg and is distributed among the developing tissues during embryogenesis [6–8]. Surai [7] reported that maternal SY supplementation significantly increased GSH-Px activity, glutathione (GSH), and Se concentrations and reduced the generation of lipid peroxides in liver of chicks for 1 to 10 days post-hatch when compared with the control group. Pappas et al. [9] indicated that maternal SY intake readily elevated the Se concentration and GSH-Px activity in blood, liver, and breast muscle of chicks for 2 to 4 weeks post-hatch in comparison with hens on the control diet.

Many researches attributed the high biological effect of SY to selenomethionine (Se-methionine (Met)), a Se analog of Met, which is the predominant form of Se in SY [10–12]. Selenomethionine is also the major form of Se in some plant material such as cereals and forage crops and therefore the most appropriate form of Se in human and animal nutrition [11, 12]. Besides, Se-Met is the only Se-amino acid that is non-specifically incorporated into tissue proteins in place of Met, allowing the build-up of Se reserves in the organism [11, 12]. However, additional research seemed necessary to compare the effects of supplemental Se-Met and SS in the material diet on antioxidant status and tissues' Se concentrations of 1-day-old chicks and on performance of the progeny during early post-hatch life.

Therefore, the main objective of the current study was to determine the effects of maternal dietary SS or Se-Met supplementation on tissues Se deposition and antioxidant property of 1-day-old chicks and on growth performance of the offspring. The movement of Se from broiler breeders to their eggs, productive performance, and Se deposition in serum and tissues of broiler breeders were also assessed.

Materials and Methods

Experimental Animals and Treatments

This project was approved and conducted under the supervision of the Zhejiang University Animal Care and Use Committee, which has adopted Animal Care and Use Guidelines governing all animal use in experimental procedures.

Two hundred and forty 39-week-old broiler breeders (Lingnan yellow) were randomly distributed to two treatments, each of which was replicated three times with 40 hens per replicate. A corn-soybean meal basal diet (Table 1) was formulated to meet the recommendations for broiler breeders of the National Research Council [13] with regard to the requirements of all nutrients except for Se. The basal diet contained 0.04 mg/kg Se. Dietary Se addition was based on calculated levels for each source. Selenium from SS (Sigma-Aldrich Chemical Co., St. Louis, MO, USA) or Se-Met (seneno-DL-methylseleno) (Sigma-Aldrich Chemical Co., St. Louis, MO, USA) was added at 0.30 mg/kg Se into the basal diet to make each treatment, respectively. During the experiment, feed (in mash form) and water were provided ad libitum. Pretreatment period was 2 weeks, and the experiment lasted 8 weeks. The facility was environmentally controlled.

Egg production and egg weight were evaluated during the 8-week experiment. Each day at approximately 12:00 h, the total eggs produced and the number of dirty, cracked, or

Table 1 Ingredients and nutrient content of the maternal basal diets (grams per kilogram, unless otherwise stated)

Ingredients	
Corn	646
Soybean meal	250
Monocalcium phosphate	18
Limestone	70
DL-Methionine	3
Salt	3
Vitamin–mineral premix ^a	10
Composition (analyzed except for (ME) ^b)	
ME (MJ/kg)	11.24
Crude protein	161.1
Calcium	30.2
Total phosphorus	6.5
Lysine	8.2
Methionine	5.5
Methionine+cysteine	8.1

Sodium selenite and selenomethionine were premixed in corn and added to the diets at 0.30 mg selenium per kilogram to achieve the appropriate treatment levels

^a Supplied the following per kilogram of diet: iron, 72 mg; copper, 7 mg; zinc, 72 mg; manganese, 90 mg; iodine, 0.9 mg; vitamin A, 10,800 IU; vitamin D3, 2,160 IU; vitamin E, 27 IU; vitamin K3, 1.4 mg; vitamin B1, 1.8 mg; vitamin B2, 8 mg; vitamin B6, 4.1 mg; vitamin B12, 0.01 mg; niacin, 32 mg; calcium pantothenate, 11 mg; folic acid, 1.08 mg; biotin, 0.18 mg

^b ME was calculated from data provided by Feed Database in China [40]

shell-less eggs were recorded and weighted for each replicate. The eggs were incubated once a week, and birth rate was measured from all hatching eggs produced throughout the 8-week experiment.

