

Supplementation of organic and inorganic selenium to diets using grains grown in various regions of the United States with differing natural Se concentrations and fed to grower–finisher swine^{1,2,3}

D. C. Mahan,^{*4} M. Azain,^{*} T. D. Crenshaw,^{*} G. L. Cromwell,^{*} C. R. Dove,[†]
S. W. Kim,^{*} M. D. Lindemann,[†] P. S. Miller,^{*} J. E. Pettigrew,^{*} H. H. Stein,^{*} and E. van Heugten[†]

^{*}North Central Coordinating Committee on Swine Nutrition (NCCC-42); and [†]Southern Regional Committee on Nutritional Systems for Swine to Increase Reproductive Efficiency (S-1022)

ABSTRACT: Grains grown in various regions of the United States vary in their innate or natural Se contents. A regional study evaluated the effects of adding inorganic Se (sodium selenite) or organic Se (Se yeast) to diets with differing innate Se contents. A 2 × 2 + 1 factorial evaluating 2 Se sources (organic or inorganic) at 2 Se levels (0.15 or 0.30 mg/kg) in 18 total replicates ($n = 360$ total pigs). A basal diet was fed without supplemental Se and served as the negative (basal) control. The study was conducted as a randomized complete block design in 9 states (Georgia, Illinois, Kentucky, Nebraska, North Carolina, Ohio, South Dakota, Texas, and Wisconsin) with each station conducting 2 replicates. Pigs were fed from 25 to approximately 115 kg BW. Similar dietary formulations were used at each station, incorporating a common source of trace mineral and Se premixes. Three pigs per treatment in 16 replicates ($n = 240$) were bled at 55, 85, and 115 kg BW and serum Se and glutathione peroxidase (GSH-Px) activities were determined. Three pigs ($n = 260$) from each treatment pen were killed at 115 kg BW and issues (liver, loin, and hair) were analyzed for Se. The corn Se content from the various states ranged from 0.026 to 0.283 mg Se/kg while the soybean meal Se

content ranged from 0.086 to 0.798 mg Se/kg. Tissue and serum Se concentrations were greater ($P < 0.01$) when supplemental organic Se was fed, whereas serum GSH-Px was greater ($P < 0.01$) as Se level increased. There were linear increases ($P < 0.01$) in loin and quadratic increases ($P < 0.01$) in liver and hair Se concentrations as dietary Se level increased within each state. There was a source × level interaction ($P < 0.01$) for each tissue resulting in a greater increase when organic Se was fed. Serum Se and GSH-Px activity increased ($P < 0.01$) when both Se sources were fed and plateaued at each state at 0.15 mg Se/kg. There was a high and significant correlation between each tissue Se, serum Se, and GSH-Px activity to dietary Se level indicating that those states having greater grain natural Se contents also had greater tissue Se concentrations. These results indicate that a large difference in corn and soybean meal Se concentrations exists between states, that the addition of organic or inorganic Se to these grains increased tissue and serum Se in each state, and that organic Se was incorporated at greater concentrations in the loin, liver, and hair tissues of grower–finisher pigs than inorganic Se.

Key words: corn, minerals, pigs, selenium, state

© 2014 American Society of Animal Science. All rights reserved.

J. Anim. Sci. 2014.92:4991–4997
doi:10.2527/jas2014-7735

¹Salaries and research support were provided by state and federal funds appropriated to the various universities involved in the collaborative project.

²Appreciation is expressed to Alltech Biotechnology, Inc. (Nicholasville, KY), for supplying the Sel Plex and partial financial support for this project, to M. R. Watts for the Se analysis, and to B. L. Bishop for statistical analysis.

³Authors and affiliation at the time of the study: DCM, The Ohio State University, Columbus; MA University of Georgia, Athens; TDC,

University of Wisconsin, Madison; SWK, Texas Tech University, Lubbock; JEP, University of Illinois, Urbana; MDL, University of Kentucky, Lexington; HHS, University of Illinois, Urbana; PSM, University of Nebraska, Lincoln; and EVH, North Carolina State University, Raleigh.

⁴Corresponding author: mahan.3@osu.edu

Received July 19, 2014.

Accepted September 1, 2014.

