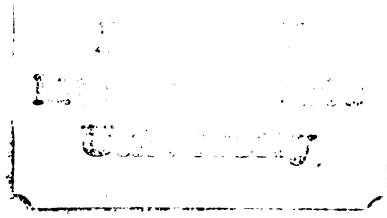

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thesis entitled

MASTITIS METRITIS AGALACTIA IN SWINE:

Role of Vitamin E and Selenium

presented by

Oswaldo E. Vale

has been accepted towards fulfillment
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Major professor

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MASTITIS METRITIS AGALACTIA IN SWINE:

Role of Vitamin E and Selenium

by

Oswaldo E. Vale

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ABSTRACT

MASTITIS METRITIS AGALACTIA IN SWINE:

Role of Vitamin E and Selenium

by

Oswaldo E. Vale

The role of vitamin E and selenium (Se) fed to gilts during gestation and lactation in preventing mastitis-metritis-agalactia (MMA) and death losses in baby pigs was determined in 10 gilts fed a basal diet in comparison to 9 gilts fed the same diet supplemented with vitamin E and Se. The corn used in the basal diet was initially high moisture shelled corn that had been stored anaerobically for several months, then dried artificially and used as dried shelled corn. Gilts fed the basal diet had more clinical signs of MMA, and farrowed and weaned smaller litters in which individual pigs were lighter in weight at birth and at 21 days of age than were baby pigs nursing gilts fed supplemental vitamin E and Se. Plasma vitamin E, Se, and glutathione peroxidase (GSH-Px) values were lower at birth and at 21 days in pigs nursing gilts fed the basal diet.

Dedicated with love to my parents,
my wife, María, and my children, María Alejandra
and Oswaldo Rafael, who encouraged me
to finish this study.

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INTRODUCTION

The mastitis-metritis-agalactia (MMA) complex is a serious and economically important disease in swine throughout the world. Swine producer associations and research groups give a high priority to additional research on MMA.[1] The disease occurs at parturition, and it was clinically so named in the USA because of the 3 main clinical signs present,[2] and referred to by the acronym MMA. However, in recent years, agalactia has been considered as the primary disturbance which directly or indirectly contributes to death losses in young pigs from birth to weaning. Illinois workers estimated that these losses account for about 75% of the preweaning losses, and amounted to over $150 million annually.[3]

The MMA complex was especially troublesome to swine producers during the 1960's. In recent years, swine practitioners and producers mention that, while MMA is still an importan disease, its incidence seems to have declined.[4] This decline may be due in part to the increased use of vitamin E and Se in swine rations. In the early 1970's, it was established that growing pigs required supplemental vitamin E and Se to prevent excessive death losses.[5-7] Thus, a carry-over effect of a vitamin E and Se deficiency to amturity may have had an effect on improving reproduction.

Whereas it is well-established that the currently used corn-soybean diet fed to growing pigs requires supplemental vitamin E and Se,[8,9] there is limited information on the requirements of these nutrients during reproduction-lactation. Vitamin E has been known to be essential in reproduction, particularly in laboratory type animals for over 60 years.[10]

Many factors have been suggested as causes of MMA.[2,4,11,12] Infectious agents were initially incriminated because of the clinical signs present and the specific pathogens isolated from natural causes.[11-16] However, attempts to reproduce MMA by giving these agents to susceptible sows were unsuccessful.[4,15,17-19] Thus, the cause is not clearly established.[17,18] A fundamental contribution to the understanding of a disease is the experimental reproduction of it as it occurs naturally. This has not been achieved for MMA in sows.

I selected research on MMA in swine to obtain training which would have direct application to my teaching and research activities in Venezuela. Only a limited amount of research has been conducted on MMA. The facilities, cooperation and expertise were available at Michigan State Universidty to do research on MMA.

LITERATURE REVIEW

Raising swine is an intensive type of livestock production, where profitability depends upon rearing large, healthy litters.[20] Newborn pigs are more prone to the hazards of diseases than other livestock. Important factors contributing to baby pig losses are nutritional deficiencies and infectious agents, or a combination of both.[21,22] Proper nutrition for baby pigs begins with an adequate nutrition for the sow during gestation and lactation. A voluminous amount of literature is available on factors causing death losses in baby pigs.[21,22] This literature review emphasizes an important disease (MMA) occurring in swine at parturition which contributes to significant death losses in young pigs.[3]

History and Incidence

During the late 1960's and early 1970's, American investigators described a prevalent and important disease in swine that occurred at or shortly after parturition. Prior to this time, isolated individual case reports of parturition disorders in sows were described. The early U.S.A investigators' attention as to treatment and prevention of the disease was focused on the most obvious

clinical signs of mastitis and metritis, and the agalactia received less attention.[4,14-16] Some controversy has existed on the disease through the years as to its cause and clinical signs.[18] This may be because of variation in clinical signs as influenced by management practices, nutritional programs, environment, and other factors.[11,12,18] Details of a 10-year investigation on a similar post-parturient problem were published in 1960 by Dr. Ringarp, at the Royal Veterinary College in Stockholm, Sweden.[11] This report was initially overlooked by early American investigators.[13-16] In Sweden, the predominant symptom was also agalactia, and the disease was called "agalactia toxemia."[11.12] Most investigators in the U.S.A. now agree that the MMA complex is primarily a lactation failure.[4,17-19]

The incidence of agalactia is difficult to determine. Backstrom and Morkoc,[4] in a recent review article, stated that in most countries during the 1960's, 7 to 17% of farrowing sows had the disease. In the U.S.A., MMA was ranked as the number one disease problem in swine production.[4] Missouri workers reported an incidence of 13.1% of swine agalactia.[23] The Michigan State University swine herd was reported to have an MMA incidence of 21.2% in 1969. This was based on a clinical diagnosis of 1.4% mastitis, 19.3% metritis, and 0.8% agalactia.[24] There is a tendency for the incidence of MMA to increase with the number of farrowings.[4,11,25] The highest incidence occurs during

the 3rd and 4th farrowings.[4,11] While there are some variations, the disease occurs at all seasons of the year.[11] Ringarp correlated the highest incidence (88.5%) of "agalactia toxemia" with poor feeding programs, but no specific deficiency could be demonstrated.

Clinical Signs

The clinical signs usually appear in the sow as soon as parturition is completed. It is not well-established as to which of the clinical signs is most important. There is agreement that the sow first appears restless and nervous.[4,11,17-19,21,22] Other miscellaneous clinical signs include inappetence, constipation and recumbency. Some sows may lie on their udders, preventing the pigs from nursing, but other sows may attempt to nurse the piglets.[18,19]

Any of the clinical signs of mastitis and metritis are highly suggestive of the disease. The mastitis (with or without metritis) is clinically characterized by edema, firmness, increased temperature and red or purple skin of the mammary glands.[4,18,19] The metritis referred to is characterized by the presence of a uterine mucopurulent discharge which is thicker than normal, and increased in quantity.[4,11,18,19] The normal uterine discharge in the sow is also a mucopurulent type of exudate, but scanty and of smaller volume.[11] A wide range of body temperatures

in sows with MMA has been reported, but this seems to be unreliable in diagnosing the disease.[11]

Clinically, one or all signs may be present, and variation of symptoms exists from sow to sow.[11,18,19] This makes the disease difficult to diagnose. The main aspect of MMA is a lactation failure during the first 24 to 60 hours post-farrowing which is not readily detectable.[11,18,19] Most authors emphasize that careful observation of the piglets and sow during nursing is essential to determine if milk is available for the litter in order to avoid losses from starvation.[4,18,22]

Microbiology

The microflora associated with MMA in sows has been well-established by isolating and identifying microorganisms from naturally occurring cases.[11,12,13,15,26,27] The microorganisms most commonly isolated were enterobacteriacea.[4,11,14-18] Among the isolates, Escherichia coli (E. coli) and Klebsiella occurred most frequently.[11,13,15,16] Escherichia coli has been consistenly found, but isolation of serotypes indicates that it is not the primary pagthogen.[15] As dicussed later, attempts to reproduce MMA using mainly E. coli have not been successful.[4,17,18] Thus, the pathogens isolated might have been secondary to other primary causes.

Other miscellaneous microorganisms occurring in isolated cases of MMA were proteus and staphylococcus.[13,15,16] Texas workers reported Mycoplasma hyogenitalium to be a cause of MMA in sows.[27]

Pathology

Sows with MMA rarely die, unless a severe toxic septicemia occurs.[18] Therefore, there are no known reports of characteristic lesions correlated with death. Some workers reported on the lesions of sows with MMA that were slaughtered.[11,25,28,29]

Grossly, mastitic changes and generalized edema of the udder were present.[11,25,29] The mammary glands were inflamed, and small foci of necrosis with serohemorrhagic or purulent exudate were noted. Necropsy of 14 sows from spontaneous cases of agalactia revealed that they were in a good state of nutrition.[11] There was subcutaneous and extensive edema of the mammary glands, and excessive serous fluid mixed with blood and fibrin present in abdominal and thoracic cavities. In more severe cases of mastitis, the udder had yellow gelatinous masses in the interstitial and subcutaneous tissue. Those cases with metritis had edematous and hemorrhagic uterine endometrium with variable quantities of mucopurulent fluid. Hepatosplenomegaly was also present.[11]

Histologically, there was marked vacuolation of uterine endometrium and mammary alveolar epithelium. Necrosis and exfoliation of the lining alveolar epithelium were also present in the mammary tissue. In other organs and tissues, there was vacuolating hepatopathy and centrolobular non-reactive necrosis of hepatocytes.[11] Fatty changes were present in the adrenal glands and the myocardium, and there was a waxy degeneration of skeletal muscle fibers. Similar lesions to the above were present in 1 sow slaughtered from 7 experimentally reproduced cases.[11] Purdue University investigators reported that, in 18 sows with MMA, the lesions were very similar to those as described by Dr. Ringarp.[29]

Experimental Production

Many attempts have been made to reproduce MMA in sows in order to clarify the cause and pathogenesis.[11,12,15,17,18] Because of the complexity of the problem and diverse opinons on factors involved, various approaches were made to reproduce the disease.

In the U.S.A., Midwestern investigators attempted to reproduce MMA or agalactia by using either coliform bacteria[15,17] isolated from natural cases, or E. coli endotoxins.[17,30,31] Iowa workers reported that intravaginal infusion of E. coli isolated at necropsy from 14 natural cases of MMA produced uterine infection in 13 of 16 sows, but clinical evidence of agalactia, mastitis or profuse

vaginal discharge was not observed.[15] In later work, Iowa researchers noted that, in the necropsies of 13 agalactic sows, coliforms were the most common bacteria associated with mastitis.[14] Other researchers reported on reproducing MMA in a small number of cases by intranasal and intravenous inoculation of cultures of Mycoplasma hyogenitalium.[25] The role of E. coli endotoxins on MMA was investigated by Wisconsin workers.[30] They injected E. coli endotoxin into the mammary gland and produced clinical changes similar to those noted in natural cases of agalactia. Workers at Illinois also noted the E. coli endotoxin resulted in marked suppression of prolactin and reduced piglet growth rate.[4,18] In contrast to the Illinois reports, Missouri workers infused E. coli endotoxin into the jejunum of 12 postparturient sows, and they exhibited no clinical signs of endotoxin absorption or lactation failure. There was no indication of endotoxemia on gross pathology or light microscopy.[32]

Ringarp also considered the role of microorganisms in agalactia toxemia.[11] He examined 127 uterine tampons and 167 milk samples from agalactic sows. The main bacteria isolated were coliforms and β-hemolytic streptococci. He concluded that no uniform infection could be demonstrated. Ringarp, because of field observations on the incidence of agalactia previously mentioned, gave more attention to the role of the diet in attempts to reproduce the disease

by feeding a poor quality feed. The feed (wheat) that he used had been damaged during harvest, and was unsuitably stored. He purchased this feed from a farm in which all 6 sows developed the disease when they farrowed during a 2-month period. The farm had no previous history of agalactia toxemia. Ringarp, using this feed experimentally, reproduced the disease in 7 of 9 sows.[11]

Importance of Vitamin E and Selenium In Swine Production and Diseases

Vitamin E was first recognized in 1922, and chemically identified in 1938.[33] This vitamin is known to have an antioxidant effect against lipid peroxides,[34-36] and for many years a deficiency has been associated with myopathy, reproduction problems, and infertility in livestock and poultry.[37] Selenium was demonstrated to be an essential micronutrient in 1957.[38] It functions through GSH-Px at subcellular levels within the mitochondria to protect the cells from peroxidative damage.[39] Thus, these nutrients work together to avoid peroxidation of vital phospholipids in cellular and subcellular membranes to preserve the integrity of tissues.[40]

The role of vitamin E and Se in swine production and diseases has been recently summarized.[41] They appear to function primarily as *in vivo* antioxidants,[35,36,39] and their requirements in swine are influenced by a number

of factors.[42] Increased resistance of chicks and lambs to E. coli and chlamydia was reported when vitamin E was supplemented in the diet.[43] Supplementation of this vitamin in swine rations was reported to enhance the immune response to E. coli bacteria.[44] Ohio investigators reported a reduced pig mortality at weaning and increase in the immune humural response when vitamin E and Se were given to weanling swine.[45] Lowered levels of GSH-Px activity and decreased microbicidal ability in circulating leukocytes and pulmonary macrophages wer reported in vitamin E and Se deficient animals.[41]

In addition, experimental work supports the theory that supplementation of these 2 nutrients increases the resistance of pigs to swine dysentery. In 3 experiments, the incubation period was shorter, and the severity of lesions was more pronounced in deficient pigs than in those supplemented with vitamin E and Se.[46,47] This research suggests that these 2 nutrients play an important role in the mechanisms of defense against disease.

