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Effect of Maternal Selenium and Methionine on Poultry Products (Egg and Meat) Qualities and Oxidative Stability

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1. Introduction

As the events during critical development periods may influence long-term or life-time structure and function of the body (Angelbeck and Du Bru 1983; Lucasand others 1990, 1996; Snoeck and others 1990; Desaiand others 1995, 1996), the impact of breeders nutrition on nutritional status of off springs has received considerable attention.

Selenium (Se) and methionine (Met) are 2 essential substances for poultry nutrition. Se is an essential component of a variety of selenoproteins, the best known of which is glutathione peroxidase (GSH-Px). 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 hydro peroxides (Brigelius-Flohe1999). Se derived from the diet of the female bird is deposited in the egg and is distributed among the developing tissues during embryo genesis (Ga´alandothers1995; Surai2000; Paton and others2002). Consequently, GSH-Px is expressed in the chicken embryo in a tissue and stage-specific manner(Wilson and others1992; Ga´aland others1995; Surai1999). Supplementary Se in the diet of the hen was shown to increase the concentration of this element in the egg and in the tissues of the chick at hatch, and to elevate the expression of GSH-Px, while reducing the generation of lipid peroxides in the liver of the day-old chick(Surai2000; Paton and others2002). Pappas and others(2005) have shown that dietary supplementation of the female chicken with Se increased Se concentrations and GSH-Px activity in blood, liver, and breast of chicks for 2 to 4 wk post hatch. Surai(2000) also revealed that the effects of maternal Se supplementation remained significantly after10 d of hatching.

Methionine is considered to be the 1st limiting factor in classical diets used for growing chickens that plays unique roles, both in protein structure and in metabolism (Baker 2006). Methionine, an essential dietary amino acid, is used to synthesize proteins and other amino acids. Cysteine and homocysteine are produced during Met metabolism. In most cells,

especially liver cells, about half of cysteine come from Met by the transsulfuration pathway (M`etayer and others 2007). Met plays a particularly important role in providing cysteine for glutathione (GSH) synthesis (Beatty and Reed 1980). These common S-containing amino acids and GSH are antioxidants (Mosharov et al., 2000). All of the elements of the antioxidant system interact with each other and form an efficient antioxidant defense. This interaction probably starts at the level of nutrient absorption and continues during metabolism (Surai, 2000). All these common sulfur-containing amino acids and GSH are antioxidants(Mosharov and others 2000).

In order to study the effects of selenium **(Se)** and methionine **(Met)** supplementation of breeder hens diets on their eggs qualities and offspring's meat quality, a total of four hundred fifty 52-week-old Lang-shan hens (dual-purpose type, an indigenous poultry breed of China) were randomly divided into 9 treatments with 5 replicates each treatment. Birds were fed corn and soybean-based diets (0.13mg Se/kg) supplemented with 0, 0.30 and 0.60 mg/kg Se from Se yeast and 3.2, 4.0 and 5.4 g of DL-Met/kg, respectively. A 30-d adapting period and 70-d experiment period were used for collecting eggs. After incubation for 21 d, 160 healthy chicks from each treatment group were randomly divided into 5 replicates and fed with the same corn and soybean-based diet similar to industry recommendations for another 3-phase feeding program. The starting phase was fed 1.20% total Lys and 2.80 Mcal/kg of ME

from 0 to 21 d, the growing phase from 22 to 42 d with 1.08% total Lys and 2.70 Mcal/kg of ME, and the finishing phase from 43 to 91 d with 0.90% total Lys and 2.60 Mcal/kg of ME. Then the effects of Se and Met supplementation of breeder hen diets on physical qualities and antioxidant capacity of the breeding eggs, meat quality and antioxidant capacity of their male offspring and Se concentration and oxidative stability of lipids in the thigh muscles of progeny were examined.

1.1 Se concentration and oxidative stability of lipids in the thigh muscles of progeny

With precooked and chilled storage, poultry meat is particularly prone to oxidation due to its high polyunsaturated fatty acid (PUFA) content(Mercier and others 1998; Racanicci and others 2004), which results in rancid flavor development and decreased quality. Se and Met to diets can increase anti oxidative capacity in animals. Unfortunately, there is little information available on the effects of Se and Met supplementation of breeders on lipid oxidation of the meat of progeny during growth. In order to study such effect, Lang-shan breeding hens (450) were obtained at 52 wk of age and randomly allotted to 9 treatments; 5 replicates of each treatment were carried out. The breeders were fed a basal corn–soybean meal diet (0.13 mg Se/kg) supplemented with 0, 0.30, or 0.60 mg/kg Se from Sel-Plex and 0.32%, 0.40%, or 0.54% Met for the 30-d adapting period and 70-d experiment period. Se and glutathione (GSH) concentrations, glutathione peroxidase (GSH-Px) activity, and the oxidative stability of muscular lipids of 90-d progeny were determined by testing the TBARS values to evaluate the effects of Se and Met supplementation of breeders on lipid oxidation of the meat of progeny during growth.

2. Experiment parameters measured

2.1 Se concentration

The Se content in progeny thigh was generally between approximately 0.075 and 0.093 mg/kg (Table 1). The main effect of either dietary Se or Met supplementation of breeders

was not significant, but a significant interaction was found between them ($P < 0.01$). Compared to the control treatment, Se concentration in progeny thigh was reduced with the supplementation of 0.60 mg Se/kg diet of breeders($P < 0.05$), but the supplementation effect was significant only when the breeders were supplemented with 0.32% Met (Table 1). Se content was increased in addition to 0.54% Met supplementation compared to 0.32% ($P < 0.05$), when breeders were supplemented with 0.6 mg Se/kg diet (Table 1).

Se mg/kg	Met %	Se content mg/kg	GSH-Px EU	GSH mmol/g pro	TBARS-6 h mg/kg	TBARS-3 d mg/kg
0.00	0.32	$0.093^{a} \pm 0.009$	$1.71^{bc} \pm 0.61$	$40.47^{ab} \pm 0.83$	$1.87^{d} \pm 0.35$	$3.83^{de} \pm 0.35$
	0.40	$0.088^{a} \pm 0.005$	$1.97^{abc} \pm 0.19$	$39.45^{ab} \pm 1.06$	$2.53^{bc} \pm 0.31$	$4.24^{abcd} \pm 0.50$
	0.54	$0.085^{ab} \pm 0.009$	$2.35^{a} \pm 0.10$	$42.26^{a} \pm 3.92$	$3.11^{a} \pm 0.25$	$4.66^{a} \pm 0.33$
0.30	0.32	$0.083^{ab} \pm 0.006$	$2.00^{ab} \pm 0.29$	$39.95^{ab} \pm 1.09$	$2.82^{ab} \pm 0.32$	$4.33^{abc} \pm 0.28$
	0.40	$0.087^{a} \pm 0.001$	$1.79^{bc} \pm 0.26$	$38.35^{b} \pm 1.06$	$2.67^{b} \pm 0.26$	$4.14^{bcd} \pm 0.19$
	0.54	$0.087^{a} \pm 0.007$	$1.92^{abc} \pm 0.26$	$41.60^{ab} \pm 1.09$	$2.53^{bc} \pm 0.55$	$4.57^{ab} \pm 0.36$
0.60	0.32	$0.075^{b} \pm 0.010$	$1.67^{bc} \pm 0.31$	$38.41^{b} \pm 2.98$	$2.21^{cd} \pm 0.29$	$4.06^{cd} \pm 0.23$
	0.40	$0.085^{ab} \pm 0.005$	$1.89^{abc} \pm 0.30$	$41.24^{ab} \pm 2.09$	$2.49^{bc} \pm 0.12$	$3.94^{cd} \pm 0.24$
	0.54	$0.092^{a} \pm 0.006$	$1.50^{c} \pm 0.04$	$41.21^{ab} \pm 2.48$	$2.03^{d} \pm 0.22$	$3.45^{e} \pm 0.33$
0.00		0.089 ± 0.008	$2.01^{a} \pm 0.43$	40.73 ± 2.48	$2.50^{a} \pm 0.60$	$4.24^{a} \pm 0.51$
0.30		0.086 ± 0.006	$1.91^{ab} \pm 0.26$	39.96 ± 1.69	$2.67^{a} \pm 0.38$	$4.35^{a} \pm 0.32$
0.60		0.084 ± 0.010	$1.68^{b} \pm 0.28$	40.29 ± 2.68	$2.24^{b} \pm 0.28$	$3.82^{b} \pm 0.37$
	0.32	0.084 ± 0.011	1.80 ± 0.42	$39.61^{b} \pm 1.94$	$2.30^{b} \pm 0.50$	4.07 ± 0.34
	0.40	0.086 ± 0.004	1.88 ± 0.24	$39.68^{b} \pm 1.83$	$2.56^{a} \pm 0.24$	4.11 ± 0.34
	0.54	0.088 ± 0.008	1.92 ± 0.39	$41.69^{a} \pm 2.53$	$2.56^{a} \pm 0.57$	4.23 ± 0.65
P-value						
Se		0.218	0.04	0.68	0.003	0.0002
Met		0.295	0.58	0.04	0.048	0.4062
Se * Met		0.009	0.04	0.26	<0.0001	0.0002

[a,b]Means within a column lacking a common superscript differ ($P < 0.05$).

Table 1. The effect of dietary Se and Met supplementation of breeders on Se and GSH content, GSH-Px activity, and stability of lipid oxidation of progeny thigh.