INTRODUCTION

Plants do not have a Se requirement but some can accumulate large quantities of Se within their matrix (Oldfield, 1999). Most grains and forages retain Se reflecting their soil conditions (Allaway et al., 1966; Allaway, 1972). Plants grown on dry, well aerated, and alkaline soils seem to absorb more Se, whereas soils under wet conditions are acidic in nature and generally have plants with lower Se contents. The soils in some states have naturally high Se contents, although such states also contain areas with moderate Se concentrations. The majority of Se in most plants is found as selenomethionine in the protein portion of the plant while the remainder is present as other organically bound as a Se metabolites or inorganic Se (Olson et al., 1970; Allaway et al., 1981). The feeding of low Se grains to pigs often results in Se deficiencies (Mahan et al., 1973). Historically, in the United States, those areas with grains of a high Se content are in the western Corn Belt and the upper barley and wheat producing states, whereas the eastern Corn Belt and the East and West Coast produce grains with lower Se contents (Mayland, 1985).

Selenium is required by swine, largely as a component of several antioxidant enzymes located in soft tissue, principally as glutathione peroxidases (**GSH-Px**). Both natural and supplemental Se (organic and inorganic) contribute to the production of these enzymes. Under deficient conditions, mortality and morbidity are high (Mahan et al., 1973). As a result, the U.S. Food and Drug Administration initially approved the addition of sodium selenite to pig diets at 0.10 mg/kg diet but this was later amended to 0.30 mg/kg diet (FDA, 1974, 1987). Later, the addition of an organic Se yeast was approved (FDA, 2002) up to a supplemental level of 0.30 mg/kg. In all states, Se can legally be added to a level of 0.30 mg/kg diet regardless of grain Se content. This could produce diets with greater Se levels than required (NRC, 1998) and possibly affect other biological functions. This study examined the effects of adding organic or inorganic Se to corn- and soybean meal-based swine diets differing in their natural organic Se within the regions of the committee members.

MATERIALS AND METHODS

Several members of the regional NCCC-042 and S-1022 swine nutrition committees participated in a study to evaluate the effects of adding inorganic or organic Se at 2 levels to the diets of grower-finisher pigs using corn and soybean meal grown in their region and to measure the subsequent growth responses, tissue Se contents, serum Se, and serum GSH-Px activity at market weight. In each state, the individual scientists followed animal care protocols approved by their institutional animal care committee. The 9 states participating in the study were

Table 1. Percentage composition of basal diets (%; as fed basis)

Ingredient	25 to 55 kg	55 to 85 kg	85 to 115 kg
Corn	69.00	72.70	76.35
Soybean meal, 48% CP	27.00	23.50	20.00
Tallow	1.00	1.00	1.00
Dicalcium phosphate	1.45	1.30	1.10
Limestone	0.90	0.85	0.90
Trace mineral premix ¹	0.05	0.05	0.05
Vitamin premix ²	0.25	0.25	0.25
Inorganic Se ³	±	±	±
Organic Se ³	±	±	±
Salt	0.30	0.30	0.30
Antibiotic ⁴	0.05	0.05	0.05

¹Each station added a common trace mineral premix that provided 4 mg Cu (sulfate)/kg, 0.14 mg I (iodate)/kg, 60 mg Fe (sulfate)/kg, 2 mg Mn (oxide)/kg, and 60 mg Zn (sulfate)/kg to the diets.

²Each station added their own vitamin premix that was typically used at their station. Each premix met or exceeded NRC (1998) requirements.

³Se sources (sodium selenite or selenized yeast) were added at 0, 0.15, or 0.30 mg/kg to treatment diets at the expense of corn.

⁴States used their own antibiotic of choice.

Georgia, Illinois, Kentucky, Nebraska, North Carolina, Ohio, Texas, South Dakota, and Wisconsin.

A basal diet without supplemental Se served as the negative control and used the corn and soybean meal available within their region. Treatment diets were supplemented with 0.15 or 0.30 mg Se/kg from sodium selenite or organic Se (Sel Plex; Alltech Biotechnology Center, Inc., Nicholasville, KY). Consequently, the experiment was a 2 × 2 + 1 factorial arrangement of treatments conducted in a randomized complete block design with 18 replicates. Each state conducted 2 replicates using pigs raised at their location and fed conventional nursery diets fortified with 0.30 mg Se/kg from sodium selenite. Diets were conventional corn-soybean meal formulations that met NRC (1998) nutrient requirements with Se as the experimental variable (Table 1). The diets were fed in 3 phases: from 25 to 55, 55 to 85, and 85 to 115 kg BW. A common trace mineral premix and the Se premixes were prepared at 1 location (The Ohio State University) and sent to each participating scientist. This was done to prevent different mineral interactions from various trace mineral supplementations. The corn and soybean meal used in each state was obtained within the region of the study, or in cases where states normally imported corn or soybean, they used the sources normally fed to grower-finisher pigs within their state. Samples of diets and the grains that made up the diets were saved and later analyzed for Se.