The Role of Vitamin E and Selenium

Prior to about 1970, vitamin E and Se were assumed to have an unimportant role in practical swine production.[48] This was summarized in the 1968 Nutrient Requirements of Swine with the statements, "The biochemical functions of

selenium have not been clarified," and "It is unlikely that practical swine diets would be deficient in vitamin E unless the diet contained excessive amounts of highly unsaturated fatty acids or oxidized fats; therefore, a supplemental source is not needed." Following the 1969 and 1970 reports from Michigan State University[5,6] and Purdue University[7] researchers on the importance of these 2 nutrients in preventing death losses in young pigs under practical conditions, the 1973 Nutrient Requirements of Swine[49] states that "It is suggested that 11 IU of vitamin E be added per kilogram of diet until more specific information is obtained." In addition to the reports on the importance of vitamin E and Se in growing pigs, Michigan State University investigators reported that the supplementation of these nutrients in swine herds under field conditions reduced the incidence of MMA in sows.[6] Experimental evidence was also available which indicated that vitamin E supplemented to sows reduced the incidence of MMA from 50 to 14%, and the addition of vitamin E also increased the survivability of baby pigs to 3 weeks of age.[50,51]

Many investigators have questioned the importance of MMA during the past decade. Some authors mentioned various factors that have changed in modern swine production,[11,20,30,42] among them, the use of improperly harvested and stored feed.[11,42,52]

Iron and Vitamin E in Swine

Iron (Fe) is a vital element for cellular respiration and oxygen transport in the tissues, and forms part of the respiratory enzymatic systems.[53] Pigs have a basic needs for Fe at birth because of an inadequate dietary source from the sow's milk.[39,54,55,56] Thus, pigs have to be supplemented with Fe in order to avoid depletion of body iron and subsequent anemia.[57]

Iron injections have at times produced death losses in baby pigs.[58-60] These losses were associated with a vitamin E and Se deficiency in baby pigs.[61,62] Evidence on the protection of vitamin E and synthetic antioxidants against iron toxicosis in pigs was reported.[61,63] The relationship of a low vitamin E level and high level of polyunsaturated fatty acids in the diet was observed in baby pigs which had died after Fe administration.[62] In addition, British researchers reported that vitamin E and Se deficient pigs had low tolerance to Fe injections.[64] Iron was demonstrated to increase the rate of free radical peroxidation and to enhance vitamin E requirements.[65] In recent years, there has been a tendency to reduce labor costs in swine production by giving a single large injection of Fe rather than repeated smaller dosages to baby pigs. This has accounted from cases of iron toxicosis in vitamin E and Se deficient pigs under practical conditions.

Vitamin E and Prostaglandin

Vitamin E, in addition to having an antioxidant role *in vivo*,[34-36] has also been reported to be involved in prostaglandin (PG) synthesis.[66] These cyclic oxygenated fatty acids (arachidonic acid) are active biological compounds which function in the maintenance of cellular homeostasis.[66] They also exert significant effects in a number of physiological and pathological processes, such as blood flow and pressure, platelet aggregation, immune response, and gastrointestinal circulatory disturbances.[66-68] Therefore, it is not unlikely that PG is also involved in several endocrinologic processes. Many investigators have emphasized that MMA is related to endocrine imbalances[4,11,17,18] and gastrointestinal disorders,[11,18] resulting in physiological and pathological effects including a lactation failure. Recent reports were published on a decreased incidence of MMA in gilts induced to farrow with prostaglandin $F_2\alpha$.[4] Reduced peristalsis of the gastrointestinal tract (GIT), coprostasis and constipation leading to endotoxemia have been reported in sows with MMA.[11,18] Even though these changes were associated with nutritional disturbances, a role of PG as regulators of gastrointestinal circulation and physiology is possible. Furthermore, several investigators reported on the role of PG in blood circulation in GIT, liver, pancreas and spleen.[67,68] An increase in substances derived from arachidonic acid was also

reported in experimental animals with endotoxic shock.[69,70] Thus, the pathophysiology of MMA appears to involve predisposing factors (vitamin E deficiency), altered endocrine functions and endotoxemia, with infectious agents playing a secondary or aggravating role.[17]

Summary

The literature is clearly supportive that MMA or agalactia toxemia is a widespread, important and complex disease in swine. The cause is not established, although the problem has existed for 20 to 25 years. The dieases is associated with production techniques, especially feeding and processing feeds. Various authors have suggested causative factors based mainly on the history and clinical information. Only limited research has been conducted to reproduce the disease as it occurs naturally. Although infectious agents have not been incriminated as a primary cause, there is evidence that they have a secondary or complicating role. European investigators have incriminated the general role of nutrition, but a specific nutrient deficiency has not been established. In growing pigs, a specific role for vitamin E and Se has been established, and the lesions of the deficiency are not too dissimilar from the lesions described in MMA. In recent years, there has also been evidence that vitamin E has a role in endocrinology, especially in the synthesis of prostaglandin

that are involved in many physiological and pathological events. Thus, research on the role of these nutrients in MMA is justified.

OBJECTIVES

The objectives of this research were:

1. To produce experimentally the MMA complex as it occurs naturally in sows.

2. To determine the role of vitamin E and Se in preventing MMA or reducing its incidence.

3. To evaluate the role of these 2 nutrients in improving the livability and health of baby pigs from birth to 3 weeks of age.

4. To determine the susceptibility of vitamin E and Se deficient neonatal pigs to Fe injections.

5. To monitor plasma levels of vitamin E, Se, Fe and GSH-Px to establish minimum levels expected in a dietary deficiency of these nutrients in sows during gestation, parturition and lactation.

6. To ascertain plasma, vitamin E, Se, Fe and GSH-Px status of baby pigs from birth to 21 days.

7. To determine the role of vitamin E and Se in swine as being of practical value in improving swine production, especially during late gestation and early growth.

MATERIALS AND METHODS

Experimental Diets

A basal high moisture shelled corn-soybean meal diet was fed to 2 groups of Yorkshire x Landrace gilts in the fall of 1982. The high moisture corn was anaerobically stored for 6 to 8 months, then dried, ground and mixed with the other ingredients of the diet. This diet was reported to enhance the production of a vitamin E and Se deficiency in pigs,[51,71] poultry,[72] and MMA in sows.[11,50,51] One group of gilts (10) was fed the basal diet, and the other group of gilts (9) was fed the basal diet supplemented with vitamin E (50 IU/Kg) and Se (0.1 ppm). The composition of diets is given in Table 1.

Experimental Animals

Nineteen crossbred gilts (Yorkshire x Landrace) were divided into 2 groups and fed the experimental diets. Gilts were bred as they came into heat. After mating, gilts were weighed and bled early in gestation, at parturition and at 21 days of lactation, and blood plasma vitamin E, Se, Fe, GSH-Px, and hematology [Hemoglobin (Hgb), Packed Cell Volume (PCV), Mean Corpuscular Hemoglobin Concentration (MCHC)] values were determined. At parturition, data on

TABLE 1. COMPOSITION OF GESTATION-LACTATION DIETS

INGREDIENTS[a]	DIETS[e] MSU BASAL	+ VITAMIN E-Se
DRIED HIGH MOISTURE SHELLED CORN	893	892.32
DEHULLED SOYBEAN MEAL	70	70
MONO-DICALCIUM PHOSPHATE	15	15
CALCIUM CARBONATE ($CaCO_3$)	12	12
SALT	5	5
VTM[b] PREMIX	5	5
VITAMIN E (PREMIX)[c]	---	.18
SELENIUM 90 (PREMIX)[d]	---	.5
Total	1,000	1,000.00

[a]INGREDIENTS ARE EXPRESSED IN PARTS PER THOUSAND.

[b]VTM, VITAMIN TRACE MINERALS PREMIX, AS IN BULLETIN 537, SWINE FEEDS AND FEEDING, MICHIGAN STATE UNIVERSITY, EAST LANSING.

[c]VITAMIN E (PREMIX) CONTAINS 275,000 IU/KG (SUPPLEMENTED TO SUPPLY 50 IU/KG DIET).

[d]SELENIUM 90 (PREMIX) CONTAINS 200 MG/KG (SUPPLEMENTED TO SUPPLY 0.1 PPM).

[e]ON ANALYSIS, MSU BASAL CONTAINED 0.78 µg/g VITAMIN E AND 0.04 µg/g Se. SUPPLEMENTED DIET CONTAINED 56.1 µg/g VITAMIN E AND 0.12 µg/gr Se.

clinical observations, length of farrowing time and rectal temperatures of each gilt were monitored daily.

There were 175 live pigs obtained from 19 litters. Each pig was weighed, ear-notched, taildocked, and given 200 mg or 200 mg of Fe* intramuscularly at birth. Pig weights were also recorded at 21 days of age. Litters were weaned at 28 days of age. All the pigs were bled at birth and at 21 days, and hematology, plasma vitamin E, Se, Fe, and GSH-Px values were determined. The 42 pigs that died from birth to 21 days of age were necropsied and tissues were collected for microbiology and pathological evaluation. Records on stillborn, malformed and dead pigs were also maintained.

Analytical Procedures

Hematology

Bleeding Procedures. Baby pigs were bled from the anterior vena cava with a sterilized 1 inch, 18-gauge needle and 15 ml syringe. Blood was collected in 10 ml heparinized test tubes. Gilts were also bled by the same technique, using a sterilized 3.5 inch, 18-gauge needle and 20 ml syringe.

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*Gleptoferron = a polysacharide complex of beta-ferric oxyhydroxide and dextran glucoheptonic acid, Burns-Biotec Laboratories, Inc.

Packed Cell Volume (PCV). Hematocrit values were determined[73] by centrifuging heparinized microhematocrit tubes containing blood samples for 5 minutes at 10,000 rpm in an International model MB microhematocrit centrifuge.[73] The percent of packed cells was recorded from an International microhematocrit reader.

Hemoglobin (Hgb). Hemoglobin values were measured by the cyanmetahemoglobin method using a Drabkin's cyanide reagent to lyse the red blood cells. The absorbance of the solution was measured in a spectrophotometer at 540 nm of wavelength.[74] Absorbance values were multiplied by a conversion factor in order to obtain the Hgb concentration values in grams per deciliter ($^{g}/dl$).

Mean Corpuscular Hemoglobin Concentration (MCHC). This blood parameter was calculated by using the following formula:

$$\text{MCHC (\%)} = \frac{\text{Hgb } (^{g}/dl)}{\text{PCV (\%)}} \times 100$$

Plasma Collection. Blood samples were centrifuged at 3,000 rpm for 15 minutes in order to separate plasma from cellular elements. Plasma samples were kept in clean 5 ml tubes, the air was displaced with nitrogen gas, and then frozen for determination of vitamin E, Se, Fe, and GSH-Px values.

Plasma Vitamin E, Selenium, Iron and GSH-Px Analyses

Vitamin E. Plasma vitamin E values were measured by a sensitive fluorometric method for tissue tocopherol as modified for plasma tocopherol.[75,76] The method consisted of precipitating the protein in 1 ml of plasma with 2 ml of absolute ethanol and displacing the air with nitrogen gas before shaking the samples for 5 seconds. Afterward, 2 ml of cyclohexane were added, air was again displaced, and tubes were shaken for 20 seconds. All the tubes were maintained on ice and treated in the same manner. Test tubes were centrifuged in a Danon/IEC model PR-6000 refrigerated centrifuge at 3,000 rpm for 15 minutes and the upper layer was pipetted into vials. Duplicates were run for standards and samples. Transmission of samples was read in an Aminco-Bowman spectrofluorometer at 296 nm excitation and 330 nm emission. Values were regressed against micrograms of α-tocopherol using a computerized curvilinear program.