2.2 GSH-Px activity

GSH-Px activity of progeny thigh was decreased ($P < 0.05$) in supplementation of 0.60 mg Se/kg diet of breeders compared to the control treatment; however, the main effect of Met supplementation was not significant(Table 1). Se and Met had a significant interaction ($P < 0.05$) with regard to GSH-Px activity (Table 1). Dietary supplementation of breeders with higher Se significantly decreased GSH-Px activity($P < 0.05$), but this effect was only significant when Met supplementation was 0.54% (Table 1). For the treatments not supplemented with Se, GSH-Px activity was increased by the supplementation of 0.54% Met compared to 0.32% ($P < 0.05$). The treatment that was not supplemented with Se and contained 0.54% of Met had the highest GSH-Px activity in progeny thigh, and the lowest GSH-Px activity was found in the groups that had Se and Met, both with the highest or the lowest levels.

2.3 GSH content

GSH content of progeny thigh was not influenced by dietary supplementation with Se ($P > 0.05$). Conversely, it was higher in subjects with the highest supplementation of Met(0.54%) ($P < 0.05$), and there was no significant difference between groups supplemented with 0.32% and 0.40% Met. Se and Met had no significant interaction($P > 0.05$) on the concentration of GSH.

2.4 TBARS content

The TBARS content, expressed as milligram MDA equivalents per kilogram meat, was increased ($P < 0.05$) with chilled storage time (Table 1). Dietary supplementation of breeders

with 0.60 mg Se/kg diet reduced ($P < 0.05$) the TBARS content of progeny thigh at both 6 h and 3d (Table 1), but its effect was significant only when dietary supplementation with Met was 0.54%. With the exception of a significant reduction ($P < 0.05$) in TBARS content at 6 h, dietary supplementation with 0.30 mg Se/kg diet, with respect to that which was not supplemented, did not result in significant variations in the se substances at 3 d when breeders were supplemented with 0.54% of Met. This leads to the supposition that 0.30 mg Se/kg was not sufficient to guarantee adequate protection against oxidative phenomena when the lipid oxidation was greater after 3d under the meat storage conditions employed in this study. However, when the breeders received the diets with 0.32% of Met,0.30 mg Se/kg dietary supplementation revealed the highest TBARS content ($P < 0.05$), and there was no evident difference ($P > 0.05$) between the control and the treatment supplemented with 0.6 mg Se/kg diet both at 6 h and 3 d. Irrespective of the effect of Se supplementation, addition of 0.54% and 0.40% Met increased TBARS content of progeny thigh ($P < 0.05$) compared to 0.32% at 6 h, and did not result in significant difference in the se reactive substances at 3d ($P > 0.05$). When the breeders were not supplemented with Se, TBARS content of progeny thigh was increased ($P < 0.05$) with the supplementation of Met at 6 h and 3 d. However, when the breeders received diets with 0.6 mg Se/kg, 0.54% Met supplementation decreased the TBARS content ($P < 0.05$) compared to 0.40% at 6 h and the other 2 levels at 3d, and there was no significant difference between treatments supplemented with 0.32% and 0.40% of Met ($P > 0.05$).

In summary, the results can be concluded as:

1. When breeders were received the higher levels of Met or Se, GSH-Px activity in their progeny's thigh muscle was decreased, while the Se concentration and the oxidative stability of muscular lipids were increased. When breeder hens were given a Met-deficient diet, supplementing with Se decreased the Se deposition in progeny thigh. With regard to lipid oxidation, 0.3 mg/kg maternal Se supplementation decreased the oxidative stability of muscle lipid and 0.6 mg/kg Se supplementation showed no difference from the control group. When breeders were fed a Se-deficient diet, the GSH-Px activity was significantly increased and the oxidative stability of progeny muscles was decreased with the supplementation of Met.
2. A significant interactive effect between maternal Se and Met on relative quantity of primary volatile oxidative compounds produced at the beginning of warmed-over flavour (WOF) was found ($P<0.05$), but the main effect of either Se or Met was not significant ($P>0.05$). Both the higher levels of Se and Met could result in the lower content of total aldehyde, hexanal, 1-pentanol, but the higher concentration of 2,3-octanedione and 2-pentyl-furan in progeny thigh muscle ($P<0.05$). There was no significant effects of maternal Se and Met supplementation on the content of volatile oxidative compounds produced at the later stage of WOF ($P<0.05$). The relative quantity of volatile oxidative compounds as a result of WOF development was significantly influenced by the chilled storage time ($P<0.01$). The contents of total aldehyde, total acids, hexanal, pentanal and 1-pentanol were found to negatively covary and decrease with increasing days of storage, while the relative quantities of total hydrocarbon, total ketone, octanal, nonanal, 1-octanol, 2-octen-1-ol, 1-octen-3-ol, 2,3- octanedione and 2-pentyl-furan increased with the storage time.

5.4 g of Met/kg treatment exhibited the highest concentrations of free fatty acids at 6 h ($P<0.05$); the contents of myristic acid, palmitoleic acid, linolenic acid and docosahexaenoic

acid at 6 h significantly increased as a result of maternal Se supplementation ($P<0.05$); the interactive effect of Se and Met on the contents of free fatty acids at 6 h was not significant ($P>0.05$). Also, there were no significant effects of dietary Se and Met supplementation of breeder hens on the concentrations of free fatty acids at 3 d. Free fatty acids and phospholipid fatty acids were all found to decrease with the prolong of storage time ($P<0.01$). The oxidative rates of phospholipid fatty acids were significantly higher than those of free fatty acids ($P<0.01$). The most decreased fatty acids from 6 h to 3 d both in free fatty acids and in phospholipid fatty acids were linolenic acid, palmitoleic acid and myristic acid.
Se is an essential component of GSH-Px, which plays an important role in the anti-oxidation system of tissue. Irrespective of Met supplementation levels, GSH-Px activity of progeny thigh was decreased with the supplementation of Se, in agreement with the results reported by Waschulewski and Sunde (1988a), who founded that when weaning rats received 0.5 mg Se/kg, the percentage of muscle Se present as GSH-Px was decreased at all of Met levels. Additionally, supplementation of the breeder's diet with Se increased the protective ability of progeny thigh against lipid oxidation, which was shown by lower TBARS values.
Met participates in methyl group metabolism and the synthesis of other sulfur amino acids, notably cysteine. Cysteineis required for the synthesis of GSH and taurine, which are essential compounds for host defense against oxidative stress (M`etayer and others 2007). GSH concentration of progeny thigh was significantly increased with the supplementation of Met. Lipid oxidation was not influenced by the supplementation of Met, except that TBARS values after 6 h were elevated with the supplementation of Met.

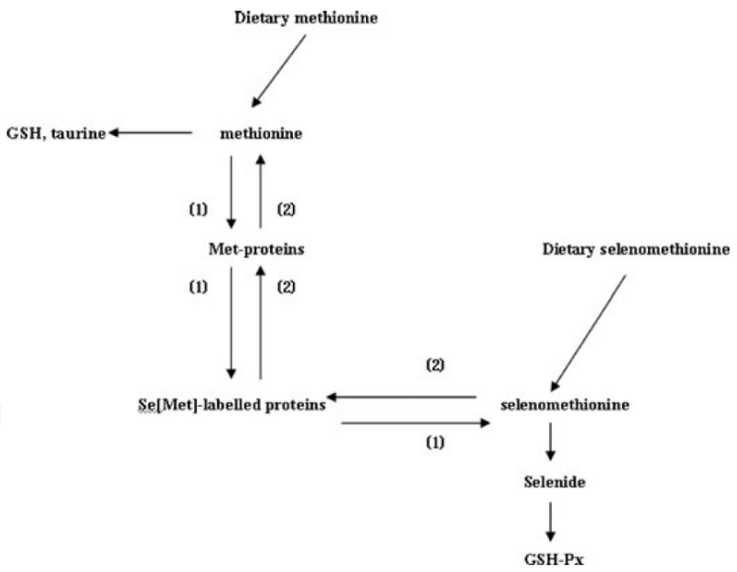


Fig. 1. Proposed schemeof the metabolism of Se (Se[Met]) and Met. Pathway 1, free Met, arising from dietary Met or from protein turnover, is incorporated into proteins for synthesis GSH-Px. Pathway 2, free Se, arisling from dietary Se [Met] or from protein turnover, is incorporated into proteins in place of Met as mediated by $tRNA^{Met}$ (McConell and Hofman 1972), so there is more Met for catabolizing GSH, taurine, and other sulfur-contalning antloxidants.

