Effects of maternal and progeny dietary selenium supplementation on growth performance and antioxidant capacity in ducklings

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ABSTRACT This study evaluated the effects of selenium (**Se**) supplementation in maternal and offspring diets on performance and antioxidant capacity of ducklings aged from 0 to 2 wk. A total of 144 female Longyan duck breeders aged 22-wk were allotted into 2 treatments and fed a control diet or a 0.16 mg Se/kg supplemented diet. At 40-wk, 120 offspring from each treatment were divided into 2 groups, with 6 replicates of 10 birds. Using a 2 × 2 factorial design, ducklings from each maternal dietary treatment were assigned to a control diet or a 0.16 mg Se/kg supplemented diet from hatch to 2-wk. Compared with Se-deficient diet, maternal diet supplemented with 0.16 mg Se/kg increased the BW of hatchlings ($P < 0.01$). There were interactions between maternal and progeny diet with 0.16 mg Se/kg in BW of ducklings aged 2 wk and BW gain (**BWG**) as ducklings from maternal Se/progeny none treatment had the lightest BW and BWG ($P < 0.01$). Maternal diet with 0.16 mg Se/kg decreased plasma concentration of uric acid and insulin-like growth factor 1 ($P < 0.01$), and progeny diet supplemented with 0.16 mg Se/kg increased the activities of glutathione peroxidase 3 (**GPx3**) in plasma and glutathione peroxidase 1 in erythrocyte ($P < 0.01$). Maternal diet with 0.16 mg Se/kg increased ($P < 0.05$) the hepatic activity of total superoxide dismutase (**T-SOD**). Progeny diet supplemented with 0.16 mg Se/kg increased ($P < 0.01$) hepatic activity of GPx3 and decreased ($P < 0.01$) the hepatic concentration of malondialdehyde. Interactions were detected between maternal and progeny diet with 0.16 mg Se/kg in hepatic activity of T-SOD and maternal and progeny diet supplemented with Se displayed the highest hepatic activity of T-SOD ($P < 0.05$). Overall, Se supplementation in the diet of duck breeders and offspring increased the antioxidant capacity of ducklings. Maternal Se supplementation increased the BW of hatchlings, whereas maternal and progeny dietary Se supplementation did not affect the BWG of ducklings aged from 0 to 2 wk. Se supplementation with additional 0.16 mg/kg in the diet of duck breeders and offspring displayed beneficial effects particularly on the antioxidant capacity in ducklings.

Key words: selenium, duckling, antioxidative biochemistry, muscle development

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INTRODUCTION

Selenium (**Se**) is a vital trace element because it is required by most body organs (brain, pancreas, liver, kidney, spleen, thymus, bursa of Fabricius, intestine, and testes) for normal cellular development and functioning (Zhao et al., 2014; Sun et al., 2015; Huang et al., 2016; Zhang et al., 2019; He et al., 2020). In addition, Se is a crucial component of selenoproteins, which have pivotal significance for optimal animal health mainly due to their antioxidant activity in tissues of all body organs (Zoidis et al., 2018). Dietary Se supplementation, therefore, can enhance the quality of meat and eggs as well as mitigate the oxidative stress caused by high ambient temperatures, immunosuppression, or mycotoxins (Galvano et al., 2001; Surai, 2002; Habibian et al., 2015; Liu et al., 2020). In poultry, Se-deficient diets led to

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impairments in the antioxidant defense system, immune system, and productivity as well as increased embryonic death and reduced hatchability (Emamverdi et al., 2019; Yang et al., 2016). Among the various avian egg components, the yolk has the highest level of Se to protect the polyunsaturated fatty acids which are highly vulnerable to oxidation (Surai, 1999; Golubkina and Papazyan, 2006). Accelerating fatty acid β-oxidation in the hatching process increases the accumulation of reactive oxygen species and free radicals, which are associated with organ malfunctions (Surai et al., 1996; Surai, 1999). In addition, maternal diets that are deficient in Se suppress protein synthesis, thereby adversely affecting the growth of skeletal muscle in their offspring (Gao et al., 2018). The main goal of the current study, therefore, was to test whether adding Se to the diet of ducklings could enhance their growth by improving the antioxidant status when their mothers were fed a diet deficient in Se.

