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I am submitting herewith a thesis written by Douglas Farmer Ellis entitled "Effect of Supplemental Dietary Selenium on Growing-Finishing Pig Performance and Hepatic and Erythrocyte Glutathione Peroxidase Activity." I recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science, with a major in Animal Science.

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Thesis
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EFFECT OF SUPPLEMENTAL DIETARY SELENIUM ON GROWING-FINISHING PIG PERFORMANCE AND HEPATIC AND ERYTHROCYTE GLUTATHIONE PEROXIDASE ACTIVITY

A Thesis

Presented for the

Master of Science

Degree

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Douglas Farmer Ellis

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ABSTRACT

Ninety-eight Yorkshire x Duroc pigs were utilized in this study to determine the effect of six supplemental dietary levels of selenium on growing-finishing pig performance and erythrocyte and hepatic glutathione peroxidase levels in the pig. The pigs were fed a basal corn-soybean meal ration containing approximately 50ppb natural selenium or the basal ration supplemented with 25, 50, 75, 100, 125 or 150 ppb Se as sodium selenite.

The pigs were weighed biweekly, feed was measured and performance data were calculated every two weeks during the trial. Blood samples were collected from eight pigs on each treatment after 70 days on experiment and at slaughter. Hemoglobin and percent packed cell volumes were determined at the time the blood samples were collected. Erythrocytes from all blood samples were prepared by washing twice with sodium-phosphate-saline buffer and then frozen at -20°C until they were analyzed for glutathione peroxidase activity. Liver samples were collected at slaughter and were stored frozen until the analysis for glutathione peroxidase could be conducted.

The results of this study indicate that there were no significant differences due to treatment on the performance criteria or carcass data that were measured in this study. Erythrocyte glutathione and enzyme units of activity were not significantly affected by treatment at 70 days of this experiment. However, pigs receiving the basal diet without any added selenium had noticeably lower levels of glutathione and enzyme units of activity on the seventieth day of the experiment. This finding suggests

that the basal control ration was possibly marginal to deficient in selenium and that the supplemental levels overcame the marginal deficiency. Erythrocyte glutathione peroxidase enzyme units of activity and enzyme units of activity per milligram of hemoglobin at slaughter were significantly ($P<.05$) affected by treatment. These differences were not linearly increased with increasing level of supplemental Se. No consistent trend was observed and the randomness of the values may be partially explained by the hypothesis of Mahan *et al.* (1977) who suggested that pigs during the finishing phase may require only 50ppb Se. Since the basal diet in this study contained approximately 50ppb natural Se, the addition of supplemental Se would not be expected to have a consistent linear effect on glutathione peroxidase activity.

No significant differences due to treatment in hepatic glutathione peroxidase were observed in this study. The fact that pigs utilized in this study were not depleted of their Se reserves before the experiment was initiated and the hypothesis of Mahan *et al.* (1977) may explain partially the results obtained for hepatic glutathione peroxidase in this study.

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CHAPTER I

INTRODUCTION

Selenium (Se) was considered for many years to be a toxic element. The concept of selenium toxicity was generally accepted as its only role in nutrition until in 1957, Schwarz and Foltz (1957) showed that sodium selenite prevented liver necrosis in rats fed torula yeast diets. Eggert *et al.* (1957) and Grant and Thafvelin (1958) demonstrated a relationship between selenium deficiency and hepatosis dietetica in swine.

Field cases of selenium and/or vitamin E deficiency were first observed in swine in this country in the late 1960's. Michel *et al.*(1969) were the first to describe the occurrence of the disease in commercial swine herds in Michigan on practical corn-soybean meal type diets. Trapp *et al.* (1970) suggested that the condition probably existed prior to the first observed cases in 1967.

In recent years selenium has not only been shown to be essential, but one of its functions has been determined (Rotruck *et al.*,1973 and Flohe *et al.*, 1973). This discovery of a specific function of selenium as a component of glutathione peroxidase in animals has been described by Hoekstra (1974). The researchers have indicated that decreases in glutathione peroxidase appear to explain partially the degenerative diseases induced by selenium deficiency. The discovery of a specific function of selenium as a component of glutathione peroxidase in animals provides a new criteria by which to assess the selenium requirements of swine.

The use of subtoxic additions of Se in the rations of growing-finishing pigs has not been shown to effect performance or carcass characteristics (Groce *et al.*, 1973b; Ku *et al.*, 1973; Ewan *et al.*, 1969; Mahan and Moxon, 1978; Doornenbal, 1975 and Wilkinson *et al.*, 1977).

The effect of supplemental dietary levels of selenium on the activity of the enzyme glutathione peroxidase in swine has not been determined or previously reported. Therefore, the objective of this study was to determine the effect of six supplemental dietary levels of Se (25, 50, 75, 100, 125 and 150 ppb) on erythrocyte and hepatic glutathione peroxidase and the performance of growing-finishing pigs.

CHAPTER II

REVIEW OF LITERATURE

Selenium (Se) was originally thought of as a toxic element, but in recent years it has been shown to be an essential trace element for some species. Early research work conducted with Se concerned its toxic effects. Several states in the midwest had naturally high levels of Se in their feed sources. These high levels resulted in toxicity in the animals consuming these feed sources.

A. EARLY WORK WITH SELENIUM

Some of the work on Se poisoning was done to determine if there was an element or compound to counteract the effects of high levels of natural selenium. Arsanilic acid was one of the compounds used. Wahlstrom and Olson (1959) determined that growing and finishing pigs on naturally high Se diets plus arsanilic acid had a higher average daily gain (ADG) and lower feed efficiency (FE) than pigs receiving the naturally high diet alone. Although there was an improvement with the addition of arsanilic acid, the effects of high levels of natural Se in the diet were not completely removed. The effect of naturally high selenium corn was investigated by Ku *et al.* (1973). Growing and finishing pigs were fed diets in which the corn contained a naturally high level of selenium. The differences in FE and ADG were not significantly different.