Selenium (Se). Selenium values were determined by a routine fluorometric method[77] which consisted of digesting 1 ml of plasma with 2 ml of nitric acid (HNO_3) and 2 ml of percholoric acid ($HClO_4$, 70%) on hot plates in Erlanmeyer flasks. The samples were neutralized and chelated with 3 ml of Ethylene Diamino Tetraacetic Acid (EDTA) (8 ml), and then complexed with 2,3 Diaminonaphthalene (DAN) (5 ml) to extract the diazoselenol into 5 ml of cyclohexane.

Deionized water was used to bring the volume up to the neck of the Erlenmeyer flasks, and the upper layer was repipetted into clean tubes. Duplicates were run for standards and samples. An Aminco-Bowman spectrophotofluorometer set at 376 nm excitation and 510 nm emission was used to read the transmission (T%) and a curvilinear regression was performed to calculate Se values in micrograms per ml (µg/ml).

Iron (Fe). Plasma Fe was determined by atomic absorption spectrophotometry using a model IL 951 Atomic Absorption Emission Spectrophotometer at 248.3 nm wavelength and an acetylene flame. The method consisted of precipitation of protein contained in 1 ml of plasma with trichloroacetic acid (TCA) and incubation at 90°C for 15 minutes. Samples were then centrifuged for 15 minutes, and Fe was measured in the supernatant.[78] Iron values were calculated in µg per deciliter (µg/dl).

Glutathione Peroxidase (GSH-Px). Plasma GSH-Px activity was determined by the coupled assay for GSH-Px.[79,80] The method consisted of mixing all the reagents with samples into 1 ml cuvettes. Hydrogen peroxide (H_2O_2) was then added, and the contents mixed. The reaction for the mixture was monitored in a Varian 634 spectophotometer and recorded in a Varian model 9176 chart recorder for about 5 minutes.

Enzyme unites were calculated as micromoles of glutathione (GSH) oxidized per minute. Increment of absorbance (ΔAb)

was recorded for blanks (ΔAb_B) and samples (ΔAb_S) by readings (R) at 0 minutes (R_1) and at 5 minutes (R_2) on the charts, and the difference ($R_1 - R_2$) was divided by the time (t). After that, ΔAb_B was subtracted from ΔAb_S and multiplied by a factor (8.0386) to obtain the enzyme units (EU) of GSH-Px per ml of sample. The formulas used are as follows:

$$\Delta Ab_B = \frac{R_1 - R_2}{t} \qquad \Delta Ab_S = \frac{R_1 - R_2}{t}$$

$$\text{GSH-Px(EU/ml)} = (\Delta Ab_S - \Delta Ab_B) \times 8.0386$$

Microbiology

During necropsy of baby pigs, microbiology tissue specimens were saved in sterile containers and sent to the Microbiology Department at Michigan State University Veterinary Clinical Center for bacteriological analyses. Tissues collected were lung, liver and heart. Isolation of enterotoxigenic E. coli from a pig with diarrhea was attempted.

Pathology

Necropsy was performed on 42 baby pigs at the Michigan State University Animal Health Diagnostic Laboratory (AHDL). Multiple tissues were collected and fixed in 10% buffered formalin. These tissues were later trimmed, parafin embedded,

sectioned and stained with hematoxylin and eosin. The procedures are described in the Manual of Histological Methods of the Armed Forces' Institute of Pathology.[81] During necropsy, randomized liver samples were saved in plastic containers and frozen for further Se analyses. The values obtained were related to the dry matter (DM) content of the samples and thus expressed as µg/g of DM.

Statistical Analyses

A linear regression ($y = a + bx$)[82] was used to calculate individual values for vitamin E and Se at each sampling. Individual values of each of the blood parameters (PCV, Hgb, MCHC, vitamin E, Se, Fe, and GSH-Px) were determined for the 2 experimental groups of animals. By using the analysis of variance,[82] treatment mean values were compared between groups, and their statistical level of significance (P) was established.

RESULTS

The reproduction performance of gilts fed the basal diet compared to gilts fed the same diet supplemented with vitamin E and Se, is summarized in Table 2. Gilts fed the supplemented diet had larger litters and heavier pigs at birth and at 21 days than gilts fed the basal diet. These parameters were significantly different ($P < 0.05$). In addition, the supplemented gilts weaned more pigs than unsupplemented gilts. The livability to 21 days of age was greater in pigs nursing the supplemented gilts. There were no differences between the 2 treatment groups in weight changes of gilts from postfarrowing until 21 days of lactation.

Incidence of MMA and Clinical Observations of Gilts and Baby Pigs

The incidence and clinical observations of MMA in gilts and baby pig death losses during lactation are summarized in Table 3. Five gilts fed the basal diet had some evidence of MMA. Of 3 gilts with signs of MMA, 2 had mastitis and 1 had metritis (Figure 1). The mastitis was characterized by a warm, firm and reddish udder. Two additional gilts had excessive pig death losses with no signs of

TABLE 2. REPRODUCTIVE PERFORMANCE OF GILTS

PRODUCTION PARAMETERS	DIETS		SEM[a]	P<[b]
	MSU BASAL	+VITAMIN E-Se		
NO. OF GILTS	10	9		
GILT WEIGHT, KG				
POST FARROWING	169.0	170.6	6.4	1.00
21 DAYS LACTATION	162.7	165.1	4.8	1.00
WEIGHT CHANGE	-6.3	-5.5		
LITTERS				
AVG. TOTAL PIGS BORN/LITTER	8.8	10.9	.68	.12
AVG. LIVE PIGS BORN/LITTER	8.1	10.2	.70	.14
AVG. LITTER WEIGHT AT BIRTH, KG	11.2	15.2	.86	.02
AVG. PIG WEIGHT AT BIRTH, KG	1.4	1.5	.02	.02
AVG. LIVE PIGS AT 21 DAYS/LITTER	6.5	8.8	.72	.11
AVG. LITTER WEIGHT AT 21 DAYS, KG	31.4	45.1	3.4	.05
AVG. PIG WEIGHT AT 21 DAYS, KG	4.8	5.1	.12	.22
LIVABILITY (BIRTH TO 21 DAYS), %	80.2	86.8	5.7	1.00
AVG. NO. OF DAYS FROM WEANING TO FIRST ESTRUS[c]	10.8 (10)	5.1 (6)	2.8	.31

[a]SEM = STANDARD ERROR OF MEAN

[b]P< = LEVEL OF SIGNIFICANCE

[c]NUMBERS IN PARENTHESES = NO. OF GILTS IN ESTRUS; THREE GILTS IN E-Se SUPPLEMENTED GROUP FAILED TO COME INTO ESTRUS AND WERE CULLED FROM THE HERD.

TABLE 3. CLINICAL OBSERVATIONS IN THE GILTS AND DEATH LOSSES OF BABY PIGS

PARAMETERS	DAM'S DIET	
	MSU BASAL	+ VITAMIN E-Se
NO. GILTS	10	9
NO. WITH MMA SIGNS[a]	3	0
NO. WITH AGALACTIA OR HYPOGALACTIA[b]	2	0
LIVE PIGS		
1 DAY	81	91
21 DAYS	65	79
MORTALITY	16 (19.8%)	12 (13.2%)

[a]TWO (2) GILTS HAD CLINICAL SIGNS OF MASTITIS AND ONE (1) HAD METRITIS.

[b]THESE GILTS HAD EXCESSIVE PIG DEATH LOSSES.

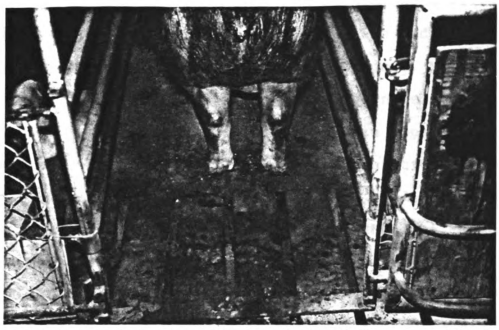

Fig 1 - Metritis in a gilt fed the basal diet. Note blood clots in vulva and purulent material on the buttocks and floor.

Fig 2 - Pig with syndactylism born to a gilt fed the basal diet. Note the curling of the phalanges.

mastitis or metritis. There were also 2 supplemented gilts that did not milk as well as expected. This was indicated by a slower growth rate of the pigs until 21 days. Mortality from birth to 21 days was greater in pigs nursing gilts fed the basal diet. There was no difference in appetite of gilts fed either diet at or following parturition of gilts in both groups fluctuated greatly, and no difference could be ascertained as to the influence of the diet. Some of the gilts fed the basal diet seemed to have more difficulties during farrowing than the supplemented gilts. General observations on the length of time for farrowing did not appear to be different between the treatment groups. There were no obvious differences in the gilt behavior at parturition between the 2 groups, with the exception of 1 unsupplemented gilt which appeared to be more nervous at parturition, and lost the entire litter within 48 hours. This gilt was given 3 piglets from another sow, and raised them successfully.

Vitamin E, Selenium, Iron, Glutathione Peroxidase and Hematology Values in Gilts During Gestation and Lactation

The plasma vitamin E and Se values of gilts fed either diet were similar during the early gestation (Table 4). However, they were significantly different ($P < 0.05$) by late gestation and after 21 days of lactation. The plasma

TABLE 4. PLASMA VITAMIN E, SELENIUM (Se), IRON (Fe), GLUTATHIONE PEROXIDASE (GSH-Px) AND HEMATOLOGY VALUES[a] IN GILTS DURING GESTATION AND LACTATION

PRODUCTION PERIOD	DIETS		SEM[b]	P<[c]
	MSU BASAL	+VITAMIN E-Se		
EARLY GESTATION (30 DAYS)				
NO. OF GILTS	5	5		
Se, µg/ML	0.15	0.19	.011	.15
VITAMIN E, µG/ML	2.74	2.14	.035	1.00
LATE GESTATION (100 DAYS)				
NO. OF GILTS	5	5		
Se, µG/ML	0.13	0.17	.005	.001
VITAMIN E, µG/ML	0.87	1.91	.094	.0003
GSH-Px, EU[d]/ML	1.21	1.01	.010	1.00
Fe, µG/DL	135.68	133.20	.051	1.00
LACTATION (21 DAYS)				
NO. OF GILTS	10	9		
Se, µG/ML	0.08	0.20	.007	.0001
VITAMIN E, µG/ML	0.76	2.02	.127	.0001
GSH-Px, EU[d]/ML	0.47	0.95	.057	.0004
Fe, µG/DL	102.80	89.60	4.76	.16
Hgb, G/DL	11.5	10.9	.36	1.00
PVC, %	34.7	32.3	1.05	.25
MCHC, %	33.3	35.2	.53	.07

[a]VALUES ARE EXPRESSED AS AVERAGES ($\bar{x}$). INDIVIDUAL VALUES ARE IN APPENDIX TABLE A-1.

[b]SEM = STANDARD ERROR OF MEAN.

[c]P< = LEVEL OF SIGNIFICANCE.

[d]EU = ENZYME UNITS AS MICROMOLES OF GSH OXIDIZED PER MINUTE.

GSH-Px activity values were significantly lower ($P < 0.05$) in gilts fed the basal diet at 21 days of lactation as compared to values of gilts fed the supplemented diet. These GSH-Px values were also lower in gilts fed the basal diet at 21 days of lactation in comparison to values during late gestation. Iron values were similar in gilts fed both diets during late gestation and decreased in both groups by 21 days of lactation. The values stayed higher in gilts fed the basal diet. Hemoglobin and PCV values were similar in both treatment groups at 21 days of lactation. However, the MCHC values were higher ($P < 0.07$) in gilts fed additional vitamin E and Se.

Blood Plasma Parameters, Performance and Hematology of Baby Pigs at Birth and at 21 Days of Age

Average plasma vitamin E, Se, Fe, and GSH-Px values in baby pigs at birth and at 21 days nursing gilts fed either diet are summarized in Table 5. Baby pigs nursing gilts fed the basal diet had lower plasma Se values at 1 day and at 21 days than pigs nursing gilts fed the supplemented diet. These plasma Se values were considered to be lower than the borderline for a Se deficiency which is 0.05 ppm. Plasma GSH-Px activity values were similar in both groups at birth and at 21 days. The GSH-Px and Se mean values were positively correlated. Iron values

TABLE 5. PLASMA VITAMIN E, SELENIUM (Se), IRON (Fe), AND GLUTATHIONE PEROXIDASE (GSH-Px) VALUES[a] OF PIGS AT BIRTH AND AT 21 DAYS OF AGE.

