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Dietary selenium regulation of transcript abundance of selenoprotein N and selenoprotein W in chicken muscle tissues

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Abstract Selenium (Se), selenoprotein N (SelN) and selenoprotein W (SelW) play a crucial role in muscle disorders. Se status highly regulates selenoprotein mRNA levels. However, few attempts have been performed on the effect of dietary Se supplementation on muscle SelN and SelW mRNA levels in birds. To investigate the effects of Se on the regulation of SelN and SelW mRNA levels in muscle tissues, one-day-old male chickens were fed either a commercial diet or a Se-supplemented diet containing 1.0, 2.0, 3.0 or 5.0 mg/kg sodium selenite for 90 days. Muscle tissues (breast, flight, thigh, shank and cardiac muscles) were collected and examined for Se content and mRNA levels of SelN and SelW. Moreover, Selenophosphate synthetase-1 (SPS-1) and selenocysteine-synthase (SecS) mRNA levels were analyzed. Significant increases in SelN mRNA levels were obtained in breast, thigh and shank muscles treated with Se, with maximal effects at 3.0 mg Se/kg diet, but 2.0 mg Se/kg diet resulted in peak levels of Sel N mRNA in flight muscles. Changes in SelW mRNA abundance in thigh and shank muscles increased in response to Se supply. After reaching a maximal level, higher Se supplementation led to a reduction in both SelN and SelW mRNAs. However, SelN and SelW mRNA levels displayed a different expression pattern in different skeletal and cardiac muscles. Thus, it suggested that skeletal and cardiac muscles SelN and SelW mRNA levels were highly regulated by Se supplementation and different muscle tissues showed differential sensitivity. Moreover, Se supplementation also regulated the levels of SPS1 and SecS mRNAs. The mRNA levels of SPS1 and SecS were enhanced in the Se supplemented groups. These data indicate that Se regulates the expression of SelN and SelW gene and affect the mRNA levels of SecS and SPS1.

Keywords Chicken · Selenium Selenoprotein N · Selenoprotein W · Selenophosphate synthetase-1 · Selenocysteine synthase · mRNA expression · Muscle tissues

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Introduction

Selenium (Se) is an essential nutritional trace element. Deficiencies due to its dietary restriction or misabsorption have been implicated in a number of disorders in livestock and man. The beneficial role of Se has been known for several decades. Se is incorporated into selenoproteins, which have been

shown to be mandatory for most beneficial properties of the trace element, aside from other less characterized low-molecular-weight Se compounds (Irons et al. 2006). The incorporation of Se as the amino acid selenocysteine (Sec) in 25 proteins by a highly elaborate cotranslational mechanism has defined the human selenoproteome (Kryukov et al. 2003), in which the precise function of about half of the proteins is still unknown (Reeves and Hoffmann 2009). Since Se exerts its biologic function largely through selenoproteins, it is important to identify and determine selenoproteins functions.

Selenoprotein N (SelN) was identified by Lescure et al. (1999). SelN is encoded by the SPEN1 gene and contains a Se atom in the form of a selenocysteine residue. SelN function remains unknown. This protein is the first selenoprotein shown to be responsible for a genetic disorder. Mutations in the SEPN1 gene result in a spectrum of early onset muscular disorders including rigid spine muscular dystrophy (RSMD1) (Allamand et al. 2006; Okamoto et al. 2006), the classical form of minicore disease (MmD) (Ferreiro et al. 2002), desmin-related myopathy with Mallory body-like inclusions (MB-DRM) (Ferreiro et al. 2004), and congenital fiber type disproportion (CFTD) (Clarke et al. 2006). These phenotypes are now referred to as SEPN1-related myopathy (SEPN1-RM1) (Tajsharghi et al. 2005; Schara et al. 2008; Maiti et al. 2008). These muscular diseases are characterized by early onset of hypotonia which predominantly affect axial muscles. For the moment, biochemical data have demonstrated that SelN is a 65-kDa transmembrane glycoprotein localized in the membrane of the endoplasmic reticulum (Petit et al. 2003). Expression level is much higher in fetal as opposed to adult tissues, with cellular studies showing that the high SEPN1 expression found in cultured myoblasts decreases progressively during the differentiation steps of myogenesis (Petit et al. 2003). Furthermore, SEPN1 zebrafish mutants obtained by morpholino injection, exhibited strong developmental defects such as abnormalities in muscles development and architecture (Deniziak et al. 2007; Jurynec et al. 2008). These data clearly pointed to a possible role for SelN during muscle development.

