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Effects of supplemental selenium on swine

John Erby Wilkinson

To the Graduate Council:

I am submitting herewith a thesis written by John Erby Wilkinson entitled "Effects of supplemental selenium on swine." I have examined the final electronic copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science, with a major in Animal Science.

Marvin C. Bell, Major Professor

We have read this thesis and recommend its acceptance:

Frank B. Massincupp, Curtis C. Melton

Accepted for the Council:
Carolyn R. Hodges

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

To the Graduate Council:

I am submitting herewith a thesis written by John Erby Wilkinson entitled "Effects of Supplemental Selenium on Swine." 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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We have read this thesis and recommend its acceptance:

Frank B. Masincupp

Curtis C. Melton

Accepted for the Council:

Hilton A. Smith

Vice Chancellor
Graduate Studies and Research

EFFECTS OF SUPPLEMENTAL SELENIUM ON SWINE

A Thesis

Presented for the

Master of Science

Degree

The University of Tennessee, Knoxville

John Erby Wilkinson

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ABSTRACT

Effects of dietary selenium on reproductive performance and blood selenium status of sows and their progeny and of dietary selenium, copper and zinc on growth characteristics, selected blood parameters, and carcass traits in growing-finishing swine were evaluated in three experiments.

In the first experiment, forty-eight Duroc barrows and gilts were allotted by sex and weight to six dietary treatments. They were fed a 16% crude protein diet containing .08 ppm selenium, 12.5 ppm copper, and 82 ppm zinc alone or supplemented with: .1 ppm selenium; 125 ppm copper; 80 ppm zinc; .1 ppm selenium plus 80 ppm zinc. As each animal reached 100 kg a blood sample was taken but no treatment differences ($P>.05$) for RBC selenium-75 uptake, plasma selenium, copper and zinc, whole blood selenium, or hematocrit were found. Overall average daily gain was not significantly different between treatments.

In the second experiment, four groups of six Duroc sows were fed basal corn-soybean meal or corn-soybean meal-tankage diets each with or without .1 ppm supplemental selenium. The basal diets contained .1 ppm natural selenium. Changes from the 28th day of gestation through the 56th day of lactation in whole blood and plasma selenium concentrations and RBC selenium-75 uptake indicated greatest demand for selenium came immediately after parturition but treatment differences ($P<.05$) were evident only at the 112th day of gestation, and 28th day of lactation. At the 28th day of lactation, the selenium supplemented sows had higher

plasma (P<.05) and whole blood (P<.01) selenium concentrations than the unsupplemented sows. Seventy-two of the progeny of these sows were allotted to treatments as in experiment # 1. Upon reaching 100 kg, the pigs were slaughtered and blood and carcass data collected. The selenium content of the last rib longissimus muscle was increased by the addition of selenium to either the sow diet (P<.05) or the growing-finishing diet (P<.001). Plasma selenium was increased (P<.05) by additional selenium in the growing-finishing diet but was not significantly affected by dietary selenium levels of the sow. Average daily gain and longissimus dry matter were significantly increased in pigs fed additional zinc. Feed efficiency was not significantly affected by treatment.

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CHAPTER I

INTRODUCTION

Microelement nutrition of swine has been investigated extensively in recent years with copper, zinc and selenium being among the most carefully evaluated micronutrients. While universally recognized as dietary essentials for swine, numerous facets of the multifarious biochemical functions, reactions and interactions of copper, zinc and selenium remain unelicited. Furthermore, the effects of many micronutrients on ultimate carcass and meat quality is an important but unknown entity.

Conflicting evidence suggests that supplemental copper as copper sulfate (Castell et al., 1975; Gipp et al., 1973) or zinc as zinc proteinate (Elgin, 1975; Masincupp, 1974) may improve average daily gain and feed efficiency in growing-finishing swine. While copper, zinc and selenium share the duality of essentiality and potential toxicity, the absolute range between dietary requirement and excess is at least an order of magnitude smaller for selenium than for either zinc or copper. Nevertheless, the limits of safety are sufficiently broad and the necessity of adequate dietary selenium so overwhelmingly important that routine supplementation of swine rations with selenium is clearly indicated where a deficiency may exist.

Such supplementation of practical swine rations with inorganic selenium has been approved only recently. Much of the research leading

to Food and Drug Administration approval necessarily involved minimum requirements and maximum tolerances. Studies on the levels of dietary selenium consistent with optimal growth, development and reproduction of swine consistently utilized low levels of dietary selenium. Conversely, the interactions of selenium with zinc, copper and other micronutrients, as well as heavy metals, have generally been demonstrated by utilizing high dietary levels of one or both elements.

Studies were, therefore, conducted to evaluate the nature of selenium supplementation above the required dietary level for swine but still within Food and Drug Administration regulations. The effects of copper and zinc supplementation of diets for growing-finishing swine were also evaluated in the studies. Specifically these studies were designed to evaluate: (1) the effects of supplementing inorganic selenium to diets naturally containing .1 ppm selenium on sow reproductive performance, blood selenium status and ultimate effect on the selenium status of their offspring; (2) the effects of supplemental selenium, copper and zinc singly and in combination on average daily gain, feed efficiency and selected blood parameters in growing-finishing swine; and (3) the effects of dietary copper, zinc and selenium on certain carcass and meat characteristics of swine.

CHAPTER II

REVIEW OF LITERATURE

I. SELENIUM

Selenium has been recognized as an essential dietary element for a relatively short time. Since 1957 when Schwarz and Foltz established the necessity of small amounts of selenium in the diets of rats, (Ullrey, 1974) "selenophobia" has greatly subsided. Subsequently the nature of selenium nutrition and its implications for human and animal health have been investigated and debated extensively. Nevertheless, a certain "dilemma" (Scott, 1973) and "schizophrenia" (Frost, 1976) regarding selenium persists both within the scientific community and the political arena.

Due to the voluminous nature of the literature on selenium, the background and inorganic chemistry of selenium will be discussed briefly with the emphasis placed on literature pertinent to the focus of the research herein presented. Rosenfeld and Beath (1964), Scott (1973) and Ullrey (1974) are excellent reviews of the historical aspects of selenium, while Muth et al. (1967) provide a comprehensive review of selenium chemistry.

History of Selenium

Elemental selenium, closely related to atomic sulfur and tellurium, was discovered in 1817 by the Swedish chemist, Berzelius, as a

precipitate of sulfuric acid (Frost, 1976; Scott, 1973). The effects of selenium toxicity, however, were recognized long before Berzelius' discovery and another 150 years passed before the symptoms and cause were associated.

Evidence of selenium toxicity may be found as early as the writings of Marco Polo (Rosenfeld and Beath, 1964) wherein an affliction of horses which came to be known as "alkali disease" or "blind staggers" was reported. Documented in Columbia by Father Pedro Simon in 1560 and in Mexico over 200 years ago, the disease was first reported in the United States by Dr. T. C. Madison in 1857 (Rosenfeld and Beath, 1964). Characterized by loss of hair and teeth, sloughing of the hooves and nails and a specific paralysis (Scott, 1973), the disease was quite prevalent for several years in some western states. Not until 1931, however, was the relationship between "alkali disease" and excessive selenium intake defined (Rosenfeld and Beath, 1964).

Until the late 1950's, toxicity remained the only recognized aspect of selenium nutrition. Complicated by the close functional relationship of selenium and vitamin E, the elucidation of the essentiality and biochemical function of selenium, nonetheless, proceeded rapidly following the work of Schwarz and Foltz in 1957 which demonstrated that sodium selenite would prevent liver necrosis in rats. Interest in selenium has steadily increased until more papers are presently being published on selenium than any other trace element. Frost (1975, 1976) published excellent reviews of the recent trends and history of selenium research.

Distribution of Selenium

While soil selenium concentrations vary markedly from region to region and somewhat with time in a given area, the lack of a consistent relationship between selenium levels in soils and the selenium concentration in the vegetation grown thereon dictates that in a nutritional sense the importance of selenium distribution is to be found in the vegetation. The lack of a soil-plant relationship of selenium concentration possibly stems from the nature of the selenium compounds found in different soils (Natl. Acad. of Sci., 1971) and caution must be observed to prevent the depletion of "biologically active" selenium from croplands (Frost, 1976). Rosenfeld and Beath (1964) provide an excellent review of the geological aspects of selenium distribution and Johnson et al. (1967) have reviewed the factors affecting the selenium content of plant materials.

While selenium accumulating plants with a selenium content of up to 1000 ppm (Natl. Acad. of Sci., 1971) are potentially toxic to livestock, the major problem with primary feedstuffs for swine is selenium deficiency. Bell et al. (1975), Ku et al. (1973), Kubota et al. (1967) and Ullrey (1974) have shown both the variability and general deficiency of selenium in corn, soybeans, sorghum grain and forages in certain areas of the United States. Mean values of selenium in corn are relatively high in South Dakota, Nebraska and North Dakota but relatively low in Michigan, Illinois, Indiana, Ohio, New York and Tennessee (Bell et al., 1975; Ullrey, 1974). Other feedstuffs show a similar variation in selenium content with fish and fish byproducts being

generally high in selenium (Allen, 1974) and grains often low. Marked variations in selenium content of primary feedstuffs are also noted within states. With the mass movements of primary feedstuffs for swine and the difficulty of selenium analysis, the selenium content of most swine feed is unknown. The problem is enhanced in total or partial confinement systems by the fact that corn and soybean meal contain far less ∝-tocopherol than pasture (Ullrey, 1974). The most biologically active form of vitamin E is ∝-tocopherol.