II. SELENIUM IN SWINE

Selenium Deficiency on Growth

After Se was discovered as an essential nutrient for body functions, studies were conducted to determine the deficiency signs of Se in different species. Luniha (1969) discovered several possible reasons for Se deficiency: (1) Feed sources obtained from areas deficient in selenium. (2) Deficiencies in vitamin E cause increased need for selenium. (3) Certain strains and/or breeds of swine may have higher requirements. (4) Use of crossbred sows to get larger litters and faster growing pigs. (5) The availability of Se in the diet. (6) Reduced feeding of sows during gestation, resulting in decreased Se intake. The relationship of vitamin E and Se seemed to be a very important factor in Se deficiency. Ewan *et al.* (1969) designed two experiments which utilized a basal diet and the basal diet containing Se, vitamin E, or a combination of the two nutrients. The diets were fed to pigs until 56 and 84 days of age on two respective experiments. Pigs fed diets containing no supplemental vitamin E or Se had a very high mortality rate. Pigs fed diets supplemented with vitamin E, Se, or both had growth and FE results that were similar. There was a significant decrease in the mortality rate in the supplemental diets as compared to the diets containing no supplementation. Groce *et al.* (1971) conducted a total of 3 experiments with growing and finishing pigs using combinations of different levels of vitamin E and selenium. Vitamin E and Se supplementation resulted in no significant effects due to the treatments on the FE or ADG of the pigs. Wastell *et al.* (1972) developed a series of diets using either torula

yeast or promosoy as a base. These diets were supplemented with either Se, vitamin E, or both and fed to growing and finishing pigs. Pigs receiving torula yeast diets supplemented with both vitamin E and Se gained significantly faster than those receiving torula yeast diets with only one of the two supplements. The promosoy diets resulted in no significant difference in the pigs' gains among different supplemented diets. Groce *et al.* (1973b) found no significant differences among treatments of Se and/or vitamin E supplementation compared to no supplementation. Growing and finishing pigs were used and different levels of vitamin E and Se were combined to make up the diets. Piper *et al.* (1975) incorporated peas into diets containing different levels of vitamin E and Se or no supplementation. The study was conducted during the growing and finishing phase of the pigs' life cycle and a significant difference in FE and ADG was not observed until the finishing period. The difference was between the unsupplemented diet and the rest of the diets. Glienke and Ewan (1977) studied the effect of vitamin E and Se supplemented torula yeast diets and unsupplemented torula yeast diets on weanling pig performance. The pigs were fed the diets for 6 weeks, with the supplemented diets having significantly improved ADG and FE over the unsupplemented diets.

Selenium Effects on Retention

To determine the degree at which Se was retained by the body of the pig, studies were undertaken to define the Se retention and excretion patterns. Groce *et al.* (1971) found Se excretion in feces and urine increased significantly with increasing levels of Se in the diet. Using

weanling pigs, they observed 20 fold more Se in the urine and 3 fold more in the feces when 0.5 ppm selenium was fed in the diet versus 0.1 ppm. Groce *et al.* (1973a) compared the excretion of Se by pigs consuming diets containing seleniferous corn, supplemental Se and vitamin E. There was significantly more Se excreted in the feces and less through the urine in the pigs fed seleniferous corn. Vitamin E supplementation lowered the excretion of Se in pigs receiving the seleniferous corn diet, but not the selenium supplemented diet. In another study by Groce *et al.* (1973a) it was shown that vitamin E increased the urinary Se excretion by pigs. Hitchcock *et al.* (1978) utilized weanling pigs from sows that had received no supplemental Se during lactation or gestation to conduct a Se retention study. The diets for the pigs contained different levels of Se and arsanilic acid. Selenium supplementation caused a significant increase in Se intake, percentage Se retention and absolute Se retention. This seemed to be due to a significant decrease in selenium excreted in the feces. Arsanilic acid added to the diets resulted in a decrease in the percentage of the fecal Se excretion by pigs receiving diets not supplemented with selenium, but had the opposite effect when pigs consumed diets containing supplemental selenium.

Selenium Effects on Carcass Characteristics

Selenium and vitamin E deficiency or selenium deficiency alone causes certain conditions to occur in the body of a deficient animal. Some of the basic signs are; necrosis of the liver, whitening of muscle fibers, and yellowing of subcutaneous fat. Ewan *et al.* (1969) fed weanling pigs a torula yeast basal diet, or the basal diet supplemented

with Se, vitamin E, or both. Examination of sacrificed pigs on unsupplemented diets revealed yellowing of subcutaneous fat and necrosis of the liver. Hyaline degeneration of the skeletal muscle was observed in 66 percent of the unsupplemented pigs and swollen muscle bundles with a loss of striation were observed in 30 percent of the unsupplemented pigs. Supplemented pigs displayed no deficiency signs. Ewan *et al.* (1969) conducted another test involving two experiments. Weanling pigs were utilized in both experiments. In the first, torula yeast diets either supplemented or not with Se and vitamin E were used. The pigs that were sacrificed at 56 days of age from the unsupplemented diet showed yellow subcutaneous fat, hepatic lobular necrosis and hyaline degeneration of the skeletal muscle. In the second experiment the pigs were on either torula yeast or purified soybean protein base diets supplemented without or with Se and vitamin E. All pigs on unsupplemented diets died before 84 days of age. Upon examination, hemorrhagic necrosis of the liver and paleness in skeletal and cardiac muscle were observed. Groce *et al.* (1971) found that a .16 ppm difference in dietary Se for a group of test rations resulted in a significantly higher level of Se in the muscle of animals on the diets containing elevated levels. Wastell *et al.* (1972) fed growing pigs rations with either a torula yeast or a promosoy base supplemented without or with Se and/or vitamin E. The pigs fed unsupplemented torula yeast diets were found to have liver fibrosis, hyalinization of skeletal muscle and yellow-brown discoloration of body fat after being on test 3 to 4 weeks. The pigs on supplemented diets did not display significant tissue changes. Groce *et al.* (1973b) used a corn-soybean basal diet

