Evaluation of the efficacy of 2-hydroxy-4-methylselenobutanoic acid on growth performance and tissue selenium retention in growing pigs

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ABSTRACT: The aim of this study was to compare the efficacy of a new organic Se (2-hydroxy-4-methylselenobutanoic acid [HMSeBA]) source (SO) with sodium selenite (SS) and selenized yeast (SY) at various dietary levels for growth performance and tissue Se deposition in growing pigs. A total of 112 crossbred (Pietrain × [Large White × Landrace]) gilts were allotted at an average body weight of 26.73 kg to 7 dietary treatments with 8 replicate pens of 2 pigs per pen. Pigs were fed basal diets unsupplemented or supplemented either with SS, SY, or SO each at 0.1 or 0.3 mg Se/kg of diet for 32 d. Feed intake and BW were recorded during the experimental period. At the end of the experiment, blood, liver, and psoas major muscle of all gilts were collected for total Se and relative bioavailability determination. No differences were observed on final BW, ADG, ADFI, and G:F among dietary treatments. All Se-supplemented groups exhibited greater total Se contents in plasma ($P < 0.01$) and liver ($P < 0.01$) compared with unsupplemented control group. However, Se retention in psoas major muscle was improved only when organic Se source (SY or SO) was added to diets ($P < 0.01$). Regardless the Se level, the Se deposition in muscle was greater ($P < 0.01$) in pigs supplemented with SO than those supplemented with SY. Slope ratio assay confirmed the greater bioavailability of Se from organic compared with inorganic Se and also revealed that the relative bioavailability of Se from HMSeBA for plasma, liver, and muscle Se response was 170, 141, and 162%, respectively, for SY. This study shows a potential advantage of HMSeBA supplementation in the increase of Se contents in pig tissues, indicating that this new organic Se source could be an alternative source of Se in swine nutrition.

Key words: bioavailability, growing pigs, selenium, tissue Se deposition, 2-hydroxy-4-methylselenobutanoic acid

INTRODUCTION

Selenium is an essential trace element that has a large number of biological functions in humans and animals. It has been well established that Se is an integral component of glutathione peroxidase, a crucial antioxidative enzyme, which catalyzes the destruction of hydrogen peroxide generated during oxidative metabolism in human and animal cells (Rotruck et al., 1973). Since then, Se was identified as an essential component in vertebrate cells of thioredoxin reductase, essential enzyme for maintaining the cellular redox balance (Tamura and Stadtman, 1996), iodothyronine deiodinases involved in the regulation of thyroid function (Pallud et al., 1997; Brown and Arthur, 2001), and in several other selenoproteins (Burk et al., 2003; Kryukov et al., 2003; Lu and Holmgren, 2008; Mariotti et al., 2012).

In pigs, as well as in other domestic animal species, Se metabolism depends closely of Se forms and sources. Generally, organic Se has greater bioavailability and rates of tissue retention than inorganic Se (Vendeland et al., 1994; Mahan et al., 1999; Juniper et al., 2008, 2011; Liao et al., 2012; Speight et al., 2012). Many studies have established that selenomethionine and selenized yeast (SY) are the most appropriate source of Se for use in animal nutritional supplements because of their greater bioavailability and lower toxicity (Tinggi, 2003; Utterback et al., 2005; Wang and Xu, 2008; Skrivan et al., 2012).

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Recently, a new organic Se source based on the 2-hydroxy-4-methylselenobutanoic acid (HMSeBA), well known as hydroxy-analogue of selenomethionine, has been characterized and its dietary efficacy in poultry nutrition has been also demonstrated (Briens et al., 2013; Jlali et al., 2013). Therefore, the objective of this study was to compare the efficacy of HMSeBA (SO) as a new Se source with sodium selenite (SS) and SY for growth performance and tissue Se deposition in growing pigs.