II. BIOCHEMICAL FUNCTION OF SELENIUM

Although a variety of biochemical roles of selenium have been postulated and might be expected (Hoekstra, 1974), the primary function of selenium now recognized is as a component of glutathione peroxidase, an enzyme which shares with vitamin E the action of antioxidant. As such, selenium plays an important part in maintaining membrane stability by preventing oxidative damage to cells resulting from lipid peroxidation.

Mechanism of Lipid Peroxidation

Lipid peroxidation and breakdown has been studied extensively both in pure systems (Farmer, 1946; Labuza, 1972; Loury, 1972) and in natural foods. An overall view of the autocatalytic free radical mechanism of these reactions is given in Figure 1. Like all free radical mechanism, three steps are involved in the reaction.

Initiation

Initiator $\longrightarrow$ R. A.

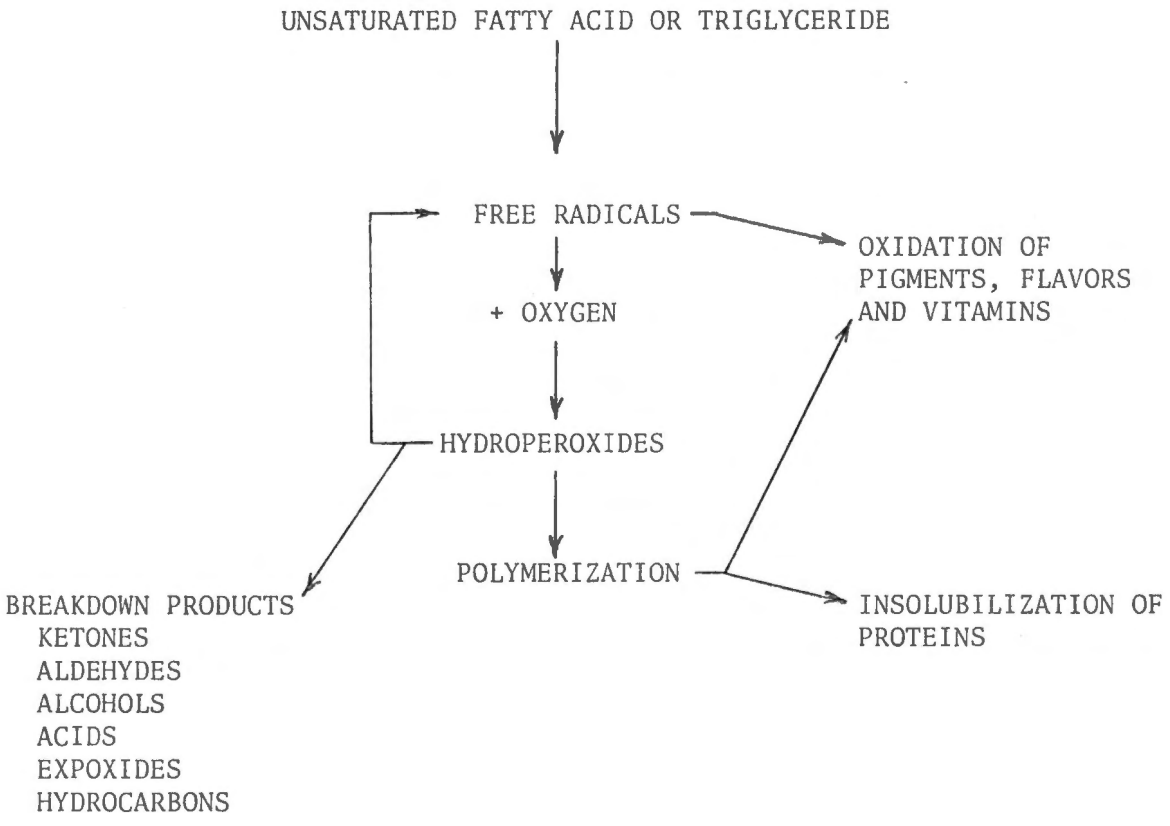

Figure 1. Postulated mechanism of lipid peroxidation (from Labuza, 1972).

Propagation

$$\left.\begin{array}{l} R. + O_2 \rightarrow ROO. \\ ROO. + RH \rightarrow ROOH + R. \end{array}\right\} \quad \begin{array}{l} B. \\ C. \end{array}$$

Termination

$$\left.\begin{array}{l} ROO. + ROO. \\ ROO. + R. \\ R. + R. \end{array}\right\} \text{Non Radical Products} \quad \begin{array}{l} D. \\ E. \\ F. \end{array}$$

In lipid peroxidation, the O_2 in reaction B always combines with the double bond of an unsaturated fatty acid rather than randomly adding to a saturated chain. This can be expected based on energy and resonance stabilization considerations. The hydroperoxide formed in reaction C undergoes scission to yield a variety of stable and free radical products. The stable compounds include those which give the off flavor and odor to rancid foods, even at very low levels. Parts per million or parts per billion are often enough to elicit rancidity (Labuza, 1972). The free radical moieties either catalyse further peroxidation or react in a terminal reaction (D, E, and F). Five other characteristics of the reaction given by Labuza (1972) are:

1. The rate increased drastically but not directly as the number of double bonds increased. Tappel (1973) stated that fatty acids with 2, 4, 5 and 6 double bonds had relative rates of peroxidation of 1, 4, 6 and 8 respectively.
2. The calculated quantum number was greater than 1.
3. Various compounds in minute amounts either accelerated or inhibited the reaction markedly.

4. With pure material, there is a long induction period.

5. The reaction has a moderately high activation energy.

Kinetics or Lipid Peroxidation

The initial formation of peroxides is crucial to the ultimate shelf life of lipids. The high activation energy (35 to 65 K cal/mole) of the reaction dictates an intermediate moiety. Singlet oxygen has been implicated. Myoglobin, metals, metal complexes and increased temperature have been cited as possible sensitizers for the formation of singlet oxygen. Once the first peroxides are formed, the reaction process becomes a monomolecular decomposition into free radicals which is believed to be catalyzed by metals.

$$\text{ROOH} + M^{+n} \longrightarrow \text{ROO.} + H^{+} + M^{+(n-1)} \qquad \text{G.}$$

$$\text{ROOH} + M^{+(n-1)} \longrightarrow \text{RO.} + OH^{-} + M^{+n} \qquad \text{H.}$$

This eventually changes into a biomolecular reaction which is also catalyzed by metals.

$$2\ \text{ROOH} \longrightarrow \text{ROO.} + RO_2 + H_2O \qquad \text{I.}$$

Thus, metals play a key role in the initiation of lipid peroxidation. Labuza (1972) states:

1. Metals are present in the necessary amounts in most foods to promote autoxidation.

2. The hydroperoxide probably forms a metal complex before decomposing.

3. Metals having a valence change $+3 \rightleftharpoons +2$ are probably most active.

4. The oxidation rate may be altered by competition, chelating complexes, and solvent effects. Obviously the complete environment of a metal will determine its activity in lipid peroxidation.

Saturation, Location and Temperature Effects on Lipid Peroxidation

The amount of fat is not important in lipid peroxidation, but rather the nature of the fat and its location relative to the matrix of the food. Most of the fat in the body is in the form of triglycerides, which consist of a glycerol base with three fatty acid esters replacing the hydroxyl groups. These fatty acids may be either saturated or unsaturated primarily determines the oxidative instability of the lipid fraction. In simple stomach animals the ratio of unsaturated/saturated fatty acids approximates dietary intake, although the extremes of abnormally high or low dietary unsaturated fats are not achieved in the depot fat. By adjusting rations, the unsaturated/saturated acid ratio can be minimized and thus reduce the susceptibility of the meat to oxidative rancidity.

The topography of the fatty tissues and the storage temperature also affect the rate of rancidity development. Superficial fat, as expected, is more susceptible to oxidative rancidity (Ingr, 1972; Kopecky, 1972b; Mehenhall, 1972) probably due to the greater oxygen tension in that portion. Lower temperatures significantly retard oxidative rancidity and thereby increase shelf life (Kopecky, 1972a; Kopecky, 1972b; Pap, 1972). A 1.5 to 1.7 fold increase in storage life

for each 5°C decrease in temperature between -5°C to -30°C has been suggested (Pap, 1972).

Inhibition of Lipid Peroxidation by Selenium and Vitamin E (∝-Tocopherol)

The biological role of vitamin E is closely related to that of selenium even though the mechanism is quite different. In preventing a variety of pathological conditions associated with lipid instability, the two have a mutual "sparing" action. Since selenium is a mineral and tocopherol a vitamin, the metabolism and action are expectedly quite different.

Vitamin E is a natural fat soluble antioxidant and hydrogen donor which prevents reactions associated with intracellular fatty acid peroxidation by acting as a free radical trap. Dietary requirements are directly related to the amount of unsaturated fat in the diet and the tissue level of vitamin E has been shown to influence oxidative rancidity. In swine fed high levels of copper, vitamin E supplementation significantly increase the oxidative stability of depot fat (Amer and Elliot, 1973). In turkeys given 10 or 100 I.U. of vitamin E orally or injected, thiobarbituric (TBA) values were significantly decreased by tocopherol supplementation. Injection was more effective than oral treatment and 100 I.U. had a greater effect than 10 I.U. Treatment X storage time and treatment X meat type interactions were also noted (Webb et al. 1972a; Webb et al. 1972b). It, therefore, seems plausible that dietary vitamin E supplementation has possibilities as a shelf life extender for pork.