	DAM'S DIET			
	MSU BASAL		+ VITAMIN E-Se	
PARAMETERS	PIGS AT 1 DAY	PIGS AT 21 DAYS	PIGS AT 1 DAY	PIGS AT 21 DAYS
NO. OF LITTERS	10	9	9	9
TOTAL NUMBER OF PIGS	81	65	91	79
Se, µG/ML	0.033 (81)	0.049 (65)	0.060 (89)	0.073 (79)
VITAMIN E, µG/ML	1.16 (78)	0.90 (65)	1.81 (91)	2.14 (79)
GSH-Px, EU[b]/ML	0.118 (78)	0.315 (65)	0.163 (91)	0.369 (79)
Fe, µG/DL	64.92 (40)	137.47 (58)	73.16 (77)	111.4 (68)

[a]VALUES ARE EXPRESSED AS AVERAGES WITHIN AND BETWEEN LITTERS. INDIVIDUAL VALUES ARE IN APPENDIX TABLES A-2, A-3, A-4, A-5. MEAN VALUES WITHIN LITTERS ARE IN APPENDIX A-6.

[b]EU = ENZYME UNITS AS MICROMOLES OF GSH OXIDIZED PER MINUTE.

( ) = NUMBER OF SAMPLES.

in the pigs nursing gilts fed the basal diet were higher than in pigs nursing supplemented gilts at 21 days of age.

The growth rate and hematological values of baby pigs given either 200 mg or 400 mg of Fe are summarized in Table 6. In both treatment groups, pigs injected with the higher dosage (400 mg) had a lower weight gain at 21 days. Pigs given either Fe dosage had increased Hgb and PCV values from birth to 21 days, but the differences were not significant ($P > 0.05$). The livability of pigs nursing gilts fed the basal diet was lower as compared to pigs nursing gilts fed the supplemented diet. Furthermore, the livability of pigs given 400 mg of Fe was reduced in both groups in comparison to pigs given 200 mg. Blood plasma vitamin E, Se and GSH-Px values of pigs nursing gilts fed either diet in general increased from birth to 21 days of age. The differences were significant ($P < 0.05$) for pigs nursing gilts fed the supplemented diet. Plasma Fe values of pigs nursing gilts fed the basal diet were significantly higher ($P < 0.05$) than in pigs nursing gilts fed the supplemented diet at birth and at 21 days. There was no significant interaction between Fe and dietary treatments.

The incidence of deaths, stillborn, malformed pigs and mortality rates are summarized in Table 7. Gilts fed either diet had no difference as to the percentage of stillborn and mummies. The mortality of pigs at 21 days was higher in gilts fed the basal diet than in gilts fed the supplemented diet.

TABLE 6. PLASMA VITAMIN E,[a] SELENIUM (Se), IRON (Fe), GLUTATHIONE PEROXIDASE (GSH-Px), HEMATOLOGY VALUES AND WEIGHT OF PIGS AT 1 DAY AND AT 21 DAYS OF AGE.

	DIETS							
	MSU BASAL		+ VITAMIN E-Se					
	IM Fe, MG/PIG		IM Fe, MG/PIG			SIGNIFICANCE[c]		(P<)
PARAMETER	200	400	200	400	SEM[b]	D	Fe	DxFe
NO. OF PIGS								
1 DAY	40	42	47	46				
21 DAYS	33	32	42	37				
WEIGHT, KG								
1 DAY	1.36	1.34	1.47	1.47	.02	.02	1.00	1.00
21 DAYS	5.17	4.48	5.52	5.00	.23	.22	.05	1.00
Hgb, G/DL								
1 DAY	9.9	10.0	10.1	9.5	.12	.20	.17	.28
21 DAYS	11.5	11.7	12.0	12.3	.13	.05	.25	1.00
PCV, %								
1 DAY	30.7	31.4	31.5	29.5	.46	1.00	1.00	.17
21 DAYS	36.3	37.1	35.0	35.4	.41	.18	1.00	1.00
LIVABILITY, %								
TO 21 DAYS	82.5	76.2	89.4	80.4				
Se, µG/ML								
1 DAY	.035	.031	.060	.128	.002	.0001	.28	1.00
21 DAYS	.046	.045	.072	.073	.001	.0001	1.00	1.00
VITAMIN E, µG/ML								
1 DAY	1.13	1.19	1.82	1.81	.078	.0005	1.00	1.00
21 DAYS	1.04	.97	2.47	2.23	.050	.0001	1.00	.21

TABLE 6. (Continued)

PARAMETER	DIETS: MSU BASAL, IM Fe, MG/PIG 200	MSU BASAL, IM Fe, MG/PIG 400	+VITAMIN E-Se, IM Fe, MG/PIG 200	+VITAMIN E-Se, IM Fe, MG/PIG 400	SEM[b]	SIGNIFICANCE[c]: D	SIGNIFICANCE[c]: Fe	(P<): DxFe
GSH-Px, EU[a]/ML								
1 DAY	.11	.11	.15	.16	.004	.00005	1.00	1.00
21 DAYS	.30	.29	.38	.39	.009	.0001	1.00	1.00
Fe, µG/DL								
1 DAY	63.0	63.2	70.0	76.2	1.59	.0029	1.00	1.00
21 DAYS	128.2	148.7	104.1	111.4	3.6	.00008	.03	1.00

[a] VALUES ARE EXPRESSED AS AVERAGES ($\bar{x}$).

[b] SEM = STANDARD ERROR OF MEAN.

[c] D = LEVEL OF SIGNIFICANCE FOR DAM'DIET; Fe = LEVEL OF SIGNIFICANCE FOR IRON VALUES; DxFe = LEVEL OF SIGNIFICANCE FOR INTERACTION OF D AND Fe.

[d] EU = ENZYME UNITS AS MICROMOLES OF GSH OXIDIZED PER MINUTE.

TABLE 7. INCIDENCE OF STILLBORN, MALFORMED PIGS, AND MORTALITY FROM BIRTH TO 21 DAYS OF AGE

DIETS[a]	No. Gilts	At Birth					Birth to 21 Days	
		Total Pigs	Live Pigs	Stillborn and Mummies	Malformed Pigs	Mortality[b] %	No. Dead Pigs	Mortality %
MSU BASAL	10	88	81	7	1	8	16	19.8
+ Vitamin E-Se	9	98	91	7	1	7	12	13.2

[a]Dried high moisture shelled corn-soybean diets

[b]Mortality of stillborn and mummies.

Microbiology

The microorganisms isolated from pigs that died are given in Table 8. A heavy growth of E. coli was isolated in tissues of 4 of 7 pigs that had lesions of polyserositis, accumulation of excessive seroganguinous fluid and serofibrinous adhesions in body cavities. One of the 4 pigs also had β-hemolytic streptococci. However, the strain of E. coli isolated in all pigs was not demonstrated to be enteropathogenic. Attempts to isolate enterotoxigenic E. coli from a pig with diarrhea were not successful.

Gross Pathology

The gross lesions and Se analyses of liver samples of pigs necropsied are summarized in Tables 8 and 9 respectively. Twenty-seven of 42 baby pigs that died between birth and 21 days of age were necropsied. Pigs from both treatment groups had enlargement of lymph nodes and yellowish coloration of subcutaneous tissue. However, a yellowish to brownish tinge was more often observed in pigs nursing gilts fed the basal diet. These pigs also had a generalized edema and serofibrinous polyserositis. Accumulation of excessive serosanguinous fluid in body cavities and pericardial sac were also commonly seen. These lesions were also present in pigs nursing gilts fed the supplemented diet, but they were less prominent. There was 1 malformed

TABLE 8. SUMMARY OF GROSS LESIONS AND MICROBIOLOGY IN TISSUES AND ORGANS AT NECROPSY. THESE PIGS WERE INJECTED INTRAMUSCULARLY WITH 200 MG OR 400 MG OF IRON.

DIETS	NO. OF DEAD PIGS[a]	NO. OF PIGS NECROPSIED	GROSS LESIONS AND MICROBIOLOGY
MSU BASAL	23	15	PALENESS AND BROWNISH DISCOLORATION OF CARCASSES. ENLARGEMENT AND DARKNESS OF LYMPH NODES AND SPLEEN. DEHYDRATION AND DIARRHEA (2 CASES). SEROSANGUINOUS FLUID IN THORACIC AND ABDOMINAL CAVITIES (7 CASES). SEROFIBRINOUS PLEURITIS, PNEUMONIA AND PERITONITIS (6 CASES). HYDROPERICARDIUM AND SEROFIBRINOUS PERICARDITIS (7 CASES). *E. COLI* WAS ISOLATED FROM 4 OF 4 PIGS. SUPERFICIAL ABSCESSES AND LACERATION OF SKIN (2 CASES). MALFORMED PIGS (SYNDACTYLIA) (1 CASE). THIS PIG HAD LESIONS IN THE LIVER CONSISTENT WITH HEPATOSIS DIETETICA.
+ VITAMIN E-Se	19	12	YELLOWISH COLORATION OF CARCASSES. ENLARGED LYMPH NODES. SEROSANGUINOUS FLUID AND FIBRINOUS EXUDATE IN THORAX AND ABDOMEN (2 CASES). URIC ACID CRYSTALS IN KIDNEYS (2 CASES). MALFORMED PIGS (1 CASE).

[a]ONLY PIGS THAT DIED FROM BIRTH TO 21 DAYS OF AGE.

TABLE 9. LIVER SELENIUM (Se) VALUES OF 14 SAMPLES FROM NECROPSIED PIGS.

DIET	TOTAL PIGS	NO. DEAD PIGS	NO. PIGS NECROPSIED	NO. LIVER SAMPLES	Se[a] µG/G DM
MSU BASAL	87	23	15	8	1.07
+ VITAMIN E-Se	97	19	12	6	1.30

[a]VALUES OF SELENIUM ARE EXPRESSED AS AVERAGES ON DRY MATTER BASIS. INDIVIDUAL VALUES ARE IN APPENDIX TABLE A-7.

pig (syndactylism) (Figure 2) born to a gilt fed the basal diet. At necropsy, this pig had gross lesions suggestive of hepatosis dietetica. The Se concentration in liver samples analyzed were similar in pigs from both treatment groups (Table 9). Lesions suggesting Fe toxicity were not observed grossly in pigs from either group.

Histopathology

The gross lesions were confirmed microscopically, and confined to body cavities, serous surfaces, reticuloendothelial system, heart, and skeletal muscles (Table 10). In body cavities and serous surfaces, a serofibrinous and suppurative generalized polyserositis was present in 7 pigs nursing 4 different gilts fed the basal diet. However, these changes were also seen in 2 pigs nursing gilts fed the supplemental diet. A serofibrinous pericarditis and epicarditis with bacterial colonies (Figure 3) were present in pigs nursing gilts fed the basal diet. Pulmonary edema with interstitial pneumonia (Figure 4) and serofibrinous pleuritis with bacterial colonies were also observed (Figure 5).

In pigs of both groups, a heavy iron pigment (hemosiderin) was observed in the spleen, subcapsular sinuses of lymph nodes and the Kupffer cells of the liver. It was less severe in pigs nursing the supplemental gilts.

TABLE 10. SUMMARY OF HISTOPATHOLOGICAL CHANGES FROM PIGS NECROPSIED.

DIETS	NO. OF PIGS NECROPSIED	MICROSCOPIC CHANGES
MSU BASAL	15	SEROFIBRINOUS AND SUPPURATIVE GENERALIZED POLYSEROSITIS. DEGENERATIVE MYOCARDITIS WITH LOSS OF STRIATIONS. PULMONARY EDEMA WITH SEROFIBRINOUS PLEURITIS AND PNEUMONIA. SUBEPICARDIAL HEMORRHAGES WITH EPICARDITIS AND PERICARDITIS AND BACTERIAL COLONIES. CENTROLOBULAR HEPATIC NECROSIS AND HEMORRHAGES WITH EXTRAMEDULARY ERYTHROPOIESIS. SEVERE VACUOLATING HEPATOPATHY. HEAVY DEPOSITION OF IRON IN LIVER, SPLEEN AND LYMPH NODES.
+ VITAMIN E-Se	12	SEROFIBRINOUS POLYSEROSITIS. VACUOLATING HEPATOPATHY. FOCAL PYELITIS IN KIDNEYS. MILD DEPOSITION OF IRON IN LYMPH NODES AND SPLEEN.