**MATERIALS AND METHODS**

All experimental procedures were carried out according to legislation governing the ethical treatment of animals, and investigators were certified by the French government to conduct animal experiments under authorization B-35-275-32 given to INRA-UMR1079 PEGASE (Saint-Gilles, France).

**Animals and Experimental Design**

A total of 112 crossbred ([Large White × Landrace] × Pietrain) gilts originated from the nursery of the INRA-UMR1079 PEGASE (Saint-Gilles, France) were used in this experiment. The gilts initially averaged 26.73 ± 3.15 kg BW and allotted to the 7 dietary treatments on the basis of weight and litter during 32 d. The experiment was conducted in 8 replicate pens of 2 pigs per pen (equalized among treatments within replicates). The dietary treatments were composed with the same basal diet (Table 1) but differed only in Se sources and levels (Table 2): control diet without Se supplementation, and control diet supplemented with 0.1 or 0.3 mg/kg of SS (SS-0.1 and SS-0.3; Micogan Se 1% BPM, DSM Nutritional Product AG [Basel, Switzerland]), SY (SY-0.1 and SY-0.3; Sel-Plex 2000, Alltech [Nicholasville, KY]), or SO (SO-0.1 and SO-0.3; HMSeBA, Selisseo, Adisseo [Antony, France]). All Se additives were added to a premix then used for the manufacturing of the complete feedstuff. All gilts were housed in a totally slotted floor in an environmentally controlled building with 2.25 m² (0.85 × 2.65 m) pen space per 2 gilts. Water and diets were provided ad libitum throughout the experimental period. Gilts were weighed individually at the beginning and at the end of experimental period and feed intake per pen was monitored weekly.

**Sampling**

The representative feed samples were taken for chemical (DM, CP, and mineral matter) and total Se analysis. At the end of the study and after 12 h of feed withdrawal, all gilts were randomly slaughtered (INRA-UMR1079-PEGASE). Individual blood samples for all gilts (n = 112) were collected in heparin tubes during exsanguinations and after euthanized by electrocution. The blood samples were centrifuged at 3000 × g at 4°C for 10 min, and plasma was aliquoted and stored at −20°C until total Se analysis. Samples of liver and tenderloin (psosas major) muscle (50 g per tissue) were collected from all pigs (112 samples/tissue) then immediately stored at −20°C until further analysis.

**Total Se Analysis**

Total Se concentrations in diet, plasma, liver, and muscle were determined according to the method previously described by Bierla et al. (2008) and Vacchina et
RESULTS

Diet Se Concentrations and Growth Performance

The Se content in each diet is summarized in Table 2. The results showed that, considering the analyzed Se level in the control diet, the expected Se levels were confirmed in the experimental diets. The effects of dietary treatments (Se source and level) on pig growth performance responses are presented in Table 3. Final BW and ADG did not differ among treatments. Likewise, no difference in ADFI and G:F were observed among the treatments groups during the study.

Plasma and Tissue Se Concentrations

The Se concentrations in plasma and tissues (expressed per DM or fresh matter product) are summarized in Table 4. No effect of dietary treatments on moisture contents was observed either in liver or muscle. Indeed, the average values of moisture were 72.58 and 74.88% in liver and muscle, respectively. Pigs fed diets supplemented with Se had greater plasma Se concentrations ($P < 0.05$) than those fed control diet without Se added. At 0.1 mg Se/kg of diet, plasma Se concentration did not differ among pigs supplemented with inorganic Se (SS) and those supplemented with organic Se (SY and SO). However, the increase of dietary Se level to 0.3 mg/kg of diet increased ($P < 0.05$) the plasma Se concentration only in pigs supplemented with Se from SY and SO. The