Tappel (1965) postulated that selenium, like vitamin E, was important in maintaining membrane stability, but early in vitro studies indicated selenium was not effective in preventing red blood cell hemolysis. Inclusion of glucose in the incubation medium, however, reversed the initial findings and showed that dietary selenium indeed could prevent red cell hemolysis and hemoglobin oxidation. The glucose dependent nature of the protection and the effect on hemoglobin differed from the action of vitamin E, indicating a distinction in function which could account for the failure of selenium or vitamin E to completely replace the other. Furthermore, the results implicated selenium in the metabolic pathway shown in Figure 2. Selenium was eventually shown to be a component of the last enzyme in the reaction sequence, glutathione peroxidase. The enzyme, which consists of four subunits is believed to contain four selenium atoms per molecule (Hoekstra, 1974).

The discovery of the relationship of selenium and glutathione peroxidase permitted postulation regarding the relationship of selenium and vitamin E. The current theory, shown schematically in Figure 3, is that selenium as a component of glutathione peroxidase destroys hydroperoxides formed in the oxidation of unsaturated fatty acids, while vitamin E decreases the initial formation of the hydroperoxides. The postulated relationship suggests several hypothesis including the following:

(1) Tissues of high H_2O_2 production are especially prone to degeneration when the body is low in both selenium and vitamin E.

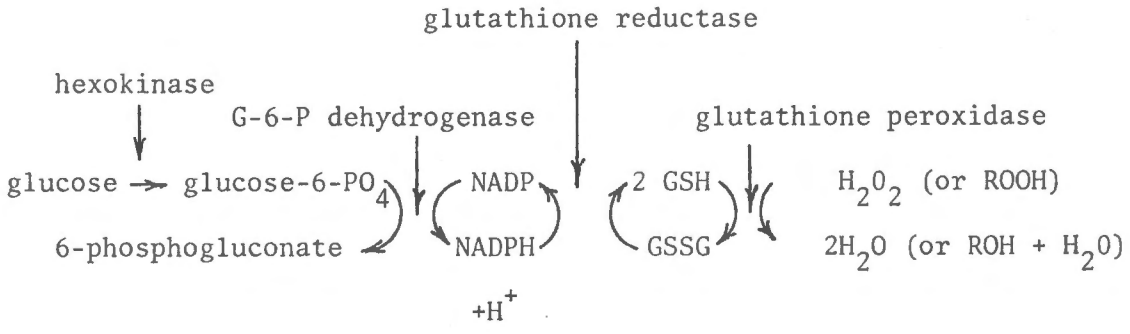

Figure 2. Glucose-dependent pathway of lipid hydroperoxide destruction (from Hoekstra, 1974).

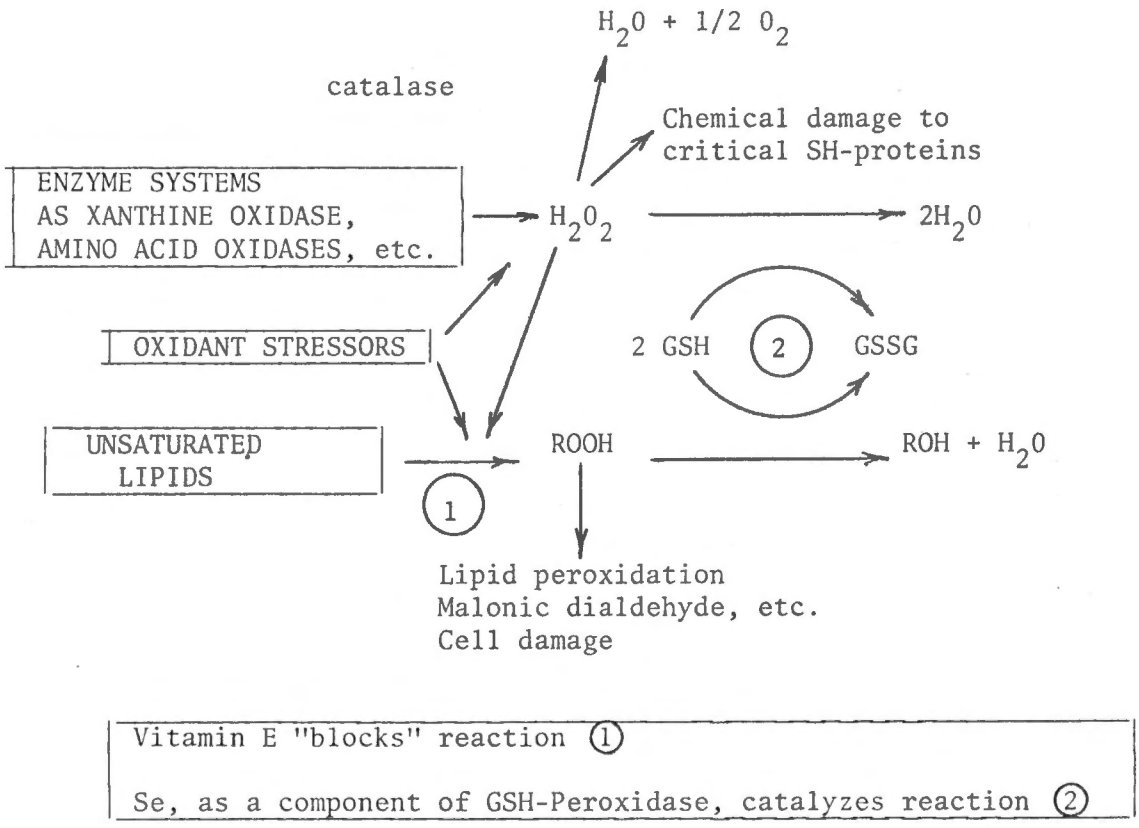

Figure 3. Schematic representation of the postulated functions of selenium and vitamin E and mechanism of their interrelationship (Hoekstra, 1974).

(2) If the body is low in vitamin E but has adequate selenium, the capacity to destroy the hydroperoxides formed is exceeded in some tissues naturally low in glutathione peroxidase.

(3) The capacity to destroy hydroperoxides that are formed may be exceeded in some tissues when vitamin E is adequate but selenium inadequate in the body.

(4) The sparing action of selenium and vitamin E is readily explained by the postulated mechanism (Hoekstra, 1974).

Glutathione peroxidase activity in plasma and red blood cells is a function of dietary selenium level (Omaye and Tappel, 1974). However, the chemical nature of the tissue selenium is of utmost importance if it is to play a role in preventing lipid peroxidation in the meat. The relationship between selenium levels and glutathione peroxidase activity in muscle is unknown. Whether selenium itself or as a nonenzyme, non-protein moiety has any antioxidant characteristics is also unknown, but not impossible. If selenium is active only as a component of glutathione peroxidase, only short term protection against rancidity can be expected. Since the glutathione peroxidase activity is glucose dependent and glycolysis is complete soon after slaughter, glutathione peroxidase activity must rapidly decrease post mortem. Furthermore, lipid peroxidation damages proteins and inactivates enzymes. Thus, the only role for glutathione peroxidase in preventing lipid peroxidation in meat is by delaying the initiation of peroxide formation until a temperature is achieved at which autoxidation of lipids in inhibited. As the

kinetics of the free radical mechanism suggests, the short term protection could, nonetheless, be important. Hafeman and Hoekstra (1976) have shown that lipid peroxidation proceeds exponentially *in vivo* in terminal vitamin E, selenium deficiency. Whether a similar situation exists in meat is not known.

III. SELENIUM IN SWINE

Pathology of Selenium Deficiency

Since 1957, a number of pathological conditions in various species have been shown to arise from selenium-vitamin E deficiency (Frost, 1975). While selenium and vitamin E have a mutual "sparing" action, neither can completely replace the other in preventing some of these pathological states (Hoekstra, 1974). In swine, as with other species, the selenium deficiency manifests itself in a variety of ways. Hepatosis dietetica, mulberry heart and sudden death are all indicative of inadequate dietary selenium in swine. Pigs weighing 20-40 kg are most commonly involved and often die suddenly. At necropsy, the pigs show a bilateral paleness of the skeletal muscles with the *quadriceps femoris, gracilis, adductor, psoas* and *longissimus dorsi* muscles being most affected. Muscle fibers exhibit loss of striations, vacuolization fragmentation and mineral deposition. The character of the liver is greatly changed with lobules having undergone marked degeneration and necrosis. Edema in various tissues is often noted and dystrophy of the myocardium sometimes observed (Ullrey, 1974).

Selenium deficiency is difficult to detect in live animals, but stress is believed to enhance the consequence (Ullrey, 1974). Reproduction is severely reduced and death may ensue in selenium deficient sows (Mahan et al., 1974).