To study the effects of maternal Se source on the Se status, antioxidant property, and performance of the offspring, eggs (120 from each replicate) were collected during the eighth week of treatment. These eggs were incubated using standard conditions (37.5°C and 55% relative humidity) in a forced-draft incubator with automatic egg turning (model FT-KCFC10, The 41st Electronic Inst., Qingdao, Shandong, People's Republic of China). Weights of eggs collected for incubation were recorded daily. After incubation for 21 days, 180 healthy chicks from the same parental treatment group were divided into three replicates, with 60 birds per replicate. Other chicks were used for analysis of tissues Se and GSH-Px. All the offspring were fed the same basal diet containing 0.04 mg Se/kg with no added Se. The basal diet was formulated to meet or exceed requirements of broilers according to National Research Council [13] except for Se (Table 2). Feed and water were provided ad libitum throughout the experiment. The experiment lasted 56 days. Mortality, temperature, and feed intake were recorded daily. At dietary phase change and at the end of the experiment, broilers were deprived of feed for 12 h and weighed per replicate. Average daily gain (ADG), average daily feed intake (ADFI), and feed conversion ration (FCR) were calculated.

Table 2 Formulation and proximate analysis of the progeny basal diets (grams per kilogram, unless otherwise stated)

Items	Starter (1 to 21 days)	Grower (22 to 42 days)	Finisher (43 to 56 days)
Ingredients			
Maize	591.9	638.6	657.6
Soybean meal	360.0	310.0	290.0
Soy oil	10.0	14.0	20.0
Monocalcium phosphate	15.0	12.0	10.0
Limestone	12.0	15.0	12.0
Salt	3.0	3.0	3.0
L-Lysine HCl	1.3	1.0	1.0
DL-Methionine	1.8	1.4	1.4
Vitamin–mineral premix ^a	5.0	5.0	5.0
Composition (analyzed except for (ME)^b)			
ME (MJ/kg)	11.88	12.18	12.43
Crude protein	205.1	186.6	179.3
Lysine	10.4	9.2	8.4
Methionine	4.8	3.4	3.2
Methionine+cysteine	8.2	6.5	5.6
Calcium	9.3	9.1	7.7
Total phosphorus	6.5	5.8	5.4

^a Supplied per kilogram of diet: retinyl acetate, 3,440 µg; cholecalciferol, 100 µg; DL- α -tocopheryl acetate, 10 mg; menadione, 3.0 mg; thiamine, 1.5 mg; riboflavin, 3.5 mg; pyridoxine, 3.0 mg; cobalamin, 15 µg; niacin, 30 mg; folic acid, 0.5 mg; pantothenic acid, 10 mg; biotin, 150 µg; iron, 80 mg; copper, 8 mg; manganese, 60 mg; zinc, 40 mg; iodine, 0.33 mg; ethoxyquin, 100 mg

^b ME was calculated from data provided by Feed Database in China [40]

Sample Collection and Preparation

At the end of the broiler breeders' experiment, four eggs per replicate from each treatment were randomly collected. Each individual egg was broken out, and the albumen and yolk were separated and weighed. The albumen and yolk were homogenized with an electric blender at chilled conditions and stored frozen in 10-ml plastic tubes until Se analysis could be conducted. Then four hens per replicate from each treatment were randomly selected, fasted for 12 h, and blood samples were taken from the main wing vein. A 10-ml sample was extracted per bird and allowed to coagulate at room temperature for 1 h. Serum was separated by centrifugation at 4°C, 1,000×g for 20 min and transferred into 1.0-ml microcentrifuge tubes. After blood collection, the birds were killed. Samples of about 10 g of breast muscle and whole liver, kidney, and pancreas were collected and placed in liquid nitrogen. At 1 day of age, thirty chicks per replicate from each treatment were randomly selected and killed by cervical dislocation. Samples of whole breast muscle, liver, kidney, and pancreas were collected and placed in liquid nitrogen. All samples were marked with their treatments, replicate number, and sampling date. Frozen tissues and serum samples were stored at -80°C until further analysis.