For relative bioavailability values, Se deposition data were subjected to multiple nonlinear regression analyses to verify the fundamental validity assumptions of the slope-ratio assay (Littell et al., 1997), which include testing equality of the intercept and equality of the common intercept to the mean of the basal diet. The multiple nonlinear regression analysis was performed using the following equation:

$$Y = a + a_0X_0 + \beta_1 (SS + \beta_2\beta_1\, SY + \beta_3\beta_1\, SO)$$

where $Y$ is the response variable (Se concentrations in plasma, liver, or muscle), $a$ is the intercept, $a_0X_0$ is a correction for negative control diet, and $\beta_1$, $\beta_2$, and $\beta_3$ are the slopes of the lines in response to SS, SY, and SO doses, respectively. The SS, SY, and SO are the supplemental doses of SS, SY, and HMSeBA in the diets, respectively. The slope of each Se source was directly determined by using the PROC MLIN procedure of SAS and the relative biological values (the ratio between slopes) were presented with their confidence interval. Statistical significance was set at $P \leq 0.05$ and tendencies were considered at $P < 0.10$.

Table 2. Se sources and levels supplemented in diets

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Se source</th>
<th>Supplemental Se, mg/kg</th>
<th>Total Se, mg/kg of diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>Basal diet</td>
<td>0.0</td>
<td>0.11 ± 0.01</td>
</tr>
<tr>
<td>SS-0.1</td>
<td>Basal diet + sodium selenite</td>
<td>0.1</td>
<td>0.20 ± 0.01</td>
</tr>
<tr>
<td>SS-0.3</td>
<td>Basal diet + sodium selenite</td>
<td>0.3</td>
<td>0.38 ± 0.02</td>
</tr>
<tr>
<td>SY-0.1</td>
<td>Basal diet + Se-enriched yeast</td>
<td>0.1</td>
<td>0.22 ± 0.01</td>
</tr>
<tr>
<td>SY-0.3</td>
<td>Basal diet + Se-enriched yeast</td>
<td>0.3</td>
<td>0.42 ± 0.02</td>
</tr>
<tr>
<td>SO-0.1</td>
<td>Basal diet + 2-hydroxy-4-methylselenobutanoic acid</td>
<td>0.1</td>
<td>0.21 ± 0.01</td>
</tr>
<tr>
<td>SO-0.3</td>
<td>Basal diet + 2-hydroxy-4-methylselenobutanoic acid</td>
<td>0.3</td>
<td>0.41 ± 0.02</td>
</tr>
</tbody>
</table>

1Control = basal diet; SS-0.1 = basal diet supplemented with 0.1 mg Se/kg from sodium selenite; SS-0.3 = basal diet supplemented with 0.3 mg Se/kg from sodium selenite; SY-0.1 = basal diet supplemented with 0.1 mg Se/kg from Se-enriched yeast; SY-0.3 = basal diet supplemented with 0.3 mg Se/kg from Se-enriched yeast; SO-0.1 = basal diet supplemented with 0.1 mg Se/kg from 2-hydroxy-4-methylselenobutanoic acid; SO-0.3 = basal diet supplemented with 0.3 mg Se/kg from 2-hydroxy-4-methylselenobutanoic acid.

2Values are means of 8 samples.

Statistical Analysis and Relative Bioavailability Determination

All Data were analyzed using GLM procedure of SAS (v. 9.1.3; SAS Inst. Inc., Cary, NC). The fixed effect of dietary treatment, random effect of pen, and initial BW added as a covariate were included in the model. The pen was used as experimental unit for growth performance trait analysis and Se concentrations in plasma, liver, and muscle. Comparisons of least square means were performed by Tukey-Kramer test when overall treatment effect was significant.
Organic selenium and selenium bioavailability

Circulating concentrations of Se were increased when the pigs were supplemented with increasing levels of Se from SY ($P < 0.05$) and SO ($P < 0.05$) in comparison with those fed Se-supplemented diets from SS. Moreover, the liver Se concentrations were also greater ($P < 0.05$) in pigs fed diets supplemented with Se in comparison with those fed control diet containing only the native Se offered by the ingredients of basal diet. Compared with SS, the liver Se concentrations increased ($P < 0.05$) as the dietary Se level from SY and SO increased in pig diets. However, at 0.1 mg Se/kg of diet, the liver Se concentration was similar between pigs fed SS and those fed SY, whereas with the same supplemented level of Se in diet, the addition of HMSeBA increased the hepatic levels of Se in comparison with SS ($P < 0.05$) and SY ($P < 0.05$). The supplementation with 0.3 mg Se from HMSeBA resulted in greatest liver Se ($P < 0.05$).