Selnoprotein W (SelW) is another selenoprotein linked to a muscular disease. In previous work, the chicken SelW cDNA has been sequenced and it was found that SelW is expressed ubiquitously in various tissues (Li et al. 2010). Like SelN, SelW is highly expressed in proliferating myoblasts, decreasing during differentiation, and accordingly, it was shown to protect proliferative myoblasts from oxidative stress during muscle growth and differentiation (Loflin et al. 2006). SelW is a protein highly expressed in muscles from Se-supplemented animals, but almost undetectable in those presenting WMD, a muscle disease characterized by alteration of both cardiac and skeletal muscle fibers (Whanger et al. 1993). The crucial role of Se and selenoproteins in animal and human health, in particular in muscle disorders, has been well established, but the underlying molecular mechanisms remain poorly understood.

SecS widely distributes in ancient bacteria and eukaryote tissues containing selenoproteins. Research showed that SecS participates the complex machinery of Sec synthesis (Hatfield et al. 2006). It transfers monoselenophosphate, the active form of Se, onto the seryl-tRNA$^{[Ser]Sec}$ to generate selenocysteyl-tRNA$^{[Ser]Sec}$ in prokaryotes and eukaryotes (Xu et al. 2007a, b). SPS1 is an additional protein possibly implicated in the pathway of Sec biosynthesis (Low et al. 1995). SPS-1 forms a complex with several proteins involved in Sec biosynthesis and has been reported to interact with SecS both in vitro and in vivo (Small-Howard et al. 2006).

However, the expression pattern of SelN, SelW, SPS1 and SecS mRNAs induced by Se supplementation in the avian muscle tissues remains unknown. Therefore, the present study focus on evaluating the regulation of dietary Se intake on SelN, SelW, SPS1 and SecS transcripts abundance in chicken muscle tissues.

Materials and methods

Animals and diets

Care and treatment of experimental animals was approved by the Institutional Animal Care and Use Committee of Northeast Agricultural University. Fifty male chickens (1-day-old; Weiwei Co. Ltd., Harbin, China) were randomly divided into five groups (10 chickens/group) and fed either the commercial granulated diet or the Se-supplemented granulated diet containing 1.0, 2.0, 3.0 or 5.0 mg of Se/kg of diet (as Na_2SeO_3) for 90 days. The basal commercial

granulated diet was shown by analysis to contain 0.145 mg of Se/kg. On the 90th day of the experiment, all of the chickens were fasted overnight. Following euthanasia with sodium pentobarbital, skeletal muscles (breast muscle, flight muscle, thigh muscle, shank muscle) and cardiac muscle were quickly removed, blotted and then rinsed with ice-cold sterile deionized water, frozen immediately in liquid nitrogen and stored at −80°C until required. Se content and SelN, SelW, SPS-1, and SecS mRNA expression levels were measured in these tissues.

Determination of Se concentration in tissues

Se content in the tissues was estimated by the method described by Hasunuma et al. (1982). The assay is based on the principle that Se contained in samples is converted to selenous acid in response to acid digestion. The reaction between selenous acid and aromatic-o-diamines, such as 2,3-diamino-naphthalene (DAN), leads to the formation of 4,5-benzopiazselenol, which displays a brilliant lime-green fluorescence when excited at 366 nm in cyclohexane. The fluorescence emission in extracted cyclohexane was measured by a fluorescence spectrophotometer with an excitation and emission wavelengths of 366 and 520 nm, respectively. The Se content was calculated by reference to a standard curve.

Determination of the SelN, SelW, SPS-1, and SecS mRNA level by quantitative RT-PCR

Isolation of total RNA and detection of SelN, SelW, SPS-1, and SecS mRNA level were performed on the procedure described by (Li et al. 2011). Primer Premier Software (PREMIER Biosoft International, USA) was used to design specific primers based on known chicken sequences. SelN transcripts were quantified by qRT-PCR using the following primers: 5′-TGGTTTCTTGGGAATCTCTCAC-3′/5′-AGCG GGTTTTGACGAAGG-3′. SelW, SPS-1, SecS and GADPH transcripts were quantified by qRT-PCR using the primers described by Sun (Sun et al. 2011).

qRT-PCR was performed on an ABI PRISM 7,500 Detection System (Applied Biosystems, USA). The mRNA relative abundance was calculated according to the method of (Pfaffl 2001), accounting for gene-specific efficiencies and was normalized to the mean expression of GADPH.

Statistical analysis

Statistical analysis of Se concentration and mRNA level was performed using SPSS statistical software for Windows (version 13; SPSS Inc., Chicago, IL, USA). A significant value ($P < 0.05$) was obtained by one-way analysis of variance. All data showed a normal distribution and passed equal variance testing. The correlation was assessed using Pearson's correlation coefficient. Data are expressed as mean ± standard deviation.