Biochemical Determinations

The Se assay for the samples was performed by hydride generation atomic fluorescence spectrometry [14]. The GSH-Px, superoxide dismutase (SOD), GSH, malondialdehyde (MDA), and total protein were determined using the method of Lawrence and Burk [15], Panckenko et al. [16], Beutler et al. [17], Placer et al. [18], and Lowry et al. [19], respectively. The total antioxidant capability (T-AOC) was examined by assay kits purchased from Nanjing Jiancheng Institute of Bioengineering (Nanjing, Jiangsu, People's Republic of China) and the procedures accordingly. The T-AOC and enzyme activity of GSH-Px and SOD were expressed as units per milligram of protein (U/mgprot) in tissues and as units per milliliter (U/ml) in serum, respectively. The GSH and MDA concentrations were expressed as milligrams per gram of protein and nanomole per milligram of protein in tissues and as milligram per liter and nanomole per milliliter in serum, respectively.

Statistical Analysis

The experimental data were analyzed using the unpaired *t* test procedure of the SPSS 16.0 for Windows. Values of $p < 0.05$ were taken as significant. Replicate was considered as the experiment unit for performance determined. All results were expressed as means ± standard deviation.

Results

Egg production and egg weight were not significantly ($p > 0.05$) affected by dietary treatments. Selenomethionine supplementation significantly ($p < 0.05$) improved the birth rate of broiler breeders more so than the equivalent amount of SS, increasing it by 5.73 % (Table 3).

Broiler breeders receiving the Se-Met-supplemented diets had higher ($p < 0.01$) serum and tissues (except pancreas) Se concentrations than those consuming the SS-supplemented diets (Table 4).

Eggs from hens fed the Se-Met diets contained significantly ($p < 0.01$) more albumen and yolk Se than did eggs from hens fed the SS diets (Table 5).

Table 3 Effects of different selenium sources on productive performance of broiler breeders

Items	SS	Se-Met
Egg production (%)	70.87±1.34	70.21±0.97
Egg weight (g)	54.09±0.87	53.35±0.69
Birth rate (% hatching eggs)	85.07±2.61	90.80±2.20*

Values are means±SD, $n=3$

SS sodium selenite, *Se-Met* selenomethionine

* $p<0.05$

The concentration of Se in the tissues (except pancreas) of 1-day-old chicks was significantly ($p<0.01$) increased by maternal Se-Met intake in comparison with maternal SS intake (Table 6).

Compared with maternal SS supplementation, maternal Se-Met supplementation generally increased the antioxidant status of 1-day-old chicks, which was exhibited as follows: significantly ($p<0.05$) increased GSH-Px activity in breast muscle; significantly increased SOD activity in breast muscle ($p<0.01$) and kidney ($p<0.05$); significantly ($p<0.01$) increased kidney GSH concentration; significantly ($p<0.05$) increased T-AOC in breast muscle and liver; significantly ($p<0.05$) decreased liver and pancreas MDA concentrations (Table 7).

Average daily gain and ADFI of the progeny were not affected ($p>0.05$) by dietary maternal Se source in any period of growth or in the overall period. Maternal Se-Met supplementation significantly ($p<0.05$) improved the FCR of the offspring during growing phase (22 to 42 days of age) and finishing phase (43 to 56 days of age) post-hatch as well as for the cumulative period (1 to 56 days of age), but not starting phase (1 to 21 days of age) ($p>0.05$), compared with maternal SS supplementation. Maternal Se-Met intake significantly ($p<0.05$) decreased the mortality of the chicks during the first week postnatal and the overall mortality during the 8-week growing period in comparison with maternal SS intake (Table 8).

Discussion

Birth rate is one of the most important parameters of chick producers' demand. In our experiment, birth rate from hens supplemented with Se-Met was higher than that from hens

Table 4 Selenium concentration in serum and tissues of broiler breeders (milligram per kilogram, wet weight basis)

Items	SS	Se-Met
Serum	0.367±0.021	0.975±0.078**
Liver	0.315±0.026	0.596±0.032**
Kidney	0.361±0.024	0.644±0.016**
Breast muscle	0.124±0.013	0.163±0.010**
Pancreas	0.261±0.018	0.256±0.016

Values are means±SD, $n=3$

SS sodium selenite, *Se-Met* selenomethionine

** $p<0.01$

Table 5 Selenium concentration in eggs (milligram per kilogram, wet weight basis)