supplemented with Se, vitamin E, or both to determine the effect of Se and vitamin E in the ration on organ levels of the two nutrients. They observed rations supplemented with vitamin E caused a significant increase in Se level in the kidney when compared to pigs receiving unsupplemented diets. There was a significant difference in Se levels of the longissmus muscle and liver when the ration Se level varied from 0.1 to 0.15 ppm.

Piper *et al.* (1975) investigated the effectiveness of using peas as a replacement for corn in growing and finishing swine rations. Their diets were designed to contain added Se, vitamin E, or a combination of both to the basal diet. The pigs on the diet containing neither Se not vitamin E were the only ones that showed deficiency signs. Upon examination of the pigs showing deficiency signs; massive necrosis of hepatic lobules, renal lesions, severe cholemic nephrosis and degenerative nutritional cardiac myopathy were observed.

Michel *et al.* (1969) designed a ration scheme in which a basal diet of torula yeast was supplemented with Se, vitamin E, methionine or combinations of different levels of the three. The diet with no supplementation resulted in liver necrosis and fibrosis, ulcerations in the stomach and nutritional myopathy upon examination of the pigs fed the diet. Eggert *et al.* (1957) used a torula yeast basal diet supplemented with Se or vitamin E for growing pigs. Sixty-six percent of the pigs on the unsupplemented diet died. The pigs displayed no outward signs of sickness before death, but upon examination, liver necrosis and yellow discoloration of body fat was found. There were no Se-vitamin E deficiency signs found in the sacrificed pigs on the other two diets.

Selenium Effects on Reproduction

Mahan *et al.* (1974) studied the effect of diets low in Se on sows during the reproductive cycle. During the first parity the sows fed diets containing Se had significantly higher conception rates than did the sows fed semi-purified diets with no Se added. In the second parity none of the sows receiving the semi-purified diet carried their litters to term. Sows that died while on the semi-purified diet displayed definite signs of vitamin E-Se deficiency. Mahan *et al.* (1975) observed that feeding diets containing 3 different Se levels resulted in Se being concentrated most in the kidney at all 3 levels of supplementation. Pigs farrowed from sows on the lowest Se level displayed deficiency lesions and esophagogastric ulcers at 56 days of age. Mahan and Moxon (1976, 1977) studied the transfer of Se from the sow to the prenatal pigs and how it effected the pigs performance. Sow diets contained two levels of Se and one with no Se added. Liver Se levels of pigs in all groups dropped from birth to weanling. This suggested that there was a transfer from the sow, but it was inadequate for the pig.

Diehl *et al.* (1975) administered injections of Se instead of supplementing the feed. All pigs receiving injections of 1.65 mg Se/Kg body weight died within 12 hours after injection. Toxicity symptoms such as vomiting, muscular incoordination, dyspnea and prostration were observed. The pigs not given an injection were still receiving 0.05 ppm Se in the feed, so there was more of a problem with toxicity than deficiency. Levander and Morris (1970) showed vitamin E to elicit the best protection against selenium poisoning if used at high enough levels.

Herigstad *et al.* (1973) utilized basal rations of torula yeast or whole milk powder as the protein source. These diets were supplemented with different levels of Se with the intention of determining toxic levels. Lethal levels were 9.4 mg/Kg for sodium selenite and 13.6 mg/Kg for selenomethionine. Some of the gross toxicosis signs were vomiting, transitory diarrhea, gasping respiration and coma. Histological signs included pulmonary and interstitial edema, congestion of renal medulla and subendocardial ecchymoses.

Niyo *et al.* (1977) studied the effect of giving Se and vitamin E by injection to young pigs, Vitamin E-Se deficiency signs were seen only in pigs receiving no supplemental vitamin E or selenium. Deaths in the unsupplemented group were not observed to occur until after weaning. Mahan *et al.* (1973) allotted 6 week old pigs to diets with supplemental Se, vitamin E, or a combination of the two added to the basal diet. Supplementation was given as an injection. Eighty percent of the pigs receiving no supplementation and 50 percent of those receiving only vitamin E died. These deaths were determined to be related to Se-vitamin E deficiency. In a second study 4 week old pigs were used. The treatments were the same except the level of supplemental vitamin E was increased. The death rate of pigs on only vitamin E supplementation decreased, but so did the death rate for those receiving no supplementation.

Relationships of Se Intake and Carcass Characteristics

Doornenbal (1975) attempted to relate offal Se level at slaughter to a weight group. This was accomplished by slaughtering animals at different weights up to commercial slaughter weight. Their results

revealed no pattern of Se level with increasing weight of the pigs. Wilkinson *et al.* (1977) devised rations in which selenium, copper, zinc, or a combination of two of them were supplemented in the diets. They observed no significant treatment difference for live weight, hot carcass weight, length, carcass weight as a percent of live weight, loin eye and fat thickness. Ku *et al.* (1972) found that Se level contained in pork chops from a variety of herds was highly correlated (r = .95) to the selenium in the feed. Ewan (1971) fed diets supplemented with Se, vitamin E, or a combination of the two supplements. They observed higher Se levels in the kidney and liver of pigs on the Se supplemented diets as compared to the diets not supplemented with selenium. It was also determined that there was a significantly higher calcium level in pigs on the nonsupplemented diets. This was thought to be due to calcification of necrotic areas.