Results

Se content in skeletal and cardiac muscles

Skeletal and cardiac muscles Se concentrations were analyzed (Fig. 1) in chickens fed diets containing increasing amounts of Se ranging from 1.0 to 5.0 mg Se/kg diet. Our results showed that Se concentrations in thigh, shank and cardiac muscles were higher than those of breast and flight muscles in chickens fed the basal diet. Se concentrations in both skeletal and cardiac muscles of chickens fed Se-supplemented diets were increased compared to those of chickens fed the basal diet. In chickens fed Se-supplemented diets, breast, thigh and shank muscles Se concentration increased in a concentration-related manner. Compared to the control group, thigh and shank muscles Se concentration significantly increased ($P < 0.05$) in chickens fed 2.0–3.0 mg Se/kg diet, but it was no further significant increase ($P > 0.05$) between 3.0 and 5.0 mg Se/kg diet. Compared to the control group, Se concentration in breast muscle significantly increased ($P < 0.05$) in chickens fed 3.0 and 5.0 mg Se/kg diet. When chickens were fed the diets containing 1.0–3.0 mg Se/kg, flight and cardiac muscles Se concentration dose dependently increased with dietary Se content. In contrast, tissue Se content in flight and cardiac muscles of chickens fed diets containing 3.0–5.0 mg Se/kg reached a plateau ($P > 0.05$).

The Pearson correlation coefficients of Se concentrations in blood and in the different muscle tissues are presented in Table 1. Se concentrations in the different muscle tissues were significantly correlated with the blood concentrations for the different Se supply (all, $P < 0.01$). The ratio of increase in different muscle tissues/increase in blood is presented in Fig. 2. Our

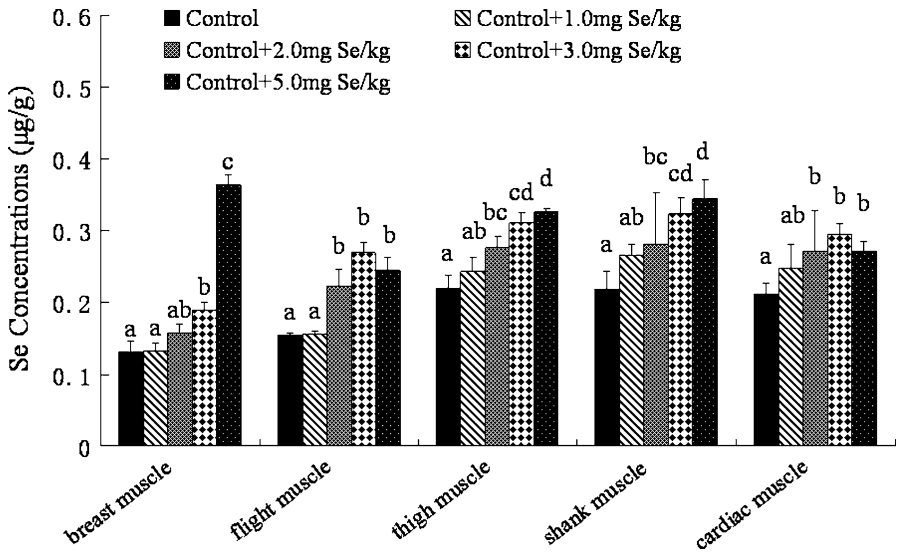

Fig. 1 Skeletal and cardiac muscles (μg/g, wet wt of tissue) Se concentrations in chickens fed diets containing various concentrations of Se. Bars represent mean ± standard deviation (n = 3/group). Within the groups treated with various levels of Se, bars sharing a common letter are not significantly different ($P > 0.05$)

results showed that the ratio of Se concentrations in muscles and blood was decrease except for an increase in breast from chickens fed 5.0 mg Se/kg and flight from chickens fed 2.0 and 3.0 mg Se/kg, suggesting no specific or preferential accumulation of Se, neither in cardiac nor in skeletal muscles.

Effect of Se dietary supplementation on SelN and SelW mRNA levels

To investigate whether SelN and SelW mRNA levels relate to Se dietary supplementation, we measured skeletal and cardiac muscles SelN and SelW mRNA levels in chickens fed with diets containing increasing amounts of Se, ranging from 1.0 to 5.0 mg Se/kg. Results of SelN and SelW mRNA relative expression measured by quantitative RT-PCR in skeletal and cardiac muscles are presented in Figs. 3, 4, 5, and 6.

The basal SelN expression level in chicken different muscle tissues was normalized to the breast muscle of same concentration group. When compared to the breast muscle, SelN expression in the control group decreased as follows: cardiac, thigh, shank, flight and breast muscles (Fig. 3). Of note, the SelN mRNA abundance in thigh muscle was the highest among muscle tissues of chicken fed diets containing 1, 3, 5 mg/kg Se (all, $P < 0.05$). When chickens fed diets containing 2.0 mg/kg Se, the SelN mRNA abundance in cardiac muscle was the highest among muscle tissues (Fig. 3).