Pigs of crossbreeding (genetics program varied by station) were allotted ($n = 360$ total pigs) to experimental diets on the basis of gender, weight, and litter to 5 treatments at an average weight of approximately 25 kg. The

Table 2. Effect of the innate Se of state feed grains and diets on grower–finisher pig performance, tissue Se, serum Se, and glutathione peroxidase (GSH-Px) activity

Item	State									SEM	P-value	R ^{2,1}
	Georgia	Illinois	Kentucky	Nebraska	North Carolina	Ohio	South Dakota	Texas	Wisconsin			
Feed Se, mg/kg												
Dietary Se, avg. ²	0.057	0.062	0.057	0.333	0.045	0.063	0.355	0.181	0.171	–	–	–
Corn	0.026	0.028	0.017	0.283	0.074	0.044	0.234	0.132	0.075	–	–	–
Soybean meal, 48% CP	0.152	0.176	0.129	0.543	0.086	0.132	0.789	0.365	0.498	–	–	–
No. of replicates	2	2	2	2	2	2	2	2	2	–	–	–
Pig weights, kg												
Initial	24.0	23.4	30.0	25.6	25.7	22.7	26.2	28.9	23.9	0.3	0.01	–
Final	121.5	112.0	124.6	127.3	114.1	106.1	121.9	109.3	107.9	3.2	0.01	–
Tissue Se, mg/kg ³												
No. of pigs	6	6	6	6	6	6	6	6	6	–	–	–
Loin Se	0.130	0.126	0.096	0.345	0.082	0.119	0.527	0.289	0.264	0.017	0.01	0.99 (0.01)
Liver Se	0.489	0.285	0.279	0.687	0.340	0.238	0.722	0.535	0.536	0.022	0.01	0.78 (0.04)
Hair Se	0.456	0.328	0.279	0.256	0.278	0.123	1.022	0.735	0.556	0.037	0.01	0.86 (0.06)
Serum values												
No. of pigs	6	6	6	⁵	6	6	6	6	6	–	–	–
Serum Se (avg.), mg/kg	0.123	0.072	0.087	–	0.119	0.050	0.181	0.162	0.116	0.006	0.01	0.61 (0.01)
Serum GSH-Px (avg.) ⁴	0.872	0.682	0.578	–	0.966	0.457	1.059	0.787	0.774	0.059	0.01	0.27 (0.04)

¹Correlation coefficient (R^2) of the average dietary Se from each station to the average of the collected measurements. Significant P -values are in parenthesis.

²The Se diet analysis is the average within each station across all treatments and phases.

³Tissues collected at harvest.

⁴GSH-Px activity/milliliter = micromoles of GSH-Px oxidized per minute using the molar extinction coefficient of 6.22×10^3 for NADPH.

⁵Samples not collected at this station.

number of pigs per pen was constant within replicate but varied from 3 to 6 between states. Diets were provided ad libitum in meal form throughout the experiment.

Diets were changed at 55 and 85 kg BW with pig gains and feed consumption measured. Three randomly selected pigs from each treatment pen were bled in 16 replicates from the vena cava before diet changes and at the end of the experiment. The blood was placed on ice, centrifuged at the respective laboratory, serum separated, frozen at -20°C , and stored for later analysis. When the study was completed, the samples were packed in dry ice and sent overnight to the Ohio station for determination of GSH-Px activity and serum Se concentration.

At approximately 115 kg BW, 3 pigs per treatment pen ($n = 270$) were harvested either at a local commercial abattoir or at the respective university meat laboratory facility. Before killing, a 3- to 5-g sample of hair was removed along the topline over the shoulder area of each pig and stored for later Se analysis. After pigs were killed, 50- to 100-g samples of loin muscle and liver were collected and frozen (-20°C) for later analysis. Samples were packed in dry ice and sent overnight to the Ohio station for Se analysis.

