Efficacy of Dietary Sodium Selenite and Calcium Selenite Provided in the Diet at Approved, Marginally Toxic, and Toxic Levels to Growing Swine1,2,3

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ABSTRACT

A 2 × 3 factorial experiment conducted in three replicates of a randomized complete block design compared the effects of calcium selenite and sodium selenite at three different levels of Se (.3, 5, or 15 ppm) in the diets of growing swine on performance and tissue Se concentrations. Ninety pigs averaging 12.5 kg of BW were given ad libitum access to corn-soybean meal diets fortified with one of the treatment Se sources and dietary levels for a 35-d experimental period. Growth and feed intake were similar in pigs fed .3 and 5 ppm of Se but were lower (P < .01) in those fed 15 ppm from either Se source. Serum Se increased (P < .01) as dietary Se level increased with no difference between Se sources at each dietary Se level. Liver, kidney, and longissimus muscle Se concentrations increased (P < .01) as the dietary level of Se increased and were similar when either Se source was provided. These results indicate that calcium selenite was as effective as sodium selenite using the measurement criteria of growth, serum, and tissue Se concentrations and glutathione peroxidase activities of growing swine when fed at approved, marginally toxic, and toxic dietary Se levels.

Key Words: Pigs, Selenium, Selenosis

Introduction

Both inorganic sodium selenite and selenate are approved by the Food and Drug Administration (FDA, 1987) for inclusion in the diets of farm animals. The selenite form is, however, more commonly used because of its lower relative cost. Although the contribution of indigenous Se from grain sources to livestock diets is not regulated by FDA, other sources of inorganic Se salts have not been approved and cannot be incorporated into diet formulations. It has been reported that both sodium salt forms of Se (i.e., selenite and selenate) are of potential danger to humans because of their rapid water solubility when in direct contact with skin and mucous membranes; hence, toxic responses might occur (Echevarria et al., 1988). Calcium selenite is also considered toxic if it is inhaled or swallowed or when it comes into contact with the skin, but its solubility is less rapid than that of sodium selenite and thus presents less immediate danger to humans.

The amount of research conducted with sodium selenite has been extensive, but the efficacy of this sodium salt compared to that of

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other inorganic Se forms has been studied much less. Research with young chicks and lambs has demonstrated similar Se bioavailabilities when calcium or sodium selenite have been fed during both short (< 10 d) and long (40 or 80 d) time periods (Echevarria et al., 1988; Henry et al., 1988; Tarla et al., 1989).

Because there is no research available comparing sodium selenite and calcium selenite in swine, this study investigated the relative efficacy of sodium selenite and calcium selenite at three different Se levels.

Experimental Procedure

A $2 \times 3$ factorial arrangement of treatments as a randomized complete block design experiment was conducted in three replicates with growing swine to compare the efficacy of two inorganic Se sources, each at three dietary levels, on pig gain, feed intake, and subsequent retention of tissue Se. Reagent grade calcium selenite (47.4% Se) and sodium selenite (45.2% Se) were each added to the diet at .3, 5, or 15 ppm of Se. Supplemental Se was added at the approved (FDA, 1987), marginally toxic, or toxic dietary levels to compare their relative efficacy within the wide range. Cumulative biological responses had been previously ascribed to Se at each of these dietary levels (Meyer et al., 1981; Mahan and Moxon, 1984).

Pigs were weaned at 23 ± 2 d of age and fed a conventional corn-soybean meal mixture fortified with .3 ppm of Se for a 2- to 3-wk period before the start of the trial. Ninety pigs with an average BW of 12.5 kg were allotted by litter, sex, and weight to the dietary treatments in groups of five pigs per pen. Diets were formulated from corn and soybean meal to a dietary lysine level of 1.15% (Table 1). Both Se sources were dissolved in distilled water and added to the treatment diets in appropriate quantities at the time the diet was prepared. Subsequent feed samples that were collected and analyzed for dietary Se indicated that total values ranged from .06 to .09 ppm of Se above the supplemental treatment levels, suggesting that this quantity is the indigenous Se contribution from the basal ingredients. Diets were available ad libitum to the pigs in a five-hole metal feeder for a 35-d experimental period. Pigs were housed in partially slotted, concrete floor nursery pens with a covered hover area (25%).