The basal SelW expression level in chicken different muscle tissues was normalized to the breast muscle of same concentration group. When compared to the breast muscle, SelW expression in the control group was the highest in cardiac muscle and lowest in flight muscle (Fig. 4). Of note, the SelW mRNA abundance in thigh muscle was the highest in muscle tissues of chicken fed diets containing 1, 3 and 5 mg/kg Se (all, $P < 0.05$). When chickens fed diets containing 2 mg/kg Se, the SelW mRNA abundance decreased as follows: shank, cardiac, thigh, flight and breast muscles (Fig. 4). The basal SelN and SelW expression of breast and flight muscles in different concentration group retained the lowest level.

Effect of Se dietary supplementation on SelN mRNA levels in the different muscle tissues normalized to the homologous control group of same tissue is showed in Fig. 5. When compared to the homologous control group, a significant increase in the SelN mRNA levels was observed in breast, thigh and shank

Table 1 Pearson correlation coefficients between blood Se and different muscle tissues Se for the different Se supplementations

	Breast muscle	Flight muscle	Thigh muscle	Shank muscle	Cardiac muscle
Whole blood	0.867**	0.776**	0.862**	0.870**	0.718**

* $P < 0.05$, ** $P < 0.01$

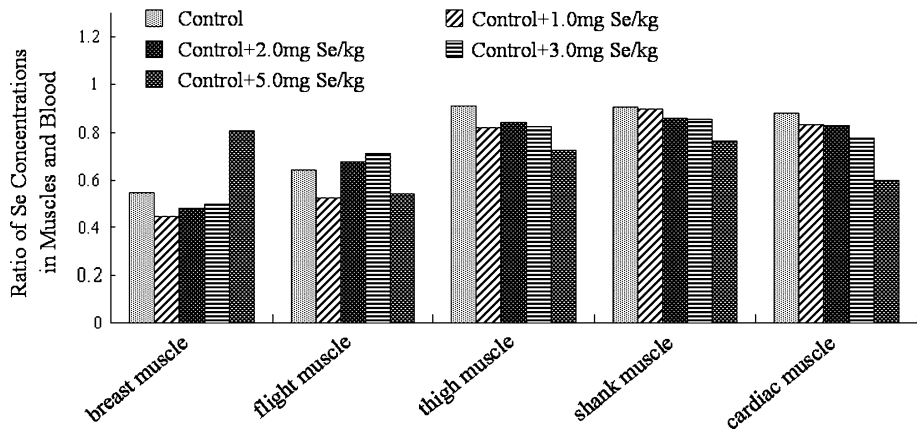

Fig. 2 Ratio of Se concentrations in muscles and blood from chickens fed diets containing various concentrations of Se

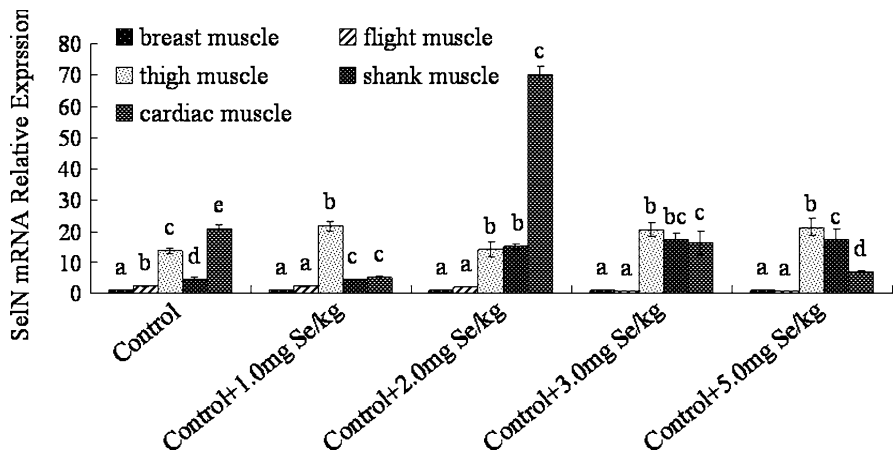

Fig. 3 Effects of the same concentrations Se on the abundance of SelN mRNA in skeletal and cardiac muscles of chicken. *Bars* sharing a common letter are not significantly different ($P > 0.05$). Data are normalized to the breast muscle of same concentration group, internal control