Selenium Effects on Blood Enzymes

Selenium was discovered to be an essential trace mineral for the pig, but its functional location in the body was not known. Studies were undertaken to determine the metabolic function in which Se was involved. Ewan and Wastell (1970) undertook two experiments involving diets fed to weanling pigs with varying levels of Se and/or vitamin E. The pigs on the diets containing no supplementation of either nutrient had increased activity of serum glutamic-oxaloacetic transaminase (SGOT), serum glutamic-pyruvic transaminase (SGPT) and lactic dehydrogenase (LDH) over the other diets. Groce *et al.* (1971) found no significant differences in hemoglobin, hematocrit or SGOT among pigs on diets

containing different levels of Se and/or vitamin E. Watsell *et al.* (1972) used growing and finishing pigs fed different levels of vitamin E and/or Se to determine the effect on SGOT and LDH levels. There was a significantly higher level of both SGOT and LDH activity in the nonsupplemented pigs, indicating tissue damage. Mahan *et al.* (1973) injected different levels of Se and vitamin E into weanling pigs. Pigs not receiving supplemented Se had significantly higher SGOT up until 10 weeks of age, but there was no difference by the fourteenth week. Diehl *et al.* (1975) used weanling pigs to study the effect of different levels of injections of Se on selenium deficiency. The SGOT levels increased in all treatments within 12 hours after injection, but decreased over the test period. The decrease was greater with increased level of the Se injection. Mahan *et al.* (1974) studied the effect of low Se and vitamin E levels on the reproductive performance of sows and the performance of their progeny. The sows diet consisted of a control with low Se, the control with added Se, and a semi-purified diet with very low selenium. The sows were taken through two reproductive cycles on the study. The sows on the semi-purified diet had significantly higher SGOT and SGPT levels than the other two diets. During the second cycle none of the sows on the semi-purified diet carried their litters to term. The pigs from the control fed sows had SGOT levels that were, overall, higher than those from sows fed the supplemented diet. There was a distinct difference in death rate. The pigs from sows on the control had a mortality of 11 to 21 percent whereas none of the pigs died from sows on the supplemented diet. In another test using reproducing sows Mahan *et al.* (1977) used diets containing different levels of Se to

study the effects on the sow during gestation and lactation. They observed a lower concentration of serum Se during lactation as compared to gestation. During lactation the Se level in the milk varied directly with the Se in the feed. The SGOT level of the sow was unaffected by the treatments. The Se level in the livers of sacrified pigs from birth to weaning decreased in all sow treatment groups. This indicated that the mammary transfer of Se to the pig was not adequate to meet the needs of the young pig. Wilkinson *et al.* (1977) found evidence that the lactating sow had a greater demand for Se than the gestating sow.

III. SELENIUM FUNCTION AND UTILIZATION

There was a long time span between the discovery of Se being essential for the pig and the actual function in which Se is thought to be involved. The early name for the Se containing compound involved was factor 3. Schwarz and Foltz (1957) compared inorganic, organic and elemental Se to factor 3 in the ability of the compounds to prevent liver necrosis in rats. None of the compounds had more than 30 percent of the activity of factor 3. Elemental Se was almost inactive, while inorganic Se had improved activity to prevent liver necrosis than organic Se did. Diplock *et al.* (1971) studied the effect of antioxidants on the oxidation state of Se in rat liver. The rats were dosed with ^{75}Se a week before sacrificing. Upon fractionation of liver samples it was determined that the SE in the mitochondria and microsomal portion had a significant increase in selenide and selenite when antioxidants were added. It was concluded that the degree of reduction of selenium was due to the addition of antioxidants during isolation.

Stadtman (1977) listed several Se containing enzymes as being formate dehydrogenase and glycine reductase in bacteria and glutathione peroxidase (GSH-PX) in mammals. Following the discovery that Se was a part of the enzyme glutathione peroxidase (GSH-PX), Flohe *et al.* (1973) determined the Se content of GSH-PX and reported that it had 4 atoms of Se per molecule of enzyme. Rotruck *et al.* (1973) demonstrated that the effects of Se and vitamin E were different. They also showed through a series of tests that Se could be an intregal part of GSH-PX and not a loosely bound cofactor. Lawrence and Burk (1978) collected liver samples from a variety of species of mammals and determined the GSH-PX activity. The GSH-PX activity in the pig was 2.5 ± .05 units per gram of liver. The units were moles of reduced nicotinamide adenine dinucleotide phosphate.

CHAPTER III

METHODS AND PROCEDURES

I. EXPERIMENTAL ANIMALS AND TREATMENT

Ninety-eight Duroc x Yorkshire crossbred pigs were chosen for the study. The pigs had an initial average weight of 26.8 kilograms (Kg) and were marketed at 104.5 Kg. Random allotment was used to obtain 3 gilts and 4 barrows per pen. The study utilized 14 5 X 20 foot pens in the growing-finishing barn at The University of Tennessee Blount Farm. The barn was a pole-type, open to the South year-round and able to be open to the North in the summer. One two-holed self feeder was provided in each pen and the waterer was situated at the lower end of the pen. The solid concreted floor had a slight slope to an outside gutter for drainage purposes. The pens were scraped, to remove fecal material, daily.

