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EFFECT OF VARYING DIETARY SELENIUM LEVELS ON TISSUE COMPOSITION, BLOOD COMPOSITION AND PERFORMANCE OF GROWING SWINE FED SELENIFEROUS GRAINS

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It is established that selenium is an essential micronutrient as well as a natural toxicant for domestic livestock. However, reports of selenium toxicosis in swine are limited and not well documented. The level at which selenium becomes toxic to swine is thought to be about 8 ppm. This value was derived from the initial selenium research of the 1930's, with considerable extrapolation from other species used in this determination.

Since that time, diet composition has become much more complex, nutrient level of diets has increased and feed additives are commonly used. It is not known what effect these factors or other nutritional interrelationships may have on the level at which selenium becomes toxic. Due to the variability of selenium content in feedstuffs and because selenium is now approved as a feed additive, it is important to better define the level at which selenium becomes toxic to swine.

This research was conducted to determine the effect of varying dietary selenium levels on tissue and blood composition and performance of growing swine fed seleniferous grains.

Experimental Procedure

Twenty-four weaned crossbred pigs initially averaging 17.6 lb were individually fed in a 6-wk growth trial. Three barrows and three gilts were allotted to each of four dietary selenium levels. Dietary treatments I through IV were formulated to contain 0, 3, 6 or 9 parts per million (ppm) of selenium, respectively, as organic selenium. The 21% protein diets were composed of corn, wheat, oats and soybean meal. In diets II, III and IV, wheat and oats were replaced with seleniferous wheat and oats at levels to obtain the desired dietary selenium levels. Dietary composition is shown in table 1.

Results

The effect of selenium level on pig performance is shown in table 2. The level of dietary selenium had no effect on pig performance. Average daily gain and average daily feed intake numerically decreased with added selenium levels, but these differences were not significant. Feed conversion was not affected by dietary treatment.

Selenium concentration in the liver, kidney, heart, spleen, diaphragm muscle and hair increased linearly with increasing level of dietary selenium (table 3). In a similar experiment, it was found that generally pigs with toxicosis had greater concentrations of selenium in liver than in kidney and those not developing toxicosis had greater concentrations of selenium in

Table 1. Percent Composition of Diets

Diet	I	II	III	IV
Selenium level				
Calculated (ppm)	0	3	6	9
Actual analysis (ppm)	.47	2.58	5.60	8.40
Seleniferous wheat	--	12	24	35
Control wheat	35	23	11	--
Seleniferous oats	--	6	13	20
Control oats	20	14	7	--
Control corn	17	17	17	17
Soybean meal	25	25	25	25
Dicalcium phosphate	1.6	1.6	1.6	1.6
Limestone	.9	.9	.9	.9
Trace mineralized salt	.3	.3	.3	.3
Vitamin antibiotic premix[a]	.2	.2	.2	.2

[a] Supplied per lb of diet: vitamin A, 1818 IU; vitamin D, 182 IU; vitamin E, 4 IU; vitamin K, 1.6 mg; riboflavin, 2 mg; pantothenic acid, 8 mg; niacin, 12.8 mg; choline, 80 mg; vitamin B_{12}, 8 mcg; sulfamethazine, 50 mg; selenium, 72 mcg; aureomycin, 50 mg, and penicillin, 25 milligrams.

Table 2. Effect of Selenium Level on Pig Performance[a,b]

	Selenium level (ppm)			
	0	3	6	9
Avg daily gain	1.32	1.30	1.30	1.19
Avg daily feed	2.40	2.31	2.31	2.16
Feed/gain	1.79	1.78	1.77	1.80

[a] All weights in pounds.
[b] Six individually-fed pigs per treatment, average initial wt, 17.6 lb.