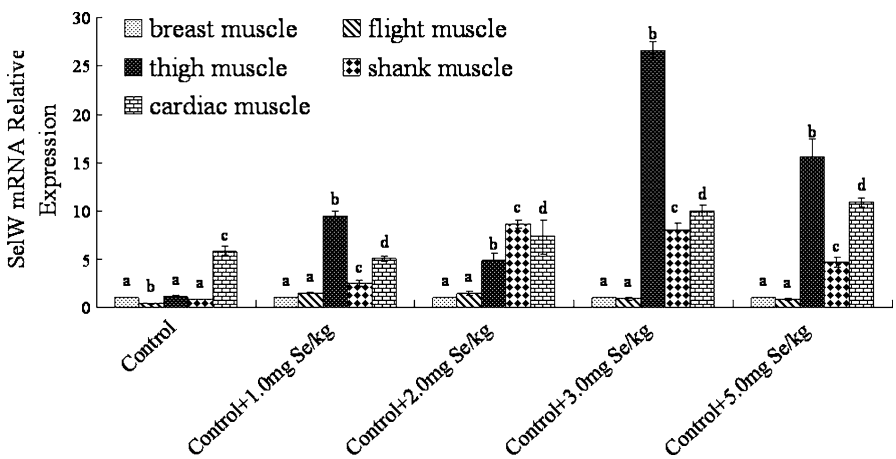

Fig. 4 Effects of the same concentrations Se on the abundance of SelW mRNA in skeletal and cardiac muscles of chicken. *Bars* sharing a common letter are not significantly different ($P > 0.05$). Data are normalized to the breast muscle of same concentration group, internal control

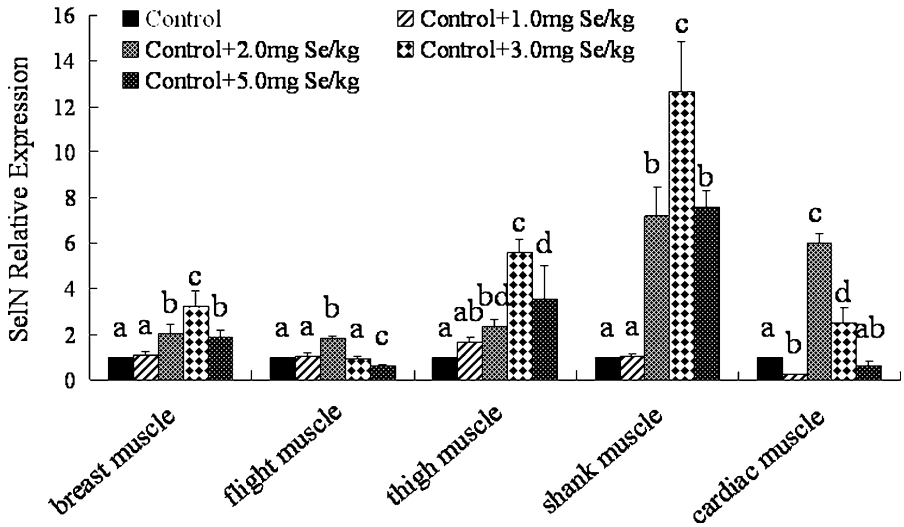

Fig. 5 Effects of different concentrations of Se on SelN mRNA levels in skeletal and cardiac muscles of chicken. *Bars* sharing a common letter are not significantly different ($P > 0.05$). Data are normalized to the homologous control group of same tissue

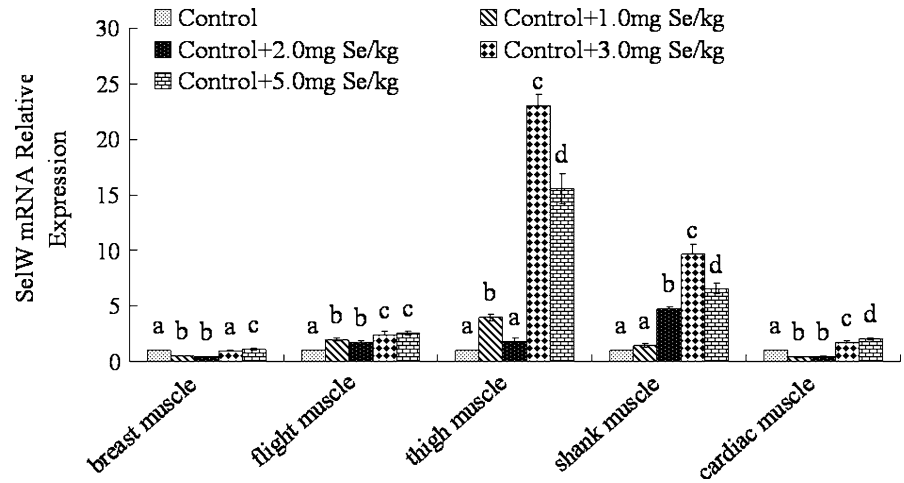

Fig. 6 Effects of the same concentrations Se on the abundance of SelW mRNA in skeletal and cardiac muscles of chicken. *Bars* sharing a common letter are not significantly different ($P > 0.05$). Data are normalized to the homologous control group of same tissue