Requirements

Due to the close biochemical association of vitamin E and selenium, the exact dietary requirement of each is difficult to ascertain and depends on the constitution of the diet. Using practical swine diets containing low levels of natural selenium (approximately .05 ppm) supplemented with .05, .1, .2, .5, and 1.0 ppm selenium as sodium selenite, Groce et al. (1971, 1973a, 1973b) and Hitchcock (1973) have shown that .1 ppm supplemental selenium, or .15 ppm total dietary selenium, satisfies the selenium requirement for growing-finishing swine. Retention of selenite-selenium reached a maximum at the .1 ppm level. Higher levels or inorganic selenium resulted in higher absorption, but increased secretion negated any net gain in selenium retention (Groce et al., 1973a). Death loss, gross pathology and histopathological lesions associated with selenium deficiency were prevented and whole blood, serum, liver, kidney, myocardium and longissimus muscle selenium concentrations increased by .1 ppm supplemental selenium as sodium selenite. Longissimus muscle selenium concentrations reached a plateau at .33 ppm which corresponded to a total dietary selenium level of .15 ppm (Groce et al., 1973b). However, studies in which all selenium was in the natural form a linear correlation of .95 between dietary selenium

(.027 to .493 ppm) and longissimus muscle selenium (.034 to .521 ppm) has been established (Ku et al., 1973). These conflicting results were explained by a study in which diets naturally high in selenium were compared to diets naturally low in selenium but supplemented with selenite to give the same total dietary selenium concentration. The longissimus muscle selenium concentrations were significantly higher in pigs fed the natural diets, suggesting that organic selenium is more available, while inorganic selenium is not utilized above the required level (Ku et al., 1973). Nonetheless, supplementation of .1 ppm selenium as sodium selenite appears to meet this requirement in practical diets for growing-finishing swine.

Selenium requirements of reproducing sows appears to be similar to that of growing-finishing swine. In a comparison of reproductive performance of 108 sows fed corn soybean-meal diets those receiving supplemental selenium and vitamin E produced a significantly higher number of pigs per litter (Ullrey, 1974). Addition of 10 ppm selenium to reproduction diets, however, impaired reproductive efficiency (Ullrey, 1974).

The most extensive investigation of selenium requirements of reproducing swine has been conducted by Mahan et al. (1974, 1975). Comparison of a basal diet containing .04 ppm selenium with the same diet supplemented with .1 ppm selenium as sodium selenite and a semi-purified diet containing .011 ppm selenium demonstrated the critical demands of gestation and lactation on selenium nutrition. Sows fed the basal diet with and without supplemental selenium had similar litter

sizes in the first parity, but in parity II, the unsupplemented sows had significantly smaller litters. Only 3 of 9 sows fed the semipurified diet farrowed in parity I and none farrowed in parity II. Those sows which did produce progeny had small litters and were extremely weak at parturition.

The histopathology of the sows corresponded to the reproduction pattern with the sows fed the basal and basal plus selenium diets exhibiting no lesions indicative of selenium deficiency, while the sows fed the semipurified diet had lesions in the skeletal muscle and stomach characteristic of selenium deficiency. Offspring of the sows fed the basal diet exhibited signs of selenium deficiency much earlier than those of sows fed supplemental selenium when both groups of pigs were fed similar low selenium diets (Mahan et al., 1974).

Sow tissue levels tended to reflect the level of dietary selenium. Serum selenium levels were much lower and more variable during gestation and lactation in sows fed the basal diet than those fed the basal plus selenium diet. Organ and muscle selenium concentrations were likewise much higher in the supplemented sows. Selenium concentration of the milk was generally about twice as high for the supplemented sows than for the unsupplemented sows with colostrum being much higher in selenium content than later milk. The differences in milk were reflected in the serum, organ and muscle selenium concentration of the progeny—the pigs from the unsupplemented sows having much lower selenium concentration in all tissues sampled.

From the data presented, .15 ppm total dietary selenium appears to be adequate for swine of all ages in all physiological states. Inorganic

and/or organic selenium appears to be equally capable of supplying adequate dietary selenium, though the availability of organic selenium, especially above the required level, appears to be greater than the inorganic selenium.

IV. COPPER AND ZINC

Copper in Swine

Copper, which is necessary for hemoglobin production and a variety of other biochemical functions, is a dietary essential for swine. Like selenium, copper deficiency is an area problem, but in many cases copper deficiency is complicated by interactions with other micronutrients. Nonetheless, 5-10 ppm dietary copper seems sufficient for swine (Maynard and Loosli, 1969).

In recent years, the emphasis in copper nutrition in swine has focused not on requirements but on the effects of feeding high levels of copper on average daily gain, feed efficiency and fat characteristics. Braude (1965) reviewed published reports on high level copper feeding in England and Wallace (1967, 1968a) reviewed the same work in the United States. Wallace (1968a) concluded that:

(1) Baby pigs generally responded dramatically to copper feeding with an average increase in gain of 22.1% and feed efficiency improvement of 8.3%.

(2) Growing pigs' feed conversion was improved by 2.3% and gain increased by 6.5%.

(3) Growing-finishing pigs' gain and feed conversion improved 3.6% and 1.1% respectively.

Wallace further concluded that the maximum response was attained with 125 ppm to 250 ppm supplemental copper. Source of the copper was not important but copper sulfate or copper oxide was recommended. Castell et al. (1975) conducted an extensive investigation of the effects of supplemental copper on growing-finishing pigs in Canada and concluded that .05% to .08% dietary copper sulfate pentahydrate improved gain by 1.9% and feed efficiency by 2.1%.

Although the majority of reports suggests some advantage of high level copper feeding of swine, conflicting evidence does exist (Gipp et al., 1973; Myres and Bowland, 1973; Wallace, 1968a). It has been postulated that dietary copper at the 125 ppm to 250 ppm level may function as a bactericidal agent (Wallace, 1968a) which may explain the variability of results. Sanitation and general herd health may drastically influence the experimental outcome. Most of the ingested copper appears in the feces and that which is absorbed tends to accumulate in the liver (Wallace, 1968b) further suggesting the advantageous function of copper supplementation occurs at the intestinal level (Wallace, 1968b).

Studies on the effect of high level of copper feeding on carcass quality have yielded conflicting results and data is insufficient to draw any conclusions (Wallace, 1968b).

Effect of Copper on Lipids

The most significantly and widely reported physiological effect of feeding supplemental copper is the effect on lipids. Numerous workers have reported a softening of the depot fat (Bowland and Castell, 1964,

1965; Taylor and Thonke, 1964) or increased iodine number (DeGoey et al., 1971; Taylor and Thonke, 1964) and alterations in the depot fat associated with increased proportions of unsaturated fatty acids (Amer and Elliot, 1973; Elliot and Bowland, 1968, 1969, 1970; Moore et al., 1968) when high levels of copper were fed to swine. Elliott and Bowland (1970) showed the alterations in depot fat to be greater when animal protein rather than plan protein was fed. Their findings are consistent with work demonstrating that copper is absorbed to a greater extent when animal products are the dietary protein source (Wallace, 1968b). Myres and Bowland (1973) showed the lipid alteration was not due to increased synthesis of unsaturated fatty acids in the adipose tissue. Feeding high levels of dietary copper, however, resulted in an increased activity of the liver stearic acid desaturase system which could account for some of the lipid alteration (Thompson et al., 1973).

Zinc in Swine

Swine have a dietary requirement for zinc of approximately 50 ppm. The requirement can be altered by a variety of dietary factors including phytic acid and calcium concentrations and the source of oil or fat, protein and phytic acid (Plumlee et al., 1960). In swine, deficiency results in a dermatitis known as parakeratosis which is characterized by specific skin lesions, retarded growth and lower feed efficiency (Maynard and Loosli, 1969). Prasad (1966) has an excellent review of zinc metabolism which will not be discussed herein.

Recent work (Elgin, 1975; Masincupp, 1974) has yielded conflicting evidence that supplemental zinc in the form of zinc proteinate may

improve average daily gain and feed efficiency in growing-finishing swine.

V. INTERACTIONS OF SELENIUM, COPPER AND ZINC

Like most micronutrients, selenium, copper and zinc interact with a variety of other dietary constituents which alters the nature and/or extent of their metabolic influence. Matrone (1974) and Mills (1974) provide reviews of some of the chemical and dietary considerations of trace element interactions.

The interrelationship of copper and zinc have been reported by numerous workers (Ritchie et al., 1963; Suttle and Mills, 1967; Wallace, 1968b). High level copper feeding (250ppm) of swine may result in copper toxicity if supplemental zinc is not also included in the diet (Ritchie et al., 1963). The incidence and severity of parakeratosis, however, may be reduced by supplemental copper (Wallace, 1968b) when zinc is deficient in the diet. Improved average daily gain and feed efficiency from copper supplementation has been shown to be dependent on concurrent zinc supplementation (Bunch et al., 1963). Wallace (1968b) terms the evidence "conclusive" that zinc must be included in swine diets high in copper to prevent copper toxicity and achieve the maximum response in growth parameters.

Selenium has been shown to interact with a variety of toxic metals including cadmium, mercury, thallium and silver (Parizek et al., 1974) as well as zinc and copper (Jensen, 1975a, 1975b). High levels of either copper or zinc may induce selenium deficiency in animals fed diets

considered selenium adequate (Jensen, 1975b) as well as prevent selenium toxicity in animals fed high levels of selenium (Jensen, 1975a). In either case, copper and zinc supplementation appear to depress selenium utilization.

Copper may also indirectly influence selenium nutrition through the relationship of copper to iron. In pigs fed 250ppm copper, microcytic hypochromic anemia developed in five weeks (Gipp et al., 1973). When such iron deficiency anemia was induced in New Zealand white rabbits, glutathione peroxidase activity dropped to 26% of the initial levels and did not return to normal until five weeks after the hemoglobin levels were corrected by dietary iron supplementation (Rodvien et al., 1974).