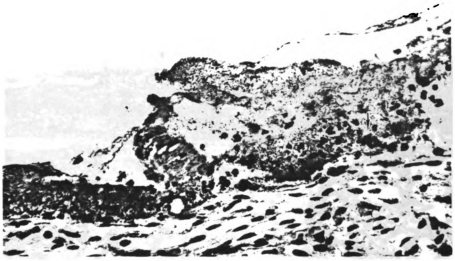

Fig 3 - Serofibrinous pericarditis and epicarditis with bacterial colonies (arrow) in a pig from a gilt fed the basal diet.

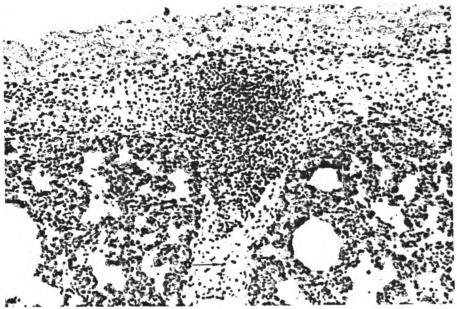

Fig 4 - The same pig as in Fig 3. Pulmonary edema (arrow) and interstitial pneumonia (a) with accumulation of inflammatory cells.

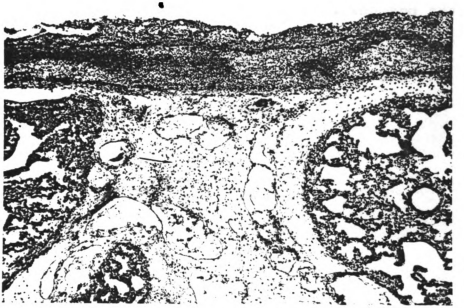

Fig 5 - A lower magnification of the same lung as Fig 4. Note the serofibrinous pleuritis with bacterial colonies and interlobular edema (arrow).

Centrolobular hemorrhagic necrosis along with diffuse vacuolating hepatopathy and extramedullary erythropoiesis were also present in the pig with syndactylia (Figures 6 and 7).

Degenerative myocarditis with calcification and granulation of fibers were observed in pigs nursing gilts fed the basal diet (Figures 8 and 9). Loss of striations of skeletal muscle fibers was also present.

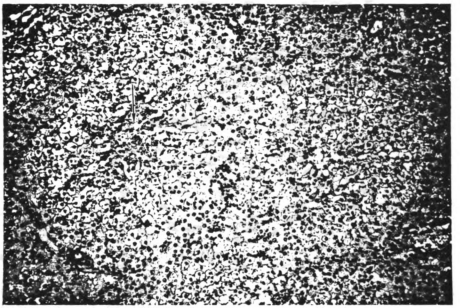

Fig 6 - Centrolobular hemorrhages (arrow) and vacuolation of hepatocytes. Liver of pig with syndactylism. Note also the extramedullary erythopoiesis (lower center).

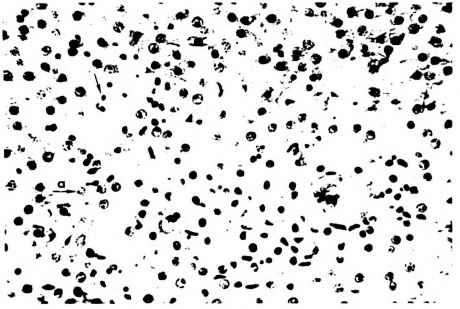

Fig 7 - Section from the same liver as Fig 6 (higher magnification). Necrosis of hepatocytes evidenced by pyknosis (arrow) and karyorrhexis of nuclei (a).

Fig 8 - Fragmentation and granulation of cardiac fibers. Note loss of striations.

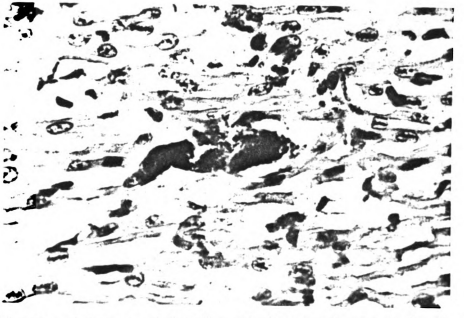

Fig 9 - Higher magnification of the same heart as in Fig 8. Note calcification of cardiac fibers.

DISCUSSION

This research confirms evidence that vitamin E and Se have a role in reproduction in swine.[50,83] It also provides information that a dietary deficiency of these nutrients is related to the MMA problem in swine. Additional evidence was obtained that the quality of feed as reported by Ringarp[11,42] has an important role in increasing the requirements of swine for vitamin E and Se.

Gilts' Performance

The general reproductive performance of gilts supported previous observations that vitamin E and Se increased the number of live pigs born and surviving to 3 weeks of age.[50] These nutrients also reduced the incidence of MMA in gilts which confirms previous experiments and observations.[50,51] From the general behavior of gilts in both groups, it is concluded that assessment on whether or not gilts had MMA was difficult. Whereas the clinical manifestations (mastitis and metritis) were not as evident as anticipated to occur according to the literature,[11,18,19] agalactia was evidenced in the poor growth rate of baby pigs. It was the single most important factor that reduced the growth rate and caused death losses.[4,18,21] This agrees

with Ringarp's[11] emphasis on the lack of milk production and death losses of young pigs as emphasized by Leman.[3] It was of interest that vitamin E and Se supplemented gilts farrowed 2 more and weaned 2.3 more pigs per litter than gilts fed the basal diet. While this was not significant ($P = 0.11$), it is considered an economically important factor in the profitability to swine producers.[3] Although the number of observations in this research was limited, the data suggest that pigs carried to term are reduced when gilts are fed a diet deficient in vitamin E and Se. This confirms the observations of Ohio researchers.[84,85] However, it disagrees with previous observations[86] in which no impairment in reproductive performance of sows occurred when a corn-soybean diet was fed without additional inorganic Se.

Blood Analyses of Gilts

Blood parameters obtained from both groups of gilts confirmed that plasma vitamin E and Se values depend directly upon dietary intake of these nurtients.[84] These values decreased in gilts fed either diet during gestation which confirms the previous observations[85] that in gravid gilts plasma vitamin E and Se values decline during pregnancy. Vitamin E, Se and GSH-Px values tended to decrease during lactation in gilts fed the basal diet, while in gilts fed the supplemented diet tended to increase. In general,

there was a positive correlation between plasma Se values and GSH-Px activity. This confirms previous observations.[87] Plasma Fe values were higher in gilts fed the basal diet during lactation. These gilts had lower MCHC values than gilts fed the supplemented diet. The biological explanation of this observation is not known. However, a defective utilization of Fe and a decreased Fe binding capacity might be involved.

Progeny Performances

Excessive losses at parturition and a higher mortality of baby pigs (19.8%) during lactation occurred in gilts fed the basal diet. This supports previous observations that piglets nursing Se deficient sows had a higher mortality than piglets nursing Se supplemented sows.[84] Although baby pigs in both groups had a reduced weight at 21 days of age when higher dosages of Fe were given, tolerance to the different Fe dosages was observed which confirms previous work.[57] This reduction of pig weight was significantly ($P = 0.05$) different between pigs in each treatment group. The biological implications of this result are not known. While clinical signs of Fe toxicosis in baby pigs were not observed, a greater incidence of infections was noted in pigs nursing gilts fed the basal diet. This supported the observations that a dietary deficiency of vitamin E and Se increases susceptibility to

infections or reduces the defense mechanisms.[41-47] It was of interest that most of the pigs from which E. coli was isolated were pigs which had been injected with 400 mg of Fe. This confirms earlier observations that Fe injections exacerbate E. coli growth.[88,89]

Blood Analyses of Baby Pigs

Plasma vitamin E and Se values in baby pigs suggest that their plasma antioxidant status depends directly on the dietary status of the dams. This antitoxidant status was not influenced by Fe injections given to pigs which confirms previous observations in rats that oxidative stress associated with elevated dietary Fe intake did not increase these values in blood plasma.[90] However, a significant increase of Fe values in plasma of pigs nursing gilts fed the basal diet was observed at 21 days as compared to pigs nursing gilts fed the supplemented diet. Plasma Se and GSH-Px values at birth and at 21 days in pigs from either treatment group confirm that genetic factors control this status within red blood cells and the dependency of baby pigs on Se and GSH-Px status of the dam.[91] Of particular interest was that plasma vitamin E values did not change much from birth to 21 days and tended to be higher in pigs nursing gilts fed the supplemented diet. This may be due to the fact that pigs nursing supplemented gilts obtained more vitamin E from colostrum.

Borderline values for vitamin E were present in the baby pigs nursing gilts fed the basal diet (0.90 µg/ml). The Se borderline values for the gilts were estimated to be about 0.80 µg/ml. Borderline values for Se were confirmed to be similar to the data previously published on the borderline values for Se in pigs (0.03 - 0.07 µg/ml).[72]

This research provides preliminary information on borderline vitamin E values expected during a dificiency of this nutrient in swine. It also confirms that death losses in neonatal pigs and preweaning mortality have not been improved lately as indicated in previous reports.[21,93] Finally, this research suggests that future investigations should be directed toward a better understanding of non-infectious factors influencing the incidence of MMA in swine. This is in agreement with previous observations on the role of infectious agents in MMA.[93]

SUMMARY

The role of vitamin E and Se fed to gilts during gestation and lactation in preventing MMA and death losses in baby pigs was determined in 10 gilts fed a basal diet in comparison to 9 gilts fed the same diet supplemented with vitamin E and Se. The corn used in the basal diet was initially high moisture shelled corn that had been stored anaerobically for several months, then dried artificially and used as dried shelled corn. Gilts fed the basal diet had more clinical signs of MMA, farrowed and weaned smaller litters in which individual pigs were lighter in weight at birth and at 21 days of age than were baby pigs nursing gilts fed supplemental vitamin E and Se. Plasma vitamin E, Se and GSH-Px values were lower at birth and at 21 days in pigs nursing gilts fed the basal diet, and these pigs had a higher incidence of infections.

Baby pigs nursing gilts fed the basal diet gained less weight following Fe injections than pigs nursing supplemented gilts. There was also a high death loss from birth to 21 days of age in pigs nursing gilts fed the basal diet. Supplementing vitamin E and Se to a corn-soybean basal diet composed of dried high moisture corn reduced the incidence of MMA in gilts, and improved the health and performance of baby pigs from birth to 21 days of age.

LIST OF REFERENCES

REFERENCES

1. King NB: Contribution and needs of animal health and disease research. Am J Vet Res 42: 1096-1097, 1981.

2. Martin, CE, Elmore RC: Mammary glands, in Leman AD (ed): Diseases of Swine. Ames, Iowa, Iowa State University Press, 1981, pp 155-169.

3. Leman AD, Knudsen C, Rodeffer HE, et al: Reproductive performances of swine on 76 Illinois farms. J Am Vet Med Assoc 161: 1248-1250, 1972.

4. Backstrom L, Morkoc A: Postparturient dysgalactia in the sow. Paper of Swine Herd Health Programming Conf University of Minnisota. St. Paul, Minnesota, 1982, pp 2-29.

5. Michel RL, Whitehair CK, Keahey KK: Dietary hepatic necrosis associated with selenium-vitamin E deficiency in swine. J Am Vet Med Assoc 155: 50-59, 1969.

6. Trapp AL, Keahey KK, Whitenack DL, et al: Vitamin E-Se deficiency in swine: Differential diagnosis and nature of field problems. J Am Vet Med Assoc 157: 289-299, 1970.

7. Van Vleet JF, Carlton W, Olander HJ: Hepatosis dietetica and mulberry heart disease associated with selenium deficiency in Indiana swine. J Am Vet Med Assoc 157: 1208-1212, 1970.

8. Teague, HS, Aherne FX, Baker DH, et al: Nutrient Requirements of Swine. Nat Res Counc (NRC), Washington, D. C., 1979, pp 7-9.

9. Miller ER, Ullrey DE: Swine feeds and feeding. Swine Tech. Bull. 537. Dept Anim Sci. Michigan State University. 1975, pp 6-8.

10. Follis, RH Jr: The pathology of nutritional diseases. Charles C. Thomas. Spring, Ill 1946, pp 119-127.

11. Ringarp N: Clinical and experimental investigation into a postparturient syndrome with agalactia in sows. Act Agr Scand Suppl 7, pp 11-119, 1960.

12. Ringarp R: Toxemia agalactia in sows after farrowing. Vet Bull 29: 510-511, 1959.

13. Ross RF, Orning AP, Woods RD, et al: Bacteriologic study of sow agalactia. Am J Vet Res 42: 949-955, 1981.

14. Ross RF, Zimmerman BJ, Wagner WC, et al: A field study of coliform mastitis in sows. J Am Vet Med Assoc 167: 231-235, 1975.