muscles in chickens fed diets containing 2.0–5.0 mg Se/kg ($P < 0.05$). The greatest increases in SelN mRNA expression were observed in breast, thigh and shank muscles in chickens fed the diet containing 3 mg Se/kg ($P < 0.05$). Compared to the diet containing 3 mg Se/kg, a significantly decrease of SelN mRNA expression was observed in breast, thigh and shank muscles in chickens fed the diet containing 5.0 mg Se/kg. The change in SelN mRNA levels induced by Se supplementation was not significant in flight muscle. The 2.0 mg Se/kg diet caused an increase in flight muscle SelN mRNA levels of approximately 1.8-fold ($P < 0.05$) whilst those fed 5 mg Se/kg diet caused a decrease of 65% ($P < 0.05$) compared to the control group. However, SelN mRNA expression decreased by about 26% ($P < 0.05$) and 61% ($P > 0.05$) in cardiac muscle fed diet containing 1.0 and 5.0 mg Se/kg compared to the control group. Cardiac muscle from chickens fed 2.0 and 3.0 mg Se/kg diets had significant increases ($P < 0.05$) (6 fold and 2.5 fold respectively) in SelN mRNA levels (Fig. 5).

Effect of Se dietary supplementation on SelW mRNA levels in muscle tissues normalized to the homologous control group of same tissue is showed in Fig. 6. When compared to the homologous control group, a significant increase in the SelW mRNA levels was observed in flight, thigh and shank muscles in chickens fed diets containing 2.0–5.0 mg Se/kg ($P < 0.05$). The largest increases in SelW mRNA

expression were observed in flight, thigh and shank muscles in chickens fed the diet containing 3.0 mg Se/kg ($P < 0.05$). Compared to the diet containing 3.0 mg Se/kg, a significantly decrease of SelW mRNA expression was observed in thigh and shank muscles in chickens fed the diet containing 5.0 mg Se/kg. The 1.0 and 2.0 mg Se/kg diet caused a decrease in breast and cardiac muscles ($P < 0.05$) whilst those fed 3.0 or 5.0 mg Se/kg diet caused an increase compared to the control group. However, the changes in SelW mRNA levels induced by Se supplementation were limited in breast, flight and cardiac muscles.

The Pearson correlation coefficients between SelN, SelW mRNA levels and tissue Se are presented in Table 2. SelN mRNA levels in thigh, and shank muscles were significantly correlated with tissue Se content for the different Se supplementations (all, $P < 0.01$). SelW mRNA levels in flight, thigh and shank muscles were significantly correlated with tissue Se for the different Se supplementations (all, $P < 0.01$).

Effect of Se dietary supplementation on SPS1 and SecS mRNA levels

SPS-1 mRNA abundance measured by quantitative RT-PCR normalized to the homologous control group of same tissue is shown in Fig. 7. It was a significant increase ($P < 0.05$) in SPS-1 mRNA abundance in thigh and shank muscles of Se-supplemented group chicken compared to the control group. The greatest increases in SPS1 mRNA expression were observed in chickens fed the diet containing 3.0 mg/kg Se in shank muscle and 5.0 mg/kg Se in thigh muscle ($P < 0.05$). The change of SPS-1 mRNA abundance was consistent with the Se- supplemented diet group in cardiac muscle. However, Se dietary supplementation had little impact on SPS1 mRNA levels in flight, and breast muscles.

SecS mRNA abundance measured by quantitative RT-PCR normalized to the homologous control group of same tissue is shown in Fig. 8. It was a significant

Table 2 Pearson correlation coefficients between SelN, SelW, SPS1 and SecS mRNA levels and tissue Se concentration for the different Se supplementations

Parameter	Breast muscle	Flight muscle	Thigh muscle	Shank muscle	Cardiac muscle
SelN	0.243	−0.087	0.773**	0.768**	0.353
SelW	0.600	0.648**	0.791**	0.864**	0.354
SPS1	0.662**	0.558*	0.912**	0.688**	0.170
SecS	0.954**	0.075	0.802**	0.705**	0.464