CHAPTER III

MATERIALS AND METHODS

All hogs used in these experiments were supplied by The University of Tennessee Agricultural Experiment Station at Blount Farm. Seventy-two of the finished hogs were slaughtered in The University of Tennessee Meats Laboratory. A total of 3 experiments were conducted and each will be discussed separately.

I. EXPERIMENTAL METHOD

Experiment #1

Forty-eight Duroc pigs including twenty-four barrows and twenty-four gilts weighing 25-30 kg were allotted by sex and weight to 6 dietary treatments. The treatments were: (1) basal 16% crude protein rations, (2) basal plus .1 ppm selenium, (3) basal plus 150 ppm copper, (4) basal plus 80 ppm zinc, (5) basal plus .1 ppm selenium plus 150 copper, and (6) basal plus .1 ppm selenium plus 80 ppm zinc. Composition of diet is shown in Table I. All supplemental selenium was in the form of a commercial selenite premix formulated as prescribed by FDA regulations (Schmidt, 1974). Supplemental copper was in the form of reagent grade copper sulfate pentahydrate and zinc was supplied as zinc proteinate. Two pens containing two barrows and two gilts each were included in each treatment. Each pen measured approximately 1.5 x 6.0 meters. Feed and water were provided ad libitum in concrete-floored, open-fronted buildings.

TABLE I

COMPOSITION OF BASAL DIETS

Ingredients	Ref. No.	% by weight		
		Sow diet 1	Sow diet 2	Growing-finishing diet
Corn, yellow, grain ground	4-02-992	79.0	82.0	74.7
Soybean meal	5-04-607	12.5	5.0	15.0
Tankage	5-00-385	-	5.5	5.0
Alfalfa meal	1-00-023	5.0	5.0	3.0
Dical. phosphate	6-01-080	1.1	0.5	1.0
Limestone	6-01-069	1.4	1.0	-
Salt		0.5	0.5	0.5
Vitamin-mineral premix		0.5	0.5	0.7
Antibiotics		-	-	0.1

Individual animal weights were initially recorded at 14-day intervals; but as the pigs approached 100 kg, they were weighed every seven days. From those weighing 100 kg or more a heparanized blood sample (approximately 30 ml) was taken by vena cava puncture. Each sample was analyzed for red blood cell selenium-75 uptake within four hours after collection (Wright and Bell, 1963) and the results expressed as percentage of dose per ml of packed cells. Plasma and whole blood were frozen and later analyzed for stable selenium using a slight modification of the A.O.A.C. flurometric procedure (A.O.A.C., 1975). A .5g sample was digested in 6 ml concentrated nitric acid and 2 ml of 70% perchloric acid before being dissolved in HCl. The p^H was adjusted to make the solution slightly acid and the solution incubated in 2,3 diamino naphthaline. After washing with decaline, the fluorescence was read 525 mu. Each ration was similarly analyzed for stable selenium. Plasma and feed copper and zinc concentrations were determined by atomic absorption spectroscopy after being ashed and dissolved in 6 N HCl (A.O.A.C., 1975).

Experiment #2

Twenty-four Duroc sows were randomly allotted to 4 dietary treatments within 14 days after breeding and fed the respective diets through lactation. Visual appraisal was used to minimize size difference between treatments and in pens within treatments. The diets utilized were: (1) basal corn-soybean meal ration, (2) basal corn-soybean meal-tankage ration, (3) ration 1 plus .1 ppm selenium, and (4) rations 2 plus .1 ppm selenium. Composition of the basal diets is included in

Table I (page 26). All supplemental selenium was in the form of a commercial selenite premix. Sows were fed twice daily in groups of three on grass lots during gestation and individually in concrete-floored farrowing stalls throughout lactation. Pigs were fed a 20% crude protein ration ad libitum from approximately 21 days of age until weaning at approximately 56 days of age.

Blood samples were collected from the sows in heparanized syringes by vena cava puncture on the 28th, 70th and 112th day of gestation and the 28th and 56th day of lactation. Red blood cell selenium-75 uptake and plasma and whole blood stable selenium was determined as previously described. For each sow, records were kept of the number of pigs born, number of live pigs born, number of live pigs after 3 days, number of pigs weaned and the birth weight and weaning weight of each pig.

Experiment #3

After weaning, the progeny of the sows in experiment #2 were placed on a 16% crude protein diet until they reached 40 kg. At that time, seventy-two of the barrows and gilts were randomly selected and allotted by weight and sex to one of the six dietary treatments used in experiment #1. The pigs were weighed every seven days until they reached 100 kg at which time they were slaughtered.

After 24 hours of fasting, the finished hogs were weighed, stunned by electric shock and killed by exsanguination. Blood samples were taken as the blood drained from the heart and major blood vessels. The whole blood was analyzed for stable selenium and hematocrit and the plasma analyzed for copper, zinc and selenium as described previously.

The hair was partially removed from the carcasses mechanically and the remainder by hand. The carcasses were hung by the achilles tendon. The head, front feet and viscera were removed and the carcasses before being chilled 24 hours at $4^{o}C$.

The chilled carcasses were measured for length from the 1st rib to the aitch bone and fat thickness over the 1st rib, last rib and last lumbar rib.

The mean of the three fat measurements was used as an overall average of fat thickness for each carcass.

After the loins were removed from each carcass, a 2.5 cm chop was removed at the 10th rib of the left loin. Loin eye area was measured to the nearest .05 cm^2 by tracing the muscle outline on acetate paper and following the outline with a planometer. The chops were covered with cellophane to prevent moisture loss before being placed in a lighted cooler at $4^{o}C$. After 1, 24, 96, and 168 hours of light exposure, the color of each chop was evaluated using a Hunter Color Difference Meter. The X, Y, and Z coordinates were mathematically coverted to x and y values as prescribed by C.I.E. procedure (Color-Eye Instruction Manual, 1967).

After 168 hours at $4^{o}C$, the excess fat was removed from each chop and the longissimus muscle was ground and mixed by twice passing through a .3 cm grinding plate. Duplicate 2g samples of longissimus muscle were dry ashed and analyzed for copper and zinc concentrations and 10g samples were wet ashed in 40 ml of concentrated nitric acid prior to selenium analysis (A.O.A.C., 1975). Wet ashing permitted greater uniformity in

sampling which is crucial when analyzing small samples. Dry matter and ether extract were determined on duplicated 3g samples (A.O.A.C., 1975) and oxidative rancidity of two 5g aliquots evaluated (Turner et al., 1954). The thiobarbituric acid test for oxidative rancidity involved boiling the 5g samples for 30 minutes in 10 ml of 20% trichloracetic acid in 2M phosphoric acid and 5 ml of .01M thiobarbituric acid. The solution was cooled in an ice bath and the excess fat removed. After centrifugation at 2400rpm for 10 minutes, the optical density was read at 538 um.

II. STATISTICAL ANALYSIS

The results were analyzed statistically according to the nature of the data. The Statistical Analysis System (SAS) was utilized for computational purposes.

Significant differences between treatments for the growing-finishing hogs were determined by analysis of variance (Steel and Torrie, 1960) using the following model:

$Y_{ij} = u + t_i + e_{ij}$ where Y_{ij} is the Jth pig in the ith treatment,

u is the overall mean

t_i is the treatment where i = 1, ...6.,

e_{ij} is the error term.

Significantly different means were separated by using Duncan's Multiple Range Test (Steel and Torrie, 1960).

Data from the sows was evaluated by analysis of variance using the following similar model:

$Y_{ijk} = u + t_i + B_j + e_{ijk}$ where Y_{ijk} is the kth sow in the ith treatment at the jth bleeding time,

u is the overall mean

t_i is the treatment where i = 1, ...4.,

B_j is the bleeding time where j = 1, ...5.,

e_{ijk} is the error term.

Duncan's Multiple Range Test was used to separate significantly different means.

CHAPTER IV

RESULTS

I. EXPERIMENT #1

A summary of the blood data for the growing-finishing pigs is presented in Table II. No significant differences were found between treatments for any blood parameter. Average daily gain for the growing period (20-60 kg), finishing period (60-100 kg), and combined average daily gain is given in Table III. These data were based on 47 observations. One pig in the control group lost weight over a 14 day period as the result of illness unrelated to diet and the corresponding average daily gain value was, therefore, deleted from the analyses. In the growing period pigs fed the basal plus selenium diet had a significantly lower average daily gain than those fed the basal plus selenium plus copper diet. The great variability within the basal plus selenium group, however, suggests the depression of growth rate may have been of nondietary origin. No significant differences between groups were found during the finishing period or in the overall growing-finishing period.