Laboratory and Statistical Analyses

Diet samples for each phase obtained from each station were pooled in equal proportions and diets, corn, and soybean meal were finely ground (Cyclotec 1093;

Tectator, Höganäs, Sweden) using a 1-mm screen. Hair samples were cleaned in ethanol, rinsed with distilled water, and dried before Se analysis. Serum, hair, liver, and loin tissue were wet ashed in nitric and perchloric acid and analyzed for their Se contents using the fluorometric method (AOAC, 2000). Bovine liver from the National Institute of Standards and Technology was used as the Se standard. Serum GSH-Px activity was determined by spectrophotometry (Spectronic 20+, model 333182; Thermo Electron Corp.) using the procedure of Lawrence and Burk (1976).

The ANOVA was conducted using the GLM procedure of SAS (SAS Inst. Inc., Cary, NC) as a $2 \times 2 + 1$ arrangement of treatments with the basal diet as the negative control. Pen means and the average loin, liver, and hair Se content per pen from pigs killed was the experimental unit for measurement traits. The statistical model included the effects of state, Se source, Se level, blocks (replicates), the source \times level interaction, and the 3-way interaction of state, source, and level with the treatment \times block as the error term. Block was considered random. There were no 3-way interactions and therefore these are not reported. The basal or negative control treatment response was contrasted to the inorganic and organic Se sources and each Se source was analyzed independent of the other treatments to compare the innate Se treatment between the various states. The contrasts used the LSD and incorporated the basal diet with both Se sources and levels. Correlation coefficients (R^2)

Table 3. Main effects of dietary Se source (S) on grower finisher pig performance responses and serum measurements

Item	Se source		SEM	Se level, mg/kg			SEM	P-value ¹		
	Organic	Inorganic		0.00	0.15	0.30		S	L	S × L
No. of pens	36	36	–	18	36	36	–	–	–	–
No. of pigs	144	144	–	72	144	144	–	–	–	–
Pig data										
Initial wt., kg	25.5	25.4	0.1	25.6	25.6	25.4	0.1	0.56	0.16	0.32
Final wt., kg	115.3	115.2	0.8	116.1	115.2	115.3	0.8	0.90	0.92	0.08
ADG, kg	0.92	0.92	0.08	0.93	0.92	0.93	0.08	0.49	0.42	0.07
ADFI, kg	2.55	2.56	0.03	2.57	2.55	2.55	0.02	0.65	0.92	0.32
G:F	0.363	0.364	0.003	0.365	0.362	0.365	0.002	0.82	0.30	0.54
Tissue Se concentration, mg/kg										
Loin Se 0.392		0.240	0.011	0.221 ^a	0.285 ^b	0.340 ^c	0.011	0.01	0.01	0.01
Liver Se 0.681		0.586	0.026	0.456 ^a	0.598 ^b	0.669 ^c	0.026	0.01	0.01	0.01
Hair Se 0.819		0.568	0.018	0.448 ^a	0.634 ^b	0.754 ^c	0.016	0.01	0.01	0.01
Serum Se, mg/L										
Period 1 0.137		0.133	0.002	0.106 ^a	0.130 ^b	0.140 ^c	0.002	0.16	0.01	0.03
Period 2 0.152		0.144	0.002	0.109 ^a	0.143 ^b	0.155 ^c	0.002	0.01	0.01	0.35
Period 3 0.165		0.154	0.002	0.121 ^a	0.156 ^b	0.163 ^c	0.002	0.01	0.01	0.48
Avg. 0.156		0.148	0.002	0.114 ^a	0.147 ^b	0.156 ^c	0.002	0.01	0.01	0.09
Serum GSH-Px activity ^{1,2}										
Period 1	0.783	0.810	0.022	0.721	0.804	0.789	0.020	0.61	0.05	0.50
Period 2	0.877	0.898	0.020	0.786 ^a	0.904 ^b	0.870 ^b	0.020	0.15	0.04	0.35
Period 3	0.873	0.882	0.018	0.833 ^a	0.884 ^b	0.872 ^b	0.018	0.57	0.06	0.12
Avg.	0.817	0.844	0.016	0.768 ^a	0.837 ^b	0.823 ^b	0.016	0.47	0.05	0.44

^{a-c}Se level superscripts that differ within row are significantly different ($P < 0.01$).

¹ P = probability value at the end of each production phase or diet change.

²Glutathione peroxidase (GSH-Px) activity/milliliter = micromoles of GSH-Px oxidized per minute using the molar extinction coefficient of 6.22×10^3 for NADPH.

between stations were conducted between the dietary Se levels to each of the various measurements collected.