* $P < 0.05$, ** $P < 0.01$

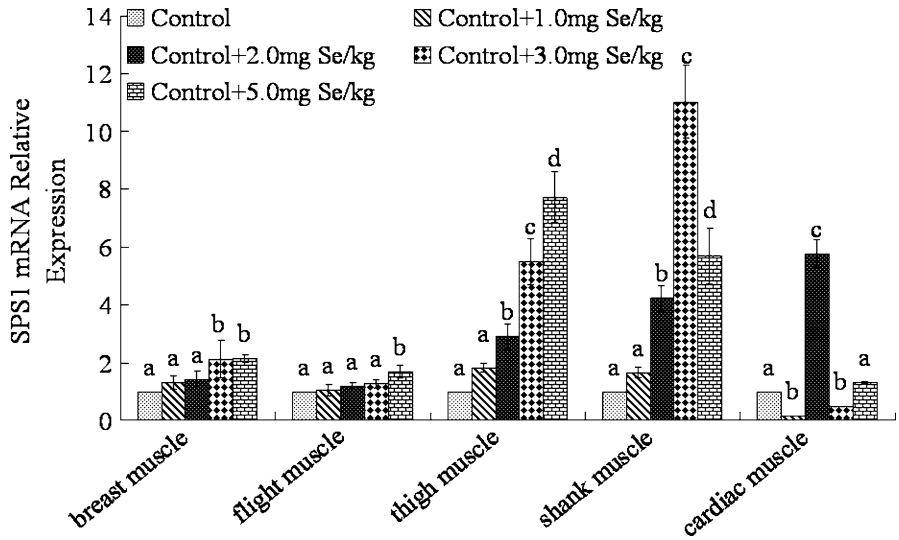

Fig. 7 Effects of different Se concentrations on SPS1 mRNA levels in skeletal and cardiac muscles of chicken. *Bars* sharing a common letter are not significantly different ($P > 0.05$). Data are normalized to the homologous control group of same tissue

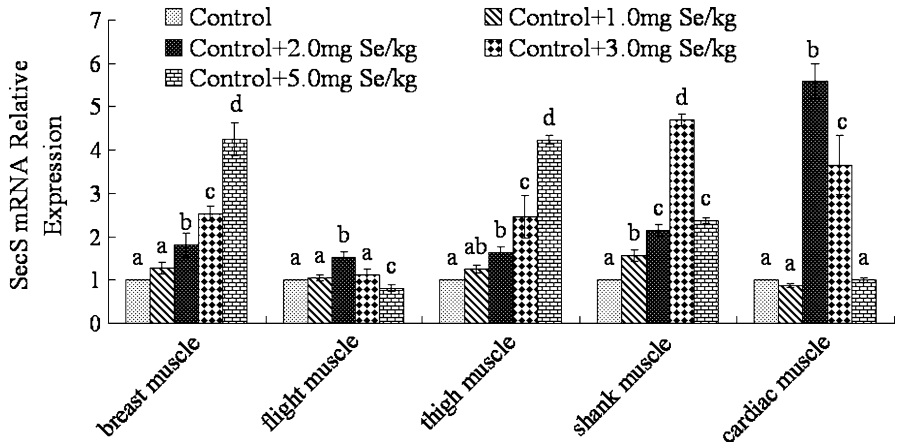

Fig. 8 Effects of different Se concentrations on SecS mRNA levels in skeletal and cardiac muscles of chicken. Bars sharing a common letter are not significantly different ($P > 0.05$). Data are normalized to the homologous control group of same tissue

increase ($P < 0.05$) of SecS mRNA abundance in breast, thigh, shank and cardiac muscles of Se-supplemented group chicken compared to the control group. The greatest increases in SecS mRNA expression were observed in chickens fed the diet containing 2.0 mg/kg Se in cardiac muscle, 3.0 mg/kg Se in shank muscle and 5.0 mg/kg Se in breast and thigh muscles ($P < 0.05$). SecS mRNA abundance in flight muscle showed minor variation in Se- supplemented diet group. The 2.0 mg Se/kg diet caused an increase in flight muscle SecS mRNA levels of approximately 1.5-fold ($P < 0.05$) whilst those fed 5.0 mg Se/kg diet caused a decrease of 81% ($P < 0.05$) compared to the control group. However, after reaching the maximal level, further increases of sodium selenite dose actually led to a reduction in SecS mRNA expression.

The Pearson correlation coefficients between SPS1, SecS mRNA levels and tissue Se are presented in Table 2. SPS1 mRNA levels in breast, flight, thigh, and shank muscles were significantly correlated with tissue Se content for the different Se supplementations (all, $P < 0.05$). SecS mRNA levels in breast, thigh and shank muscles were significantly correlated with tissue Se content for the different Se supplementations (all, $P < 0.01$).

Discussion

In the current study, we showed that the content of Se in skeletal and cardiac muscles was increased with Se dietary supplementation. Tissue Se content was consistent with blood Se, but the increase of muscle tissues Se content was lower compared with blood Se content. Therefore, it was showed that Se does not preferentially accumulate in muscle tissues in chicken. Se was absorbed in priority in the gastrointestinal tract of chicken, therefore, the content of Se in most tissues of the gastrointestinal tract of chicken especially in small intestine was higher than that of muscle tissues (Li et al. 2010). This data indicated that the preferential accumulation of Se was in blood and the gastrointestinal tract compared with muscle tissues in chicken. Moreover, Se concentrations in various muscles were significantly different in control condition, but also independently of the Se supply: thigh, thank and cardiac muscles concentration being higher than breast and flight muscles. This difference was modified only at the highest Se dietary concentration, for which a preferential accumulation in breast muscle is observed.