MATERIALS AND METHODS

Duck Breeder Husbandry

All procedures employed in this study were approved by the Animal Care and Use Committee of Guangdong Academy of Agricultural Sciences, Guangzhou, China, with the approval number of GAASISA2018-1030. The experimental birds were obtained from the original breeding farm of Longyan laying duck. A total of 144 female Longyan duck breeders at the age of 22 wk with the same genetic background and comparable BW (1.40 ± 0.01 kg) were allotted randomly into 2 treatments, each with 6 replicates of 12 birds. The experimental ducks were housed in individual galvanized battery cages (length 40 cm × width 27.8 cm × height 55 cm, Guangzhou Huanan Poultry Equipment Co., Ltd, Guangzhou, China). Each battery cage was equipped with a feeder and nipple drinker. In a previous study, we found that supplementing the diet of duck breeders of the same breed with 0.16 mg Se/kg (with a final concentration of 0.27 mg Se/kg) maximized the fertility and hatchability (Xia et al., 2020). In the present study, therefore, the experimental duck breeders were corresponded to diets (containing 0.11 mg Se/kg) either supplemented with 0.16 mg Se/kg (with a final concentration of 0.27 mg Se/kg) in the form of sodium selenite (99.0% Sodium Selenite, Guangdong Newland Feed Science & Technology Co. Ltd, Guangdong, China) or without Se supplementation. The nutrient levels of ME, CP, Ca, Available P, Lys, Met, and Se in basal diet were covered according to our previous findings for laying ducks (Xia et al., 2019a,b, 2020). From hatch to 22 wk of age, the duck breeders used here received diets containing 0.15 to 0.3 mg Se/kg in commercial diets. Chemical and calculated analysis of the basal diet is shown in Table 1. The Se content of diets was determined using fluorometric method as described previously by Cantor and Tarino (1982). Water was available ad libitum and 85 g of feed were introduced twice daily at 7:00 AM and 3:00 PM throughout the experimental period (in total 170 g/bird/d). In addition to ambient daylight, 4 h of artificial light (15 lx/m^2) were provided from 6:30 PM to 10:30 PM to give in total 16 h light: 8 h dark. Male duck breeders of the same breed of females were caged and given a commercial diet containing 10.87 MJ ME/kg, 170 g/kg crude protein, 8.00 g/kg Ca, 3.80 g/kg available phosphorus, and 0.3 mg/kg Se.

At the age of 40 wk, all female duck breeders were artificially inseminated twice (with a 3-d interval) with

Table 1. Composition and nutrient levels in the basal diet of laying duck breeders and ducklings aged from hatch to 2 wk (%, as fed basis).

Item[1]	Selenium supplementation in duck breeder (mg/kg)		Selenium supplementation in duckling (mg/kg)	
	0	0.16	0	0.16
Corn, %	52.30	52.30	61.90	61.90
Soybean meal, %	26.10	26.10	27.8	27.8
Wheat bran, %	10.15	10.15	6.40	6.40
Limestone, %	8.70	8.70	0.77	0.77
Calcium hydrogen phosphate, %	1.30	1.30	1.60	1.60
Lysine, %	0	0	0.05	0.05
DL-methionine, %	0.15	0.15	0.18	0.18
Salt, %	0.30	0.30	0.30	0.30
Premix[1], %	1.00	1.00	1.00	1.00
Total, %	100	100	100	100
Nutrient composition				
ME, MJ/kg	10.45	10.45	11.71	11.71
CP, %	18.0	18.0	19.0	19.0
Ca, %	3.60	3.60	0.85	0.85
Total P, %	0.60	0.60	0.66	0.66
Available P, %	0.35	0.35	0.40	0.40
Total Lys, %	0.95	0.95	1.05	1.05
Total Met, %	0.40	0.40	0.45	0.45
Total Met+Cys, %	0.70	0.70	0.76	0.76
Selenium[2], mg/kg	0.11	0.27	0.15	0.31

[1]The premix provided the following per kilogram of diet: vitamin A 5,500 IU, vitamin D_3 400 IU, vitamin E 10 IU, vitamin K 2.0 mg, vitamin B_1 3.0 mg, vitamin B_2 4.6 mg, vitamin B_6 2.2 mg, vitamin B_{12} 0.02 mg, choline 500 mg, D-calcium pantothenate 7.4 mg, folic acid 1.0 mg, biotin 0.08 mg, Fe 80 mg, Cu 10 mg, Mn 39 mg, Zn 52 mg, I 0.26 mg.

