SUPPLEMENTING WEANLING PIGS WITH HIGH CONCENTRATIONS OF ZN AND THE ZN AVAILABILITY OF ZN SOURCES FOR WEANLING PIGS

by

Timothy Charles Schell

Dissertation submitted to the Faculty of the Virginia Polytechnic Institute and State University in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

in

Animal Science

Approved:

E. T. Kornegay, Chairman

G. E. Bunce

F. C. Gwazdauskas

C. J. Pfeiffer

C. M. Wood

September, 1994

Blacksburg, Virginia

SUPPLEMENTING WEANLING PIGS WITH HIGH CONCENTRATIONS OF ZN AND THE ZN AVAILABILITY OF ZN SOURCES FOR WEANLING PIGS

by

Timothy C. Schell

Committee Chairman: E. T. Kornegay

Animal Science

ABSTRACT

Thirteen trials (n=930) were conducted to investigate the supplementation of weanling pigs with high levels of Zn and to compare the availability of Zn from several Zn sources for weanling pigs. In the first four trials, supplementing Zn by injecting Zn acetate either i.m. or i.p. at various times near weaning did not improve postweaning growth performance compared with pigs that were not injected. Additionally, stressing pigs by regrouping and then injecting Zn acetate did not improve growth performance. Serum Zn concentrations were increased in all of the trials by the injection of Zn. In the next five trials, feeding 3,000, 2,000 or 1,000 mg Zn/kg of diet from $ZnSO_4$, Zn-lysine or Zn-methionine did not improve growth performance immediately after weaning compared with pigs fed diets with 105 mg Zn/kg of diet. Feeding 3,000 mg Zn/kg of diet as ZnO ($P < .05$) improved growth performance above that of pigs fed 3,000 mg Zn/kg of diet from the other sources,

but did not improve growth performance compared to controls. Lower tissue Zn concentrations suggested a lower availability of Zn from ZnO compared with $ZnSO_4$, Zn-lysine and Zn-methionine. There was little difference in Zn availability among the other sources. In the next three trials, feeding diets with different levels of lysine had little influence on the availability of Zn from Zn-lysine compared to $ZnSO_4$. Results indicate that Zn from Zn-lysine is not absorbed in conjunction with the lysine component of the complex. Additionally, there were no differences in the availability of Zn from $ZnSO_4$ compared to Zn-lysine. In the last trial, Zn from ZnO was less available ($P < .05$) to Zn deficient pigs than $ZnSO_4$, Zn-lysine or Zn-methionine when rib bone Zn concentration was used as an indicator of Zn availability. In summary, supplementing weanling pigs with high levels of Zn immediately before or after weaning does not appear to improve growth performance. Furthermore, Zn from ZnO is less available to weanling pigs than Zn from $ZnSO_4$, Zn-lysine or Zn-methionine.

ACKNOWLEDGEMENTS

I would like to express my sincere appreciation to Dr. E. T. Kornegay for his advice, expertise, understanding and time. The freedom he allowed me in determining the area of my research has made my work both enjoyable and meaningful.

I would like to thank the members of my committee Dr. G. E. Bunce, Dr. F. C. Gwazdauskas, Dr. C. J. Pfeiffer and Dr. C. M. Wood for their time and efforts. They have all broadened my knowledge more than they will ever know.

I would also like to express my sincere appreciation to Dr. T. Goodale, Dr. L. Peters, and Dr. J. Eaton for listening when needed and helping when possible.

I am greatly indebted to Gary Apgar for his assistance countless times. Without his help, this research project would not have been possible or enjoyable.

The efforts and patience of Lisa Flory, Marilyn Rohl and Don Conner in laboratory assistance are greatly appreciated.

Also, I would like to thank Cindy Hixon for her time and invaluable technical assistance as well as her sense of humor.

I would also like to thank Gene Ball, Ricky Dove and Mike Graham for their assistance at the Swine Center numerous times.

To the department of Animal and Poultry Sciences, I am grateful for financial and moral support.

Mostly, I would like to thank the many other graduate students who were quick to lend a helping hand when needed. Their support and friendship throughout my graduate career were greatly appreciated.

Finally, I would like to thank my mother and brother for their patience and support.

I hope that someday each of these individuals will know how much they have influenced my life.

TABLE OF CONTENTS

LIST OF TABLES

CHAPTER IV

CHAPTER V

LIST OF FIGURES

CHAPTER IV

Chapter I

Introduction

The primary goal of pork producers is the efficient production of a product acceptable to the consumer. The increased competitiveness of poultry and beef products in the marketplace has put increased pressure on swine producers to improve their efficiency of production. Additionally, pressures from groups with environmental concerns represent a new challenge to producers. As regulations on the amount of animal waste that can be applied to land increase, more pressure is placed on the animal industry to increase the efficiency of nutrient utilization by animals. Therefore, the swine industry currently faces two major challenges: maintaining a secure spot in the marketplace and addressing the environmental challenges of the future.