RESULTS

Dietary Se analysis of the innate basal treatment is reported in Table 2. The treatment diets supplemented with organic or inorganic Se sources were analyzed and found to be within 10% of calculated experimental limits and are not reported. Supplementing the diets at 0.15 or 0.30 mg/kg did not affect pig ADG, ADFI, or G:F compared to the nonfortified Se basal diet (Table 2).

The main effect of loin, liver, and hair Se concentrations demonstrated that there was a difference in these tissues between Se sources (Table 3). The organic Se source had greater Se concentrations in the tissues measured than inorganic Se treatments ($P < 0.01$). As Se level increased, an increase ($P < 0.01$) occurred in loin, liver, and hair Se concentrations with the basal diet having the lowest Se concentration. A Se source × Se level interaction ($P < 0.01$) resulted for each tissue with the organic Se levels demonstrating a greater increase than when the inorganic Se source was provided.

The main effects of serum Se reported in Table 2 demonstrated that at 55 kg BW there was no difference in serum Se concentrations when either Se source was fed;

however, there was an effect at the end of period 2 (85 kg BW) and period 3 (115 kg BW) when the organic and inorganic Se sources ($P < 0.01$) were fed. As the dietary Se level increased, there was an increase in serum Se ($P < 0.01$) at each level and period. There was a Se source × Se level interaction ($P < 0.03$) in period 1 where the organic Se source was greatest when the level increased, but there were no significant interactions in period 2 and 3.

The main effect of treatments on serum GSH-Px activity from period 1 presented in Table 3 demonstrated no significant Se source response or a source × level interaction. However, as the level of dietary Se increased during periods 2 and 3, there was an increase ($P < 0.01$) in serum Se with no further increase above 0.15 mg Se/kg level. This indicates that a plateau occurs at the 0.15 supplemental Se level.

The treatment responses of tissues from the different states presented in Table 4 demonstrated that Se concentrations of each tissue were the lowest when the basal diet was fed ($P < 0.01$). There were no 3-way interactions for the loin, liver, or hair tissues. When the regression of the basal diet within each of the inorganic or organic Se levels was compared, there was a linear increase ($P < 0.01$) in loin, liver, and hair Se as dietary levels increased for all states but the greatest increase occurred from the addition of organic Se.

The treatment effects on serum Se concentration for the individual states are presented in Table 5. Serum Se

Table 4. Effect of Se source and level on grower–finisher pig tissue responses at each state

Measurement	State	Dietary Se source and level					SEM	P-value		
		Basal (0)	Organic Se, ppm		Inorganic Se, ppm			Regression ¹		
			0.15	0.30	0.15	0.30		Basal vs. the rest	Basal with organic	Basal with inorganic
No. of pigs		54	54	54	54	54				
Loin Se, mg/kg	Georgia	0.130	0.230	0.355	0.138	0.141				
	Illinois	0.126	0.287	0.448	0.159	0.163				
	Kentucky	0.096	0.204	0.328	0.136	0.123				
	Nebraska	0.345	0.445	0.532	0.348	0.349				
	North Carolina	0.082	0.187	0.278	0.093	0.109				
	Ohio	0.119	0.294	0.450	0.148	0.173				
	South Dakota	0.527	0.621	0.690	0.517	0.527				
	Texas	0.289	0.391	0.453	0.296	0.341				
	Wisconsin	0.267	0.358	0.496	0.283	0.268				
	Avg. ²	0.221	0.335	0.448	0.235	0.244	0.019	0.01	0.01 L	0.01 L
Liver Se, mg/kg	Georgia	0.489	0.593	0.671	0.498	0.501				
	Illinois	0.285	0.593	0.738	0.522	0.562				
	Kentucky	0.279	0.571	0.728	0.498	0.550				
	Nebraska	0.678	0.680	0.798	0.619	0.755				
	North Carolina	0.340	0.521	0.614	0.456	0.483				
	Ohio	0.238	0.613	0.660	0.517	0.602				
	South Dakota	0.722	0.887	1.009	0.830	0.862				
	Texas	0.535	0.560	0.650	0.597	0.579				
	Wisconsin	0.536	0.629	0.741	0.576	0.544				
	Avg. ²	0.456	0.627	0.734	0.568	0.604	0.019	0.01	0.01 L	0.01 L
Hair Se, mg/kg	Georgia	0.456	–	–	–	–				
	Illinois	0.328	0.576	0.732	0.426	0.474				
	Kentucky	0.279	0.493	0.794	0.451	0.535				
	Nebraska	0.256	1.063	1.332	0.749	0.815				
	North Carolina	0.278	0.526	0.764	0.333	0.491				
	Ohio	0.123	0.251	0.343	0.132	0.136				
	South Dakota	1.022	1.317	1.594	1.076	1.038				
	Texas	0.735	0.787	0.888	0.674	0.681				
	Wisconsin	0.556	0.748	0.890	0.535	0.548				
	Avg. ²	0.448	0.72	0.917	0.547	0.590	0.037	0.01	0.03 Q	0.01 L