The interaction of dietary Met and Se can be explained because of the common metabolic pathways of these amino acids. The Se used in this experiment was mainly present in the

form of Se[Met]. Se[Met] can be an excellent analogue for Met in biochemical reactions because of the similar covalent radio of Se and S. As McConnell and Cho(1965) mentioned, Se[Met] is transported by the same intestinal transport system as Met, it is readily esterified to tRNA Met (Hoffman and others 1970), and it can substitute for Met during protein synthesis in eukaryotes (McConnell and Hoffman 1972). Thus Se[Met] can be metabolized by the same enzymes that incorporate Met into protein. It is also likely that Se[Met] follows the same catabolic pathways as Met until 1 of the C-Se bonds is broken (Sunde1984). These common pathways thus can be combined into a diagram of Se[Met] and Met metabolism (Figure 1) that could be used to illustrate and summarize the 2 fates of dietary Se[Met] and Met. When breeders received the Se-deficient diet, dietary supplementation with Met would release Se[Met] from body proteins, which could be converted to selenide by either of the 2 Met catabolic pathways as described by Steeleand Benevenga (1978) and Esakiand others(1982), thus Se from Se[Met] would be available for co-translational incorporation into GSH-Px and other seleno proteins as proposed by Sunde and Evenson (1987). The same effect of Met on the utilization of the deposited tissue Se for GSH-Px synthesis was reported by Waschulewski and Sunde (1988b). The ability of progeny thigh for preventing lipid oxidation was decreased concomitant with increased GSH-Px activity and decreased Se concentration. It was suggested that Met used for catabolizing to GSH, taurine, and other sulfur-containing antioxidants could be reduced (pathway1). When breeders were given sufficient Se (0.6 mg Se/kg) then decomposition of Met to GSH, taurine, and other sulfur-containing antioxidants was increased substantially as dietary Met was raised from deficient to adequate levels, and the protective ability of progeny thigh against lipid oxidation was significantly increased. It was supposed that the catabolism of Met increases the utilization of Se from dietary Se[Met] for protein synthesis, and thus increase the Se concentration of muscle (pathway2), which was contrary to the result of Waschulewski and Sunde (1988a) who found that when weanling rats were given 0.5 mg Se as Se[Met] per kilogram, muscle Se concentration was significantly decreased with the supplementation of Met. The difference would be related to the supplemented Met levels, the type of experiment animals, and the transport of nutriment from hen to embryo andthen to progeny. Similarly, when breeder stook adequate (0.54%) Met, dietary supplementation of breeders with Se substantially decreased the utilization of dietary Se[Met] for GSH-Px synthesis, and it led to preferential incorporation of Se[Met] into tissues in a form other than as GSH-Px. This resulted in elevated muscle Se levels and lower GSH-Px activity in Se-adequate treatment relative to the Se-deficient treatment. Also, dietary Se[Met] supplementation substantially increased the catabolism of Met for the synthesis of GSH, taurine, and other sulfur-containing anti oxidants, therefore increased the protective ability of muscle tissue against lipid oxidation (pathway 2). When breeders received a Met-deficient diet, supplementation of 0.3 mg Se[Met]/kg tended to increase the utilization of Se for GSH-Px synthesis and accelerate the catabolism of Se-labeled muscle proteins, which gave rise to more free Met for synthesis of muscle proteins and thereby decreased muscle anti oxidative ability (pathway 1). This was contrary to the result of Sunde and others(1981) and Waschulewski and Sunde (1988b) who reported that Se availability for GSH-Px synthesis from dietary Se[Met] was reduced when supplemented at less than 0.5 mg Se/kg in rats fed on a Met-deficient diet. However, increments of dietary Se[Met] to 0.6 mg/kg lessened this tendency; there was more Se for incorporation into muscle proteins instead of GSH-Px and more Met for synthesis of GSH, taurine, and other sulfur-containing anti oxidants. As a result, decreased GSH-Px activity and increased ability of protection against lipid oxidation were revealed (pathway 2).

Meat quality and antioxidant capacity of their male offspring

Meat quality (like color and drip loss) affected the acceptability at the time of consumer purchase to an extent. Meat discoloration was believed to be related to the effectiveness of the oxidation processes (Faustman and Cassens, 1990). Changes associated with oxidation include unpleasant tastes and odors, discoloration, protein solubility, and even potential formation of toxic compounds (Baron and Andersen, 2002). Lipid oxidation reduced the shelf life of meat and decreased nutritive and sensory quality of meat. Protein oxidation reduced meat product quality (Decker et al., 1993). Therefore, the meat industry has great interest in improving meat quality and optimizing meat color and water-holding capacity, limiting meat discoloration and loss of fluids (drip loss) because it implies a financial loss.
Four hundred fifty 52-wk-old Lang-shan breeding hens (dual-purpose type, an indigenous poultry breed of China) were randomly divided into 9 treatments with 5 replicates each treatment. They were fed corn-soybean diets with 0, 0.30, and 0.60 mg of Se/kg from Se yeast and 3.2, 4.0, and 5.4 g of dl-Met/kg, respectively. After incubation, 250 chickens each treatment were randomly divided into 5 replicates and fed the same diet. At 21 d old, 10 male chicks in each treatment were slaughtered. Color, water-holding capacity, and oxidative stability were examined to elucidate the effects of Se and Met supplementation of the maternal diets on of their male offspring meat at the early stage.

3. Experimental parameters measured

3.1 Se content

The influence of Se yeast, Met, and their interactions on the Se content in 21-d-old male offspring breast meat was remarkable ($P < 0.01$; Table 2). The Se content significantly increased with the increase of the maternal Se yeast ($P < 0.01$). However, the Se content in 5.4 g of Met/kg treatments were significantly less than those of 3.2 and 4.0 g of Met/kg treatments ($P < 0.01$).

Se (mg/kg)	Met (g/kg)	Se (μg/100 mg)	MDA (mg/kg)	Protein carbonyls (nm/mg)
0	3.2	$0.42 \pm 0.04^{d,D}$	$0.43 \pm 0.02^{a,A}$	$5.53 \pm 0.29^{a,A}$
	4.0	$0.49 \pm 0.06^{c,C}$	$0.30 \pm 0.03^{d,CD}$	$4.31 \pm 0.34^{bc,BC}$
	5.4	$0.40 \pm 0.05^{d,D}$	$0.29 \pm 0.03^{d,D}$	$2.69 \pm 0.28^{e,D}$
0.3	3.2	$0.55 \pm 0.03^{b,B}$	$0.37 \pm 0.03^{b,B}$	$4.54 \pm 0.48^{b,B}$
	4.0	$0.60 \pm 0.04^{b,B}$	$0.29 \pm 0.07^{d,D}$	$3.88 \pm 0.38^{cd,BC}$
	5.4	$0.55 \pm 0.01^{b,B}$	$0.27 \pm 0.05^{d,D}$	$2.87 \pm 0.35^{e,D}$
0.6	3.2	0.71 ± 0.02^{Aa}	$0.29 \pm 0.04^{d,D}$	$4.45 \pm 0.78^{bc,B}$
	4.0	$0.69 \pm 0.04^{a,A}$	$0.33 \pm 0.06^{c,BC}$	$4.04 \pm 0.61^{bcd,BC}$
	5.4	$0.59 \pm 0.04^{b,B}$	$0.29 \pm 0.04^{d,D}$	$3.62 \pm 0.50^{d,C}$
0		$0.44 \pm 0.06^{c,C}$	$0.34 \pm 0.07^{a,A}$	4.18 ± 1.23^{a}
0.3		$0.57 \pm 0.04^{b,B}$	$0.31 \pm 0.05^{b,B}$	3.76 ± 0.80^{b}
0.6		$0.66 \pm 0.06^{a,A}$	$0.30 \pm 0.04^{b,B}$	4.03 ± 0.69^{ab}
	3.2	$0.56 \pm 0.13^{b,A}$	$0.36 \pm 0.06^{a,A}$	$4.84 \pm 0.72^{a,A}$
	4.0	$0.59 \pm 0.10^{a,A}$	$0.31 \pm 0.04^{b,B}$	$4.08 \pm 0.47^{b,B}$
	5.4	$0.51 \pm 0.09^{c,C}$	$0.28 \pm 0.03^{c,C}$	$3.06 \pm 0.55^{c,C}$
P-value				
Se		0.0001	0.0001	0.0338
Met		0.0001	0.0001	0.0001
Se × Met		0.0033	0.0001	0.0002

[a–d]Means in a column with different superscripts are significantly different ($P < 0.05$).
[A–D]Means in a column with different superscripts are significantly different ($P < 0.01$).

Table 2. Effects of the Se yeast and Met supplementations of the maternal diets on Se, malondialdehyde (MDA), and protein carbonyl content of their male offspring breast meat (n = 10)

3.2 Protein and lipid oxidation

There were significant effects of interactions between maternal Se yeast and Met on carbonyl content (P < 0.01; Table 2). The carbonyl content significantly decreased with increase of Met supplementation (P < 0.01) and the carbonyl content of 0 mg of Se/kg treatments were higher than those of 0.3 mg Se/kg treatments (P < 0.01). Moreover, 0.30 and 0.60 mg of Se/kg treatments significantly decreased MDA content compared with those of 0 mg of Se/kg treatments (P < 0.01). The 4.0 and 5.4 g of Met/kg treatments significantly decreased MDA content compared with those of 3.2 g of Met/kg treatments (P < 0.01).