15. Ross RF, Lauren LC, Maynard LS: The role of certain bacteria in mastitis-metritis-agalactia in sows. J Am Vet Med Assoc 154: 1368, 1969.

16. Armstrong CH, Hooper BE, Martin CE: Microflora associated with agalactia syndrome of sows. Am J Vet Res 29: 1401-1407, 1968.

17. Elmore RG: Pathophysiology of lactation failure in sows. Proc 100th Annual Meeting Vet Med Assoc, 1982, pp 1-20.

18. Wagner WC: Mastitis-Metritis-Agalactia. Vet Clin of North Am. Larg Anim Prac 4:333-341, 1982.

19. Wagner WC: What can we do about MMA? Hog Farm Manag 20:33-35, 1983.

20. Pond WG: Modern Pork Production. Sci Am (May) 1983, pp 96-103.

21. Brookbank NH: Disorders of the lactating sow and newborn pig. Vet Rec 70: 1148-1158, 1958.

22. Le Roy GB: Agalactia of the sow and bacterial infections. Ve Professional Top Swine. University of Illinois, 1982, pp 3-5.

23. Threlfall WR, Martin CE: Swine agalactia in Missouri. Vet Med. Small Anim Clin 67: 423, 1973.

24. Peterson DH: MMA in the MSU swine herd. Rep of Swine Res Dept Anim Sci, Michigan State University, East Lansing 99: 6-9, 1969.

25. Penny RHC: The agalactia complex in the sow. A review. Aust Vet J 46: 153-156, 1970.

26. Ross RF, Speat ML: Role of the sow as a reservoir of infection for Mycoplasma hyosynoviae. Am J Vet Res 34: 373-378, 1973.

27. Switzer WP: Swine mycoplasma. J Am Vet Med Assoc 151: 1656-1661, 1967.

28. Martin CE, McDowell WS: Lactation failure (mastitis-metritis-agalactia), in Dunne HW (ed): Diseases of Swine. Ames, Iowa, Iowa State University Press, 1975, pp 953-960.

29. Martin CE, Hooper BE, Armstrong CH, et al: A clinical and pathologic study of the mastitis-metritis-agalactia syndrome of sows. J Am Vet Med Assoc 151: 1629-1634, 1967.

30. Nachreiner RF, Ginther OJ: Porcine agalactia: Hematologic, serum chemical and clinical changes during the preceding gestation. Am J Vet Res 31: 799-809, 1972.

31. Nachreiner RF, Ginther OJ: Swine agalactia. J Am Vet Med Assoc 154: 1369, 1969.

32. Balke JME, Elmore RG, Berg JN, et al: Lactation failure in swine: The effect of feed intake and the intestinal infusion of E. coli. Proc CRWAD, Chicago, 1982, p 39.

33. Benton W, Benton H: Encyclopaedia Britannica. Macropaedia. Vol XI, Chicago Ill, 1981, pp 835-1949.

34. Machlin LJ: Vitamin E: A comprehensive treatise. Basic and Clin Nutr, Vol I, Marcel Dekker, Inc., New York, 1980, pp 1-5.

35. Marks J: Vitamins in health and disease: A guide to vitamins. Tech Publish Co, Ltd, Cambridge, England, 1975, pp 62-65.

36. de Duve C, Hayaiski O: Tocopherol, oxygen and biomembranes. Biomedical Press. Elsevier, North Holland, Amsterdam, New York, 1978, pp 23-71.

37. Taylor TG: The importance of vitamins to human health. Int Med Publisher, London, 1978, pp 101-107.

38. Schwarz K, Foltz CM: Selenium as an integral part of Factor 3 against dietary necrotic liver degeneration. J Am Chem Soc 79: 3293, 1957.

39. Fontenot JP, Coppock CE, Goodrich RD, et al: Mineral tolerance of domestic animals. Acad of Sci, Washington D. C., 1980, pp 242-252, 392-401.

40. Hanahan DJ, De Luca H: The fat soluble vitamins. Handbook of lipid research, Vol 2, Plenum Press, New York and London, 1978, pp 133-197.

41. Van Vleet JF: Current knowledge of Selenium-Vitamin E deficiency in domestic animals. J Am Vet Med Assoc 176: 321-324, 1980.

42. Cunha TJ: Swine feeding and nutrition. Academic Press, New York, 1977, pp 127-130.

43. Nockels CF: Protective efforts of supplemental vitamin E against infection. Fed Proc, 38: 2134-2138, 1979.

44. Ellis RP, Vorhies MW: Effect of supplemental vitamin E on the serologic response of swine to an E. coli bacterin. J Am Vet Med Assoc 168: 231-232, 1976.

45. Peplowski MA, Mahan DC, Murray FA, et al: Effect of dietary and injectable vitamin E and selenium in weanling swine antigenetically challenged with sheep red blood cells. J Anim Sci 51: 344-351, 1981.

46. Teige J Jr, Nordstoga K, Aursjo J: Influence of diet on experimental swine dysentery. 1. Effects of a vitamin E and selenium deficient diet supplemented with 6.8% cod liver oil. Act Vet Scand 18:384-396, 1977.

47. Teige J Jr, Saxegaard F, Froslie A: Influence of diet on experimental swine dysentery. 2. Effects of a vitamin E and selenium deficient diet supplemented with 3% cod liver oil, vitamin E or selenium. Act Vet Scand 19: 133-146, 1978.

48. Cunha TJ, Becker DE, Bowland JP, et al: Nutrient Requirements of Swine. Nat Acad Sci, Washington D. C., 1968, pp 12-13.

49. Cunha TJ, Baumgardt BR, Bell JM, et al: Nutrient Requirements of Swine. Nat Res Counc (NRC), Washington D. C. , 1973, pp 11-12.

50. Ullrey DE: Vitamin E and MMA. Rep of Swine Res Dept of Anim Sci, Michigan State University, East Lansing, 99: 10-13, 1969.

51. Ullrey DE: Vitamin E (Selenium and Choline) in reproduction and MMA. Rep of Swine Res Dept of Anim Sci, Michigan State University, East Lansing 99: 10-13, 1969.

52. Young LG, Lun A, Pos J, et al: Vitamin E stability in corn and mixed feed. J Anim Sci 40: 495-499, 1975.

53. Beck WS: Hematology. The Mitt Press, Cambridge, 1978, pp 165-173.

54. Whitehair CK, Miller ER: Nutritional deficiencies, in Leman AD (ed): Diseases of Swine. Ames, Iowa, Iowa State University Press, 1981, pp 656-670.

55. Miller ER: Baby pig anemia and the need for iron. Cont Anim Health News Conf, 1980.

56. Miller ER, Waxler GL, Ku PK, et al: Iron requirements of baby pigs reared in germ-free or conventional environments on a condensed milk diet. A Anim Sci 54: 106-115, 1982.

57. Cook RW: Iron Tolerance in the Young Pig. M. S. Thesis. Dept of Path, Michigan State University, East Lansing, 1974.

58. Arpi T, Tollerz G: Iron poisoned in piglets. Autopsy findings in experimental and spontaneous cases. Acta Vet Scand 6: 360-373, 1965.

59. Behrens H: Pigs poisoned by iron injection. J Am Vet Med Assoc 132: 169-170, 1958.

60. Blandford TB, Lodge GA: Acute hepatic necrosis following an iron dextran injection. Vet Rec 78: 117, 1966.

61. Lannek N, Lindberg P, Tollerz G: Lowered resisance to iron in vitamin E-deficient piglets and mice. Nature 195: 1006-1007, 1962.

62. Henriksson K: Poisoning of piglets during iron therapy. Finsk Veterinartids Krift 68: 293-297, 1962.

63. Tollerz G, Lannek N: Protection against iron toxicity in vitamin E-deificnet piglets and mice by vitamin E and synthetic antioxidants. Nature 201: 846-847, 1964.

64. Ullrey, DE: Vitamin E for swine. J Anim Sci 53: 1039-1052, 1981.

65. Worthington R, Bonnie S: Contemporary developments in nutrition. The CV Mosby Co., St. Louis, 1981, pp 135-158.

66. Gryglewski RJ, Vane JR: The evolution of ideas in prostaglandin research, in Ruzyllo E (ed): Materia Medica Polona, The Polish J of Med and Phar 12: 159-168, 1980.

67. Gallavan RH Jr, Jacobson ED: Prostaglandins and the splanchnic circulation. Proc Soc Exp Biol Med 170: 391-397, 1982.

68. Takeuchi K, Svanes K, Critshlow J, et al: Prostaglandins stimulate and inhibit acid secretion in amphibian fundic mucosa. Proc Soc Exp Biol Med 170: 398-404, 1982.

69. Reines HD, Halushka PV, Cook JA, et al: Plasma thromboxane concentrations are raised in patients dying with septic shock. Lancet 2: 174-175, 1982.

70. Lipinski B, Macklin LJ: Enhanced susceptibility to endotoxin-induced intravascular coagulation in vitamin E deficiency. Med Sci 9: 122-123, 1981.

71. Sharp BA, Young LG, van Dreumel AA: Effect of supplemental vitamin E and selenium in high moisture corn diets on the incidence of mulberry heart disease and hepatosis dietetica in pigs. Can J Comp Med 36: 393-397, 1972.

72. Moran ET, Carlson HC, Pettit JR: Vitamin E-Selenium deficiency in the duck aggravated by the use of high-moisture corn and molding prior to preservation. Avian Dis 18: 536-543, 1974.

73. McGovern JJ, Jones AR, Steinberg AG: The hematocrit of capillary blood. New Eng J Med 253: 308, 1955.

74. Crosby WH, Munn JI, Furth FW: Standardizing a method for clinical hemoglobinometry. U.S. Armed Forces Med J 5: 693, 1954.

75. Whetter PA, Ku PK: Procedure for plasma/serum α-tocopherol determinations. Dept of Anim Sci, Michigan State University, East Lansing, 1982.

76. Taylor SL: Sensitive method for tissue tocopherol analysis. Lip 11: 530-538, 1976.

77. Whetter PA, Ullrey DE: Improved fluorometric method for determiing selenium. J Assoc Off Anal Chem 61: 927-930, 1978.

78. Olson AD, Hamlin WB: A new method for serum iron and total iron binding capacity by atomic spectrophotometry. Clin Chem 15: 438-444, 1969.

79. Paglia DE, Valentine WN: Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. J Lab and Clin Med 70: 158-169, 1967.

80. Lawrence RA, Sunde RA, Schwartz GL, et al: Glutathione Peroxidase Activity in rat lens and other tissues in relation to dietary selenium intake. Exp Eye Res 18: 563-569, 1974.

81. Luna LL: Manual of Histologic Staining Methods of the Armed Forces Institute of Pathology. McGraw-Hill Book Co., New York, 1968.

82. Fredman D, Pisani R, Purves R: Statistics. W. W. Norton and Co., Inc., New York, 1978, pp 146-166.

83. Whitehair CK: Role of vitamin E and selenium in swine diseases. J Am Vet Med Assoc 158: 1853-1854, 1971.

84. Mahan DC, Pehale LH, Clive JH, et al: Efficacy of supplemental selenium in reproductive diets on sow and progeny performance. J Anim Scie 39: 536-543, 1974.

85. Piatowski TL, Mahan DC, Cantor AH, et al: Selenium and vitamin E in semipurified diets for gravid and nongravid gilts. J Anim Sci 48: 1357-1365, 1979.

86. Wilkinson JE, Bell MC, Masincupp FB: Effects of supplemental selenium on swine. I. gestation and lactation. J Anim Sci 44: 224, 1977.

87. Sivertsen T, Karlsen JT, Froslie A: The relationship of erythrocyte glutathione peroxidase to blood selenium in swine. Act Vet Scand 18: 494-500, 1977.

88. Klasing KC, Knight CD, Forsyth DM: Effects of iron on the anticoli capacity of sow's milk in vitro and in ligated intestinal segments. J Nutr 110: 1914-1921, 1980.

89. Kadis S, Dressen DW, Shotts EB: Relationship between administration of iron to baby pigs and their susceptibility to enterotoxic colibaciosis. Res Inv Rep. Iowa, 1983, pp 33-34.

90. Lee YH, Layman DK, Bell RD, et al: Response of glutathione peroxidase and catalase to excess dietary iron in rats. J Nutr 111: 2195-2202, 1981.

91. Jørgensen Fogd P, Hyldgaard-Jensen J, et al: Glutathione peroxidase activity in porcine blood. Act Vet Scand 18: 323-334, 1977.