[2]Selenium contents of basal diet in duck breeder and duckling were measured in the mixed feed. Other nutrient levels are calculated values.

100 μL of diluted fresh semen (diluted with 0.9% saline solution in a proportion of 1: 1 volume/volume). 50 eggs per replicate (egg weight > 63 g, with no soft shells, cracks, dirtiness, or double yolks) were collected from the third through tenth day following the first insemination (Xia et al., 2020). All eggs were incubated in the same incubator (Bengbu Sanyuan Incubation Equipment Co., Ltd, Anhui, China) at 37.2°C to 38.0°C and 60 to 75% relative humidity for 28 d.

Duckling Husbandry

After 28 d of incubation, 120 healthy hatched ducklings from each duck breeder treatment group (with or without Se supplementation) were collected and divided randomly into 2 groups, each with 6 replicates of 10 birds. Using a 2 × 2 factorial arrangement of treatments, the offspring of duck breeders from each treatment were randomly assigned to experimental diets of 0.16 mg Se/kg supplementation in the form of sodium selenite or without Se supplementation. The ducklings were fed the 4 dietary treatments from hatch to 2 wk of age. Chemical and calculated analysis of the basal diet is shown in Table 1. All hatchlings were reared in an environmentally controlled room. The temperature was maintained at 34°C on d 1 and decreased by 2 to 4°C each week till 26°C was reached on d 14. The light regimen was 24 h light/0 h dark a day.

Tissue Sampling and Storage

At the end of the experiment with ducklings aged 2 wk, two birds were randomly selected from each replicate and blood samples were collected from the left-wing vein in 5 mL vacutainer tubes at 10:00 am after an overnight fast for 12 h. Within 30 min of collection, the blood was centrifuged (1,200 × g for 10 min) to separate plasma, and the collected plasma was stored in 0.5 mL Eppendorf tubes at −20°C until analysis. Erythrocytes were ruptured by mixing 3 volumes of a Tris-Hcl (1 mmol/L, pH 8.0) buffer solution with one volume of blood, agitated mildly in an ice bath, centrifuged (12,000 × g for 5 min) at 5°C to separate the supernatant cytosol, and stored at −80°C for enzyme activity analyses. The birds were then euthanized by cervical dislocation and exsanguinated. The livers and pectoralis muscles were then collected, excised, washed with 0.9% saline solution, frozen by direct plunging into liquid nitrogen, and stored at −80°C until analysis.

Growth Performance

Ducklings were weighed at the beginning and end of the experiment (at hatch and at 2 wk of age). Live BW and feed consumption (the difference between the added and refused feed amounts) were measured for each pen in order to calculate the body weight gain and feed conversion ratio (**FCR**, kg of feed consumed/kg of live body weight gain). Mortality and health status were recorded daily throughout the experimental period and dead birds were weighed to adjust the calculated FCR.

Chemical Analysis of Plasma, Erythrocytes, and Liver

The plasma activities of glutathione peroxidase (**GPx**), total superoxide dismutase (**T-SOD**), and plasma contents of malondialdehyde (**MDA**) and uric acid (**UA**) were determined spectrophotometrically in duplicates using kits (Jiancheng Bioengineering Institute, Nanjing, China) according to Huang et al. (2015). Plasma concentration of insulin-like growth factor 1 (**IGF-1**) was measured by radioimmunoassay as previously described by Song et al. (2011) with rabbit anti-human IGF1 serum (Tianjing Nine Tripods Medical & Bioengineering Co., Ltd, China).

A quantity of 40 mg of frozen liver was homogenized on ice in 4 mL of homogenization buffer (0.05 mol/L Tris-Hcl, pH 7.4, 1 mmom/L EDTA, 0.25 mmom/L sucrose) with an Ultra-Turrax T8 (IKA, Staufen, Germany) for 5 s at 3,000 × g. The homogenate was centrifuged at 12,000 × g for 10 min at 4°C, and the supernatant was stored at −80°C. The concentrations of MDA and activities of T-SOD, GPx, and T-AOC in the liver and the prepared erythrocyte cytosol of ducklings were measured with kits (Jiancheng Bioengineering Institute, Nanjing, China).