Se (mg/kg)	Met (g/kg)	L* value	a* value	b* value	Drip loss (%)
0	3.2	$57.00 \pm 3.89^{bcd,BC}$	$2.18 \pm 0.18^{bc,B}$	$7.87 \pm 0.85^{a,A}$	$2.16 \pm 0.20^{a,A}$
	4.0	$61.45 \pm 2.36^{a,A}$	$1.91 \pm 0.32^{c,B}$	$7.33 \pm 0.50^{ab,AB}$	$1.91 \pm 0.23^{b,AB}$
	5.4	$54.10 \pm 2.62^{d,C}$	$2.98 \pm 0.33^{a,A}$	$6.24 \pm 0.25^{c,C}$	$1.57 \pm 0.30^{de,CD}$
0.3	3.2	$59.78 \pm 1.94^{ab,AB}$	$2.30 \pm 0.30^{a,B}$	$6.82 \pm 0.57^{bc,BC}$	$1.83 \pm 0.16^{bc,BC}$
	4.0	$57.13 \pm 3.96^{bcd,BC}$	$2.25 \pm 0.30^{bc,B}$	$6.62 \pm 0.45^{bc,BC}$	$1.63 \pm 0.25^{cd,BCD}$
	5.4	$55.89 \pm 1.75^{cd,BC}$	$2.85 \pm 0.38^{a,A}$	$6.23 \pm 0.65^{c,C}$	$1.20 \pm 0.15^{f,E}$
0.6	3.2	$57.05 \pm 2.43^{bcd,BC}$	$2.25 \pm 0.36^{bc,B}$	$6.69 \pm 0.42^{bc,BC}$	$1.72 \pm 0.18^{bcd,BC}$
	4.0	$55.93 \pm 1.34^{cd,BC}$	$2.88 \pm 0.38^{a,A}$	$6.74 \pm 0.13^{bc,BC}$	$1.37 \pm 0.19^{ef,DE}$
	5.4	$58.45 \pm 3.01^{bc,AB}$	$3.10 \pm 0.39^{a,A}$	$7.86 \pm 0.77^{a,A}$	$1.33 \pm 0.26^{f,DE}$
0		57.52 ± 4.23	$2.36 \pm 0.54^{b,B}$	$7.15 \pm 0.88^{a,A}$	$1.88 \pm 0.34^{a,A}$
0.3		57.60 ± 3.10	$2.47 \pm 0.42^{b,B}$	$6.56 \pm 0.59^{b,B}$	$1.55 \pm 0.32^{b,B}$
0.6		57.14 ± 2.49	$2.74 \pm 0.51^{a,A}$	$7.10 \pm 0.96^{a,A}$	$1.47 \pm 0.27^{b,B}$
	3.2	57.94 ± 3.05^{a}	$2.24 \pm 0.28^{b,B}$	7.13 ± 0.81	$1.90 \pm 0.26^{a,A}$
	4.0	58.17 ± 3.59^{a}	$2.35 \pm 0.52^{b,B}$	6.90 ± 0.78	$1.63 \pm 0.31^{b,B}$
	5.4	56.15 ± 3.02^{b}	$2.97 \pm 0.37^{a,A}$	6.78 ± 0.97	$1.37 \pm 0.28^{c,C}$
P-value					
Se		0.8247	0.0005	0.0050	0.0338
Met		0.0238	0.0001	0.1896	0.0001
Se × Met		0.0001	0.0012	0.0001	0.0002

[a–f]Means in a column with different superscripts are significantly different ($P < 0.05$).
[A–E]Means in a column with different superscripts are significantly different ($P < 0.01$).

Table 3. Effects of the Se yeast and Met supplementation of the maternal diets on color and drip loss of their male offspring breast meat (n = 10)

3.3 Meat color

The maternal Met significantly influenced L* value (0.01 < P < 0.05) and a* value (P < 0.01; Table 3). Supplementation of Met at 5.4 g/kg significantly increased a* value compared with 3.2 and 4.0 g of Met/kg (P < 0.01). The 0.60 mg of Se/kg treatment significantly increased a* value compared with 0 and 0.30 mg of Se/kg treatments (P < 0.01) and 0 mg of Se/kg treatment significantly increased b* value compared with 0.3 and 0.6 mg of Se/kg treatments (P < 0.01). Moreover, the interactions of the maternal Met and Se yeast significantly influenced L*, a*, and b* values (P < 0.01).

3.4 Drip loss

The Se yeast and Met supplementation significantly decreased drip loss (P < 0.01) (Table 3). Supplementation of Se at 0.30 and 0.60 mg/kg decreased drip loss compared with 0 mg of Se/kg treatment.

In summary, the results can be concluded as:

1. Supplementation of Met at 5.4g/kg significantly increased International Commission on Illumination a* value compared with 3.2 and 4.0 g of Met/kg (P<0.01). Supplementation of Se at 0.6 mg/kg significantly increased a* value compared 0 and 0.3 mg of Se/kg (P<0.01) and 0 mg of Se/kg significantly increased b* value compared with 0.30 and 0.60 mg of Se/kg (P<0.01).

2. Selenium supplemented at 0.30 and 0.60 mg/kg decreased drip loss compared with 0 mg of **Se**/kg and 4.0 and 5.4 g of **Met**/kg decreased drip loss compared with 3.2 g of **Met**/kg, respectively.
3. The carbonyl content of the myofibrillar protein significantly decreased with the increase of **Met** supplementation ($P<0.01$) and the carbonyl content of the 0 mg of **Se**/kg treatment was higher than the 0.3 mg of **Se**/kg treatment ($P<0.01$). Selenium supplementation at 0.30 and 0.60 mg/kg significantly decreased MDA content compared with that of 0 mg of **Se**/kg ($P<0.01$) and 4.0 and 5.4 g of **Met**/kg supplementation significantly decreased MDA content compared with that of 3.2 g of Met/kg($P<0.01$). However, the higher levels of **Met** and **Se** (5.4 g/kg **Met** and 0.60 mg/kg **Se**) significantly increased reactive sulfhydryl content in the muscle.
4. The intermediate levels of **Met** and **Se** (4.0 g/kg Met and 0.30 mg/kg **Se**) significantly increased heat-induced gel hardness and water-holding capacity of the gel and the higher levels of **Met** and **Se** (5.4 g/kg **Met** and 0.60 mg/kg **Se**) decreased gel hardness and water-holding capacity of the gel to an extent.

Whether the Se was in an inorganic form or part of an organic molecule in the maternal diets would influence the Se content transferred to the developing embryo and the 2-wk-old chickens after hatch (Paton et al., 2002; Pappas et al., 2005). Maternal Se yeast could significantly increase the Se content of serum, liver, and muscle tissues in newborn chickens and quails (Surai, 2000). Pappas et al. (2005) reported that the high levels of maternal Se could significantly increase Se content of serum, liver, and muscle tissues from 2- to 4-wk-old chickens. In this study, all of the progeny were fed the diet with the same Se concentration throughout the experiment. Therefore, the differences in the Se content of the breast meat were due solely to the different Se content of the maternal diets.

Selenomethionine, the Se-containing analog of Met, is thought to be the common form of Se in foodstuffs of plant origin. Beilstein and Whanger (1986) reported that Selenomethionine supplementation could increase tissue Se levels substantially. This study also showed that 4.0 g of Met/kg supplementation of the maternal diet could increase Se content of the chicken meat. It might be due to the fact that tissue Se was present as selenomethionine when Se and Met were provided at high levels in the diets.

The oxidative stability of meat depends upon the balance between anti- and prooxidants. One approach to enhancing the oxidative stability of meat is to add antioxidants either into the diet of the animal or directly during processing. Selenium is an essential component of the antioxidant enzyme GSH-Px (Surai and Dvorska, 2002a, b) and Met plays a particularly important role in providing Cys for GSH synthesis (Beatty and Reed, 1980). Supplementing broiler diets with 0.25 mg/kg of Se substantially increased GSH-Px activity in breast (2.1-fold) and leg (4.1-fold) muscle and as a result decreased lipid peroxidation was detected (2.5-fold in breast muscle and 3.3-fold in leg muscles) after 4 d of storage at 4°C compared with the control group (Devore et al., 1983). Ryu et al. (1995) reported that the dietary Se from 1 to 8 mg/kg revealed only minor improvements in the oxidative stability of 42-d-old chicken meat and 8 mg/kg of Se supplementation in combination with 100 IU of α-tocopherol was more effective in reducing lipid oxidation during refrigerated storage. Several authors have measured protein oxidation in meat and meat products and have related this parameter to lipid oxidation (Mercier et al., 1998, 2004; Ventanas et al., 2006). In this study, the Se yeast and Met supplementation significantly decreased protein carbonyl and MDA content. Based on the fact presented above that the hen diets could affect Se

intake in the chicks and should modulate antioxidant enzyme GSH-Px activities, it is possible to suggest that GSH-Px contributes to the overall antioxidant defense of muscle, decreasing tissue susceptibility accomplished by organic Se supplementation of the hen diets.

The oxidative state of muscle pigments plays an important role in meat color. Redness is related to myoglobin content and its chemical state in meat (Mancini and Hunt, 2005). Ryu et al. (1995) reported that the dietary Se and α-tocopherol levels did not affect 42-d old chicken meat color. However, this present study indicated that the Se yeast and Met supplementation of the maternal diets could increase meat color stability to an extent. It might be due to the fact that that Se yeast and Met supplementation of the maternal diets could decrease protein oxidation to an extent.

Meat oxidation could decrease hydrolysis sensitivity, weaken protein degradation, and reduce water reservation among myofibrils, which increase juice loss of meat (Elisabeth and Steven, 2005). Drip is a dilute solution of the sarcoplasmic proteins. Factors that affect the state of myofibrillar proteins, like protein and lipid oxidation, will also affect drip loss. Carbonyl group formation is the main chemical modification of amino acids during oxidation. Lipid oxidation also could increase cell membrane permeability and induce juice loss (Cheah et al., 1995).