Seven rations were utilized in this study with two replications per ration. The composition of the experimental diets is presented in Table 1. All the rations were the same except the six experimental rations had no vitamin E and varying levels of selenium. A vitamin-mineral premix was used that contained no vitamin E or selenium. The selenium was added by used sodium selenite and making a 2.27 kilogram premix with corn to be added to 454.5 Kg of a given ration to provide the appropriate level of selenium for each diet.

Table 1. Percentage Composition of the Basal Diet

Ingredient	International reference no.	Percent
Corn, dent yellow grain, gr 2 mm 54 wt. (4)	4-02-931	-74.9
Soybean	5-04-604	21.5
Calcium phosphate dibasic	6-01-080	1
Limestone	6-02-632	1
Salt		.5
Vitamin-trace mineral premix[a]		.5
Se premix[b]		.5
Antibiotic[c]		.1

[a]Provided the following per Kilogram of diet: Vitamin A, 3,300 Iu; Vitamin D, 660 Iu; riboflavin, 3.3 mg; nicotinic acid, 17.6 mg; d-pantothenic acid, 13.2 mg; choline chloride, 110 mg; Vitamin B_{12}, 19.8 mg; zinc, 74.8; manganese 37.4 mg; iodine, 2.7 mg; copper, 9.9 mg; iron, 59.4 milligrams.

[b]Sodium selenite premixed with finely ground corn to give the added level of Se in experimental diets.

[c]Pfizer TM 50, 50 g/ton.

II. DATA AND SAMPLE COLLECTION

The pigs were weighed and feed consumption was measured every 2 weeks. Blood samples were taken 70 days after initiation of the study and at slaughter. Four pigs from each pen were bled and slaughtered when they reached market weight. The blood was taken by anterior vena cava puncture, with a minimum of 15 ml taken at a bleeding.

III. PROCESSING AND ANALYSIS OF SAMPLES

Hemoglobin was determined by the method of Crosby *et al.* (1954) and microhematocrits were determined by the method of McGovern *et al.* (1955) on heparinized whole blood collected at each bleeding. Whole blood and serum samples were stored at -20°C until further analyses could be performed. Samples of kidney, liver, myocardium, spleen, and skeletal muscle were obtained at slaughter and stored in plastic bags and frozen at -20°C until analyzed for GSH-PX (glutathione H_2O_2 oxido-reductase, EC. 1. 11. 1. 9).

At each bleeding, 0.4ml of blood was washed twice with 4ml of saline-phosphate buffer (Draper and Csallany, 1969) and then centrifuged at 1200 X g for 15 minutes. After removing the buffer, plasma and buffy coat, the remaining cells were frozen at -20°C until analyzed for GSH-PX. The livers were removed from pigs at slaughter, rinsed with deionized distilled water and blotted dry and the wet tissue weight was recorded. Samples of liver (50-100g) were taken from the right medial lobe and stored until the GSH-PX analysis could be conducted.

Livers were homogenized in 4 volumes of 0.15 M KCl, on ice, for 30 seconds at 10,000 rpm using a homogenizer[1] equipped with a PT10 generator.

At the time of analysis of erythrocyte GSH-PX, frozen erythrocytes were hemolyzed with 4ml of water and were allowed to equilibrate to room temperature.

Glutathione Peroxidase Assay

Both erythrocyte hemolysates and liver homogenates were analyzed for GSH-PX activity by a modification of Mill's procedure 2 (1959). The enzyme assay tubes were incubated at 37°C and contained: 1.0ml of 2.0 mM reduced glutathione, 1.0ml of 0.40 M sodium phosphate buffer (pH 7.0) also containing 4 X 10^{-4} M EDTA, 0.50ml of 0.01 m NaN_3 (to inhibit catalase), 0.30ml of 20 percent (w/v) liver homogenate (or a portion of erythrocyte hemolysate containing approximately 2mg of hemoglobin), and water to bring the total volume to 4.0ml. After a 5 minute preincubation, 1.0ml of 1.25 mM H_2O_2 (prewarmed to 37°C) was added. Thereafter, at 3 minute intervals 1.0 ml aliquots of incubation mixture were removed and added to 4.0ml metaphosphoric acid precipitation solution.[2] Glutathione in the protein free filtrate was determined by mixing 2.0ml of filtrate

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[1]Polytron, Brinkman Instruments, Catlague Road, Westbury, New York 11590.

[2]Metaphosphoric acid solution. Add: 30 g NaCl, 1.67 g glacial metaphosphoric acid (a mixture of HPO_3 and $NaPO_3$) and 0.2 g EDTA to 100ml of H_2O.

with 2.0ml of 0.4 M Na_2NPO_4 and 1.0ml of DTNB reagnet.[3] A_{412} was recorded within 2 minutes after mixing. A blank (with H_2O_2 or 0.15 M kc_1 substituted for enzyme source) was carried through the incubation simultaneously with the samples since nonenzymatic GSH oxidation by H_2O_2 occurred during incubation. Both enzymatic and nonenzymatic reactions proceed at rates directly proportional to GSH concentration. An enzyme unit of activity was defined as a decrease in the log [GSH] of 0.001 per minute after the decrease in log [GSH] per minute of the nonenzymatic reaction was subtracted.

IV. STATISTICAL ANALYSIS OF DATA

The data were analyzed by the General Linear Model (GLM) procedure and Duncan's Mean Separation procedure of the Statistical Analyses System (SAS) (Barr *et al.*, 1978).

The GLM procedure is a multivariate regression analysis, determining differences by significant F tests. The Duncan's Mean Separation procedure was used to determine the differences among means in variables where a significant difference due to treatment was indicated.