¹Regression analysis of each source for level conducted includes the basal diet (L = linear and Q = quadratic responses).

²The average value represents the mean by treatment at each station.

concentrations increased at each station as Se level increased for the overall experimental period ($P < 0.01$). Within individual states, the results demonstrated that those states having the greater innate dietary Se concentrations also had greater serum Se concentrations, but the interactions between states were not significant.

The treatment effects for all states on serum GSH-Px activity resulted in greater responses ($P < 0.01$) when dietary Se levels increased (Table 5). However, as the dietary inorganic Se level increased there was a greater increase ($P < 0.01$) in GSH-Px activity from the basal to the Se treatment diets that tended to increase ($P = 0.09$) when the organic Se source was fed. Those states with greater innate corn, soybean meal, and dietary Se concentrations generally had greater serum GSH-Px activities. The difference between the basal to the 0.15 Se dietary supplemental Se level was greater in those states with the lower

innate grain or dietary Se concentrations but the states effect and their interactions were significant (Table 4).

The effect of the natural innate Se concentration in the corn and soybean meal in the basal diet within each state was evaluated for each measurement. The tissues (loin, liver, and hair) had greater Se concentrations in those states with the greater corn, soybean meal, and dietary Se contents. There was a corresponding high and significant ($P < 0.01$ to <0.06) correlation ($R^2 = 0.78$ to 0.99) for these tissues when compared to the diets fed in those states. This indicates that pigs consuming corn and soybean meal diets with higher innate Se contents have a greater Se tissue and serum Se status and that supplemental Se may not be needed in these diets, at least to the upper limit of 0.30 mg/kg.

When serum Se and serum GSH-Px activities from feeding the basal diets were compared for each state there were significant increases in serum Se ($P < 0.01$) and se-

Table 5. Effect of Se source and level on grower–finisher pig serum Se and glutathione peroxidase (GSH-Px) activity responses at each state

Measurement	Station	Dietary Se source and level, ppm					SEM	P-value		
		Basal	Organic Se		Inorganic Se			Regression ¹		
			0.15	0.30	0.15	0.30		Basal vs. the rest	Basal with organic	Basal with inorganic
No. of pigs		48	48	48	48	48				
Serum Se, mg/L (avg.)	Georgia	0.123	0.148	0.152	0.138	0.154				
	Illinois	0.072	0.116	0.154	0.130	0.140				
	Kentucky	0.087	0.140	0.165	0.143	0.133				
	Nebraska ²	–	–	–	–	–				
	North Carolina	0.119	0.154	0.150	0.142	0.153				
	Ohio	0.050	0.137	0.150	0.132	0.127				
	South Dakota	0.181	0.191	0.207	0.186	0.188				
	Texas	0.162	0.174	0.176	0.170	0.182				
	Wisconsin	0.116	0.128	0.150	0.121	0.126				
	Avg.	0.114	0.149	0.163	0.145	0.150	0.007	0.05 L	0.01 L	0.01 L
Serum GSH-Px activity (avg.) ³	Georgia	0.872	0.821	0.806	0.817	0.828				
	Illinois	0.682	0.705	0.871	0.924	0.919				
	Kentucky	0.578	0.796	0.816	0.835	0.741				
	Nebraska ²	–	–	–	–	–				
	North Carolina	0.966	0.974	0.804	0.944	0.834				
	Ohio	0.457	0.863	0.823	0.839	0.743				
	South Dakota	1.059	1.117	1.060	1.017	1.053				
	Texas	0.787	0.609	0.513	0.603	0.775				
	Wisconsin	0.774	0.708	0.732	0.769	0.864				
	Avg.	0.772	0.824	0.803	0.844	0.845	0.052	0.01	0.09 Q	0.01 Q

¹Regression analysis of each source for level conducted includes the basal diet (L = linear and Q = quadratic responses).