II. EXPERIMENT #2

The effects of treatment and sampling time on the selenium status of sows as measured by RBC^{75} selenium uptake, plasma selenium and whole blood selenium is given in Tables IV, V and VI. Since the treatment X sampling time interaction was not significant, the data was pooled by

TABLE II

EFFECTS OF TREATMENT ON BLOOD SELENIUM, COPPER, AND ZINC ON GROWING-FINISHING SWINE (EXPERIMENT #1)

	Blood Analysis					
Treatment	% ^{75}Se Uptake	Plasma Se(mg/l)	Plasma Cu(mg/l)	Plasma Zn(mg/l)	Whole Blood Se (mg/l)	Hematocrit
Basal	6.8	.197	2.08	1.42	.241	42.1
Basal plus Se	5.8	.221	2.25	1.12	.274	40.8
Basal plus Cu	5.0	.202	2.09	1.29	.244	41.4
Basal plus Zn	6.4	.192	2.11	1.29	.277	41.9
Basal plus Se plus Cu	5.2	.211	2.06	1.52	.249	41.8
Basal plus Se plus Zn	7.0	.217	2.06	1.37	.312	43.1

TABLE III

EFFECTS OF TREATMENT ON GROWTH RATE OF GROWING-FINISHING SWINE (EXPERIMENT #1)

	ADG (kg/day)		
Treatment	Growth	Finishing	Total
Basal	.70	1.04	.86
Basal plus Se	.62[a]	.97	.80
Basal plus Cu	.69	1.07	.86
Basal plus Zn	.75	1.06	.89
Basal plus Se plus Cu	.79[b]	.91	.85
Basal plus Se plus Zn	.74	.89	.82

[a,b]Means with different superscripts withing columns are different ($P<.05$).

TABLE IV

EFFECTS OF TREATMENT AND DAY ON SOW PLASMA SELENIUM

Plasma selenium (mg/l)

Treatment	Gestation			Lactation	
	28	70	112	28	56
Basal corn-SBM	.330[a]	.247[a,b]	.235[a,b]	.204[b]	.272[a,b]
Basal corn-SBM-tankage	.310[a]	.261[a,b]	.228[b,c]	.193[c*]	.289[a,b]
Basal corn-SBM plus Se	.288	.294	.242	.233[+]	.258
Basal corn-SBM-tankage plus Se	.285[a]	.280[a]	.295[a]	.212[b]	.284[a]
Mean	.301[a]	.270[a,b]	.248[b]	.211[c]	.276[a,b]

[a,b,c] Means with different superscripts within a line are different (P<.05).

[+,*] Means with different superscripts within a column are different (P<.05).

TABLE V

EFFECTS OF TREATMENT AND DAY ON SOW WHOLE BLOOD SELENIUM

Whole blood selenium (mg/l)

Treatment	Gestation			Lactation	
	28	70	112	28	56
Basal corn-SBM	.344^{a}	.309^{a,b}	.387^{a}	.236^{b}	.308^{a,b}
Basal corn-SBM-tankage	.343^{a}	.332^{a}	.350^{a}	.288^{b*}	.298^{a}
Basal corn-SBM plus Se	.322^{a,b}	.374^{a}	.382^{a}	.283^{b+}	.300^{a,b}
Basal corn-SBM-tankage plus Se	.326^{a,b}	.310^{a,b}	.354^{a}	.273^{b}	.276^{b}
Mean	.335^{a,b}	.331^{b}	.369^{a}	.256^{c}	.295^{d}

^{a,b,c}Means with different superscripts within a line are different (P<.05).

$^{+,*}$Means with different superscripts within a column are different (P<.05).

TABLE VI

EFFECTS OF TREATMENT AND DAY ON SOW ^{75}SELENIUM RBC UPTAKE

Selenium-75 uptake (% of dose)	Gestation			Lactation	
	28	70	112	28	56
Basal corn-SBM	4.1^{a}	4.9^{a}	4.7^{a}	10.1^{b+}	10.4^{b}
Basal corn-SBM-tankage	3.8^{a}	4.8^{a}	4.9^{a}	6.8^{a,b}	10.4^{b}
Basal corn-SBM plus Se	4.0^{a}	4.9^{a}	6.5^{a+}	7.5^{a,b}	10.7^{b}
Basal corn-SBM-tankage plus Se	3.3^{a}	5.2^{a,b}	4.5^{a,b}	7.4^{b,c}	8.4^{c}
Mean	3.8^{a}	5.0^{a}	5.2^{a}	7.9^{b}	10.2^{c}

^{a,b,c}Means with different superscripts within a line are different (P<.05).

$^{+,*}$Means with different superscripts within a column are different (P<.05).

sampling time and included in Tables IV, V and VI (pp. 35, 36 and 37) as "mean." Lactation tended to increase the demand for selenium as reflected in all 3 blood parameters while treatment had no significant effects on any parameter except during the period 3 days before and 28 days after parturition.

RBC selenium-75 uptake increased dramatically during lactation, after remaining relatively constant throughout gestation. RBC selenium-75 uptake was higher ($P<.05$) for all 4 groups 56 days postpartum than at any time prepartum, while at 28 days postpartum only sows on the basal corn-soybean meal diet were significantly different from gestation levels. The only treatment effect in the gestation period was the group of sows on the corn-soybean meal plus selenium diet which has an increased RBC selenium-75 uptake ($P<.05$) relative to other groups on the 112th day of gestation. The basal corn-soybean meal group showed a similar treatment effect just after farrowing. When the data from all 4 groups were pooled by sampling time as shown in Figure 4 and Table VI (page 37), no differences existed during the gestation period for RBC selenium-75 uptake, while significant differences were evident both at 28 and 56 days postpartum.

Plasma selenium levels tended to decrease from breeding through 28 days of lactation before increasing to near gestation levels by 56 days postpartum. Variation among the selenium supplemented groups was less marked with the only significant variation ascribed to sampling time being depressed plasma selenium in the corn-soybean meal-tankage plus selenium sows 28 days postpartum. Plasma selenium levels for sows in

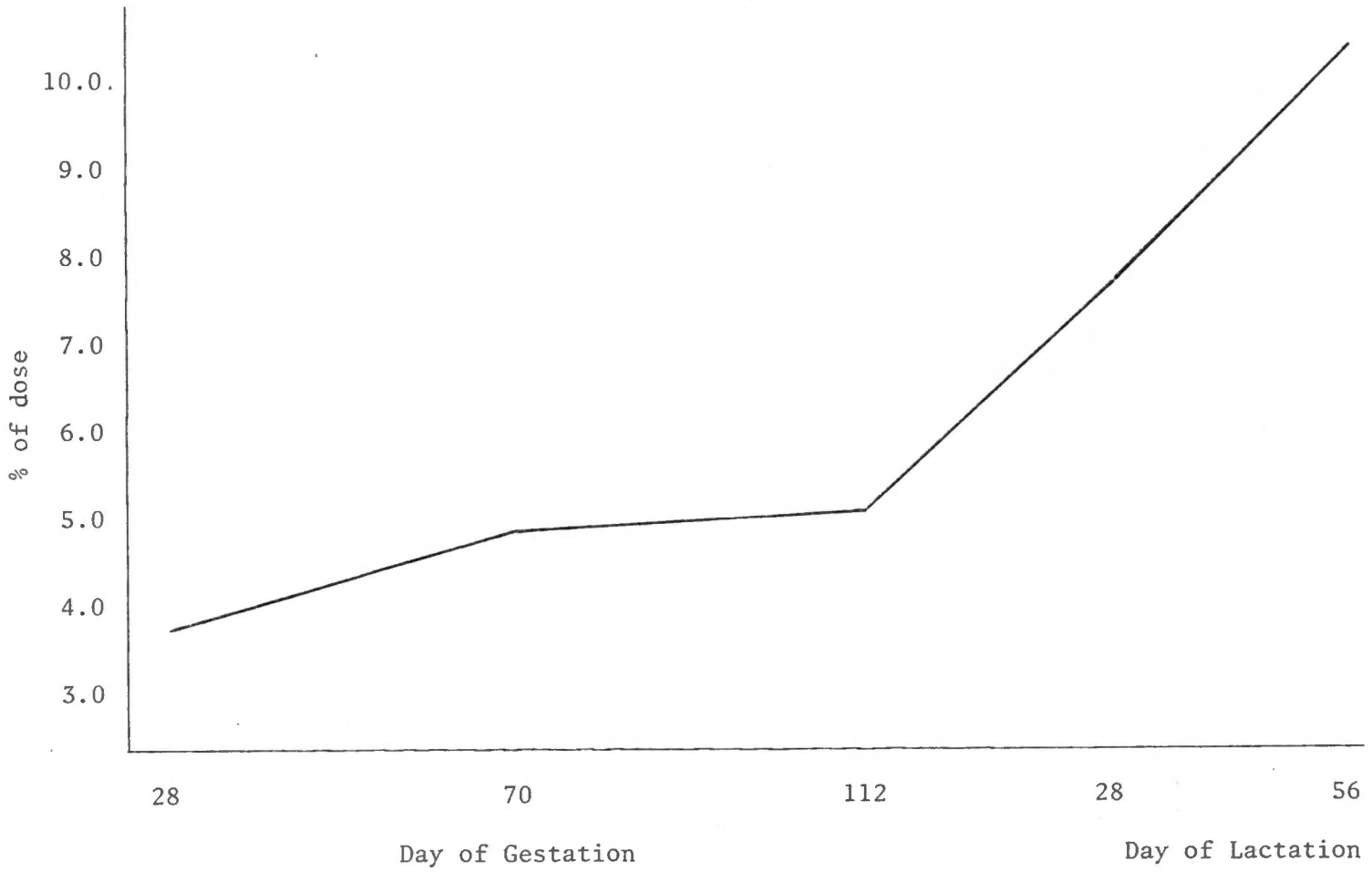

Figure 4. Mean selenium-75 RBC uptake of gestation and lactating sows.

both unsupplemented groups, however, had dropped ($P<.05$) from initial levels by the 112th day of gestation and remained depressed through 28 days postpartum. At the 28th day of lactation the selenium supplemented groups had a significantly higher plasma selenium concentration than the unsupplemented groups. Tankage had no significanct effect on plasma selenium levels. The data, pooled by sampling time, showed a significant decrease in plasma selenium from initial levels by 3 days prepartum (Figure 5) followed by another significant drop during the first 28 days of lactation, before recovery to gestation levels by 56 days postpartum. The recovery may have been due to lower selenium content of the milk (Mahan et al., 1975) and/or decrease milk production.