Many methods of improving the efficiency of pork production have been investigated over the last several decades, but the area that has probably received the most attention is that of significantly improving the low level of growth performance exhibited by pigs shortly after weaning. Some recent reports suggesting that feeding high concentrations of zinc to pigs after weaning can reduce scouring and improve postweaning growth performance have been of keen interest to producers. Research results are

mixed as to whether these therapeutic levels of zinc can actually improve growth. Several studies have reported growth performance benefits when feeding 2,500 to 3,000 mg of zinc per kg of diet during the first two weeks after weaning (Kavanagh, 1992; Poulsen, 1992). However, other researchers have found no benefits to feeding high levels of zinc (Fryer et al., 1992). Therefore, the benefits of supplementing weanling pig diets with high concentrations of zinc remains questionable.

However, because producers are showing an interest in adopting the practice of feeding high concentrations of zinc and because this practice can lead to high levels of mineral deposition on land, there is an increased need to find zinc sources that are highly available to swine in an effort to reduce waste.

Currently, several organic mineral sources are being marketed as being more available to swine than traditional inorganic sources. However, the data from trials investigating the availability of zinc from these organic sources are inconclusive. Some researchers have shown an increased availability of zinc from amino acid-mineral complexes compared with traditional inorganic sources (Hahn and Baker, 1993; Wedekind et al., 1992). Others have shown no increased availability of zinc when organic mineral complexes have been fed to livestock, compared to

traditional sources (Aoyagi and Baker, 1993; Hill et al., 1986).

The research reported in this dissertation was conducted to investigate the effectiveness of supplementing pigs with pharmacological levels of zinc to alleviate the poor growth performance immediately after weaning. In conjunction with this objective, this research examined the availability of several inorganic and organic sources of zinc to weanling pigs.

Chapter II

Literature Review

Introduction

Since Todd et al. (1934) first demonstrated that zinc was an essential component of animal diets, zinc has been shown to be an integral part of many metabolic processes. To date, over 200 proteins containing zinc have been identified (Hambidge et al., 1986). The first report recognizing the need for zinc in swine was the finding that zinc reversed and prevented parakeratosis (Tucker and Salmon, 1955). Today, swine producers recognize the role of zinc in maintaining optimal growth. Exactly how zinc affects growth is not well understood. The relationship between zinc and growth as well as the other functions of zinc are reviewed in this chapter. Also, the absorption and storage of zinc in the body and the availability of zinc from sources commonly fed to swine are discussed.

Functions

Zinc has three main biological roles: as an acid catalyst, a structural ion and a control ion (Williams, 1989). Thus, zinc acts in numerous metabolic processes throughout the body. The unique properties of zinc dictate its role in metabolism. Zinc has a relatively small size

and concentrated charge. Additionally, zinc does not have a variable valence, which prevents it from susceptibility to free radical formation. The small size and high reactivity of zinc allow zinc enzymes to have a high rate of reaction as well as low selectivity (Williams, 1989). Proteins involved in digestion and hormone action often contain zinc as these functions require low selectivity or quick action. Moreover, the properties of zinc govern the many roles zinc plays in biological processes.

Growth

Animals deficient in zinc experience reduced feed intake and growth rates. King (1990) reported that the growth rate of an animal will slow before tissue zinc stores are depleted. Chesters and Quarterman (1970) reported a 41% reduction in the feed intake of rats fed a zinc deficient diet compared with animals fed a zinc-adequate diet. Force feeding the zinc deficient rats a zinc deficient diet resulted in death.

The role of zinc in feed intake was further demonstrated by Hughes and Dewar (1971), who found that zinc-depleted chicks showed a "specific zinc appetite" by discriminating between a diet with a high zinc concentration over a diet with a low zinc concentration. These results illustrate that zinc can be a growth limiting nutrient. The specific mechanism by which zinc regulates growth has not

been fully elucidated. However, much is known about the roles zinc plays in some of the different processes which comprise overall growth. These include the role of zinc in protein synthesis and enzyme functions.

DNA and RNA function. Zinc is an important component of DNA and RNA polymerases (Chesters, 1989). Consequently, zinc plays a role in controlling protein synthesis. Additionally, Prasad and Oberleas (1974) reported decreased activity of thymidine kinase activity in mice fed a zinc deficient diet for 3 d. Similarly, Williams and Chesters (1970) reported a reduction in the incorporation of thymidine into DNA in rats that were fed a zinc deficient diet for 5 d compared to pair fed control rats. During the same period, incorporation of labelled lysine into protein was not affected. The researchers concluded that a reduction in protein synthesis is not the primary response to zinc deficiency, but a reduction in DNA synthesis appeared to be the primary response. Grey and Dreosti (1972) found similar results using regenerating liver tissue of rats fed a zinc deficient diet for 3 d. In their study, thymidine incorporation into DNA was reduced while leucine incorporation into protein was not. Again, it appears that a reduction in DNA synthesis is the primary response to initial zinc deficiency.