Postmortem changes include a decrease of the antioxidant defense system and an increase in the degree of lipid and protein oxidation. Glutathione peroxidase activity would be elevated if it was maintained postmortem. Therefore, we might expect a stabilizing effect of dietary Se supplementation during meat storage. Indeed, supplementing broiler diets with 0.25 mg/kg of Se substantially increased GSH-Px activity in breast (2.1-fold) and leg (4.1-fold) muscle and as a result decreased lipid peroxidation was detected (2.5-fold in breast muscle and 3.3-fold in leg muscles) after 4 d of storage at 4°C compared with the control group (Devore et al., 1983). It seems likely that a stabilizing effect of Se is associated with maintaining muscle membrane integrity. Edens (1996) reported that drip loss was decreased when organic Se was fed to broilers. Using a mode system based on red blood cell membrane stability, Edens (2001) confirmed a membrane-stabilizing effect of organic Se. This study indicated that the Se yeast and Met supplementation of the hen diets could decrease drip loss. It might be due to the fact that Se content in the meat could elevate and maintain the GSH-Px activity in meat and muscle membrane integrity.

Physical qualities and antioxidant capacity of the Breeding Eggs

The antioxidant system of chicken embryo is based on natural antioxidants (e.g., vitamin E, carotenoids, GSH; Surai et al., 1996, 2001a, b; Surai, 1999) and antioxidant enzymes [e.g., GSH-Px and catalase as well as antioxidant enzyme cofactors (Se, Zn, Mn, and Fe; Surai et al., 1999). Of these, vitamin E, carotenoids, and metals, including Se, are obtained from the maternal diet. Increasing antioxidant supplementation of the maternal diet can substantially increase their concentrations in developing chick tissues and significantly decrease their susceptibility to lipid peroxidation (Surai and Speake, 1998; Surai et al., 1999). Selenium is an integral part of GSH-Px and GSH-Px plays an important role in antioxidant defense in poultry (Surai and Dvorska, 2002a,b). The Se concentration in eggs depends on both Se content and form of dietary Se used in the maternal diet. Payne et al. (2005) reported that a Se-enriched yeast diet was more effective for increasing the egg Se content than a sodium selenite diet. Wakebe (1998) found that adding 0.3 mg of Se/kg from Se-Met to layers increased GSH-Px activity in both the yolk and the white of eggs.

Methionine, an essential dietary amino acid, is used to synthesize proteins and other amino acids. Moreover, Met plays a particularly important role in providing Cys for GSH synthesis (Beatty and Reed, 1980). These common S-containing amino acids and GSH are antioxidants (Mosharov et al., 2000).

Four hundred fifty 52-wk-old Langshan layer hens (dual-purpose type, an indigenous poultry breed of China) were randomly divided into 9 treatments with 5 replicates in each treatment. Birds were fed corn-soybean diets (0.13 mg of Se/kg) supplemented with 0, 0.30, and 0.60 mg/kg of Se from Se yeast and 3.2, 4.0, and 5.4 g of dl-Met/kg, respectively. Se Concentration, GSH-Px Activity, and GSH Concentration, Lipid Oxidation, Protein Carbonyl were examined to evaluate the effect of Se yeast and Met supplementation of the maternal diet on antioxidant activity of the breeding eggs.

4. Experiment parameters measured

4.1 Se concentration

As can be seen from Table 4, the inclusion of Se yeast in the diet significantly increased the Se concentration in the yolk ($P < 0.01$). Supplementation of Met at 4.0 and 5.4 g /kg in the diets significantly decreased the Se concentration in the egg yolk compared with 3.2 g of Met/kg ($P < 0.01$). Moreover, a combination of the 0.6 mg of Se/kg and 3.2 g of Met/kg treatments had the maximal Se concentration and adding 3.2 g of Met/kg alone had the minimal Se concentration.

Item	Met (g/kg)	Se (μg/g)	GSH-Px (U/g)	GSH (mg/g)	MDA (μg/g)
Se (mg/kg)					
0	3.2	$0.48 \pm 0.03^{g,E}$	$29.40 \pm 2.85^{b,B}$	$1.13 \pm 0.14^{de,BC}$	$6.34 \pm 0.79^{a,A}$
	4.0	$0.63 \pm 0.03^{ef,D}$	$17.63 \pm 3.47^{d,DC}$	$1.29 \pm 0.04^{ab,A}$	$3.71 \pm 0.65^{cd,DE}$
	5.4	$0.62 \pm 0.03^{f,D}$	$19.82 \pm 2.09^{d,C}$	$1.18 \pm 0.07^{bdce,ABC}$	$4.15 \pm 0.43^{c,CD}$
0.3	3.2	$0.84 \pm 0.03^{b,B}$	$36.42 \pm 3.34^{a,A}$	$1.23 \pm 0.08^{abcd,ABC}$	$5.07 \pm 0.59^{b,B}$
	4.0	$0.66 \pm 0.03^{d,D}$	$13.82 \pm 1.92^{e,DE}$	$1.30 \pm 0.09^{a,A}$	$4.18 \pm 0.56^{c,DC}$
	5.4	$0.66 \pm 0.02^{de,D}$	$25.60 \pm 3.31^{c,B}$	$1.11 \pm 0.14^{e,C}$	$3.14 \pm 1.13^{d,E}$
0.6	3.2	$0.95 \pm 0.04^{a,A}$	$17.88 \pm 3.84^{d,DC}$	$1.27 \pm 0.14^{abc,AB}$	$5.12 \pm 0.51^{b,B}$
	4.0	$0.85 \pm 0.03^{b,B}$	$26.91 \pm 3.96^{bc,B}$	$1.16 \pm 0.12^{cde,ABC}$	$4.87 \pm 0.35^{b,BC}$
	5.4	$0.80 \pm 0.03^{c,C}$	$12.68 \pm 1.42^{e,E}$	$1.12 \pm 0.04^{e,BC}$	$3.91 \pm 0.28^{c,DE}$
0		$0.57 \pm 0.07^{c,C}$	$22.28 \pm 5.89^{b,B}$	1.20 ± 0.11	$4.74 \pm 1.32^{a,A}$
0.3		$0.72 \pm 0.09^{b,B}$	$25.28 \pm 9.84^{a,A}$	1.21 ± 0.13	$4.13 \pm 1.11^{b,B}$
0.6		$0.87 \pm 0.07^{a,A}$	$19.15 \pm 6.78^{c,C}$	1.18 ± 0.12	$4.63 \pm 0.65^{a,A}$
	3.2	$0.80 \pm 0.21^{a,A}$	$27.90 \pm 8.44^{a,A}$	$1.21 \pm 0.13^{a,AB}$	$5.51 \pm 0.86^{a,A}$
	4.0	$0.71 \pm 0.11^{b,B}$	$19.45 \pm 6.41^{b,B}$	$1.25 \pm 0.11^{a,A}$	$4.25 \pm 0.70^{b,B}$
	5.4	$0.69 \pm 0.08^{b,B}$	$19.37 \pm 5.86^{b,B}$	$1.14 \pm 0.09^{b,B}$	$3.74 \pm 0.81^{c,C}$
P-value					
Se		0.0001	0.0001	0.5464	0.0034
Met		0.0001	0.0001	0.0012	0.0001
Se × Met		0.0001	0.0001	0.0025	0.0001

$^{a-g}$Means in a column with different superscripts are significantly different ($P < 0.05$).
$^{A-E}$Means in a column with different superscripts are significantly different ($P < 0.01$).

Table 4. Effects of Se yeast and Met supplementation of the maternal diets on contents of Se, glutathione (GSH), and malondialdehyde (MDA) and glutathione peroxidase (GSH-Px) activity in the yolk of eggs

4.2 GSH concentration

The Met supplementation of the diets significantly influenced the GSH concentration in both the yolk and the albumen of eggs ($P < 0.01$; Table 4 and 5). The 5.4 g of Met/kg treatment decreased the GSH concentration in the egg yolk compared with 3.2 and 5.4 g of Met/kg, whereas the 5.4 g of Met/kg treatment significantly increased the GSH concentration in the

egg albumen compared with 3.2 and 4.0 g of Met/kg. Moreover, there was significant effect of the interactions between Se and Met on the GSH concentration in the egg yolk. However, increasing Se supplementation in the maternal diet did not significantly influence the GSH concentration in both the yolk and the albumen of eggs.