As for tenderloin (psoas major) muscle, compared with control diet, Se retention was improved only when organic Se source (SY or SO) was added to pig diets. Thereby, muscle Se concentration remains similar between pigs fed the control diet and those fed diets supplemented with Se from SS regardless of the dietary Se level. However, for both organic Se sources (SY and SO), the increase of Se level from 0.1 to 0.3 mg/kg in pig diets increased ($P < 0.05$) the Se concentrations in the loin muscle. Compared with pigs fed diets supplemented with Se from SY, pigs fed Se as HMSeBA had greater muscle Se concentrations in psoas major muscle ($P < 0.05$).

Bioavailability of Se within Se Sources

The relative bioavailability of Se from each Se source in plasma, liver, and muscle are summarized in Table 5. Compared with SS, SY and SO were more efficient ($P < 0.01$) to improve Se concentrations in plasma, liver, and muscle. Moreover, within the organic Se source, HMSeBA was more effective in enhancing

Table 3. Growth performance of growing pigs fed diets with different concentrations and sources of Se

<table>
<thead>
<tr>
<th>Item</th>
<th>Control</th>
<th>SS-0.1</th>
<th>SS-0.3</th>
<th>SY-0.1</th>
<th>SY-0.3</th>
<th>SO-0.1</th>
<th>SO-0.3</th>
<th>SEM</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of pens</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BW, kg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial</td>
<td>26.79</td>
<td>26.91</td>
<td>26.84</td>
<td>27.00</td>
<td>26.34</td>
<td>26.76</td>
<td>26.48</td>
<td>0.34</td>
<td>0.81</td>
</tr>
<tr>
<td>Final</td>
<td>52.89</td>
<td>53.65</td>
<td>52.22</td>
<td>50.72</td>
<td>52.55</td>
<td>52.58</td>
<td>52.33</td>
<td>0.94</td>
<td>0.51</td>
</tr>
<tr>
<td>ADG, kg</td>
<td>0.831</td>
<td>0.855</td>
<td>0.810</td>
<td>0.761</td>
<td>0.819</td>
<td>0.820</td>
<td>0.813</td>
<td>0.030</td>
<td>0.50</td>
</tr>
<tr>
<td>ADFI, kg</td>
<td>1.834</td>
<td>1.874</td>
<td>1.792</td>
<td>1.734</td>
<td>1.801</td>
<td>1.804</td>
<td>1.788</td>
<td>0.040</td>
<td>0.44</td>
</tr>
<tr>
<td>G:F</td>
<td>0.452</td>
<td>0.458</td>
<td>0.451</td>
<td>0.437</td>
<td>0.453</td>
<td>0.455</td>
<td>0.455</td>
<td>0.010</td>
<td>0.78</td>
</tr>
</tbody>
</table>

1 Each pen with 2 gilts.

2 C = basal diet; SS-0.1 = BD supplemented with 0.1 mg Se/kg from sodium selenite; SS-0.3 = BD supplemented with 0.3 mg Se/kg from sodium selenite; SY-0.1 = BD supplemented with 0.1 mg Se/kg from Se-enriched yeast; SY-0.3 = BD supplemented with 0.3 mg Se/kg from Se-enriched yeast; SO-0.1 = BD supplemented with 0.1 mg Se/kg from 2-hydroxy-4-methylselenobutanoic acid; and SO-0.3 = BD supplemented with 0.3 mg Se/kg from 2-hydroxy-4-methylselenobutanoic acid.