92. Underwood EJ: Trace elements in human and animal nutrition. Academic Press, New York, 1971, pp 40-41.

93. Ross RF: Vaccination for control of mastitis and agalactia of the sow. Res Inv Rep. Iowa 1983, pp 31-32.

VITA

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APPENDIX

TABLE A-1. PLASMA SELENIUM (Se), VITAMIN E, GLUTATHIONE PEROXIDASE (GSH-Px) ACTIVITY AND IRON (Fe) INDIVIDUAL VALUES OF GILTS DURING GESTATION AND LACTATION.

PRODUCTION PERIOD	GILT NO.	Se, µg/ml	VITAMIN E µg/ml	GSH-Px EU/ml	Fe µg/dl
		MSU BASAL DIET[a]			
Gestation[c] (30 Days)	209-2	.139	2.95		
	211-4	.144	1.30		
	103-4	.163	2.48		
	208-1	.192	2.10		
	148-1	.099	4.76		
Gestation[c] (100 Days)	209-2	.134	1.15		
	211-4	.133	.60		
	103-4	.126	.77		
	208-1	.131	.83		
	148-1	.118	.99		
Lactation (21 Days)	209-2	.136	1.10	.423	122.1
	211-4	.093	.97	.659	99.0
	103-4	.061	.68	.608	100.8
	208-1	.079	.33	.442	65.5
	148-1	.076	.35	.486	95.3
	206-1	.079	.78	.346	86.7
	146-3	.056	.86	.193	102.0
	211-2	.123	N.D.	.824	90.9
	147-5	.056	1.43	.299	137.4
	107-1	.055	1.14	.441	128.4
		+ VITAMIN E-Se DIET[b]			
Gestation[c] (30 Days)	208-3	.197	2.04		
	209-1	.065	1.43		
	211-1	.203	2.05		
	148-2	.208	2.06		
	103-1	.133	3.11		
Gestation[c] (100 Days)	208-3	.176	1.53		
	209-1	.169	1.61		
	211-1	.177	2.10		
	148-2	.186	2.10		
	103-1	.140	2.21		
Lactation (21 Days)	208-3	.218	2.54	.771	89.1
	211-1	.176	2.65	1.093	99.6
	148-2	.211	1.00	.973	109.8
	146-1	.164	1.73	.778	84.0
	214-1	.153	1.76	.611	82.5
	206-2	.232	2.93	1.471	58.8
	211-3	.196	1.66	.892	71.1
	147-2	.203	1.69	.695	100.2
	107-3	.224	2.18	1.235	111.0

[a]Dried shelled corn-soybean

[b]Dried shelled corn-soybean supplemented with E-Se.

[c] = GSH-Px and Fe values were not determined in either group.

N.D. = Non-detectable.

TABLE A-2. PLASMA SELENIUM (Se), VITAMIN E, GLUTATHIONE PEROXIDASE (GSH-Px) ACTIVITY AND IRON (Fe) INDIVIDUAL VALUES OF BABY PIGS AT BIRTH NURSING GILTS FED THE BASAL DIET.

No. of Pig	Se μg/ml	Vitamin E μg/ml	GSH-Px EU/ml	Fe μg/dl
140-1	.107	.36	.161	63.6
140-2	.034	.53	.198	62.8
140-3	.048	.13	.124	54.0
140-10	.057	.47	.164	53.3
140-11	.079	.21	.138	59.1
140-12	.070	.63	.146	69.8
140-13	.104	.32	.125	50.9
141-1	.042	.30	.080	62.70
141-2	.059	.40	.098	a
141-3	.035	.50	.069	a
141-4	.022	.40	.029	a
141-5	.037	.40	.074	53.10
141-6	.035	.65	.015	58.80
141-7	.034	.76	.113	a
141-8	.016	.58	.072	a
141-10	.030	.30	.095	80.40
141-11	.036	.50	.068	a
141-12	.031	.20	.045	a
145-1	.039	1.53	.088	59.70
145-2	.039	2.49	.096	62.40
145-3	.031	2.33	.072	a
145-4	.047	1.88	.064	60.60
145-5	.049	.65	.088	a
145-6	.019	1.48	.056	a
145-10	.029	1.36	.104	a
145-11	.022	1.52	.088	a
145-12	.026	2.20	.080	64.80
145-13	.023	2.68	.016	65.40
145-14	.027	1.05	.064	a
145-15	.032	1.65	.072	a
145-16	.029	1.58	.072	a

a = values not expressed; not enough plasma

TABLE A-2. (continued)

No. of Pig	Se µg/ml	Vitamin E µg/ml	GSH-Px EU/ml	Fe µg/dl
146-1	.028	2.68	.152	a
146-2	.029	1.72	.144	a
146-3	.023	2.99	.144	a
146-4	.024	2.76	.136	a
146-5	.022	3.40	.168	a
146-6	.025	2.36	.192	a
146-10	.032	3.00	.217	70.20
146-11	.038	2.44	.201	a
a = values not expressed; not enough plasma				
147-1	.023	1.87	.128	68.40
147-2	.021	a	.096	a
147-3	.014	.42	.056	59.40
147-10	.033	.39	.104	94.80
147-11	.035	1.76	.104	85.20
a = values not expressed; not enough plasma				
148-1	.024	a	.072	46.92
148-2	.032	a	.112	28.02
148-3	.027	a	.128	a
148-10	.017	1.21	.088	a
148-11	.021	1.95	.144	39.42
148-12	.017	.64	.088	34.92
148-13	.018	.59	.080	45.65
148-14	.028	1.03	.072	45.65
148-15	.014	2.65	.064	a
148-16	.026	1.44	.144	a
148-17	.054	1.09	.096	a
148-18	.043	3.12	.088	a
a = values note expressed; not enough plasma				
149-1	.041	.67	.195	96.60
149-2	.026	.72	.120	88.20
149-3	.019	.45	.188	65.10
149-4	.029	.99	.182	64.50

TABLE A-2. (continued)

No. of Pig	Se µg/ml	Vitamin E µg/ml	GSH-Px EU/ml	Fe µg/dl
150-1	.029	1.25	.120	78.35
150-2	.035	1.31	.088	92.15
150-3	.027	.10	.120	a
150-4	.027	.63	.064	a
150-5	.034	.33	.096	a
150-6	.033	.70	.112	a
150-10	.029	1.79	.096	a
150-11	.029	.10	.120	68.45
150-12	.024	.76	.128	64.25
150-13	.027	1.70	.112	63.35
150-14	.033	2.34	.128	54.85

a = value not expressed; not enough plasma

154-1	.021	.50	.088	74.52
154-2	.032	.52	.104	a
154-3	.038	.52	.104	a
154-4	.044	.48	.168	51.42
154-5	.021	.50	.120	a
154-6	.021	.13	.088	a
154-10	.031	.73	.088	62.22
154-11	.030	.95	.104	a

a = value not expressed; not enough plasma

163-10	.036	.91	.133	a
163-11	.026	1.81	.158	a
163-12	.023	1.81	.124	a

a = value not expressed; not enough plasma

TABLE A-3. PLASMA SELENIUM (Se), VITAMIN E, GLUTATHIONE PEROXIDASE (GSH-Px) ACTIVITY AND IRON (Fe) INDIVIDUAL VALUES OF BABY PIGS AT 21 DAYS OF AGE NURSING GILTS FED THE BASAL DIET.

No. of Pig	Se µg/ml	Vitamin E µg/ml	GSH-Px EU/ml	Fe µg/dl
140-1	.060	1.00	.410	166.5
140-2[a]	--	--	--	--
140-3	.056	1.35	.410	79.3
140-10	.063	1.29	.487	224.5
140-11	.046	.74	.344	109.4
140-12	.043	.85	.315	183.3
140-13	.063	.30	.328	143.3
(a) Pig 140-2 died before 21 days of age.				
141-1	.043	.56	.275	261.0
141-2	.041	.38	.307	167.6
141-3	.040	.50	.195	79.2
141-4	.039	.41	.228	194.4
141-5	.040	.36	.169	114.4
141-6	.055	.28	.201	170.2
141-7	.050	.38	.317	170.8
141-8	.037	.17	.251	175.2
141-10[a]	--	--	--	--
141-11	.057	.28	.252	32.7
141-12	.071	.55	.235	90.7
(a) Pig 141-10 died before 21 days of age.				
145-1	.038	1.90	.305	151.8
145-2	.039	.73	.297	a
145-3	.043	1.79	.233	145.8
145-4	.036	1.50	.273	83.1
145-10	.037	2.48	.289	140.7
145-11	.043	2.07	.210	179.4
145-12	.044	1.00	.185	a
145-13	.044	.82	.265	117.9
145-14	.041	1.09	.153	184.2
145-16	.041	1.99	.169	a
a = value not expressed; not enough plasma				
146-1	.060	1.11	.337	147.9
146-2	.061	1.44	.273	174.6
146-3	.058	1.29	.281	152.7
146-4	.060	1.62	.265	153.6
146-5	.056	1.17	.337	127.5
146-6	.045	1.54	.209	111.0
146-10	.046	1.42	.249	115.2

TABLE A-3. (continued)

No. of Pig	Se µg/ml	Vitamin E µg/ml	GSH-Px EU/ml	Fe µg/dl
147-1	.067	1.27	.426	88.8
147-2	.059	.83	.297	130.8
147-3	.066	1.09	.362	119.7
147-10	.062	1.03	.321	135.0
147-11	.080	1.64	.378	96.3
148-1	.038	1.40	.313	165.3
148-3	.037	.97	.129	144.9
148-10	.035	1.59	.113	138.0
148-11	.033	1.45	.289	150.6
148-12	.052	1.52	.201	159.0
148-13	.039	1.08	.305	131.7
148-14	.052	1.01	.350	166.2
148-15	.040	1.67	.357	108.6
149-1[a]	--	--	--	--
149-2[a]	--	--	--	--
149-3[a]	--	--	--	--
149-4[a]	--	--	--	--

(a) The entire litter died within 3 days of birth.

150-1	.031	.90	.339	129.6
150-2	.028	1.01	.320	a
150-3	.026	.90	.148	156.3
150-4	.037	.85	.360	a
150-4	.027	.83	.326	a
150-6	.026	.79	.307	a
150-10	.029	.70	.212	a
150-12	.043	.90	.365	242.4
150-13	.031	1.05	.195	a

a = value not expressed; not enough plasma

154-1	.053	1.14	.341	98.7
154-2	.030	.70	.211	133.2
154-3	.050	.88	.217	128.1
154-4	.036	.93	.424	71.0
154-5	.033	1.10	.158	a
154-10	.039	.88	.347	76.5
154-11	.042	1.34	.503	81.6

a = value not expressed; not enough plasma

TABLE A-3. (continued)

No. of Pig	Se µg/ml	Vitamin E µg/ml	GSH-Px EU/ml	Fe µg/dl
163-10	.061	N.D.	.595	127.8
163-11	.059	.08	.463	118.2
163-12	.064	.02	.396	232.8

N.D. = Non-Detectable values

TABLE A-4. PLASMA SELENIUM (Se), VITAMIN E, GLUTATHIONE PEROXIDASE (GSH-Px) ACTIVITY AND IRON (Fe) INDIVIDUAL VALUES OF BABY PIGS AT BIRTH NURSING GILTS FED THE VITAMIN E AND Se SUPPLEMENTED DIET.