Se (mg/kg)	Met (g/kg)	GSH-Px (U/g)	GSH (mg/g)	Carbonyl group (nm/mg)
0	3.2	$292.00 \pm 20.51^{a,A}$	$2.56 \pm 0.12^{c,C}$	$5.01 \pm 0.39^{a,A}$
	4.0	$168.91 \pm 12.01^{de,CD}$	$2.72 \pm 0.16^{bc,BC}$	$4.64 \pm 0.48^{abcd,AB}$
	5.4	$151.78 \pm 16.61^{e,D}$	$2.97 \pm 0.30^{ab,AB}$	$4.37 \pm 0.50^{bcd,AB}$
0.3	3.2	$295.66 \pm 31.70^{a,A}$	$2.75 \pm 0.23^{bc,BC}$	$4.87 \pm 0.49^{abe,AB}$
	4.0	$178.57 \pm 21.46^{d,CD}$	$2.72 \pm 0.18^{bc,BC}$	$4.26 \pm 0.45^{cd,B}$
	5.4	$228.23 \pm 23.73^{c,B}$	$3.20 \pm 0.21^{a,A}$	$4.53 \pm 0.45^{abcd,AB}$
0.6	3.2	$252.29 \pm 17.97^{b,B}$	$2.77 \pm 0.27^{bc,BC}$	$4.74 \pm 0.52^{abc,AB}$
	4.0	$296.93 \pm 14.58^{a,A}$	$2.91 \pm 0.38^{ab,AB}$	$4.16 \pm 0.32^{d,B}$
	5.4	$187.72 \pm 13.05^{d,C}$	$2.96 \pm 0.15^{ab,AB}$	$4.39 \pm 0.63^{bcd,AB}$
0		$204.23 \pm 65.77^{c,B}$	2.75 ± 0.27	4.67 ± 0.52
0.3		$234.15 \pm 54.95^{b,A}$	2.89 ± 0.30	4.55 ± 0.51
0.6		$245.66 \pm 48.06^{a,A}$	2.88 ± 0.28	4.43 ± 0.54
	3.2	$279.98 \pm 30.57^{a,A}$	$2.69 \pm 0.23^{b,B}$	$4.87 \pm 0.46^{a,A}$
	4.0	$214.81 \pm 61.51^{b,B}$	$2.78 \pm 0.27^{b,B}$	$4.35 \pm 0.46^{b,B}$
	5.4	$189.26 \pm 39.40^{c,C}$	$3.05 \pm 0.25^{a,A}$	$4.43 \pm 0.51^{b,B}$
P-value				
Se		0.0001	0.0870	0.2159
Met		0.0001	0.0001	0.0006
Se × Met		0.0001	0.1262	0.5271

[a–e]Means in a column with different superscripts are significantly different ($P < 0.05$).

[A–D]Means in a column with different superscripts are significantly different ($P < 0.01$).

Table 5. Effects of Se yeast and Met supplementation of the maternal diets on the contents of glutathione (GSH) and carbonyl group and glutathione peroxidase (GSH-Px) activity in the albumen of eggs

4.3 GSH-Px activity

As can be seen from Tables 4 and 5, the inclusion of Se yeast and Met in the diets significantly increased or decreased the GSH-Px activity in eggs ($P < 0.01$). The inclusion of Se yeast in the maternal diets increased GSH-Px activity in both the yolk and the albumen of eggs. However, adding 0.6 mg of Se/kg decreased the GSH-Px activity in the egg yolk compared with 0 and 0.3 mg of Se/kg. The GSH-Px activity in the albumen of eggs significantly decreased with increasing Met supplementation, whereas the albumen GSH-Px activity of the 3.2 g of Met/kg treatment was significantly higher than those of the 4.0 and 5.4 g of Met/kg treatments. In the egg yolk, the inclusion of the 0.3 mg of Se/kg and 3.2 g of Met/kg treatments had the maximal GSH-Px activity, whereas a combination of the 0.6 mg of Se/kg and 5.4 g of Met/kg treatments had the minimal GSH-Px activity. In the egg albumen, a combination of the 0.3 mg of Se/kg and 3.2 g of Met/kg treatments had the maximal GSH-Px activity and adding 5.4 g of Met/kg alone had the minimal GSH-Px activity.

4.4 Lipid oxidation

The influence of Se yeast, Met, and their interactions on the MDA content in the egg yolk was remarkable ($P < 0.01$; Table 2). The MDA content in the egg yolk significantly decreased with Met supplementation ($P < 0.01$). However, adding 0.3 mg of Se/kg to the diets significantly decreased the MDA content in the egg yolk compared with the 0 and 0.6 mg of Se/kg treatments. In the egg yolk, a combination of 0.3 mg of Se/kg and 5.4 g of Met/kg had the minimal MDA content and adding 3.2 g of Met/kg alone to the diet had the maximal MDA content.

4.5 Protein oxidation

Table 5 showed that Met supplemented at 4.0 and 5.4 g/kg (irrespective of Se supplementation) decreased carbonyl content in the egg albumen compared with the 3.2 g of Met/kg treatment. Adding 3.2 g of Met/kg alone to the diet created the maximal carbonyl content in the egg albumen, whereas a combination of the 0.6 mg of Se/kg and 4.0 g of Met/kg treatments created the minimal carbonyl content.

In summary, the results can be concluded as:

1. Supplementing 0.3 mg/kg **Se** increased egg albumen weight, but decreased egg yolk weight. However, supplementing 5.4 g of DL-**Met** increased egg albumen weigh, decreased egg yolk weight. There was no significant interaction between **Se** and **Met** on egg albumen weight, egg yolk weight, egg shape index and haugh unit ($P>0.05$), while they significantly influenced egg yolk weight, egg hell weight and egg albumen height ($P<0.05$).
2. Increasing Se yeast supplementation significantly increased **Se** concentration in the egg yolk (P<0.01) and the **Se** concentration of the 3.2 g of **Met**/kg treatment was higher than those of the 4.0 and 5.4 g of **Met**/kg treatments. Adding 0.3 mg of **Se**/kg to the diet significantly increased **GSH-Px** activity in the egg yolk compared with 0 and 0.6 mg of **Se**/kg (P<0.01) and increasing **Se** yeast supplementation significantly increased the **GSH-Px** activity in the egg albumen (P<0.01). Increasing **Met** supplementation significantly decreased the **GSH-Px** activity in the egg albumen and yolk (P<0.01). Methionine supplemented at 3.2 and 4.0 g/kg significantly increased glutathione concentration in the egg yolk compared with 5.4 g of **Met**/kg (P<0.01) and increasing **Met** supplementation increased the glutathione concentration in the egg albumen. Increasing **Met** supplementation significantly decreased **MDA** concentration in the egg yolk (P<0.01) and **Se** supplemented at 0 and 0.6 mg/kg increased the malondialdehyde concentrations in the egg yolk compared with 0.3 mg of **Se**/kg (P<0.01). Methionine supplemented at 4.0 and 5.4 g/kg significantly decreased carbonyl concentration compared with 3.2 g of Met/kg.

Selenium concentration in the egg yolk depends on both the Se content and form in the maternal diet. Several authors (Cantor, 1997; Paton et al., 2000; Surai, 2000) reported that organic Se (e.g., Se-Met) is more efficiently deposited in the egg yolk. Davis and Fear (1996) found that both sodium selenite and Se-Met fed at 2 mg/kg increased the Se concentration in the egg yolk compared with a diet that was not supplemented. Paton et al. (2002) found that increasing Se levels (irrespective of source) produced a significant linear increase in egg Se concentration and the linear responses were different for the different Se sources in all egg components. The Se used in this experiment is the Se-enriched yeast that is produced by growing the yeast *Saccharomyces cerevisiae* in a high-Se medium (AAFCO, 2003). Beilstein and Whanger (1986) and kelly and Power (1995) reported that the majority of the Se in Se yeast is Se-Met, a Se analog of Met. Selenomethionine can be an excellent analog for Met in biochemical reactions because of the similar covalent radii of Se and S. The result of this current study showed that increasing Se supplementation significantly increased Se deposition in the egg yolk. However, adding 4.0 and 5.4 g of Met/kg to the maternal diet significantly decreased the Se concentration in the egg yolk compared with 3.2 g of Met/kg. It is possible that the chemical similarity between Se-Met and Met allows the body to use them interchangeably in protein synthesis (Surai, 2002), which makes it possible to build Se reserves in the other parts of eggs and decreases Se concentration in the egg yolk during storage.

Met supplementation of the diet significantly influenced the GSH concentration in both the yolk and the albumen of eggs. It might be due to the fact that Met provided plenty of Cys during metabolism for GSH synthesis when the hens were fed an excess of Met (Beatty and Reed, 1980). However, increasing dietary Se yeast supplementation did not significantly influence the GSH concentration. A possible reason is that when an excess of Se yeast is supplemented in the diet in the form of Se-Met, this amino acid will be nonspecifically incorporated into various proteins (Surai, 2002), which decreases GSH synthesis.

Selenium is an essential component of a variety of selenoproteins, the best known of which is GSH-Px. The GSH-Px family of enzymes is a crucial player in the antioxidant system, neutralizing potential threats to the integrity of cellular macromolecules by eliminating hydrogen peroxide and detoxifying lipid hydroperoxides (Brigelius-Flohé, 1999). The Se derived from hen is deposited in eggs and is distributed among the developing tissues during embryogenesis (Gaal et al., 1995; Surai, 2000; Paton et al., 2002). Consequently, the Se supplementation of the maternal diet has an effect on the GSH-Px activity in the egg yolk. Wakebe (1998) found that adding 0.3 mg of Se/kg to the diet increased GSH-Px activity in both the yolk and the albumen of eggs. Surai (2000) reported that the GSH-Px activity in the liver of newly hatched chicks significantly decreased when the hens were fed a low level of Se diet. This current study also showed that the inclusion of Se yeast in the maternal diet increased GSH-Px activity in both the yolk and the albumen of eggs. However, adding 0.6 mg of Se/kg to the diet decreased the GSH-Px activity in the egg yolk compared with the 0 and 0.3 mg of Se/kg treatments. The mechanism of this observation is not known. A possible reason is that high dietary Se-Met is preferentially incorporated into proteins rather than used for GSH-Px when Met is limiting in the maternal diet (Waschulewski and Sunde, 1988).