Transcript Abundance of Hepatic and Muscular Genes

Total RNA was extracted from the frozen liver and pectoralis muscle samples using an extraction kit (Invitrogen, Carlsbad, CA). After gel electrophoresis, their quality was confirmed based on the optical density at 260 and 280 nm (1.7 < OD260:280 < 2.0).

Complementary DNA (**cDNA**) was prepared by reverse transcription from 2.5 μg of high-quality RNA in a final volume of 25 μL according to the manufacturer's instructions (Promega, Madison, WI). The primer sequences of the examined genes are shown in Table 2.

The mRNA expression of target genes was examined by quantitative real-time PCR using a MXPro 3500 system (Stratagene, La Jolla, CA) with 1 μL of the cDNA product in a total volume of 20 μL, which contained 10 μL of SYBR-green PCR master Mix (Takara, Biotechnology Co. Ltd, Dalian, China) and 0.5 μL (10 mM) of each primer. The following protocol was used: denaturation for 30 s at 95°C, followed by 35 cycles of 20 s at 95°C, 30 s at 57 or 60°C, and 20 s at 72°C. The transcripts were quantified using a standard curve based on 10-fold serial dilutions of cDNA.

The relative abundance of the targeted genes was normalized to avian β-actin using the ΔCt method ($R = 2^{-\Delta\Delta\ Ct}$) as described previously by Livak and Schmittgen (2001).

Table 2. Primer sequences used for quantitative real-time PCR.

Genes	Primer sequence (5′-3′)	Accession no.	Amplicon (bp)	Ta (℃)
SOD1	F: CCTGTGGTGTCATCGGAATA R: TTGAACGAGGAAGAGCAAGTA	XM_013097859.1	116	57
Gpx1	F: CAGTACATCATCTGGTCGCC R: CCTGGATCTTGATGGTTTCG	KU048803.1	213	57
SEPH	F: GTCGTCATCGAGCACTGCAA R: ATCCTCCTTCACCAGGGACA	XM_027457820.1	156	60
SEPM	F: TGGGGTGACAAGGGTTTGAC R: CTCGGGGCATGGTAAAGGAA	XM_027470226.1	246	60
TXN	F: GCCCAGGATGTTGCTACACA R: CCCCATGGCTGGAGATTAGAC	XM_027446222.1	150	60
FBXO32	F: CCTGGTTAGCCTCGGTCAAG R: CTCCTTCCGTGGGTAACACC	HM627858.1	190	60
TRIM63	F: CAACATCTACTGCGTCACCT R: GAAGCCCGTCTTGTCCTCCT	XM_013096614.3	334	60
Myf5	F: CCGGTTACTGCTCCGAGATG R: TCATAGCGCCTGGTAGGTCC	FJ938240.1	212	57
MyoD	F: GCCTCTCAGGCATTGTGGAA R: TGGTGGGGGAAGGAATTTGG	NM_001310358.1	150	57
MyoG	F: ATCCAGTACATCGAGCGCCT R: TCCACGATGGAGGAGAGTGA	NM_001310376.1	248	57
β-actin	F: GCTATGTCGCCCTGGATTT R: GGATGCCACAGGACTCCATAC	EF667345.1	174	60

Abbreviations: *FBXO32*, F-box protein 32; *Gpx1*, glutathione peroxidase 1; *Myf5*, myogenic factor 5; *MyoD*, myogenic determining factor; *MyoG*, myogenin; *SOD1*, superoxide dismutase1; *SEPH*, selenoprotein H; *SEPM*, selenoprotein M; Ta, annealing temperature; *TRIM63*, tripartite motif-containing protein 63; *TXN*, thioredoxin.

Statistical Analysis

Six replicates were used as the experimental units. The effects of maternal (MSe) and progeny (PSe) dietary Se supplementation were analyzed by two-way ANOVA using the GLM procedure of the Statistical Analysis System (SAS Institute, 2003). The statistical model used for analysis was: Yijk $=\mu$ijk + Mi + Pj + (M × P)ij +εijk, where Yijk is the replicate value of a given variable, μijk is the mean, Mi is the effect of MSe (i = 1 or 2; 0 or 0.16 mg/kg), Pj is the effect of PSe (j = 1 or 2; 0 or 0.16 mg/kg), (M × P) ij is the effect of the interaction between the 2 main effects, and εijk is the random error. The data are expressed as means with SEM, derived from the ANOVA error mean square. Where the ANOVA indicated significant main effects or interactions, the corresponding means were compared using least square difference tests.