²Samples were not collected from this station.

³GSH-Px activity/milliliter = micromoles of GSH-Px oxidized per minute using the molar extinction coefficient of 6.22×10^3 for NADPH.

rum GSH-Px activities ($P < 0.01$) within states with the greater Se and GSH-Px activities from states that had the greater innate grain or dietary Se contents. These combined results indicate that pigs consuming corn and soybean meal diets with higher innate Se contents would have a greater Se tissue and serum Se status and that supplemental Se may not be needed in the diets. Organic Se seems to be more responsive in tissue deposition and may be beneficial in increasing Se intake in humans.

DISCUSSION

Historically, it has been clearly recognized that grains in certain regions of the United States have lower Se concentrations while other regions have greater Se contents (Allaway, 1972; Cromwell et al., 1999). The results of Ku et al. (1972) demonstrated a high correlation of dietary Se to loin Se content where grains of high Se content had greater muscle Se concentrations. This was corroborated by a later regional study where samples of tissue were collected from various regions of the United States that varied in their natural innate grain Se content but where the diets also had been supplemental with dietary Se (Mahan et al., 2005). The results of Mateo et al. (2007) would concur in

that tissue and serum Se increased even when the innate Se level was moderate. The results of the current experiment where supplemental Se from an inorganic or organic Se source was added to grains with differing innate Se contents also responded to Se sources and levels. Our results are consistent with previous reports in that regional differences exist in the retention of Se by pigs based on the innate grain Se content fed and that supplementing the diet with additional Se from either inorganic or organic Se further increased tissue Se concentrations. In addition, it confirms that feeding organic Se, largely in the form of selenomethionine, increased tissue Se more than inorganic Se and is more greatly retained by body muscle, liver, and hair in greater concentrations (Mahan et al., 1996, 1999; Kim and Mahan, 2001). This greater tissue retention is attributed to the organic Se in the yeast product containing selenomethionine and that this form of Se can directly replace methionine in muscle or hair protein, whereas such is not the case when sodium selenite was the dietary Se source.

Our primary objective was to evaluate the effect of 2 Se sources when added at 50% or at the upper approved level of Se in the diets of grower–finisher pigs using corn and soybean meal mixtures but at different locations. Diet

therefore differed in their Se contents based on innate Se in the grains. We evaluated the resulting tissue Se contents to see if additional Se from either form or its level would affect pig tissue Se contents. There was a clear difference from those states having higher grain Se content that resulted in greater serum, loin, liver, and hair Se concentrations and greater GSH-Px activities. Although we cannot extrapolate beyond the production phases evaluated, our results indicate that feeding corn or soybean meal with greater innate Se contents would not hinder the performance of pigs to market weight, but it would result in greater tissue and serum Se concentrations, even when 0.30 mg/kg Se was supplemented. When supplemental Se was added, particularly from organic Se, loin and liver Se concentrations continued to increase in their Se concentration. This also indicates that we had not saturated the mechanism for Se deposition in grower–finisher pigs and chronic Se toxicity did not occur. There has been interest in the long-term effects of feeding diets of high innate Se content and this experiment does not support that issue. In contrast, those states with low grain Se content seemed to benefit more from supplemental Se with greater serum Se contents and GSH-Px activities and greater body stores of Se. Organic Se enhanced the retention of Se more than sodium selenite in all regions of the United States, not just those in regions of low grain Se content. These results indicate that adding Se to diets from different regions of the United States with differing naturally bound Se were not detrimental to the growing pig.