Items	SS	Se-Met
Egg albumen	0.089±0.005	0.119±0.007**
Egg yolk	0.459±0.005	0.590±0.005**

Values are means±SD, $n=3$

SS sodium selenite, *Se-Met* selenomethionine

** $p<0.01$

supplemented with SS. Our results were in agreement with those of Sefton and Edens [20], who reported that when SS (at 0.3 mg Se/kg) was replaced by the same amount of SY in the diet of Hubbard Ultra-Yield broiler breeders, an average 4.5 extra chicks per hen was achieved during the field trial. It was suggested that Se-Met supplementation of broiler breeder diets was very beneficial from both production and economic viewpoints. Data collected from this trial indicated that using SS versus Se-Met had no effect on egg production and egg weight of broiler breeders, which was consistent with the reports of Leeson et al. [21], Utterback et al. [22], and Payne et al. [23].

Selenium concentration in serum and tissues (except pancreas) was significantly higher for broiler breeders in the Se-Met group compared with the SS group, which was in agreement with the results reported by Leeson et al. [21], Li and Wang [24], and Pan et al. [25]. This is probably due to the differences in absorption and metabolism of inorganic and organic Se sources: organic Se (Se-Met) is actively absorbed in the intestine through the amino acid transport mechanisms, whereas inorganic Se is passively absorbed [11, 12, 26]. On the other hand, the chemical similarity between Se-Met and Met allows the body to use them interchangeably in protein synthesis because the tRNA^{Met} cannot discriminate between Met and Se-Met [11, 12]. This makes it possible to build Se reserves in the body [11, 12].

Selenium in the maternal diet can be deposited into the egg. The Se content of the egg depends on its concentration in the hen's diet and also on the form of dietary Se used, since organic Se is more efficiently deposited in the egg yolk [27, 28]. Data obtained from the present experiment exhibited that the hens feeding Se-Met resulted in greater rates of Se deposition in yolk and albumen compared with those feeding SS. The results combined with several other researchers [8, 21, 24, 29–32]. Predominant yolk Se accumulation by either sources of Se was demonstrated in the present experiment, which was in agreement with the reports of Paton et al. [8] and Leeson et al. [21]. The possible mechanism was that mineral binding lipoproteins deposited during yolk accretion [33, 34].

Table 6 Selenium concentration in tissues of 1-day-old chicks (milligram per kilogram, wet weight basis)

Items	SS	Se-Met
Liver	0.230±0.016	0.492±0.037**
Kidney	0.345±0.022	0.579±0.042**
Breast muscle	0.102±0.009	0.130±0.011**
Pancreas	0.228±0.015	0.240±0.016

Values are means±SD, $n=3$

SS sodium selenite, *Se-Met* selenomethionine

** $p<0.01$

Table 7 Effects of different selenium sources in maternal diet on antioxidant properties in tissues of 1-day-old chicks

Tissues	Treatment	GSH-Px (U/mgprot)	SOD (U/mgprot)	GSH (U/mgprot)	T-AOC (U/mgprot)	MDA (nmol/mgprot)
Breast muscle	SS	38.92±3.17	39.88±4.39	0.23±0.03	0.50±0.03	1.33±0.16
	Se-Met	42.09±2.59*	54.28±4.50**	0.25±0.03	0.56±0.06*	1.34±0.21
Liver	SS	18.49±1.90	73.63±6.67	0.86±0.12	1.03±0.14	1.14±0.12
	Se-Met	17.96±1.54	72.98±6.96	0.73±0.10	1.31±0.15*	0.99±0.14*
Kidney	SS	20.65±2.94	67.46±5.23	0.97±0.10	0.20±0.03	1.02±0.13
	Se-Met	19.92±1.18	76.22±4.21*	1.42±0.12**	0.22±0.03	0.91±0.12
Pancreas	SS	14.31±1.10	45.34±0.72	0.66±0.10	0.26±0.04	0.47±0.08
	Se-Met	13.87±1.05	46.15±1.28	0.77±0.08	0.30±0.05	0.38±0.06*

Values are means±SD, $n=3$

SS sodium selenite, *Se-Met* selenomethionine, *T-AOC* total antioxidant capability, *SOD* superoxide dismutase, *GSH-Px* glutathione peroxidase, *GSH* glutathione, *MDA* malondialdehyde