The independent variables used in the regression analysis for the performance data were treatment (Trt) and replication (Rep). The independent variables used in the regression analysis for the carcass data,

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[3]DTNB reagent. Add: 40 mg 5, 5'-dithiobis (2-nitro-benzoic acid) (Aldrich Chemical Co., Milwaukee, Wisconsin) to 100 ml of an aqueous 1 percent trisodium citrate solution. Prevent prolonged periods of light exposure and extremes in pH.

hematology data and glutathione peroxidase data were treatment, replication and sex (Sx). In addition, the glutathione peroxidase data were analyzed by regression analysis where the blank value carried throughout the laboratory analysis for each set of samples was included in the model as an independent variable in addition to treatment, replication and sex.

CRANES CREST

CHAPTER IV

RESULTS AND DISCUSSION

I. EFFECT OF VARIED SELENIUM LEVELS ON PERFORMANCE TRAITS

The data for average daily gain (ADG), average daily feed intake (ADFI), and feed efficiency (FE) are presented in Table 2. The pigs on 150ppb added Se, had a slightly lower ADG and ADFI, but FE was comparable to the other supplemental levels. Although ADG and ADFI were lower, they were not significantly lower. The supplemental Se is far below the level that would cause growth inhibition, Wahlstron and Olson (1959), and the supplemental Se is the only experimental variable in the study. Overall pig performance was similar for all groups in this study and no significant differences were found in ADG, ADFI or FE. These data support the reports by Groce *et al.* (1973b), Ku *et al.* (1973), Ewan *et al.* (1969), and Mahan and Moxon (1978), who reported that supplemental Se had no effect on performance when added to the ration.

II. EFFECT OF VARIED SELENIUM LEVELS ON CARCASS TRAITS

The data for the carcass traits is presented in Table 3. The pigs on 150ppb added Se had slightly lower hot carcass weights, chilled carcass weights and lengths than pigs on other treatments, but there were no significant differences in the carcass traits. Pigs on 50 and 150 ppb Se had lower average backfat measurements than did pigs on the other treatments, while pigs on 100 and 125 ppb Se had smaller LEA, but the values were not significantly different than the other treatments. The weights for the

Table 2. Effect of Dietary Selenium Supplementation Upon Pig Performance[a]

Item	Added Se, ppb						
	0	25	50	75	100	125	150
No. of Pigs	14	14	14	14	14	14	13
Initial wt., Kg	27.0	27.2	27.8	26.9	28.3	25.7	26.0
Final wt., Kg	104.7	105.7	106.8	105.3	105.4	104.5	100.5
Average daily gain, Kg							
Growing period	.91	.89	.94	.88	.90	.89	.86
Finishing period	.81	.82	.78	.83	.78	.82	.81
Overall	.85	.85	.85	.84	.84	.85	.76
Average daily feed intake, Kg							
Growing period	2.3	2.3	2.4	2.3	2.3	2.2	2.3
Finishing period	3.2	3.2	3.3	3.2	3.3	3.3	2.4
Overall	3.0	2.7	2.9	2.8	2.8	2.7	2.4
Feed efficiency							
Growing period	2.1	2.5	2.5	2.6	2.5	2.5	2.6
Finishing period	3.8	3.9	4.2	3.9	4.2	3.9	3.7
Overall	3.2	3.2	3.3	3.3	3.4	3.2	3.2

[a]Statistical analysis revealed no significant differences in performance data.

Table 3. Effect of Dietary Selenium Supplementation on Carcass Traits[a]

Item	Added Se, ppb						
	0	25	50	75	100	125	150
No. of pigs	8	8	8	8	8	8	8
Live Wt., Kg	109.0	109.1	105.7	108.5	109.8	107.0	102.7
Chilled wt., Kg.	78.4	78.7	76.4	77.9	80.0	75.7	73.6
Hot carcass wt., Kg.	80.1	80.2	78.2	79.5	81.4	77.3	75.2
Length, cm	82.6	81.2	80.7	81.7	80.8	81.4	79.5
Back fat, cm	3.07	3.07	2.74	2.97	3.23	3.05	2.81
LEA, sq. cm.	34.90	34.83	34.51	34.37	32.12	32.12	35.20
Fat thickness LEA, cm[b]	2.36	2.57	2.46	2.32	2.46	2.66	2.33
Muscling score[c]	9.14	9.11	9.00	9.00	8.62	9.00	9.00

[a]Statistical analysis revealed no significant differences in carcass traits.

[b]Average of 3 equally spaced measurements of external fat thickness around the LEA at the tenth rib.

[c]USDA muscling scores (8=moderately thick, 9=moderately thick plus, 10=thick minus).

organs that were collected are presented in Table 4. The pigs fed the diet containing 150ppb aded Se had lower organ weights than the other treatments, but not significantly lower. These lower values are attributed to the lower growth performance of the pigs. These pigs on the 0.75 and 100ppb added Se had larger organ weights but not significantly larger. The carcass data agreed with the results obtained by Doornenbal (1975), who reported that Se supplementation had no effect on carcass characteristics.

III. EFFECT OF VARIED SELENIUM LEVELS ON BLOOD PARAMETERS

The data for hemoglobin and hematocrit are presented in Table 5. There were no significant differences between the treatments. The values for hemoglobin and hematocrit were very random across treatments and seem to be unrelated to added selenium in the diets. These results are substantiated by Trapp *et al.* (1969) and Hitchcock *et al.* (1978), who found selenium level in the diet to have no apparent effect on hemoglobin and hematocrit values. Michel *et al.* (1969) and Trapp *et al.* (1970) indicated that standard hematological measures were of little use in diagnosing selenium-vitamin E deficiency in swine.