Table 4. Effect of Se sources and levels on Se concentrations in plasma, liver, and muscle

<table>
<thead>
<tr>
<th>Item</th>
<th>Control</th>
<th>SS-0.1</th>
<th>SS-0.3</th>
<th>SY-0.1</th>
<th>SY-0.3</th>
<th>SO-0.1</th>
<th>SO-0.3</th>
<th>SEM</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma, Se, ng/g fresh matter</td>
<td>91.24&lt;sup&gt;d&lt;/sup&gt;</td>
<td>120.43&lt;sup&gt;c&lt;/sup&gt;</td>
<td>120.68&lt;sup&gt;c&lt;/sup&gt;</td>
<td>117.46&lt;sup&gt;c&lt;/sup&gt;</td>
<td>142.12&lt;sup&gt;b&lt;/sup&gt;</td>
<td>128.10&lt;sup&gt;b&lt;/sup&gt;</td>
<td>160.66&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.15</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Liver, Se, mg/kg fresh matter</td>
<td>0.34&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.44&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.48&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.46&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.65&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.56&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.74&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.02</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DM, %</td>
<td>27.99</td>
<td>27.23</td>
<td>26.9</td>
<td>26.38</td>
<td>27.35</td>
<td>28.27</td>
<td>27.81</td>
<td>0.54</td>
<td>0.21</td>
</tr>
<tr>
<td>Psoas major muscle, Se, mg/kg fresh matter</td>
<td>0.11&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.12&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.13&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.17&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.27&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.21&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.36&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.01</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DM, %</td>
<td>25.47</td>
<td>24.59</td>
<td>25.21</td>
<td>25.49</td>
<td>24.97</td>
<td>24.91</td>
<td>25.23</td>
<td>0.40</td>
<td>0.68</td>
</tr>
</tbody>
</table>

<sup>a</sup>–<sup>e</sup>Means with different superscripts are statistically different ($P < 0.05$).

1 Each pen with 2 gilts.

2 C = basal diet; SS-0.1 = BD supplemented with 0.1 mg Se/kg from sodium selenite; SS-0.3 = BD supplemented with 0.3 mg Se/kg from sodium selenite; SY-0.1 = BD supplemented with 0.1 mg Se/kg from Se-enriched yeast; SY-0.3 = BD supplemented with 0.3 mg Se/kg from Se-enriched yeast; SO-0.1 = BD supplemented with 0.1 mg Se/kg from 2-hydroxy-4-methylselenobutanoic acid; and SO-0.3 = BD supplemented with 0.3 mg Se/kg from 2-hydroxy-4-methylselenobutanoic acid.
Se concentrations ($P < 0.01$) in plasma (+69.66%), liver (+40.52%), and muscle (+62.22%) than SY.

**DISCUSSION**

The growth performance results showed no effect of Se source (inorganic or organic) on final BW, ADG, ADFI, and G:F of pigs irrespective of the dietary level of Se (0.0, 0.1, or 0.3 mg Se/kg). These results are consistent with those of Mahan and Parrett (1996) and Mahan et al. (1999), who also demonstrated no growth performance response when either an inorganic or an organic form of Se was added at various levels to growing-finishing diets fed to pigs. In our study, the lack of difference among dietary treatments on growth performance indicates that the control diet contained a sufficient amount of Se from the dietary ingredients in the basal diet (0.11 mg per kg). Even if the Se level in the control diet was lower than the 0.20 mg Se/kg of diet recommended by NRC (2012), this level appeared sufficient to sustain performance traits in the ideal conditions of experimental facilities. This is likely to indicate that pigs can store a quantity of Se that is transferred by colostrum and milk during lactation (Mahan and Peters, 2004; Yoon and McMillan, 2006; Zhan et al., 2011), which can be used at post-weaning period for growth, thereby reducing the possible effects of Se supplementation on pig growth performance. Moreover, in our experimental conditions, the control diet exhibited a very high basal level of Se (0.11 mg/kg) compared to usual level in non-supplemented diets (approximately, 0.06 mg/kg of feed; Mahan and Parrett, 1996; Mahan et al., 1999; Mahan and Peters, 2004; Li et al., 2011), which may mask any effect on growth performance criteria. The rather high dietary vitamin E level (100 IU) might also explain the absence of response to supplementary Se on growth performance (Mahan and Parrett, 1996; Li et al., 2011).