* $p<0.05$; ** $p<0.01$

Table 8 Effects of dietary maternal selenium source on growth performance and feed utilization of the progeny from 1 to 56 days of age

Growth period	SS	Se-Met
ADG (g)		
1 to 21 days	19.36±0.15	19.21±0.42
22 to 42 days	38.61±0.65	40.40±1.28
43 to 56 days	40.55±1.88	43.38±1.42
1 to 56 days	31.88±0.68	33.20±0.81
ADFI (g)		
1 to 21 days	36.07±0.93	34.96±0.65
22 to 42 days	93.49±16.24	92.95±18.54
43 to 56 days	142.96±10.11	143.86±5.55
1 to 56 days	82.19±2.34	82.71±2.31
FCR		
1 to 21 days	1.91±0.10	1.82±0.04
22 to 42 days	2.42±0.40	2.30±0.41*
43 to 56 days	3.66±0.10	3.32±0.12*
1 to 56 days	2.68±0.09	2.50±0.07*
Mortality (%)		
1 to 7 days	5.56±2.52	0*
1 to 56 days	11.67±3.33	1.06±1.83*

Values are means±SD, $n=3$

SS sodium selenite, *Se-Met* selenomethionine, *ADG* average daily gain, *ADFI* average daily feed intake, *FCR* feed conversion ration

* $p<0.05$

During embryonic development, Se accumulated in the egg is transferred to the developing embryo and subsequently delivered to different tissues [35]. In our study, the tissues' Se concentrations of 1-day-old chicks originating from hens with Se-Met supplementation were significantly higher than those of chicks hatched from hens with SS supplementation. Our results are in good agreement with those of Hassan [36]. We ascribed the elevated Se concentration in the tissues of 1-day-old chicks to two reasons for the Se-Met-treated group. Firstly, egg yolk could act as an effective nutrition source to transfer more Se to offspring; secondly, high Se content in maternal organs was passed to offspring through egg albumen. Therefore, the use of Se-Met is an effective strategy to improve the Se concentration of the egg as well as that of 1-day-old chicks.

Selenium is an important component of the antioxidant enzyme GSH-Px and it is actively involved in the antioxidant defense systems [37]. Gunter et al. [38] reported that maternal SY supplementation significantly increased the GSH-Px activity of calves at birth in comparison with maternal SS supplementation. In the present study, the antioxidant status of 1-day-old chicks was greatly improved by maternal Se-Met intake compared with maternal SS intake. The activities of antioxidant enzymes (i.e., SOD and GSH-Px), T-AOC, and concentration of GSH were increased, while the MDA concentration was decreased. Our results indicated that Se-Met showed a higher potential to improve the extent of transferring antioxidant from mother to its offspring when compared with SS. We would also suggest that the increased tissues Se deposition of 1-day-old chicks by maternal Se-Met supplementation may be responsible for improved antioxidant defenses of chick tissues against the high oxidative stress imposed by the hatching process. Since the egg-derived antioxidants (vitamin E and carotenoids) are progressively depleted from the tissues after hatching [39], antioxidant enzymes (mainly GSH-Px) become a critical arm of antioxidant defense. Therefore, enhanced GSH-Px activity in tissues by Se-Met supplementation in the maternal diet could be considered an effective way for increasing chick antioxidant potency post-hatch. An improved antioxidant system of the chick may also enhance immune system function, which is extremely important during postnatal development of the chicken.

Selenium is an essential micronutrient required for normal growth and maintenance in poultry, but a Se deficiency results in poor growth and development and increased mortality. The present results confirmed that maternal Se-Met supplementation showed a higher potential to improve the FCR and reduce the mortality of the offspring throughout the 8-week growing period in comparison with maternal SS supplementation. In this study, all the progeny were fed the same basal diets, and because feed intake did not differ between treatments, the differences in the Se status of the progeny are due solely to differences in the Se content of the maternal diets. Therefore, the reason for the more significant decrease of mortality and improve of FCR of the progeny by maternal Se-Met supplementation is may be that Se-Met was more effective than SS in increasing Se reserves and antioxidant property of the progeny during postnatal life.