LITERATURE CITED

- Allaway, W. H. 1972. An overview of distribution patterns of trace elements in soils and plants. *Ann. N. Y. Acad. Sci.* 199:17–25.
- Allaway, W. H., E. E. Cary, and C. F. Ehlig. 1981. The cycling of low levels of selenium in soils, plants and animals. In: O. H. Muth, editor, *Selenium in biomedicine*. AVI Publishing, Westport, CT. p. 273.
- Allaway, W. H., D. P. Moore, J. E. Oldfield, and O. H. Muth. 1966. Movement of physiological levels of selenium from soils, through plants, to animals. *J. Nutr.* 88:411–418.
- AOAC. 2000. *Official methods of analysis*. 17th ed. AOAC International, Arlington, VA.
- Cromwell, G. L., C. C. Calvert, T. R. Cline, J. D. Crenshaw, T. D. Crenshaw, R. A. Easter, R. C. Ewan, C. R. Hamilton, G. M. Hill, A. J. Lewis, D. C. Mahan, E. R. Miller, J. L. Nelssen, J. E. Pettigrew, L. F. Tribble, T. L. Veum, and J. T. Yen. 1999. Variability among sources and laboratories in nutrient analyses of corn and soybean meal. *J. Anim. Sci.* 77:3262–3273.
- Kim, Y. Y., and D. C. Mahan. 2001. Effect of dietary selenium source, level, and pig hair color on various selenium indices. *J. Anim. Sci.* 79:949–955.
- Ku, P. K., W. T. Ely, A. W. Groce, and D. E. Ullrey. 1972. Natural dietary selenium, alpha-tocopherol and effect on tissue selenium. *J. Anim. Sci.* 34:208–211.
- Lawrence, R. A., and R. P. Burk. 1976. Glutathione peroxidase activity in selenium deficient rat liver. *Biochem. Biophys. Res. Commun.* 71:952–958.
- Mahan, D. C., J. H. Brendemuhl, S. D. Carter, L. I. Chiba, T. D. Crenshaw, G. L. Cromwell, C. R. Dove, A. F. Harper, G. M. Hill, G. R. Hollis, S. W. Kim, M. D. Lindemann, C. V. Maxwell, P. S. Miller, J. L. Nelssen, B. T. Richert, L. L. Southern, T. S. Stahly, H. H. Stein, E. van Heugten, and J. T. Yen. 2005. Comparison of dietary selenium fed to grower–finisher pigs from various regions of the United States on resulting tissue Se and loin mineral concentrations. *J. Anim. Sci.* 83:852–857.
- Mahan, D. C., T. R. Cline, and B. Richert. 1999. Effects of dietary levels of selenium-enriched yeast and sodium selenite as selenium sources fed to grower–finisher pigs on resulting performance, tissue selenium, serum glutathione peroxidase activity, carcass characteristics, and loin quality. *J. Anim. Sci.* 77:2172–2179.
- Mahan, D. C., J. E. Jones, J. H. Cline, R. F. Cross, H. S. Teague, and A. Grifo Jr. 1973. Efficacy of selenium and vitamin E injections in the prevention of white muscle disease in young swine. *J. Anim. Sci.* 36:1104–1108.
- Mahan, D. C., and N. A. Parrett. 1996. Evaluating the efficacy of selenium-enriched yeast and sodium selenite on tissue selenium retention and serum glutathione peroxidase activity in grower and finisher swine. *J. Anim. Sci.* 74:2967–2974.
- Mateo, R. D., J. E. Spallholz, R. Elder, I. Yoon, and S. W. Kim. 2007. Efficacy of dietary selenium sources on growth and carcass characteristics of growing–finishing pigs fed diets containing high endogenous selenium. *J. Anim. Sci.* 85:1177–1183.
- Mayland, H. F. 1985. Selenium in soils and plants. In: *Selenium responsive diseases in food animals: Proceedings of a symposium, held at the Western States Veterinary Conference, February 18, 1985, Las Vegas, Nevada*. Veterinary Learning Systems Co., Inc., Princeton Junction, NJ. p. 5–10.
- NRC. 1998. *Nutrient requirements of swine*. 10th ed. National Academy Press, Washington, DC.
- Oldfield, J. E. 1999. *Selenium world atlas*. Selenium-Tellurium Development Association, Grimbergen, Belgium.
- Olson, O. E., E. J. Novacek, E. I. Whitehead, and I. S. Palmer. 1970. Investigations of selenium in wheat. *Phytochemistry* 9:1181–1188.
- U.S. Food and Drug Administration (FDA). 1974. *Food additives permitted in feed and drinking water of animals*. Selenium. Fed. Regist. 39:1355. U.S. FDA, Rockville, MD.
- U.S. Food and Drug Administration (FDA). 1987. *Correction Food additives permitted in feed and drinking water of animals*. Selenium. Fed. Regist. 52:21001. U.S. FDA Rockville, MD.
- U.S. Food and Drug Administration (FDA). 2002. *Food additives permitted in feed and drinking water of animals; selenium yeast*. Fed. Register (no. 137/July 17) 67:46850.