Selenium concentrations in plasma and tissues were influenced by both Se sources and levels. These results agreed with previous research with pigs (Mahan and Parrett, 1996; Mateo et al., 2007; Speight et al., 2012) and even on other species such as poultry (Juniper et al., 2011; Briens et al., 2013), cattle (Juniper et al., 2008), lamb (Qin et al., 2007), and sheep (Davis et al., 2008). The Se concentrations in plasma and liver were effectively increased when pigs were fed diets supplemented with Se, regardless of the source, in comparison with pigs receiving the unsupplemented control diet. The effect of Se sources addition on plasma total Se concentration appeared dose dependent when organic forms (SY or SO) were supplied, whereas the inorganic form (SS) to provide beyond 0.1 mg/kg of feed failed to increase plasma Se level. Mahan et al. (1999) also reported that 0.05 mg/kg supplemental Se from SS allowed plasma Se increase, but that plasmatic Se concentration elevation was greater when the organic Se source was provided (Hu et al., 2011; Zhan et al., 2011).

Moreover, in psoas major muscle, only the organic Se source (SY and SO) has shown the ability to improve total Se contents. Indeed, SS additions did not increase muscle Se contents, indicating a specific effect of organic source in this tissue. Our results confirmed previous research, which indicated that a lower proportion of Se was retained in muscle when inorganic Se source was fed to growing-finishing pigs (Mahan et al., 1999) or broiler chickens (Briens et al., 2013). It is probably that higher proportion of the selenoamino acids, in particular selenomethionine, provided from organic Se sources (e.g., SY or HMSeBA) was nonspecifically incorporated into muscle protein during growing period, which is characterized by a high rate of protein synthesis. Therefore, selenomethionine from organic Se sources can be readily incorporated into structural proteins because of no distinction between Met and selenomethionine by tRNA during protein synthesis in muscle (Schrauzer, 2003).

<table>
<thead>
<tr>
<th>Item</th>
<th>Intercept</th>
<th>$a_0$</th>
<th>$b_1$SS</th>
<th>$b_2$SY</th>
<th>$b_3$SO</th>
<th>$P$-value</th>
<th>RBV$_1$, %</th>
<th>RBV$_2$, %</th>
<th>RBV$_3$, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma Se, ng/g fresh matter</td>
<td>112.00</td>
<td>-20.26</td>
<td>34.27</td>
<td>95.65</td>
<td>162.30</td>
<td>&lt;0.001</td>
<td>279.1</td>
<td>473.5</td>
<td>169.7</td>
</tr>
<tr>
<td>Tissue Se, mg/kg dry matter</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liver</td>
<td>1.534</td>
<td>-0.279</td>
<td>0.905</td>
<td>2.730</td>
<td>3.837</td>
<td>&lt;0.001</td>
<td>301.8</td>
<td>424.0</td>
<td>140.5</td>
</tr>
<tr>
<td>Muscle</td>
<td>0.496</td>
<td>-0.047</td>
<td>0.024</td>
<td>1.962</td>
<td>3.183</td>
<td>&lt;0.001</td>
<td>8049</td>
<td>13,060</td>
<td>162.2</td>
</tr>
<tr>
<td>Tissue Se, mg/kg fresh matter</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liver</td>
<td>0.415</td>
<td>-0.068</td>
<td>0.224</td>
<td>0.750</td>
<td>1.118</td>
<td>&lt;0.001</td>
<td>335.0</td>
<td>499.7</td>
<td>149.2</td>
</tr>
<tr>
<td>Muscle</td>
<td>0.123</td>
<td>-0.009</td>
<td>0.007</td>
<td>0.497</td>
<td>0.806</td>
<td>&lt;0.001</td>
<td>6810</td>
<td>11,060</td>
<td>162.4</td>
</tr>
</tbody>
</table>

1Dependent variable ($y$) = common intercept + $a_0$ × Se dose in negative control (NC) diet + [Slope (SS) × (added dietary Se concentration)] + [slope (SY) × (added dietary Se concentration)] + [slope (SO) × (added dietary Se concentration)].