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TABLE 1. EXPERIMENTAL DIET COMPOSITION

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corn</td>
<td>61.15</td>
</tr>
<tr>
<td>Soybean meal, 44% CP</td>
<td>35.00</td>
</tr>
<tr>
<td>Dicalcium phosphate</td>
<td>1.70</td>
</tr>
<tr>
<td>Limestone</td>
<td>1.20</td>
</tr>
<tr>
<td>Trace-mineral salt mixture</td>
<td>.50</td>
</tr>
<tr>
<td>Vitamin mixture</td>
<td>.20</td>
</tr>
<tr>
<td>Antibacterial agent</td>
<td>.25</td>
</tr>
</tbody>
</table>

The Na or Ca selenite product was solubilized at the appropriate concentration in 100 ml of water and added to the diet at the time of mixing.

aSupplied per kilogram of diet: 8 mg of Cu, 120 mg of Fe, 20 mg of I, 15 mg of Mn, 120 mg of Zn, and 4.22 g of NaCl.

bSupplied per kilogram of diet: 1750 IU of vitamin A, 200 IU of vitamin D$_3$, 11 IU of vitamin E, .5 mg of vitamin K, 3.0 mg of riboflavin, 10 mg of pantothenic acid, 13 mg of niacin, 3 mg of folacin, .05 mg of biotin, 15 mg of vitamin B$_{12}$, .4 g of choline, and 66 mg of BHT.

dSupplied 55 mg of carbadox per kilogram of diet.

and a rubber floor mat at one end of the pen. At d 14 and 35 of the trial, all pigs were bled via heart puncture with serum harvested from the centrifuged blood and frozen for later analyses. At d 35 the hooves of each pig were subjectively evaluated and scored from 0 to 5 for hoof lesions by three individuals and averaged. A score of 0 reflected normal hoof development with no lesions; a score of 3 was given when severe lesions or cracking of the hoof wall were present, and a score of 5 was awarded when severe lesions and complete separation of the hoof from the foot were evident. Three pigs from each pen were then randomly selected and killed by electrocution; samples of liver, longissimus muscle, and kidney were collected, frozen, and saved for later analyses.

Selenium content of diets and tissues was determined after wet-ashing in perchloric and nitric acids by the fluorometric method of Koh and Bensen (1983). Serum and liver Se-dependent glutathione peroxidase (GSH-Px) activities were analyzed by the method of Lawrence and Burk (1976). Serum α-tocopherol was analyzed by HPLC as outlined by Cort et al. (1983) following the sample preparation procedure of Hatan and Kayden (1979). Performance data for the 35-d trial used pen means as the experimental unit and were analyzed by the least squares method of Harvey (1987). Bleeding time was incorporated into the statistical model for appropriate
serum measurements. Single df comparisons were contrasted for the two Se sources and regression analyses with unequal treatment spacing adjustment increments were used to test dietary Se level.

Results and Discussion

The treatment effects of dietary Se sources at each Se level are presented in Table 2. Daily gains, feed intakes, and gain:feed ratios were similar for each Se source within dietary Se level. No difference in these performance measurements occurred between .3 and 5 ppm of Se, but when the 15 ppm of Se dietary level was provided, pig weight gains, feed intakes, and gain:feed ratio declined ($P < .01$) when Se from either source was provided. Previous research indicated that supplemental dietary Se levels from 0 to 5 ppm did not result in lowered weight gains or feed intakes during the postweaning period, but a dietary level of 15 ppm of Se resulted in poorer gain and feed performance responses (Mahan and Moxon, 1984), an effect consistent with the data of this trial.

There was an increase in hoof lesion score ($P < .01$) as the dietary Se level increased, the effect being similar for each Se source. No effect on hoof lesions occurred when either selenite source was provided at .3 ppm, and the effect was only slightly evident when the diet containing 5.0 ppm of Se was fed. When either calcium or sodium selenite source was provided at 15 ppm, hooves exhibited severe cracking and separation at the coronary band, results similar to the selenosis observations reported by Moxon (1937), Miller and Schoenig (1938), and Mahan and Moxon (1984).