RESULTS

Growth Performance

As shown in Table 3, compared with Se-deficient diet, maternal diet supplemented with 0.16 mg Se/kg increased the BW of ducklings at hatch ($P < 0.01$). Interactions were detected between maternal and progeny diets supplemented with 0.16 mg Se/kg in BW of ducklings aged 2 wk as the lightest BW and BWG were from ducklings treated with MSe/None ($P < 0.01$).

Chemical Analysis of Plasma and Erythrocytes

As shown in Table 4, compared with Se-deficient diet, maternal diet supplemented with 0.16 mg Se/kg decreased plasma concentration of UA and IGF-1 in ducklings ($P < 0.05$), while progeny diet supplemented with 0.16 mg Se/kg increased the concentration of GPx3 in plasma and GPx1 in erythrocyte ($P < 0.01$). Interactions exhibited between maternal and progeny diet supplemented with 0.16 mg Se/kg in plasma concentration of T-SOD, GPx3, and UA; ducklings from nonmaternal and progeny Se treatment had the lowest concentration of T-SOD in plasma ($P < 0.05$), while the lowest concentrations of GPx3 and UA in plasma were seen in the ducklings from MSe/None treatment ($P < 0.05$).

Antioxidative Indices in Livers

The effects of maternal and progeny dietary Se supplementation on antioxidative indices in livers of ducklings are summarized in Table 5. Compared with Se-deficient diet, maternal diet supplemented with 0.16 mg Se/kg increased hepatic concentration of T-SOD ($P < 0.05$); progeny diet supplemented with 0.16 mg Se/kg increased hepatic concentration of GPx3 ($P < 0.01$), while decreased the hepatic concentration of MDA ($P < 0.01$). Interactions were detected between maternal and progeny diet supplemented with 0.16 mg Se/kg in hepatic concentration of T-SOD; ducklings from MSe/PSe treatment had the highest hepatic concentration of T-SOD ($P < 0.05$).

Transcript Abundance of Genes Related to Antioxidation in Livers

There were no differences in the transcription abundance of genes related to antioxidation in response to dietary Se supplementation except for hepatic transcription of TXN (Table 6). Compared with Se-deficient diet,

Table 3. Effects of the maternal and progeny dietary selenium (Se) supplementation on growth performance of ducklings from hatch to 2 wk of age.

Item[1]	BW_0 (g)	BW_2 (g)	BWG (g)	ADFI (g/d/bird)	ADG (g/d)	FCR (g/g)
Treatments[2] (n = 6)						
None/None	35.8	210[a]	175[a]	24.6	12.2	2.10
None/PSe	35.8	203[ab]	167[ab]	24.2	11.9	2.04
MSe/None	36.4	195[b]	159[b]	24.0	11.6	2.06
MSe/PSe	36.4	208[a]	172[a]	24.6	11.9	2.06
SEM[3]	0.08	2.68	2.77	0.52	0.36	0.03
Main effect of MSe supplementation						
0 mg/kg	35.8[b]	206	171	24.4	12.0	2.07
0.16 mg/kg	36.4[a]	202	166	24.3	11.8	2.06
SEM[3]	0.05	2.11	2.14	0.34	0.24	0.02
Main effect of PSe supplementation						
0 mg/kg	36.1	203	167	24.3	11.9	2.08
0.16 mg/kg	36.1	205	169	24.4	11.9	2.05
SEM[3]	0.05	2.11	2.14	0.34	0.24	0.02
P-value[4]						
MSe	<0.01	0.14	0.10	0.84	0.48	0.74
PSe	0.91	0.40	0.42	0.81	0.94	0.24
MSe × Pse	0.56	<0.01	<0.01	0.31	0.42	0.22

[1]Abbreviations: BW_0, hatchling body weight; BW_2, body weight at 2 wk; BWG, body weight gain; FCR, feed conversion ratio; MSe, maternal dietary selenium; PSe, progeny dietary selenium.
[2]Means separation in treatments shows significant interaction.
[3]Pooled SEM of treatments (n = 6 replications with 10 birds per replicate).
[4]Means within a column and within an item lacking the same superscript are different ($P < 0.05$).