Whole blood selenium levels remained high throughout gestation before dropping markedly at the 28th day of lactation. As with plasma selenium, whole blood selenium tended to increase from 28 days to 56 days lactation. At the 112th day of lactation, the sows in the tankage groups had significantly higher whole blood selenium levels than those fed diets containing no tankage. Selenium supplementation had a similar effect at the 28th day of lactation, with sows in the selenium supplemented groups having higher ($P<.01$) whole blood selenium concentration than unsupplemented sows. When the data was pooled by sampling time (Figure 4, page 39), the data showed that the whole blood selenium levels were relatively constant during gestation, dropped dramatically during the first 28 days of lactation, and recovered toward gestation levels by 56 days postpartum. Whole blood selenium levels 56 days postpartum, however, were still less ($P<.05$) than gestation levels.

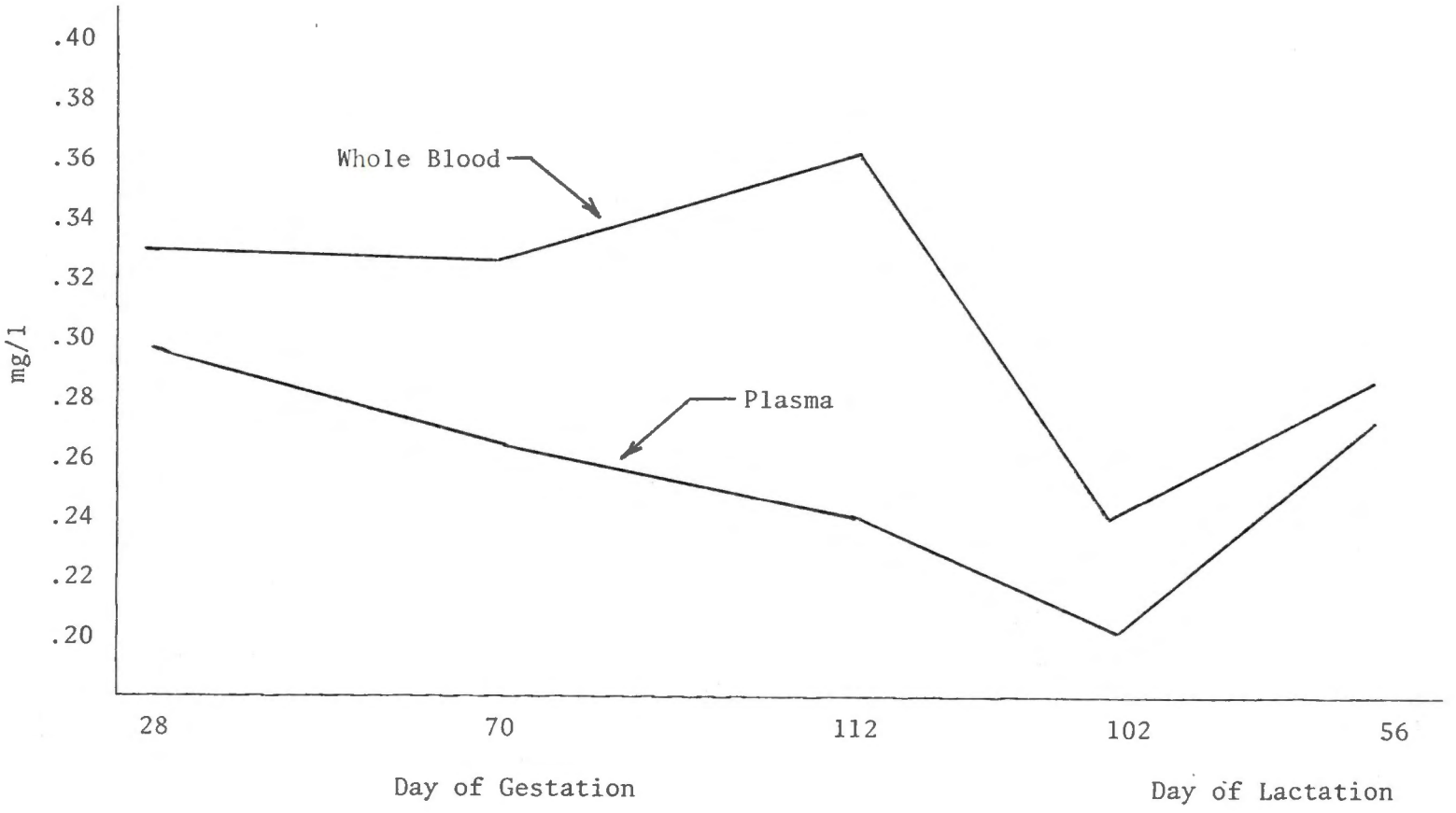

Figure 5. Mean plasma and whole blood selenium levels in gestating and lactating sows.

Sow reproductive performance is summarized in Table VII. The only significant difference between groups was a lower weaning weight for the pigs from sows on the basal corn-soybean meal diet. When the weaning weights were expressed as the weight per day of age, however, there were no significant differences although supplemented pigs tended to have a slightly higher weight per day of age at weaning.

III. EXPERIMENT #3

A summary of the carcass data is presented in Table VIII. No significant treatment differences were found for live weight, hot carcass weight, carcass weight as a percentage of live weight, length, loin eye and fat thickness.

The blood data from experiment #3 is summarized in Table IX. Whole blood selenium and plasma copper were not significantly different for any treatment. Plasma zinc tended to be higher in the zinc supplemented groups, although the results were not distinct enough to permit conclusions on the exact nature of the effects of zinc supplementation on plasma zinc concentrations. However, the effects of selenium supplementation on plasma selenium concentration were obvious with the three selenium supplemented groups having significantly higher plasma selenium levels than the three unsupplemented groups.

Selenium supplementation had a similar effect on longissimus selenium concentration (Table X). Again, all three selenium supplemented groups had high ($P<.05$) selenium concentrations than the unsupplemented groups. Selenium supplementation of the dams also resulted in an

TABLE VII

EFFECTS OF TREATMENT ON SOW REPRODUCTIVE PERFORMANCE

Treatment	Average litter						
	Pigs born	Live pigs born	Live after 3 days	Weaned	Birth wt (kg)	Weaning wt (kg)	Wt/Day at weaning (kg)
Basal corn-SBM	11.5	11.2	9.8	8.5	1.5	13.6	.24
Basal corn-SBM-tankage	10.8	10.5	9.7	6.8	1.5	15.4	.25
Basal corn-SBM-plus Se	12.8	11.8	11.0	9.5	1.5	16.9	.27
Basal corn-SBM-tankage plus Se	11.8	9.5	9.2	6.7	1.4	17.1	.28

TABLE VIII

EFFECTS OF TREATMENT ON CARCASS CHARACTERISTICS OF GROWING-FINISHING SWINE

Treatment	Live wt (kg)	Hot carcass wt (kg)	Percent yield	Length (cm)	Loin eye (cm^2)	Depot fat thickness (cm)
Basal	94.5	70.9	75	77.5	33.55	3.25
Basal plus Se	95.5	70.9	74	77.5	33.85	3.20
Basal plus Cu	93.2	68.6	74	76.9	31.40	3.40
Basal plus Zn	95.5	70.0	73	76.5	32.25	3.35
Basal plus Se plus Cu	93.2	69.5	75	76.5	33.85	3.25
Basal plus Se plus Zn	95.5	69.5	73	76.9	32.65	3.60

TABLE IX

EFFECTS OF TREATMENT ON WHOLE BLOOD SELENIUM AND PLASMA COPPER, ZINC AND SELENIUM (EXPERIMENT #3)

Treatment	Whole Blood Selenium (mg/l)	Plasma Selenium (mg/l)	Plasma Zn (mg/l)	Plasma Cu (mg/l)
Basal	.254	.174[b]	.672[a,b,c]	2.68
Basal plus Se	.250	.215[a]	.525[c,d]	2.83
Basal plus Cu	.240	.179[b]	.584[b,c,d]	2.92
Basal plus Zn	.210	.180[b]	.815[a]	2.74
Basal plus Se plus Cu	.267	.221[a]	.498[d]	2.66
Basal plus Se plus Zn	.282	.224[a]	.687[a,b]	2.71

[a,b,c,d]Means within a column with different superscripts are different ($P<.05$).