Conclusion

In summary, birth rate and Se deposition in serum and tissues of broiler breeders and Se concentrations in egg yolk and egg albumen were greatly improved by maternal Se-Met intake compared with maternal SS intake. Furthermore, maternal effect was more effective for feeding broiler breeders with Se-Met because more Se and antioxidant property were transferred from egg to its offspring and thus led to elevated tissues Se deposition and antioxidant status of 1-day-old chicks and increased performance of the progeny throughout the 8-week growing period.

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References

1. Wang C, Lovell RT (1997) Organic selenium sources, selenomethionine and selenoyeast, have higher bioavailability than an inorganic selenium source, sodium selenite, in diets for channel catfish (*Zctalurus punctatus*). *Aquaculture* 152:223–234
2. Mahan DC, Cline TR, Richert B (1999) Effects of dietary levels of selenium enriched yeast and sodium selenite as selenium sources fed to growing—finishing pigs on performance, tissue selenium, serum glutathione peroxidase activity, carcass characteristics and loin quality. *J Anim Sci* 77:2172–2179
3. Mahmoud KZ, Edens FW (2003) Influence of selenium sources on age related and mild heat stress-related changes of blood and liver glutathione redox cycle in broiler chickens (*Gallus domesticus*). *Comp Biochem Physiol B Biochem Mol Biol* 136(4):921–934
4. Rotruck JT, Pope AL, Ganther HE, Swanson AB, Hafeman DG, Hoekstra WG (1973) Selenium: biochemical role as a component of glutathione peroxidase. *Science* 179:588–590
5. Brigelius-Flohe R (1999) Tissue-specific functions of individual glutathione peroxidases. *Free Radic Biol Med* 27:951–965
6. Gaa'l T, Me'zes M, Noble RC, Dixon J, Speake BK (1995) Development of antioxidant capacity in tissues of the chick embryo. *Comp Biochem Physiol B Biochem Mol Biol* 112(4):711–716
7. Surai PF (2000) Effect of selenium and vitamin E content of the maternal diet on the antioxidant system of the yolk and the developing chick. *Br Poult Sci* 41:235–243
8. Paton ND, Cantor AH, Pescatore AJ, Ford MJ, Smith CA (2002) The effect of dietary selenium source and level on the uptake of selenium by developing chick embryos. *Poult Sci* 81:1548–1554
9. Pappas AC, Karadas F, Surai PF, Speake BK (2005) The selenium intake of the female chicken influences the selenium status of her progeny. *Comp Biochem Physiol B Biochem Mol Biol* 142(4):465–474
10. Beistein MA, Whanger PD (1986) Deposition of dietary organic and inorganic Se in rat erythrocyte protein. *J Nutr* 116:1701–1710
11. Schrauzer GN (2000) Selenomethionine: a review of its nutritional significance, metabolism and toxicity. *J Nutr* 130:1653–1656
12. Schrauzer GN (2003) The nutritional significance, metabolism and toxicology of selenomethionine. *Adv Food Nutr Res* 47:73–112
13. National Research Council (1994) Nutrient requirements of poultry, 9th edn. National Academies, Washington
14. Ga'miz-Gracia L, de Castro MD, Luque (1999) Determination of selenium in nutritional supplements and shampoos by flow injection-hydride generation-atomic fluorescence spectrometry. *Talanta* 50:875–880
15. Lawrence RA, Burk RF (1976) Glutathione peroxidase activity in selenium-deficient rat liver. *Biochem Biophys Res Commun* 71:952–958
16. Panckenko LF, Brusov OS, Gerasimov AM, Loktaeva AE (1975) Intramitochondrial localization and release of rat liver superoxide dismutase. *FEBS Lett* 55:84–87
17. Beutler E, Duron O, Kelly BM (1963) Improved method for the determination of blood glutathione. *J Lab Clin Med* 61:882–888
18. Placer ZA, Cushman LL, Johnson BC (1966) Estimation of product of lipid peroxidation (malondialdehyde) in biochemical systems. *Anal Biochem* 16:359–364
19. Lowry OH, Rosebrough NJ, Farr AL, Randall RJ (1951) Protein measurement with the folin phenol reagent. *J Biol Chem* 193:265–275
20. Sefton AE, Edens FW (2004) Sel-Plex improves semen quality in broiler breeder males in a cage environment. *Biotechnology in the Feed and Food Industry. Proceedings of the 20th Annual Symposium (Suppl. 1), 22–26 May 2004, Lexington, Kentucky, p 33*
21. Leeson S, Namkung H, Caston L, Durosoy S, Schlegel P (2008) Comparison of selenium levels and sources and dietary fat quality in diets for broiler breeders and layer hens. *Poult Sci* 87:2605–2612
22. Utterback PL, Parsons CM, Yoon I (2005) Effect of supplementing selenium yeast in diets of laying hens on egg selenium content. *Poult Sci* 84:1900–1910
23. Payne RL, Lavergne TK, Southern LL (2005) Effect of inorganic versus organic selenium on hen production and egg selenium concentration. *Poult Sci* 84:232–237