Table 4. Effects of the maternal and progeny dietary selenium (Se) supplementation on chemical analysis of plasma and erythrocytes of ducklings aged 2 wk.

	Plasma					Erythrocyte
Item[1]	T-SOD (U/mL)	GPx3 (U/mL)	MDA (nmol/mL)	UA (μmol/l)	IGF-1 (ng/mL)	GPx1 (U/mg prot)
Treatments[2] (n = 6)						
None/None	15.6[b]	1,310[b]	17.3	456[a]	55.5	279
None/PSe	18.3[a]	1,601[a]	16.3	449[ab]	50.0	367
MSe/None	18.3[a]	1,202[b]	16.1	434[b]	38.8	281
MSe/PSe	17.9[a]	1,759[a]	14.8	447[ab]	41.6	398
SEM[3]	0.64	59.5	0.82	4.58	3.75	25.9
Main effect of MSe supplementation						
0 mg/kg	17.0	1,462	16.8	452[a]	52.6[a]	328
0.16 mg/kg	18.1	1,469	15.5	439[b]	40.0[b]	343
SEM[3]	0.43	36.8	0.53	3.33	2.79	16.5
Main effect of PSe supplementation						
0 mg/kg	17.1	1,251[b]	16.6	444	45.7	280[b]
0.16 mg/kg	18.1	1,680[a]	15.5	447	46.0	381[a]
SEM[3]	0.43	36.8	0.53	3.33	2.79	16.5
P-value[4]						
MSe	0.08	0.62	0.08	<0.05	<0.01	0.48
PSe	0.06	<0.01	0.13	0.58	0.73	<0.01
MSe × Pse	<0.05	<0.05	0.88	<0.05	0.30	0.54

[1]Abbreviations: GPx3, glutathione peroxidase 3; GPx1, glutathione peroxidase 1; IGF-1, insulin-like growth factor-1; MSe, maternal dietary selenium; MDA, malondialdehyde; PSe, progeny dietary selenium; T-SOD, total superoxide dismutase; UA, uric acid.
[2]Means separation in treatments shows significant interaction.
[3]Pooled SEM of treatments (n = 6 replications with 2 birds per replicate).
[4]Means within a column and within an item lacking the same superscript are different ($P < 0.05$).

maternal diet supplemented with 0.16 mg Se/kg increased the hepatic transcription abundance of TXN ($P < 0.05$).

Transcript Abundance of Genes Related to Muscle Development in Pectoral Muscle

As shown in Table 7, interactions were detected between maternal and progeny diet supplemented with 0.16 mg Se/kg in the transcription abundance of TRIM63 in pectoral muscle; the highest transcription abundance of TRIM63 in pectoral muscle was seen in the ducklings from MSe/None treatment ($P < 0.01$).

DISCUSSION

In duck eggs, more than 90% of the total Se content mainly coming from egg yolk and albumen are utilized to meet the requirement of embryos development before hatching of ducklings (Golubkina and Papazyan, 2006). The newly hatched birds can obtain a certain amount of residual yolk to provide nutrients for growth for several days after hatching (Speake et al., 1998). The results of the current study showed that the maternal diet supplemented with 0.16 mg Se/kg increased the BW of hatchlings. This finding could be attributed to increased Se content in egg yolk and albumen leading to reduced yolk lipid peroxidation and enhanced GPx3 activity in

Table 5. Effects of the maternal and progeny dietary selenium (Se) supplementation on antioxidative indices in livers of ducklings aged 2 wk.

Item[1]	T-SOD (U/mg prot)	GPx3 (U/mg prot)	MDA(nmol/mg prot)
Treatments[2] (n=6)			
None/None	568[ab]	63.0	3.37
None/PSe	531[b]	255	2.79
MSe/None	576[a]	65.6	3.11
MSe/PSe	600[a]	276	2.43
SEM[3]	13.2	6.05	0.20
Main effect of MSe supplementation			
0 mg/kg	548[b]	168	3.05
0.16 mg/kg	587[a]	160	2.80
SEM[3]	10.3	4.00	0.15
Main effect of PSe supplementation			
0 mg/kg	571	64.3[b]	3.23[a]
0.16 mg/kg	560	264[a]	2.64[b]
SEM[3]	10.3	4.00	0.15
P-value[4]			
MSe	<0.05	0.05	0.14
PSe	0.68	<0.01	<0.01
MSe × Pse	<0.05	0.12	0.80

[1]Abbreviations: GPx3, glutathione peroxidase 3; MDA, malondialdehyde; MSe, maternal dietary selenium; PSe, progeny dietary selenium; T-SOD, total superoxide dismutase.
[2]Means separation in treatments shows significant interaction.
[3]Pooled SEM of treatments (n = 6 replications with 2 birds per replicate).
[4]Means within a column and within an item lacking the same superscript are different ($P < 0.05$).