Methionine participates in methyl group metabolism and the synthesis of other S amino acids, notably Cys. Cysteine is required for the synthesis of GSH and taurine, which are important compounds for host defense against oxidative stress (Métayer et al., 2008). This current study showed that increasing Met supplementation in the maternal diet decreased the GSH-Px activity in both the yolk and the albumen of the eggs. It is possible that adding an excess of Met to the diets leads to preferential incorporation of Se-Met into the body (e.g., eggs) in a form other than as GSH-Px (Zhao et al., 2009). Thus, the utilization of Se-Met supplementation for GSH-Px synthesis substantially decreases when the hens are fed an excess of Met. This study also indicated that a combination of the 0.3 mg of Se/kg and 3.2 g of Met/kg treatments had the maximal GSH-Px activity in the egg yolk and the second largest GSH-Px activity (slightly lower than that of the 0.6 mg of Se/kg and 4.0 g of Met/kg treatments) in the eggs, which probably means that inclusion of 0.3 mg of Se/kg and 3.2 g of Met/kg in the diet provides enough Se to the egg for the requirement for maximum Se-GSH-Px activity. It is possible that an appropriate amount of Se in the diet achieves the GSH-Px activity through Se-GSH-Px gene expression and cytosolic mRNA stabilization (Chris-tensen and Burgener, 1992) or regulates the level of GSH-Px mRNA in a posttranscriptional step (Toyoda et al., 1990).

Egg quality decreases during storage. This process is associated with biochemical changes, including lipid and protein oxidation. Lipid oxidation causes loss of nutrition. One such product is MDA, which has long been considered as an index of oxidative rancidity (Cortinas et al., 2005). Increasing Se concentration in combination with other antioxidants (vitamin E and carotenoids) could be an effective means to prevent the damaging effect of

free radicals produced in eggs (Surai, 2002). This study demonstrated that adding 0.3 mg of Se/kg to the diet significantly decreased the MDA content in the egg yolk compared with 0 and 0.6 mg of Se/kg treatments. This can be explained as a result of increased GSH concentration and GSH-Px activity and changes in lipid composition (Noble and Cocchi, 1990).

However, the MDA contents of adding 0.6 mg of Se/kg to the diet were higher than that of the 0.3 mg of Se/kg treatment. The mechanism of this observation is not known. This study also showed that Met supplementation of the diet significantly decreased the MDA content in the egg yolk. This might be due to the fact that Met provides plenty of Cys for GSH synthesis when the hens are fed an excess of Met (Beatty and Reed, 1980), which inhibits lipid oxidation and decreases MDA content in eggs.

Eggs can be divided into 3 components: albumen, yolk, and shell. Albumen, which contains approximately 67% of the protein content of the egg (Romanoff and Romanoff, 1949), provides more proteins for assimilation into tissue during embryonic development (Finkler et al., 1998). Egg storage before incubation can be associated with protein oxidation within the egg albumen. Carbonyl contents can be considered as a marker for protein oxidation because amino acid residues of proteins, such as Lys, Met, and Cys, can be oxidized to carbonyl derivatives by oxidative stresses (Butterfield et al., 1998). Therefore, protein oxidation affects the physicochemical and functional properties of egg white. This current study indicated that adding Se and Met to the hens' diets could decrease carbonyl content in the egg albumen. Adding 3.2 g of Met/kg alone to the hens' diets created the maximal carbonyl content in the egg albumen, probably due to the fact that the eggs from hens fed a low-Se diet had low GSH content in the egg albumen. However, the inclusion of 0.6 mg of Se/kg and 4.0 g of Met/kg in the hens' diets created the minimal carbonyl content in the egg albumen. This can be explained as a result of the higher GSH content and highest GSH-Px activity (Table 3), thus slowing the rate of protein oxidation (Pappas et al., 2005).

To sum up, a conclusion was drawn that **Se** yeast and **Met** supplementation of the maternal diets could improve egg quality, meat quality, meat protein functionalities and the oxidative stability of meat proteins and lipid to varying degrees.

5. References

AAFCO. 2003. Page 285 in Feed Ingredient Definitions. 57. Mineral Products. Tentative T57:163 Official Publication. Association of American Feed Control Officials Inc., Olympia, WA.

Angelbeck, J.H., Du Bru, E.F. 1983. The effect of neonatal testosterone on specific male and female patterns of phosphorylated cytosolic proteins in the rat preoptichypothalamus, cortex and amygdala. Brain Res 264(2):277–83.

Baker, D.H. 2006. Comparative species utilization and toxicity of sulfur amino acids. J Nutr 136(6):1670S–5S.

Baron, C. P., and J. H. Andersen. 2002. Myoglobin-induced lipid oxidation. A review. J. Agric. Food Chem. 50:3887–3897.

Beatty, P. W., and D. J. Reed. 1980. Involvement of the cystathionine pathway in the biosynthesis of glutathione by isolated rat hepatocytes. Arch. Biochem. Biophys. 204:80–87.

Beilstein, M. A., and P. D. Whanger. 1986. Deposition of dietary organic and inorganic selenium in rat erythrocyte proteins. J. Nutr. 116:1701–1710.

Beilstein, M. A., and P. D. Whanger. 1986. Deposition of dietary organic and inorganic selenium in rat erythrocyte proteins. J. Nutr. 116:1701–1710.

Brigelius-Flohé, R. 1999. Tissue-specific functions of individual glutathione peroxidases. Free Radic. Biol. Med. 27:951–965.

Butterfield, D. A., T. Koppal, B. Howard, R. Subramaniam, N. Hall, K. Hensley, S. Yatin, K. Allen, M. Aksenov, M. Aksenova, and J. Carney. 1998. Structural and functional changes in proteins induced by free radical-mediated oxidative stress and protective action of the antioxidants N-tert-butyl-α-phe-nylnitrone and vitamin E. Ann. N. Y. Acad. Sci. 854:448–462.

Cantor, A. H. 1997. The role of selenium in poultry nutrition. Pages 155–164 in Biotechnology in the Feed Industry. T. P. Lyons and K. A. Jacques, ed. Proc. Alltech 13th Annu. Symp. Nottingham University Press, UK.

Cheah, K. S., A. M. Chreah, and D. J. Krausgrill. 1995. Effect of dietary supplementary vitamin E on pig meat quality. Meat Sci.39:255–265.

Christensen, M. J., and K. W. Burgener. 1992. Dietary selenium stabilises glutathione peroxidase mRNA in rat liver. J. Nutr. 122:1620–1626.

Cortinas, L., A. Barroeta, C. Villaverde, J. Galobart, F. Guardiola, and M. D. Baucells. 2005. Influence of the dietary polyunsaturation level on chicken meat quality: Lipid oxidation. Poult. Sci. 84:48–55.

Daun C, Akesson B. 2004. Comparison of glutathione peroxidase activity, and of total and soluble selenium content in two muscles from chicken, turkey, duck, ostrich and lamb. Food Chem 85:295–303.

Davis, R. H., and J. Fear. 1996. Incorporation of selenium into egg proteins from dietary selenite. Br. Poult. Sci. 37:197–211.

Decker, E. A., Y. L. Xiong, J. T. Calvert, A. D. Crum, and S. P. Blanchart. 1993. Chemical, physical and functional properties of oxidized turkey white muscle myofibrillar proteins. J. Agric.

Food Chem. 41:186–189.

Desai M, Crowther, N.J., Ozanne, S.E., Lucas A, Hales, C.N. 1995. Adult glucose and lipid metabolism may be programmed during fetal life. Biochem Soc Trans 23(2):331–5.

Devore, V. R., G. R. Colnago, L. S. Jensen, and B. E. Greene. 1983. Thiobarbituric acid values and glutathione peroxidase activity in meat from chickens fed a selenium-supplemented diet. J. Food Sci. 48:300–301.

Edens, F. W. 1996. Organic selenium: From feathers to muscle integrity to drip loss. Five years onward: No more selenite. Pages 165–185 in Biotechnology in the Feed industry. Proceedings of Alltech's 12th Annual Symposium. T. P. Lyons and K. A.Jacques, ed. Nottingham University Press, Nottingham, UK.

Edens, F. W. 2001. Involvement of Sel-Plex in physiological stability and performance of broiler chickens. Pages 349–376 in Biotechnology in the Feed Industry. Proceedings of Alltech's 17th Annual Symposium. T. P. Lyons and T. A. Jacques, ed. Nottingham University Press, Nottingham, UK.

Elisabeth, H. L., and M. L. Steven. 2005. Mechanisms of waterholding capacity of meat: The role of postmortem biochemical and structural changes. Meat Sci. 71:194–204.

Esaki N, Nakamura T, Tanaka H, Soda K. 1982. Selenocysteine lyase, a novel enzyme that specifically acts on selenocysteine. Mammalian distribution and purification and properties of pig liver enzyme. J Biol Chem 257:4386–91.

Faustman, C., and R. G. Cassens. 1990. The biochemical basis for discoloration in fresh meat: A review. J. Muscle Foods 1:217–243.

Finkler, M. S., J. B. Van Orman, and P. R. Sotherland. 1998. Experimental manipulation of egg quality in chickens: Influence of albumen and yolk on the size and body composition of near-term embryos in a precocial bird. J. Comp. Physiol. B 168:17–24.

Gaal, T., M. Mezes, R. C. Noble, J. Dixon, and B. K. Speake. 1995. Development of antioxidant capacity in tissues of the chick embryo. Comp. Biochem. Physiol. B 112:711–716.

Hoffman, J.L., McConnell, K.P., Carpenter, D.R. 1970. Aminoacylation of Escherichia coli methionine tRNA by selenomethionine. Biochim Biophys Acta 199:531–4.