24. Li JK, Wang XL (2004) Effect of dietary organic versus inorganic selenium in laying hens on the productivity, selenium distribution in egg and selenium content in blood, liver and kidney. *J Trace Elem Med Biol* 18:65–68
25. Pan GL, Huang KH, Zhao YX, Qin SY, Chen F, Hu QH (2007) Effect of selenium source and level in hen's diet on tissue selenium deposition and egg selenium concentrations. *J Agric Food Chem* 55:1027–1032
26. Wolfram S, Berger B, Grenacher B, Scharrer E (1989) Transport of seleno amino acids and their sulphur analogues across the intestinal brush border membrane. *J Nutr* 119:706–712
27. Cantor AH (1997) The role of selenium in poultry nutrition. In: Lyons TP, Jacques KA (eds) *Biotechnology in the feed industry*. Proceedings of Alltech's 13th annual symposium. Nottingham University, Nottingham, pp 155–164
28. Paton ND, Cantor AH, Pescatore AJ, Ford MJ, Smith CA (2000) Effect of dietary selenium source and level of inclusion on selenium content of incubated eggs. *Poult Sci* 79(Supp. 1):40
29. Cantor AH, Scott ML (1974) The effect of selenium in the hen's diet on egg production, hatchability, performance of progeny and selenium concentration in eggs. *Poult Sci* 53:1870–1880
30. Davis RH, Fear J, Winton AC (1996) Interactions between dietary selenium, copper, and sodium nitroprusside, a source of cyanide in growing chicks and laying hens. *Br Poult Sci* 37:87–94
31. Cantor AH, Straw ML, Ford MJ, Pescatore AJ, Dunlap MK (2000) Effect of feeding organic selenium in diets of laying hens on egg selenium content. In: Sim JS, Nakai S, Guenter W (eds) *Egg nutrition and biotechnology*. CABI, New York, p 473
32. Patton ND (2000) Organic selenium in the nutrition of laying hens: effects on egg selenium content, egg quality and transfer to developing chick embryos. Ph.D. dissertation, University of Kentucky, Lexington
33. Richards MP (1977) Trace mineral metabolism in the avian embryo. *Poult Sci* 76:152–164
34. Miles RD (2000) Trace minerals and avian embryo development. *Cienc Anim Bras* 2(1):1–10
35. Gerhartz B, Kolb HJ, Wittmann J (1999) Proteolytic activity in the yolk sac membrane of quail eggs. *Comp Biochem Physiol A Mol Integr Physiol* 123(1):1–8
36. Hassan S (1986) Effect of dietary selenium on the prevention of exudative diathesis in chicks, with special reference to selenium transfer via eggs. *J Vet Med A* 33:689–697
37. Rayman MP (2000) The importance of selenium to human health. *Lancet* 365:233–241
38. Gunter SA, Beck PA, Phillips JM (2003) Effects of supplementary selenium source on the performance and blood measurements in beef cows and their calves. *J Anim Sci* 81:856–864
39. Surai PF, Ionov IA, Kuchmistova EF, Noble RC, Speake BK (1998) The relationship between the levels of α -tocopherol and carotenoids in the maternal feed, yolk and neonatal tissues: comparison between the chicken, turkey, duck and goose. *J Sci Food Agric* 76:593–598
40. China Feed Database (1999) Table of feed composition and nutritive value in China: 1998 edition. *China Feed* 4:25–31