No. of Pig	Se µg/ml	Vitamin E µg/ml	GSH-Px EU/ml	Fe µg/dl
138-1	.108	2.64	.270	66.20
138-2	.080	2.54	.132	74.40
138-3	.139	2.87	.158	66.45
138-4	.115	1.12	.170	67.35
138-5	.088	1.61	.105	63.75
138-6	.082	2.39	.146	69.45
138-7	.084	2.58	.280	55.50
138-8	.081	1.51	.193	75.00
138-9	.078	1.92	.080	66.30
138-10	.083	1.14	.127	72.30
138-11	.103	3.81	.114	64.50
138-12	.156	1.02	.145	70.80
142-1	-a	1.42	.164	67.7
142-2	-a	1.00	.161	35.4
142-3	.083	.26	.187	58.3
142-4	a	1.09	.204	51.7
142-5	.086	.66	.153	50.4
142-6	.076	.88	.195	90.0
142-7	.078	.34	.215	a-
142-10	.063	.75	.204	52.5
142-11	.061	.67	.164	45.2
142-12	.057	.50	.135	a-

a = value not expressed; not enough plasma

143-1	.041	2.07	.098	a-
143-2	.049	1.60	.082	57.65
143-3	.033	1.23	.066	a
143-4	.024	1.66	.076	62.45
143-5	.042	2.27	.076	53.15
143-10	.045	2.74	.151	54.35
143-11	.063	2.64	.109	81.65
143-12	.078	2.40	.116	a-
143-13	.026	2.49	.105	49.25
143-14	.055	3.67	.158	a-

a = value not expressed; not enough plasma

TABLE A-4. (continued)

No. of Pig	Se µg/ml	Vitamin E µg/ml	GSH-Px EU/ml	Fe µg/dl
144-1	.054	.81	.104	63.00
144-2	.037	2.40	.148	61.80
144-3	.059	2.66	.153	81.00
144-10	.046	.63	.096	95.40
144-11	.050	.99	.120	81.60
144-12	.024	1.36	.128	84.00
155-1	.034	1.20	.152	a
155-2	.036	3.84	.136	58.02
155-3	.057	.83	.136	56.82
155-4	.041	.52	.144	52.92
155-5	.045	.27	.120	a
155-10	.035	.59	.096	75.42
155-11	.045	.59	.072	57.12
155-12	.032	1.82	.104	a
155-13	.039	1.58	.120	76.92
155-14	.035	1.62	.136	117.12
155-15	.064	1.91	.144	a
155-16	.034	1.02	.072	a

a = value not expressed; not enough plasma

159-1	.052	.83	.175	69.30
159-2	.048	.99	.127	74.10
159-3	.043	1.10	.164	78.00
159-4	.057	.94	.159	105.00
159-5	.049	.62	.095	93.00
159-10	.049	.80	.121	105.30
159-11	.042	.72	.072	77.70
159-12	.044	1.46	.146	117.30
159-13	.068	1.49	.183	71.40
159-14	.039	1.92	.162	96.60
160-1	.073	1.14	.187	70.62
160-2	.009	1.00	.180	80.92
160-3	.058	.63	.198	a
160-4	.045	.86	.172	a
160-10	.052	.86	.166	50.22
160-11	.063	.73	.275	65.52
160-12	.012	1.40	.129	61.02
160-13	.107	.93	.122	62.52
160-14	.076	1.13	.166	78.12
160-15	.107	.79	.106	63.72
160-16	.101	1.21	.179	87.12
160-17	.039	.83	.169	46.02

a = value not expressed; not enough plasma

TABLE A-4. (continued)

No. of Pig	Se µg/ml	Vitamin E µg/ml	GSH-Px EU/ml	Fe µg/ml
161-1	.100	4.31	.361	a
161-2	.074	5.13	.337	a
161-10	.083	4.29	.313	117.9
161-11	.082	4.23	.225	91.8
161-12	.062	2.91	.281	92.7
161-13	.060	3.35	.241	91.2
161-14	.069	1.35	.209	63.9
a = value not expressed; not enough plasma				
162-1	.038	3.45	.289	112.5
162-2	.065	3.64	.281	78.3
162-3	.047	1.27	.193	79.8
162-4	.046	1.56	.209	66.3
162-5	.022	1.61	.137	69.3
162-6	.049	3.96	.193	90.0
162-7	.044	2.11	.137	64.2
162-8	.039	2.86	.152	72.8
162-9	.048	3.49	.169	70.5
162-10	.039	2.23	.193	62.7
162-11	.042	2.32	.161	78.3
162-12	.046	3.22	.177	94.8
162-13	.039	3.24	.177	93.9

TABLE A-5. PLASMA SELENIUM (Se), VITAMIN E, GLUTATHIONE PEROXIDASE (GSH-Px) ACTIVITY AND IRON (Fe) INDIVIDUAL VALUES OF BABY PIGS AT 21 DAYS OF AGE, NURSING GILTS FED A VITAMIN E-Se SUPPLEMENTED DIET.

No. of Pig	Se μg/ml	Vitamin E μg/ml	GSH-Px EU/ml	Fe μg/dl
138-1	.083	1.26	.233	75.3
138-2	.088	3.31	.378	a
138-3	.078	2.10	.297	a
138-4	.075	1.86	.233	86.4
138-5	.090	2.41	.297	198.8
138-6	.079	1.41	.370	119.4
138-7	.095	1.92	.257	75.0
138-8	.086	3.23	.289	164.1
138-9	.081	2.96	.233	102.3
138-10	.085	3.46	.321	165.3
138-11	.091	1.88	.426	110.4
a = value not expressed; not enough plasma				
142-1	.080	2.08	.442	119.1
142-2	.072	2.31	.402	118.2
142-3	.067	2.44	.265	178.2
142-4	.068	1.55	.337	143.4
142-5	.062	1.56	.396	131.4
142-10	.064	2.03	.265	96.6
142-11	.066	1.21	.570	79.8
142-12	.064	2.31	.361	102.3
143-1	.072	1.33	.570	116.1
143-2	.064	1.99	.321	a
143-3	.062	1.12	.394	74.1
143-4	.077	2.06	.401	149.1
143-5	.074	2.53	.595	79.2
143-10	.077	2.47	.482	123.9
143-11	.076	1.66	.426	86.1
143-12	.054	2.01	.410	127.2
143-13	.077	2.17	.579	109.1
143-14	.103	2.66	.635	a
a = value not expressed; not enough plasma				
144-1	.077	1.35	.201	134.1
144-2	.058	1.04	.257	108.0
144-3	.065	1.45	.176	104.6
144-10	.068	.92	.378	50.4
144-11	.072	1.25	.217	102.0
144-12	.070	1.58	.370	129.3

TABLE A-5. (continued)

No. of Pigs	Se µg/ml	Vitamin E µg/ml	GSH-Px EU/ml	Fe µg/dl
155-1	.066	2.19	.482	86.4
155-2	.066	2.68	.616	97.8
155-3	.072	2.41	.225	109.2
155-10	.066	1.55	.254	71.4
155-11	.074	2.10	.334	96.0
155-13	.069	2.23	.367	85.2
155-14	.069	1.95	.153	114.0
155-15	.067	1.75	.164	82.5
155-16	.054	1.86	.288	90.6
159-1	.061	2.00	.294	a
159-2	.065	1.99	.392	65.1
159-3	.079	2.12	.314	76.8
159-5	.061	2.50	.241	79.2
159-12	.058	2.10	.289	151.5
159-14	.073	2.05	.201	114.0
a = value not expressed; not enough plasma				
160-1	.071	1.87	.489	a
160-2	.076	1.59	.370	a
160-3	.099	2.67	.564	102.6
160-4	.064	1.42	.408	a
160-10	.079	2.43	.471	94.2
160-11	.087	1.72	.754	a
160-12	.073	2.85	.505	183.3
160-13	.072	1.69	.389	a
160-14	.086	2.35	.564	132.6
160-15	.069	2.08	.523	101.4
160-16	.087	2.03	.566	117.4
160-17	.067	1.47	.481	71.4
a = values not expressed; not enough plasma				
161-1	.083	2.33	.367	169.2
161-2	.097	3.59	.392	200.7
161-10	.077	2.60	.373	168.3
161-11	.067	3.71	.429	133.8
161-13	.080	2.31	.270	71.1

TABLE A-5. (continued)

No. of Pig	Se µg/ml	Vitamin E µg/ml	GSH-Px EU/ml	Fe µg/dl
162-1	.073	1.90	.291	96.6
162-2	.077	2.79	.348	119.1
162-3	.059	1.42	.441	88.2
162-4	.069	2.61	.574	83.4
162-5	.071	3.29	.441	102.0
162-6	.076	2.54	.595	117.6
162-7	.088	2.45	.457	a
162-8	.078	2.23	.280	79.5
162-9	.076	4.06	.246	94.5
162-11	.085	2.49	.585	77.7
162-12	.069	3.09	.434	a
162-13	.064	2.07	.334	91.2

a = value not expressed; not enough plasma

TABLE A-6. PLASMA SELENIUM (Se), VITAMIN E, GLUTATHIONE PEROXIDASE (GSH-Px) ACTIVITY AND IRON (Fe) VALUES[a] OF PIGS PER LITTER AT BIRTH AND AT 21 DAYS OF AGE

MSU BASAL DIET

LITTER NO.	Se µG/ML		VITAMIN E µG/ML		GSH-Px EU[b]/ML		Fe µG/DL	
	1 DAY	21 DAYS	1 DAY	21 DAYS	1 DAY	21 DAYS	1 DAY	21 DAYS
140	(7) 0.071	(6) 0.055	(7) 0.37	(6) 0.92	(7) 0.150	(6) 0.382	(7) 59.00	(6) 151.0
141	(11) 0.034	(10) 0.047	(11) 0.45	(10) 0.38	(11) 0.069	(10) 0.243	(4) 63.75	(10) 146.6
145	(13) 0.032	(10) 0.041	(13) 1.72	(10) 1.53	(13) 0.074	(10) 0.238	(5) 62.58	(7) 143.2
146	(8) 0.027	(7) 0.055	(8) 2.67	(7) 1.37	(8) 0.169	(7) 0.278	(1) 70.20	(7) 140.3
147	(5) 0.025	(5) 0.067	(4) 1.11	(5) 1.17	(5) 0.097	(5) 0.357	(4) 76.95	(5) 114.1
148	(12) 0.027	(8) 0.041	(9) 1.52	(8) 1.34	(12) 0.098	(8) 0.257	(6) 40.10	(8) 145.5
149	(4) 0.029	(0) --	(4) 0.71	(0) --	(4) 0.171	(0) --	(4) 78.60	(0) --
150	(11) 0.027	(9) 0.031	(11) 1.00	(9) 0.88	(11) 0.109	(9) 0.286	(6) 70.23	(6) 138.5
154	(8) 0.029	(7) 0.040	(8) 0.54	(7) 1.00	(8) 0.108	(7) 0.314	(3) 62.72	(6) 98.18
163	(3) 0.028	(3) 0.061	(3) 1.51	(3) 0.09	(3) 0.138	(3) 0.485	(0) ---	(3) 159.9

+ VITAMIN E-Se DIET

LITTER NO.	Se µG/ML		VITAMIN E µG/ML		GSH-Px EU /ML		Fe µG/DL	
	1 DAY	21 DAYS	1 DAY	21 DAYS	1 DAY	21 DAYS	1 DAY	21 DAYS
138	(12) 0.099	(11) 0.085	(12) 2.09	(11) 2.34	(12) 0.160	(11) 0.303	(12) 67.66	(10) 113.3
142	(7) 0.072	(8) 0.068	(10) 0.75	(8) 1.94	(10) 0.178	(8) 0.376	(8) 56.40	(8) 121.1
143	(10) 0.046	(10) 0.074	(10) 2.28	(10) 2.00	(10) 0.103	(10) 0.481	(6) 59.75	(8) 108.1
144	(6) 0.045	(6) 0.068	(6) 1.47	(6) 1.27	(6) 0.124	(6) 0.124	(6) 77.80	(8) 104.7
155	(12) 0.041	(9) 0.067	(12) 1.31	(9) 2.08	(12) 0.119	(9) 0.320	(7) 70.62	(9) 92.5
159	(10) 0.049	(6) 0.066	(10) 1.08	(6) 2.13	(10) 0.140	(6) 0.289	(10) 88.70	(5) 105.3
160	(12) 0.062	(12) 0.078	(12) 0.96	(12) 2.01	(12) 0.171	(12) 0.504	(10) 66.57	(7) 114.7
161	(7) 0.076	(5) 0.081	(7) 3.65	(5) 2.91	(7) 0.281	(5) 0.366	(5) 91.50	(5) 148.6
162	(13) 0.043	(12) 0.074	(13) 2.68	(12) 2.58	(13) 0.190	(12) 0.419	(13) 79.47	(10) 94.6

[a] VALUES ARE EXPRESSED AS AVERAGES PER LITTER. INDIVIDUAL VALUES ARE IN APPENDIX TABLES.

[b] ENZYME UNITS AS MICROMOLES OF GSH OXIDIZED PER MINUTE.

( ) = NUMBER OF SAMPLES.

TABLE A-7. LIVER SELENIUM (Se) INDIVIDUAL VALUES FROM 14 PIGS NECROPSIED

Sample No.	Liver Sample Weight (g)	DM[a] (%)	Se µg/g DM[a]
	BASAL DIET		
145-6	5.054	19.22	1.23
146-11	5.167	22.18	1.44
148-17	5.165	18.80	1.08
148-16	5.014	24.42	0.78
149-2	5.514	20.18	1.17
149-3	5.185	20.83	1.25
149-4	5.222	23.17	0.83
150-5	5.127	21.96	0.80
	+ VITAMIN E-Se DIET		
138-2	5.090	22.01	0.94
144-?	5.394	20.31	1.09
155-5	5.076	23.07	1.37
159-10	5.176	19.76	1.45
159-11	5.072	18.29	1.67
159-15	5.438	19.36	1.33

[a]DM = dry matter basis