Kelly, M. P., and R. F. Power. 1995. Fractionation and identification of the major selenium containing compounds in selenized yeast. J. Dairy Sci. 78(Suppl. 1):237. (Abstr.)

Lucas A, Morley R, Cole, T.J., Gore, S.M., Lucas, P.J., Crowle P, Pearse R, Boon, A.J., Powell R. 1990. Early diet in preterm babies and developmental status at 18 months. Lancet 335(8704):1477–81.

Lucas A, Baker, B.A., Desai M, Hales, C.N. 1996. Nutrition in pregnant or lactating rats programs lipid metabolism in the offspring. Br J Nutr 76(4):605–12.

Mancini, R. A., and M. C. Hunt. 2005. Current research in meat color. Meat Sci. 71:100–121.

McConnell, K.P., Cho, G.J. 1965. Transmucosal movement of selenium. Am J Physiol 208:1191–5.

McConnell, K.P., Hoffman, J.L. 1972. Methionine-selenomethionine parallels in rat liver polypeptide chain synthesis. FEBS Lett 24:60–2.

Mercier Y, Gattellier P, Viau M, Remingnon H, Renerre M. 1998. Effect of dietary fat and vitamin E on lipid and protein oxidation in turkey meat during storage. Meat Sci 48:301–17.

Mercier, Y., P. Gatellier, and M. Renerre. 2004. Lipid and protein oxidation in vitro, and antioxidant potential in meat from Charolais cows finished on pasture or mixed diet. Meat Sci.66:467–473.

Mercier, Y., P. Gatellier, M. Viau, H. Remignon, and M. Renerre.1998. Effect of dietary fat and vitamin E on color stability and on lipid and protein oxidation in turkey meat during storage. Meat Sci. 48:301–318.

Métayer, S., I. Seiliez, A. Collin, S. Duchêne, Y. Mercier, P. A. Geraert, and S. Tesseraud. 2008. Mechanisms through which sulfur amino acids control protein metabolism and oxidative status. J. Nutr. Biochem. 19:207–215.

Mosharov, E., M. R. Cranford, and R. Banerjee. 2000. The quantitatively important relationship between homocysteine metabolism and glutathione synthesis by the transsulfuration pathway and its regulation by redox changes. Biochemistry 39:13005–13012.

Noble, R. C., and M. Cocchi. 1990. Lipid metabolism in the neonatal chicken. Prog. Lipid Res. 29:107–140.

Oliver, C. N., B. W. Alin, E. J. Moerman, S. Goldstein, and E. R. Stadtman. 1987. Age-related changes in oxidized proteins. J. Biol. Chem. 262:5488–5491.

Pappas, A. C., T. Acamovic, N. H. C. Sparks, P. F. Surai, and R. M. McDevitt. 2005. Effects of supplementing broiler breeder diets with organic selenium and polyunsaturated fatty acids on egg quality during storage. Poult. Sci. 84:865–874.

Pappas, A. C., F. Karadas, P. F. Surai, and B. K. Speake. 2005. The selenium intake of the female chicken influences the selenium status of her progeny. Comp. Biochem. Physiol. B Biochem. Mol. Biol. 142:465–474.

Paton, N. D., A. H. Cantor, A. J. Pescatore, M. J. Ford, and C. A. Smith. 2000. Effect of dietary selenium source and level of inclusion on selenium content of incubated eggs. Poult. Sci. 79(Suppl.1):40. (Abstr.)

Paton, N. D., A. H. Cantor, A. J. Pescatore, M. J. Ford, and C. A. Smith. 2002. The effect of dietary selenium source and level on the uptake of selenium by developing chick embryos. Poult. Sci. 81:1548–1554.

Payne, R. L., T. K. Lavergne, and L. L. Southern. 2005. Effect of inorganic versus organic selenium on hen production and selenium concentration. Poult. Sci. 84:232–237.

Racanicci, A.M.C, Danielsen B, Menten, J.F.M, Reginato-d'Arce, M.A.B., Skibsted, L.H. 2004. Antioxidant effect of dittany (Origanum dictamnus) in pre-cooked meat balls during chill-storage in comparation to rosemary (Rosmarinus officinalis). Eur Food Res Technol 218:521–4.

Romanoff, A. L., and A. J. Romanoff. 1949. The Avian Egg. John Wiley and Sons, New York, NY.

Ryu, Y. C., M. S. Rhee, K. M. Lee, and B. C. Kim. 1995. Effects of different levels of dietary supplemental selenium on performance, lipid oxidation, and colour stability of broiler chicks. Poult. Sci.84:809–815.

Snoeck A, Remacle C, Reusens B, Hoet, J.J. 1990. Effect of a low-protein diet during pregnancy on the fetal rat endocrine pancreas. Biol Neonate 57(2):107–18.

Steele, R.D., Benevenga, N.J. 1978. Identification of 3-methylthiopropionic acid as an intermediate in mammalian methionine metabolism. J Biol Chem 253:7844–50.

Sunde, R.A. 1984. The biochemistry of selenoproteins. J AmOil Chem Soc 61:1891–900.

Sunde, R.A., Evenson, J.K. 1987. Serine incorporation into the selenocysteine moiety of glutathione peroxidase. J Biol Chem 262:933–7.

Sunde, R.A., Gutzke, G.E., Hoekstra,W.G. 1981. Effect of dietarymethionine on the biopotency of selenite and selenomethionine in the rat. J Nutr 111:76–86.

Surai, P. F. 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.

Surai, P. F., and N. C. Sparks. 2000. Tissue-specific fatty acid and α-tocopherol profiles in male chickens depending on dietary tuna oil and vitamin E provision. Poult. Sci. 79:1132–1142.

Surai, P. F., and J. E. Dvorska. 2002a. Effect of selenium and vitamin E on lipid peroxidation in thigh muscle tissue of broiler breeder hens during storage. Arch. Geflugelkd. 66:120.

Surai, P. F., and J. E. Dvorska. 2002b.Effect of selenium and vitamin E content of the breeder's diet on lipid peroxidation in breast muscles during storage. Proceedings of Australian Poultry Science Symposium, Sydney. 14:187–192.

Surai, P. F. 1999. Tissue-specific changes in the activities of antioxidant enzymes during the development of the chicken embryo. Br. Poult. Sci. 40:397–405.

Surai, P. F. 2002. Selenium in poultry nutrition: 2.Reproduction, egg and meat quality and practical applications. World's Poult. Sci. J. 58:431–450.

Surai, P. F., R. C. Noble, and B. K. Speake. 1996. Tissue specific differences in antioxidant distribution and susceptibility to lipid peroxidation during development of the chick embryo. Biochem. Biophys. Acta 1304:1–10.

Surai, P. F., and B. K. Speake. 1998. Distribution of carotenoids from the yolk to the tissues of the chick embryo. J. Nutr. Biochem.9:645–651.

Surai, P. F., B. K. Speake, and N. C. Spark. 2001a. Carotenoids in avian nutrition and embryonic development: 1. Absorption, availability and levels in plasma and egg yolk. Poult. Sci. 38:1–27.

Surai, P. F., B. K. Speake, and N. C. Spark. 2001b. Carotenoids in avian nutrition and embryonic development: 2. Antioxidant properties and discrimination in embryonic tissues. Poult. Sci.38:117–145.

Toyoda, H., S. Himeno, and N. Imura. 1990. Regulation of glutathione peroxidase mRNA level by dietary selenium manipulation. Biochim. Biophys. Acta 1049:213–215.

Ventanas, S., M. Estevez, J. F. Tejeda, and J. Ruiz. 2006. Protein and lipid oxidation in longissimus dorsi and dry-cured loin from Iberian pigs as affected by crossbreeding and diet. Meat Sci. 72:647–655.

Wakebe, M. 1998. Organic selenium and egg freshness. Patent 10-23864. Feed for meat chickens and feed for laying hens. Japanese Patent Office. Application Heisei 8-179629.

Waschulewski, I.H., Sunde, R.A. 1988a. Effect of dietary methionine on the tissue selenium and glutathione peroxidase (EC 1.11.1.9) activity in rats given selenomethionine. Br J Nutr 60:57–68.

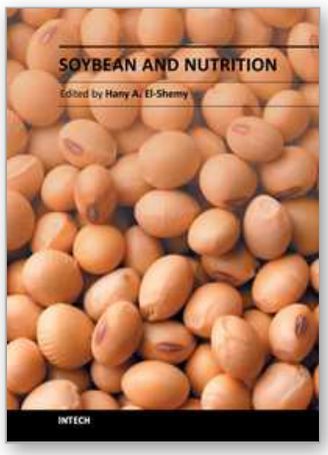
Waschulewski, I.H., Sunde, R.A. 1988b. Effect of dietary methionine on utilization of tissue selenium from dietary selenomethionine for glutathione peroxidase in the rat. J Nutr 118:367–74.

Wilson, J.X., Lui, E.M.K., Del RF. 1992. Developmental profiles of antioxidants and trace elements in chick embryo.Mech Ageing Dev 65:51–64.

Zhao, L. Y., S. Q. Xu, R. Q. Zhao, Z. Q. Peng, and X. J. Pan. 2009. Effects of selenium and methionine supplementation of breeder hen diets on selenium concentration and oxidative stability of lipids in the thigh muscles of progeny. J. Food Sci. 74:569–574.

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