TABLE X

EFFECTS OF TREATMENT ON LONGISSIMUS MUSCLE CHARACTERISTICS AND MINERAL CONTENT

Treatment	% Dry matter	% Ether extract	Oxidative Rancidity (TBA values)	Wet Basis		
				Copper (ppm)	Zn (ppm)	Se (ppm)
Basal	28.22[b]	4.83[b]	.254	6.18	15.4	.078[b]
Basal plus Se	27.95[b]	4.49[b]	.164	7.31	16.1	.111[a]
Basal plus Cu	28.47[b]	5.46[a,b]	.243	6.49	15.9	.077[b]
Basal plus Zn	29.61[a]	6.17[a]	.242	5.54	15.4	.080[b]
Basal plus Se plus Cu	27.84[b]	4.30[b]	.236	5.55	15.1	.097[a]
Basal plus Se plus Zn	28.32[b]	4.77[b]	.185	3.05	14.9	.107[a]

[a,b]Means within columns with different superscripts are different ($P<.05$).

increased (.097 vs. .082 ppm) longissimus selenium concentrations ($P<.05$). The sow effect was additive with the supplemented progeny of supplemented sows having the highest longissimus selenium content and the unsupplemented progeny of unsupplemented sows having the lowest selenium levels ($P<.05$). The pig treatment is the more dominant factor with the supplemented pigs of unsupplemented sows having higher longissimus selenium levels than the unsupplemented pigs of supplemented sows (Table XI).

Longissimus color was not significantly different for any group at any time during the study. No treatment differences were found for longissimus copper or zinc concentrations though marked variation within groups was noted. Muscle dry matter in pigs fed the basal plus zinc diet was significantly higher than in all other groups, and ether extract was higher ($P<.05$) in the basal plus zinc group than in any group except the basal plus copper fed pigs (see Table X, page 46). No significant difference between groups were noted in oxidative rancidity. However, the wide variation in the measurement of oxidation within samples suggests that the sampling procedure was inadequate.

Average daily gain was significantly higher in the basal plus zinc fed pigs than in other groups (Table XII). The increase appears to be due to greater feed consumption, however, as feed efficiency was not significantly different between groups.

TABLE XI

EFFECT OF GROWING-FINISHING TREATMENT AND SOW TREATMENT ON LONGISSIMUS SELENIUM CONCENTRATION

Pigs supplemented	Sows supplemented	Se (ppm)
YES	YES	.108[a]
YES	NO	.098[a,b]
NO	YES	.084[b,c]
NO	NO	.071[c]

[a,b,c]Means with different superscripts are different(P<.05).

TABLE XII

EFFECTS OF TREATMENT ON AVERAGE DAILY GAIN AND FEED EFFICIENCY IN GROWING-FINISHING SWINE (EXPERIMENT #3)

	Average daily gain (kg)	Feed efficiency (kg feed/kg gain)
Basal	.78[b,c]	3.10
Basal plus Se	.73[c]	3.21
Basal plus Cu	.79[b,c]	3.09
Basal plus Zn	.86[a]	3.10
Basal plus Se plus Cu	.79[b,c]	2.98
Basal plus Se plus Zn	.80[b]	3.08

[a,b,c]Means within a column with different superscripts are different ($P<.05$).

CHAPTER V

DISCUSSION

The results of these experiments indicate some positive aspects of selenium supplementation of swine diets above the .15 ppm level. Mahan et al. (1975) clearly showed the adverse effects of selenium deficiency on sow reproductive performance by using diets naturally low in selenium content over several successive reproductive cycles. While reproductive performance was not significantly affected by selenium supplementation in these experiments, the sows were evaluated over only one reproductive cycle and the basal diets contained .08 ppm selenium. Furthermore, the high plasma and whole blood selenium concentrations of the supplemented sows during lactation suggests that the additional dietary selenium was utilized by the sows in maintaining blood selenium levels and possibly in maintaining body stores of selenium. With advances in reproductive physiology which could permit rebreeding a few days after parturition, the selenium status of sows could become crucial to the ultimate long-term success of such an enterprise.

The effects of supplemental selenium fed to sows on muscle selenium levels of their progeny is also important. Muscle tends to be one of the most stable metabolic pools of many micronutrients and the acute involvement of muscle in the pathology of selenium deficiency suggests that the increased longissimus selenium levels afforded by improved selenium nutrition of the sow may be most important in many instances.

While some increase in pig tissue selenium levels could occur during gestation, consideration of the changes in body size suggest that the cause of the higher muscle selenium levels is an increased selenium content in the milk of selenium supplemented sows. The higher plasma and whole blood selenium concentration of the supplemented sows during lactation indicates that additional selenium is available for milk production and evidence suggests it appears in the milk (Mahan et al., 1975; Mahan, 1975). Furthermore, the weight of a pig increases approximately 10 times from birth to weaning. Any increase in tissue selenium levels would be negated if the constitution of milk were not a factor. A similar "dilution" effect is not as prominent in the muscle of growing-finishing pigs due to the stability of the muscle selenium pool and a change in body size of only about 6 times. Thus, the increased longissimus selenium concentration of the progeny of selenium supplemented sows in these experiments persisted at slaughter weight.

Much of the extra selenium which appears in the plasma, whole blood and milk of the selenium supplemented sows, and ultimately in the longissimus muscle of the sows progeny, may be in the form of selenoamino acids and/or selenoproteins. Selenium-75 has been shown to appear rapidly in a variety of proteins (Frost, 1975) and as such would probably form a more stable selenium pool, since excess selenite-selenium is excreted in the urine and feces. However, the exact nature of selenium in the various body pools remains unknown.

The increased selenium levels in swine muscle could have serious implications for human nutrition. The levels of selenium observed are

certainly not potentially toxic. Even chronic selenium toxicity requires a minimum of approximately 3 ppm, which gives a safety factor of 30 times. Furthermore, Frost (1975) believes selenium deficiency is a worsening problem in human nutrition. Citing a possible relationship between selenium and coronary heart disease, cancer, ubiquinone biosynthesis, and the immune response, Frost urges the immediate investigation of human selenium nutrition. If Frost's hypothesis is valid, the increase in available selenium provided by longissimus muscle from selenium supplemented pigs and the progeny of selenium supplemented sows may become a valuable source of extra selenium for humans.

The effects of selenium supplementation of growing-finishing diets on plasma selenium levels of pigs is also reflected in these experiments. Although the effects were not significant in experiment #1, the three supplemented groups had higher plasma selenium levels than the unsupplemented groups. In experiment #3, the treatment differences were highly significant ($P<.0001$) suggesting that had more pigs been included in experiment #1, the differences would have been statistically significant.

As reported by other workers (Groce et al., 1973; Hitchcock, 1973) selenium supplementation had no effect on average daily gain and feed efficiency. The failure of supplemental dietary copper to improve these growth characteristics or alter any blood, carcass or meat parameter evaluated may have been due to the addition of only 125 ppm copper. Wallace (1968a) suggested that 250 ppm copper and 100 ppm zinc were necessary for the maximum effect of copper supplementation. Since the basal diets contained 80 ppm zinc, it would appear that zinc was not the

limiting factor in preventing copper related effects. While 250 ppm copper may have given quite different results, the nature of the iron-copper may be detrimental (Gipp et al., 1973).

The variable effects of zinc proteinate on average daily gain observed in these experiments has previously been reported (Elgin, 1975; Masincupp, 1974).

The increased dry matter and ether extract observed in the zinc supplemented pigs in experiment #3 is probably an artifact of the more rapid growth and not a true zinc effect. However, the increased dry matter is probably due to the increased ether extract, since ash and protein generally remain constant while fat varies inversely with water content in muscle.

The effects of copper, zinc and selenium on lipid oxidation in porcine muscle remains unclear. In experiment #3, an attempt was made to evaluate short term effects of these three microelements. While no significant effects were noted in these studies, the method of measurement lacked sufficient sensitivity to permit any conclusions. Furthermore, muscle contains little fat relative to many processed meats and the lipids found in muscle tend to be more saturated than those found in depot fat and meat products containing such fat. Long-term shelf life of frozen and/or processed pork may depend on the concentration and chemical nature of many micronutrients and deserves further investigation.

CHAPTER VI

SUMMARY

One hundred-twenty Duroc barrows and gilts and twenty-four Duroc sows were utilized in three experiments to evaluate the effects of selenium on swine and the effects of copper and zinc on growing-finishing swine.

In experiment #1, forty-eight Duroc barrows and gilts, allotted by weight and sex to six dietary treatments, exhibited no significant variation due to treatment for RBC selenium-75 uptake, plasma selenium, copper and zinc, whole blood selenium, hematocrit, or average daily gain. The six treatments were: basal 16% crude protein diet; basal plus .1 ppm selenium; basal plus 125 ppm copper; basal plus 80 ppm zinc; basal plus .1 ppm selenium plus 125 ppm copper; and basal plus .1 ppm selenium plus 80 ppm zinc. The basal diet contained .08 ppm selenium, 12.5 ppm copper and 82 ppm zinc.

In the second experiment, four groups of six Duroc sows were fed basal-corn-tankage diets each with or without .1ppm supplemental selenium for one gestation and lactation. RBC selenium-75, plasma selenium, and whole blood selenium of the sows was measured periodically. At the 28th day of lactation, the selenium supplemented sows had higher plasma ($P<.05$) and whole blood ($P<.01$) selenium concentrations than the unsupplemented sows. Seventy-two of the progeny of these sows were allotted to treatments as in experiment #1. The selenium content of the last rib

longissimus muscle was increased by either supplemental selenium in the sow diet ($P<.05$) or the growing-finishing diet ($P<.001$). Plasma selenium concentration responded to additional selenium in the growing-finishing diet ($P<.05$). Average daily gain was increased ($P<.05$) in the zinc supplemented pigs.

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