SelN and SelW mRNAs abundance were different in different muscles in control condition. Levels of SelN and SelW mRNA were lower in breast muscle and flight muscle than other tissues in control condition. The effect of Se supplementation on SelN and SelW mRNA abundance was small in breast muscle and flight muscle, which was consistent with tissue Se content. Changes in SelN and SelW mRNA abundance in thigh and shank muscles were correlated to Se supply. Levels of SelN and SelW mRNA were increased by Se intake, while higher Se supplementation led to dose-dependent decreasing trends of the mRNA level. Here, our hypothesis is that SelN and SelW mRNA abundance in thigh and shank muscles were not further increased by 5.0 mg Se/kg diet, most

likely due to muscles toxicity induced by Se. Similar changes of selenoproteins induced by Se were found in rodents and cultured cells (Raines and Sunde 2011; Wu et al. 2010). Levels of cardiac muscle SelN and SelW mRNA were distinct with skeletal muscle. It was concluded that the sensitivity of different muscle tissues to Se was distinct.

A lack of Se associated to several muscular diseases affecting both cardiac and skeletal muscles was described in both animals and humans, including nutritional muscular dystrophy, white muscle disease (WMD) rigid/stiff lamb syndrome (RL), Keshan disease and so on (Rederstorff et al. 2006). Moreover, in chicks, exudative diathesis was another Se deficiency-related disorder. Emergence of these disorders was linked to Se content in the food and Se supplementation was shown to be protective. Therefore, there is sufficient evidence to indicate that Se is important for normal muscle metabolism. SelN may play a significant role in preventing some muscular dystrophies, a group of diseases referred to as SEPN1-RM1, where SEPN1 refers to the gene name of the SelN protein (Moghadaszadeh et al. 2001; Petit et al. 2003). SelN is central to the development of muscular architecture, and in zebrafish where SelN has been severely reduced, muscular sarcomere organization, myofiber attachment and myoseptum integrity are disrupted (Deniziak et al. 2007). The first selenoprotein described to be linked to a muscular disorder was SelW (Whanger 2000). Thus, the function of SelN and SelW may be important for muscle organization.

Numerous studies have shown that selenoprotein mRNA levels were regulated by Se dietary supplementation. Levels of all of these selenocysteine-containing proteins including the glutathione peroxidase family (GPX1, GPX2, GPX3, and GPX4), one or more iodothyronine deiodinases and two thioredixin reductases and Selenoprotein P in various tissues are affected to different extents by Se availability. Increased amounts of selenoproteins observed in response to Se supplementation were shown in several studies to correlate with increases in the corresponding mRNA levels (Allan et al. 1999; Wu et al. 2010).

SelW mRNA expression in the gastrointestinal tract, liver, pancreatic tissue; immune organs and skeletal muscle system in chickens are highly sensitive to Se supply (Li et al. 2010; Gao et al. 2011; Sun et al. 2011; Wang et al. 2011; Yu et al. 2011; Ruan et al. 2011). Pagmantidis et al. (2005) reported that SelN and SelW expression levels in human intestinal Caco-2 cells is highly sensitive to Se-depletion and Se-adequate conditions.

In this work, the effect of Se supplementation on SPS1 and SecS mRNA levels was examined. Se regulated the mRNA expression of SPS1 and SecS in chicken muscle tissues. The increase of SPS1 and SecS mRNA abundance was significant especially in thigh muscle and shank muscle of chickens fed Se-supplemented diets (Figs. 7 and 8).

SPS1 and SecS mRNA levels in breast muscle, thigh muscle and shank muscle were significantly correlated with tissue Se concentration for the different Se supplementations (Table 2).

Recent studies showed that knockdown of SPS1 in mammalian cells had no effect on selenoprotein biosynthesis (Xu et al. 2007a, b). Thus, the role of SPS1 in selenoprotein biosynthesis remains unresolved. The presence of SPS1 in organisms lacking selenoproteins indicates that this protein is involved in a pathway unrelated to selenoprotein biosynthesis. SPS1 may define a new pathway of Se utilization in animals (Lobanov et al. 2008). Here, it was concluded that SPS1 mRNA abundance was regulated by Se dietary supplementation in chicken muscle tissues. SPS1 played an important role in Se utilization even though this protein was not involved in a pathway of selenoprotein biosynthesis. SecS invariably takes place on $tRNA^{[Ser]Sec}$ (Hendrickson 2007) and play a important role in the complex machinery of insertion of Sec.

Thus, it was concluded that Se regulated of SelN and SelW mRNA abundance and showed differential sensitivity among different skeletal muscles and cardiac tissue. Sensitive and rapid responses of SelN and SelW expression to Se status indicated that they might play a role in metabolism in muscle tissues. In this work, the impact of Se-supplemented on SPS-1 and SecS mRNA expression showed that Se upregulated the mRNA expression of SPS-1 and SecS mRNA levels.

In summary, understanding the regulation of the SelN and SelW induced by Se in birds muscle tissues is an essential step in defining the association of the dietary Se level and expression of SelN and SelW gene in muscle function. However, more mechanistic studies are needed for better determining a potential role for SelN and SelW in muscle function and function of SPS1 and SecS in selenoprotein synthesis and Se utilization.

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