The values for erythrocyte GSH-PX are presented in Table 6. The results of this study indicated that there were no significant differences between treatments for the first bleeding, at 70 days of the experiment. Although there were no significant differences, there was a noticeable difference between the values in the 0 selenium added treatment and the values of the other treatments. Mahan *et al.* (1977) hypothesized that

Table 4. Effect of Dietary Selenium Supplementation on Organ Weights[a]

Item	Added Se, ppb						
	0	25	50	75	100	125	150
Kidney, g	364.3	366.2	336.7	374.8	355.5	375.9	331.5
Liver, g	3314.0	3277.0	3337.0	3475.0	3562.0	3312.0	3237.0
Heart, g	365.4	359.0	344.6	359.5	350.4	370.7	347.1
Spleen, g	195.2	175.5	179.2	187.2	193.1	185.4	160.5

[a]Statistical analysis revealed no significant differences in organ weights.

Table 5. Effect of Selenium Supplementation on Hemoglobins and Hematocrits[a]

Item	Added Se, ppb						
	0	25	50	75	100	125	150
No. of pigs	8	8	8	8	8	8	8
70th Day of Experiment							
Hemoglobin, g/100 ml	13.65	15.15	14.20	12.75	13.25	13.00	12.80
Hematocrit, percent	38.17	37.38	37.40	37.72	37.71	37.15	35.85
Day of Slaughter							
Hemoglobin, g/100 ml	16.10	15.55	15.10	15.55	15.65	15.75	15.85
Hematocrit, percent	42.48	47.31	45.57	39.32	46.53	39.75	46.70

[a]Statistical analysis revealed no significant differences in hematological parameters.

Table 6. Effect of Selenium Supplementation on Erythrocyte GSH-PX

Item	Added Se, ppb						
	0	25	50	75	100	125	150
No. of pigs	8	8	8	8	8	8	8
70th day of Experiment							
μM GSH oxidized/min.	129.00	147.62	133.12	142.25	144.87	125.50	147.87
Enzyme units activity	98.14	176.25	156.37	168.00	217.12	174.37	167.87
μM GSH oxidized/min./mg Hb	47.96	52.76	50.48	55.06	55.81	49.42	59.13
Enzyme unit activity/mg Hb	36.81	57.68	59.75	65.68	82.77	68.08	65.99
Day of Slaughter							
μM GSH oxidized/min.	156.87	185.75	175.00	166.75	188.87	160.12	192.00
Enzyme units activity	170.75[b]	200.87[ab]	152.12[b]	178.62[b]	257.62[a]	141.62[b]	261.75[a]
μM GSH oxidized/min./mg Hb	51.93	60.15	58.50	53.97	60.76	51.06	60.24
Enzyme unit activity/mg Hb	55.55[b]	64.88[ab]	50.67[b]	57.06[b]	82.34[a]	54.17[b]	81.67[a]

[a,b]Values in the same row with different superscripts are significantly different ($P<.05$).

the requirement of Se for the finishing pig is 50ppb instead of the 150ppb shown to be needed in the younger, rapidly growing pig. If this hypothesis is correct, an explanation of the lower values obtained in this study for the control pigs at the 70 day bleeding could be that the basal control was marginal to deficient in Se and that all the supplemental levels overcame the marginal deficiency. The lack of difference between the other treatments suggests that the finishing pig may not need or utilize more than 50ppb Se in the diet.

On the day of slaughter there were significant differences in enzyme units activity and enzyme units of activity per mg of hemoglobin. These differences were not linearly increased with the increasing levels of Se, the 100 and 150 ppb added selenium diets had significantly ($P < .05$) increased enzyme units of activity and activity per mg of hemoglobin than did pigs receiving the 0, 50, 75 or 125 ppb added Se. The inconsistent nature of the responses and the lack of noticeable differences between treatments in the other 2 variables may be partially attributed to the variation in slaughter day. Since the pigs were slaughtered on 4 different days, the effect of induced stress due to handling during slaughter may have also caused the randomness of the values.

In addition, the inconsistency of the results may be partially attributed to the hypothesis of Mahan (1977) who suggested that pigs during the finishing phase may require only 50ppb. Since the basal diet contained approximately 50ppb, the addition of supplemental Se would not be expected to have a consistent linear effect on GSH-PX activity.

During the GSH-PX analysis, there appeared to be a variation in the values for the blank. In an attempt to compensate for this variation,

an analysis was undertaken holding the blanks constant throughout the analyses for GSH-PX. Table 7 presents the data with the blanks used as independent variables. The analysis of the data utilizing the blanks as independent variables did not alter the magnitude of the responses obtained in the amount of GSH-PX or enzyme units of activity.

The adjustment procedure resulted in a slight difference in mean separation which can be seen in Table 7. The reason for this difference can be attributed to the adjustment procedure. The procedure decreased the difference in the minimum to maximum values, resulting in the values being closer together. The same inferences can be made concerning 0 Se added diet versus the other treatments as in the data with the blanks used as dependent variables.