Serum GSH-Px activity was not influenced by Se source. Higher GSH-Px enzymatic activity resulted as the dietary level of Se increased from .3 to 15 ppm, significant ($P < .05$) only at the 35-d period. Serum Se at both 14 and 35 d increased ($P < .01$) as the dietary level of Se increased. Within each dietary Se

| TABLE 2. TREATMENT EFFECT OF SODIUM-SELENITE OR CALCIUM-SELENITE AT THREE SUPPLEMENTAL DIETARY LEVELS OF SELENIUM ON SWINE PERFORMANCE AND TISSUE RESPONSES |
|----------------|-----------------|-----------------|
| **Item**       | **Na**          | **Ca**          | **Na**          |
|                 | **5.0**         | **15.0**        | **SE**          |
| Weight, kg      |                 |                 |                 |
| Initial         | 12.5            | 12.5            | 12.6            | 12.6            |
| Final           | 33.2            | 32.8            | 31.5            | 31.5            |
| Daily gain, g   | 574             | 562             | 545             | 393             |
| Daily feed intake, g | 1,165          | 1,123           | 1,070           | 1,151           |
| Gain:feed ratio | .493            | .500            | .509            | .468            |
| Feet lesion score | .05            | .05             | .13             | .25             |
| Serum concentrations GSH-Px, units/ml$^-^c$ | .625 | .652 | .625 | .652 |
| 14-Day          | .515            | .580            | .669            | .603            |
| 35-Day          | .648            | .603            | .602            | .697            |
| Se, ppm         | .110            | .099            | .428            | .406            |
| 14-Day          | .175            | .173            | .646            | .646            |
| 35-Day          | .235            | .240            | .230            | .265            |
| α-Tocopherol, μg/ml | .220          | .230            | .265            | .260            |
| Liver GSH-Px, units/g$^-^c$ | 1.28          | 1.231           | 1.134           | 1.217           |
| Tissue Se, ppm  | .600            | .652            | .625            | .652            |
| Liver           | 623             | 657             | 4.275           | 4.038           |
| Kidney          | 1.653           | 1.853           | 3.144           | 3.133           |
| Longissimus muscle | .285         | .241            | .443            | .347            |

$^a$ Dietary Se level response ($P < .01$).

$^b$ Feet lesion scores ranged from 0 to 5; 0 = no lesions and 5 = severe separation of the hoof at the corneal band.

$^c$ GSH-Px = glutathione peroxidase. One unit equals 1 μmol of reduced nicotinamide adenine dinucleotide phosphate (NADPH) oxidized/min.

$^d$ Dietary Se level response ($P < .05$).
level the responses were similar for the two Se sources. There was a serum GSH-Px activity ×
time interaction (P < .01) and serum Se concentration × time interaction (P < .01) from
the 14- to the 35-d measurement period. Both serum GSH-Px activity and serum Se in-
creased to higher levels as the dietary Se level increased. Although serum α-tocopherol was
higher when calcium rather than when sodium selenite was provided and as the dietary Se
level increased, these responses were not significant (P > .15).

Liver GSH-Px activity was similar for both Se sources at the various dietary Se levels. Liver,
kidney, and longissimus muscle tissue Se concentrations increased (P < .01) as the dietary Se
level increased, but within each tissue the Se values were similar for both Se sources. Kidney
had the highest Se concentration, followed by the liver and longissimus muscle tissue, when .3
ppm of Se was supplemented, whereas when the 5 and 15 ppm of Se levels were fed the liver
increased in Se concentration and surpassed kidney Se content. Longissimus muscle Se con-
centration also increased as the dietary Se level increased, but the greatest increase occurred
between the .3 to 5 ppm of Se level rather than between the 5- to 15-ppm level.

These results suggest that tissue retention and thus the bioavailability of Se from either
calcium or sodium selenite was similar for growing swine, and that both selenite sources
were equally effective in influencing pig growth and feed intake at each of the three
dietary Se levels evaluated.

Implications

Sodium selenite and selenate are currently the only approved inorganic forms of Se that
can be added to livestock diets. Calcium selenite was demonstrated to be as effective as
sodium selenite when evaluated by perform-
ance, glutathione peroxidase activity, and

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