Table 3. Effect of Selenium Level on Selenium Content of Organs and Tissue[a]

	Selenium level (ppm)			
	0	3	6	9
Liver[b]	.82	3.17	5.12	6.79
Kidney[b]	1.72	3.44	5.12	6.62
Heart[b]	.43	1.85	2.88	4.10
Spleen[b]	.53	1.51	2.54	3.49
Muscle[b]	.32	1.63	2.78	3.72
Hair[b]	.80	4.07	8.53	10.51
Liver (% body wt)[b]	2.51	2.65	2.73	2.94

[a] Parts per million selenium, wet basis.
[b] Linear effect ($P<.01$).

kidney than in liver, indicating our level of approximately 9 ppm may have been approaching the toxic level. It was noted that within a treatment black hair gave a higher selenium value than did white or red hair. Depending on hair color, the selenium level ranged from 2.59 ppm (white) to 16.48 ppm (black) when feeding diet III and 4.04 ppm (red) to 17.80 ppm (black) when feeding diet IV.

Other researchers have found that the liver becomes necrotic and degenerates due to selenium poisoning. In this experiment rather than degenerating as expected, the liver/body weight ratio was increased with increasing selenium level (table 3). This enlargement may be an indication of the attempt by the animal to adapt to a foreign compound.

The effect of selenium level on blood composition is shown in table 4. The selenium levels of the blood increased linearly with increasing selenium level. Other researchers have found that animals may become anemic due to selenium toxicosis. The Packed Cell Volume (PCV) and hemoglobin levels of the blood may be used as an indication of anemia. In our experiment, high levels of dietary selenium did not produce an anemic condition as indicated by the PCV and hemoglobin level, which did not differ among treatments. An increase in serum bilirubin, the major end product of hemoglobin decomposition, may be used as an indication of liver damage. The low levels of bilirubin present suggest no liver damage in these animals.

The effect of selenium level on blood enzyme activity is shown in table 5. Certain enzymes are indicative of either the selenium status of the body or of tissue damage. Because selenium is an essential component of the enzyme glutathione peroxidase (GSH-PX), the level of this enzyme might be expected to increase with increasing selenium level. However, there was no effect on cellular or serum glutathione peroxidase in this experiment. An increase in serum glutamic oxalacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT) is used as an indication of liver damage. However, the SGOT and SGPT levels were not different, indicating no liver damage at any of the selenium levels fed in this experiment.

Table 4. Effect of Selenium Level on Blood Composition

	Selenium level (ppm)			
	0	3	6	9
Selenium, ppm[a]	.24	.88	1.79	2.42
Packed Cell Volume	42.0	43.0	42.0	42.0
Hemoglobin, g/100 ml	13.2	13.5	13.3	13.3
Serum bilirubin, mg/100 ml	.22	.21	.22	.33

[a] Linear effect ($P<.01$).

Table 5. Effect of Selenium Level on Blood Enzyme Activity[a]

	Selenium level (ppm)			
	0	3	6	9
GSH-Px cells				
EU/mg protein	27.1	30.7	24.7	27.4
EU/mg hemoglobin	41.5	48.6	36.6	40.6
GSH-Px serum,				
EU/mg protein	7.4	10.6	9.5	10.7
SGOT, S-F units/ml	44.7	52.8	46.3	60.5
SGPT, S-F units/ml	20.4	24.9	25.2	24.2

[a] Glutathione peroxidase (GSH-Px), serum glutamic oxalacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT).

Summary

A 6-week growth trial was conducted to determine the effect of varying selenium levels on tissue and blood composition and performance of growing swine fed seleniferous grains.

Selenium levels of .47, 2.58, 5.6 or 8.4 ppm had no effect on pig performance as measured by average daily gain, average daily feed or feed/gain. Blood composition and enzyme activity were not affected by dietary treatment. The selenium concentrations of the blood, hair, liver, kidney, heart, spleen and diaphragm muscle were linearly increased by dietary treatment. In addition, the liver/body weight ratio was linearly increased by dietary treatment. No signs of selenium toxicity were noted. Thus, the toxic level of selenium in diets containing seleniferous ingredients appears to be greater than 8.4 ppm.