IV. VARIED SELENIUM LEVELS ON HEPATIC GSH-PX

The data for the effect of supplemental Se on hepatic GSH-PX is presented in Table 8. There were no significant differences between treatments for the variables studied. An explanation for the lack of significant difference may be due to the differences in induced stress during slaughter, since the pigs were slaughtered on four different days. Another explanation could be an adequate store of Se in the liver during the study, since the pigs were not depleted before the study began. With the Se requirement for finishing pigs being projected at 50ppb, Mahan *et al.*, (1977); the only marginal deficient diet would have been the control and it contained approximately 50ppb natural Se. Since the 0 added Se diet is approximately the projected adequate level, it is possible that stores built up before the study was initiated were sufficient to meet

Table 7. Effect of Selenium Supplementation on Erythrocyte GSH-PX When GSH Blanks or E.U. Blanks Were used as Continuous Independent Covariates

Item	Added Se, ppb						
	0	25	50	75	100	125	150
No. of pigs	8	8	8	8	8	8	8
70th day of Experiment							
μM GSH oxidized/min.	125.92	145.87	136.53	141.72	148.14	125.42	146.23
μM GSH oxidized/min./mg Hb	46.54	51.98	52.67	54.46	57.46	49.45	58.40
Enzyme units activity	102.13	176.98	152.92	172.41	211.65	173.19	169.33
Enzyme units activity/mg Hb	38.55	58.11	57.41	67.97	80.74	67.75	66.71
Day of Slaughter							
μM GSH oxidized/min.	161.55	180.31	166.19	160.19	201.51	155.72	199.80
μM GSH oxidized/min./mg Hb	52.49	58.49	55.69	51.92	65.28	49.75	63.13
Enzyme units activity	167.09[b]	202.56[ab]	163.30[b]	185.11[ab]	245.13[ab]	171.13[b]	257.76[a]
Enzyme units activity/mg Hb	55.16[b]	65.24[ab]	53.75[b]	58.80[ab]	78.66[ab]	54.27[b]	80.41[a]

[a,b]Values in the same row with different superscripts are significantly different (P<.05).

Table 8. Effect of Selenium Supplementation on Hepatic GSH-PX[a]

Item	Added Se, ppb						
	0	25	50	75	100	125	150
No. of pigs	8	8	8	8	8	8	8
μM GSH oxidized/min.	100.12	111.37	103.87	83.75	104.12	83.12	95.50
μM GSH oxidized/min./g liver	9.70	10.02	9.64	8.83	10.57	8.47	8.93
Enzyme unit activity	103.62	123.12	123.75	98.62	113.87	81.75	125.87
Enzyme unit activity/g liver	10.42	10.68	11.68	9.87	11.57	8.30	11.87

[a]Statistical analysis revealed no significant differences in hepatic GSH-PX.

the requirement of pigs receiving the control diet. Studies by Lawrence and Burk (1977), show that only 33 percent of the GSH-PX activity in the pig is selenium dependent, thus the pigs could have built up stores large enough to satisfy the Se requirement for the 33 percent of the enzyme.

The blanks used in the laboratory analysis of hepatic GSH-PX appeared to vary considerably from one group of analysis to the next. Therefore, an analysis of the data was conducted with the blanks used as independent variables. These data are presented in Table 9. The variation in blank was not significant so as to cause a difference in the interpretation of the data. The same conclusions can be made as were made from the data on Table 8.

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Table 9. Effect of Selenium Supplementation on Hepatic GSH-PX When GSH Blank or E.U. Blanks were used as Continuous Independent Covariates[a]

Item	Added Se, ppb						
	0	25	50	75	100	125	150
No. of pigs	8	8	8	8	8	8	8
μM GSH oxidized/min.	100.35	107.14	105.13	85.98	102.36	84.90	95.98
μM GSH oxidized/min./g liver	9.71	9.70	9.73	8.55	10.44	8.61	8.97
Enzyme units activity	102.85	132.17	121.09	96.16	116.60	77.32	124.41
Enzyme units activity/g liver	10.31	11.95	11.31	9.52	11.95	7.68	11.66

[a]Statistical analysis revealed no significant differences in hepatic GSH-PX.

CHAPTER V

SUMMARY

Ninety-eight Yorkshire x Duroc pigs were utilized in this study to determine the effect of six supplemental dietary levels of selenium on growing-finishing pig performance and erythrocyte and hepatic glutathione peroxidase levels in the pig. The pigs were fed a basal corn-soybean meal ration containing approximately 50ppb natural selenium or the basal ration supplemented with 25, 50, 75, 100, 125 or 150 ppb Se as sodium selenite.

The pigs were weighed biweekly, feed was measured and performance data were calculated every 2 weeks during the trial. Blood samples were collected from eight pigs on each treatment after 70 days on experiment and at slaughter. Hemoglobin and percent packed cell volumes were determined at the time the blood samples were collected. Erythrocytes from all blood samples were prepared by washing twice with sodium phosphate-saline buffer and then frozen at -20°C until they were analyzed for glutathione peroxidase activity. Liver samples were collected at slaughter and were stored frozen until the analysis for glutathione peroxidase could be conducted.

The results of this study indicate that there were no significant differences due to treatment on the performance criteria or carcass data that were measured in this study. Erythrocyte glutathione and enzyme units of activity were not significantly affected by treatment at 70 days of the experiment. However, pigs receiving the basal diet without any

added supplemental selenium had noticeably lower levels of glutathione and enzyme units of activity on the seventieth day of the experiment. This finding suggests that the basal control ration was possibly marginal to deficient in selenium and that all the supplemental levels overcame the marginal deficiency. Erythrocyte glutathione peroxidase enzyme units of activity and enzyme units of activity per milligram of hemoglobin at slaughter were significantly ($p < .05$) affected by treatment. These differences were not linearly increased with increasing level of supplemental Se. No consistent trend was observed and the randomness of the values may be partially explained by the hypothesis of Mahan *et al.* (1977) who suggested that pigs during the finishing phase may require only 50ppb Se. Since the basal diet in this study contained approximately 50ppb natural Se, the addition of supplemental Se would not be expected to have a consistent linear effect on glutathione peroxidase activity.

No significant differences due to treatment in hepatic glutathione peroxidase were observed in this study. The fact that pigs utilized in this study were not depleted of their Se reserved before the experiment was initiated and the hypothesis of Mahan *et al.* (1977) discussed previously may explain partially the results obtained for hepatic glutathione peroxidase in this study.

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