Table 6. Effects of the maternal and progeny dietary selenium (Se) supplementation on the relative hepatic expression levels of genes related to antioxidation in livers of ducklings aged 2 wk.

Item[1]	*SOD1* (a.u.)	*GPx1* (a.u.)	*SEPH* (a.u.)	*SEPM* (a.u.)	*TXN* (a.u.)
Treatments[2] (n = 6)					
None/None	0.94	0.99	1.02	1.06	1.03
None/PSe	1.04	0.98	0.94	0.97	0.96
MSe/None	1.03	1.04	0.97	1.05	1.19
MSe/PSe	0.95	1.05	1.05	0.96	1.24
SEM[3]	0.08	0.11	0.10	0.11	0.11
Main effect of MSe supplementation					
0 mg/kg	1.00	0.98	0.98	1.00	0.99[b]
0.16 mg/kg	0.99	1.04	1.00	1.01	1.21[a]
SEM[2]	0.06	0.08	0.07	0.07	0.07
Main effect of PSe supplementation					
0 mg/kg	0.99	1.01	1.00	1.05	1.11
0.16 mg/kg	1.00	1.01	0.99	0.97	1.08
SEM[2]	0.06	0.08	0.07	0.07	0.07
P-value[3]					
MSe	0.98	0.58	0.77	0.97	<0.05
PSe	0.91	0.99	0.99	0.37	0.91
MSe × Pse	0.29	0.92	0.39	0.99	0.56

[1]Abbreviations: *SOD1*, superoxide dismutase1; *GPx1*, glutathione peroxidase 1; MSe, maternal dietary selenium; PSe, progeny dietary selenium *SEPH*, selenoprotein H; *SEPM*, selenoprotein M; *TXN*, thioredoxin.
[2]Pooled SEM of treatments (n = 6 replications with 2 birds per replicate).
[3]Means within a column and within an item lacking the same superscript are different ($P < 0.05$).

plasma, erythrocyte, and liver of ducklings, when Se concentrations increased in the maternal diet (Xia et al., 2020). It has been reported that dietary Se supplementation enhanced GPx activity, reduced lipid peroxidation and oxidative stress as well as cell apoptosis in chickens (Yao et al., 2013), which could be reflected muscles development and growth rate. Also, studies showed that Se supplementation in the maternal or progeny diets affected the birth weight, BW gain, and FCR of offspring in livestock and poultry (Wang et al., 2011; Lemley et al., 2014). In the current study, the interaction of maternal and offspring Se supplementation were found on BW of duckling aged 2 wk and BW gain, but there was no difference in the BW and BW gain of ducklings between birds fed both maternal and progeny diets with 0.16 mg Se/kg addition and no Se supplementation. This could be due to that dietary intake of Se is not the sole determinant of the net uptake of the element into the tissue, and the regulatory mechanism of dynamic equilibrium prevents deficient retention or excessive accumulation in body tissues for example, the secretion of selenoprotein P from the liver into the circulation and the conversion of excess Se to excretory metabolites are stimulated when the Se concentration of liver is too high (Hill et al., 2003).

Se is an indispensable structural component of the GPx enzyme (Rotruck et al., 1973). The antioxidant effects of GPx activity were shown to be mediated by

Table 7. Effects of the maternal and progeny dietary selenium (Se) supplementation on the relative muscular expression levels of genes related to muscle development in pectoral muscle of ducklings aged 2 wk.