2$RBV_1 = (b_2 SY/b_1 SS) × 100; RBV_2 = (b_3 SO/b_1 SS) × 100; RBV_3 = (b_3 SO/b_2 SY) × 100.$

3SO = source; SS = sodium selenite; SY = selenized yeast; $b_1$, $b_2$, and $b_3$ are the slopes of the lines in response to SS, SY, and SO doses, respectively.
This result is probably due to differences in metabolic pathways between inorganic and organic Se (Suzuki, 2005). Indeed, inorganic forms of Se such as selenite and selenate lead to selenocysteine through selenophosphate and Ser-tRNA, which is incorporated specifically into selenoproteins such as glutathione peroxidases or thioredoxin reductase, but cannot lead to de novo selenomethionine synthesis. Conversely, organic Se sources such as SY and SO can lead to both selenomethionine and selenocysteine with their respective contents varying considerably among tissues (Juniper et al., 2011; Briens et al., 2013). The cells can incorporate indifferently Met or selenomethionine into the protein when synthesized (Schrauzer, 2001, 2003; Navarro-Alarcon and Cabrera-Vique, 2008), leading to seleno containing proteins considered as a storage form of Se especially under selenomethionine form. Surai (2002) reported that endogenous Se in selenomethionine form stored in muscle could be mobilized if necessary (e.g., stress conditions) for maintaining the cell redox homeostasis.

Relative bioavailability assessment indicated that the organic Se (SY or SO) was more available for increasing Se circulation in blood and also Se deposition in liver and muscle than inorganic Se (SS). It has been previously reported that feeding diets supplemented with Se from organic Se source resulted in a greater tissue accumulation of Se than inorganic forms of Se (Mahan and Parrett, 1996; Kim and Mahan, 2001; Taylor et al., 2005). Likewise, Beilstein and Whanger (1986) have demonstrated that selenomethionine has a greater bioavailability than inorganic Se, which could be the result of its greater incorporation into proteins. In addition, although SS can be utilized for selenoprotein biosynthesis, only selenomethionine can be incorporated nonspecifically into body proteins in place of Met (Schrauzer, 2000), allowing Se to be stored in the animal body tissue protein, such as liver and muscle in our study. Moreover, within the organic Se sources used in our study, we showed a greater bioavailability of HMSeBA to increase Se concentrations in plasma (+70%), liver (+41%), and muscle (+62%) than SY. Similarly, Briens et al. (2013) indicated that HMSeBA was 39% more efficient in increasing muscle Se deposition in broiler chickens than SY. It seems that the chemical form of Se in those different organic sources can strongly determine the amount of Se uptake and its deposition in animal tissues. Cantor et al. (1975) have indicated that biological availability of dietary Se seems to depend primarily on its chemical nature rather than on its digestion or absorption characteristics in the intestine. However, some complementary studies are needed to delineate the complete metabolic pathway of HMSeBA molecule and how it is incorporated into several proteins in various tissues.

In this study, we showed that organic Se sources (SY or HMSeBA) increased the plasma Se concentrations and improved the deposition of this trace element in both psoas major muscle and liver in comparison to mineral Se. Within the organic Se, we showed that HMSeBA was more efficient in improving the Se deposition in liver and muscle than SY, indicating a greater bioavailability of HMSeBA than SY. Therefore, HMSeBA could be considered as an alternative source of Se in animal nutrition. Finally, further studies are needed to determine the mechanistic pathways by which different sources of Se accumulate in animal tissues, and this could help to understand the causes of higher bioavailability of HMSeBA.

**LITERATURE CITED**