Item[1]	*FBXO32* (a.u.)	*TRIM63* (a.u.)	*Myf5* (a.u.)	*MyoD* (a.u.)	*MyoG* (a.u.)
Treatments[2] (n = 6)					
None/None	0.87	0.87[b]	1.19	0.73	1.07
None/PSe	0.97	1.09[ab]	1.24	0.86	1.25
MSe/None	1.05	1.36[a]	1.06	0.87	1.03
MSe/PSe	1.04	0.91[b]	1.10	0.73	1.17
SEM[3]	0.13	0.12	0.12	0.08	0.15
Main effect of MSe supplementation					
0 mg/kg	1.04	0.99	1.22	0.81	1.18
0.16 mg/kg	0.93	1.15	1.08	0.80	1.10
SEM[3]	0.09	0.08	0.08	0.06	0.10
Main effect of PSe supplementation					
0 mg/kg	0.97	1.13	1.12	0.80	1.05
0.16 mg/kg	1.00	1.01	1.18	0.81	1.22
SEM[3]	0.09	0.08	0.08	0.06	0.10
P-value[4]					
MSe	0.33	0.16	0.27	0.98	0.66
PSe	0.73	0.32	0.72	0.99	0.25
MSe × Pse	0.68	<0.01	0.99	0.11	0.89

[1]Abbreviations: *FBXO32*, F-box protein 32; *Myf5*, myogenic factor 5; *MyoD*, myogenic determining factor; *MyoG*, myogenin; MSe, maternal dietary selenium; PSe, progeny dietary selenium; *TRIM63*, tripartite motif-containing protein 63.
[2]Means separation in treatments shows significant interaction.
[3]Pooled SEM of treatments (n = 6 replications with 2 birds per replicate).
[4]Means within a column and within an item lacking the same superscript are different ($P < 0.05$).

Se, which removes potential damaging lipid hydrogen peroxides and plays a unique role in protecting cells against free radical-induced oxidative stress (Arthur, 2001). The study of Surai (2000) demonstrated that maternal dietary Se supplementation increased Se-dependent glutathione peroxidase activity in the liver of the 1-day-old and 5-day-old chicks. In addition, the finding of Xiao et al., 2016 revealed that maternal dietary Se supplementation increased the mRNA expression of GPx1 in the chick embryo liver. The findings of the present study confirm that progeny dietary Se supplementation increased the activities of GPx3 in plasma and liver as well as GPx1 in erythrocyte, indicating that the lipid hydrogen peroxides could be removed in the term of dietary Se supplementation.

Thioredoxin is well known for its role as an intracellular redox regulator of gene expression (Hirota et al., 1997) and cytosolic anti-oxidative property (Takagi et al., 1998). Maternal diet supplemented with 0.16 mg Se/kg increased the hepatic transcription abundance of *TXN* in ducklings aged 2 wk. This finding suggests that adding 0.16 mg/kg Se in the maternal diets could play a role in improving the antioxidant capacity of ducklings.

In poultry, uric acid is the end product of protein metabolism (Song et al., 2011). The lower concentration of uric acid in the plasma from the offspring of duck breeders fed Se supplementation suggested that the protein degradation was inhibited in ducklings. This goes in line with the finding in the chicken reported by Gao et al. (2018), who suggested that broiler breeder fed with 0.5 mg/kg organic or inorganic Se decreased the uric acid concentration in serum of the offspring. On the other hand, the concentration of IGF-1, an important growth stimulating factor, was reduced here. This could explain the lack of differences in the musculogenic genes transcription abundance in pectoral muscle of ducklings in the current study. Muscle ring finger 1(*Murf1*, also called *TRIM63*) and muscle atrophy F-box (*mafbfx*, also called *FBXO32*) are the 2 ubiquitin ligase that increased the muscular protein degradation (Sacheck et al., 2004; Koyama et al., 2008; Nakashima et al., 2013). Therefore, the higher mRNA expression of *TRIM63* in the in pectoral muscle of ducklings from MSe/None treatment was probably contributed to the inhibition of skeletal muscle development, causing reduced duckling growth.

In conclusion, progeny diets supplemented with 0.16 mg Se/kg or the same dose of Se supplementation in the diet of duck breeders and offspring increased the antioxidant capacity of the ducklings. Whereas maternal and progeny dietary Se supplementation did not affect the body weight gain of ducklings from hatch to 2 wk. Se supplementation with additional 0.16 mg/kg in the diet of duck breeders and offspring displayed beneficial effects particularly on the antioxidant capacity in ducklings.

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DISCLOSURES

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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