Cell division. Researchers have also found that zinc is essential for the replication of cells (Guigliano and Millward, 1984). Beyond its role in DNA formation, zinc can influence the rate of the cell cycle (Chen, 1986). Swinkels (1992) reported that serum mitogenic activity was reduced by 34% in pigs fed a zinc deficient diet compared to control animals. The rate of cell differentiation of epithelial cells (Chen, 1986), bone cells (Westmoreland, 1971) and fibroblasts (Hsu et al., 1974) have all been slowed by zinc deficiency. Reducing a zinc supply to tumor cells has also been shown to be an effective method of inhibiting these rapidly growing cells in animals (De Wys, 1972).

Hormones. Several of the hormones that regulate growth are thought to be associated with zinc metabolism. Kirchgessner and Roth (1985) were able to attribute a reduced concentration of growth hormone in the serum of rats to zinc deficiency. Serum concentrations of growth hormone were lower in zinc deficient rats than in the pair fed animals. Also, triiodothyronine concentrations have been reported to be lower in zinc deficient rats compared with pair fed controls (Morley et al., 1980). Oner et al. (1984) were able to demonstrate that somatomedin levels were also reduced in rats as a result of zinc deficiency. The researchers were able to distinguish between the reduction in somatomedin levels and the effects of reduced growth

hormone levels by injecting growth hormone into the control animals. Injection of growth hormone in the zinc deficient controls did not increase somatomedin concentrations.

Digestive functions

The zinc content of the intestinal tract is relatively high compared to other tissues of the body, which may be related to the many roles zinc plays in the intestine. Zinc status of the intestine appears to be directly related to intestinal cell development. Rats fed a zinc deficient diet show a reduction in the production of new intestinal cells when compared to pair fed controls (Southon et al., 1985). Additionally, rats fed a zinc deficient diet have shorter and narrower villi in the jejunum and ileum than pair fed control rats (Southon et al., 1986). Zinc supplementation of the deficient rats resulted in increased intestinal cell proliferation and increased villus height, width and density in the small intestine.

Along with the proliferation of intestinal cells, zinc has been shown to have protective roles in the intestine. Zinc compounds have been reported to reduce ulcer formation by stabilizing membrane integrity and thereby reducing mast cell degranulation (Pfeiffer et al., 1987).

Zinc has also been shown to be a component of many digestive enzymes such as carboxypeptidases, tripeptidase, aminopeptidase and others (Georgievskii, 1982).

Immunity

Immune function is also influenced by zinc status (Carlomagno et al. 1985; Chandra, 1985). Mice fed a diet deficient in zinc have rapid atrophy of the thymus, reduced splenocyte numbers, and depressed responses to T-cell dependent and T-cell independent antigens (Fraker et al., 1986). Atrophy of the thymus during zinc deficiency is usually of a much greater magnitude than the magnitude of the general reduction in body weight (Chandra and Au, 1980). Immune function alterations originating from atrophy of the thymus have been speculated to be caused by a lowered DNA content which may be responsible for a reduction in the development and differentiation of thymocytes (Hambidge et al., 1986). Zinc repletion of zinc deficient mice resulted in improved immune function within 5 d (Fraker et al., 1986).

Conflicting results have been reported for the influence of zinc on humoral immunity. Beach et al. (1980) reported that zinc deficiency in mice produced abnormal serum immunoglobulin profiles. Additionally, Fraker et al. (1986) reported that zinc deficiency during periods of rapid growth and development can cause severe immune disorders as seen in abnormal serum antibody profiles in 4 wk old mice fed a zinc deficient diet. In contrast, Cunningham-Rundles

et al. (1981) reported no effects of inadequate zinc intake on lymphocytic proliferation and differentiation.

Zinc also plays several other protective roles that can be considered as augmentations of the immune system. During infection, serum zinc is lowered by metallothionein shuttling zinc to the liver for storage. This is thought to lower the availability of zinc to the invading microbes, and thus inhibits their replication (Klasing, 1984). Additionally, zinc compounds can increase the stabilization of lysosomes in the presence of destabilizing agents, thereby enhancing the protection of cells (Pfeiffer and Cho, 1980).

Stress

The metabolism of zinc during stress may be related to an immune function. Zinc concentrations in the plasma of animals decrease during periods of stress (Chvapil, 1976). It is hypothesized that this decline in plasma zinc concentration may be part of an animal's defense mechanism. Chvapil (1976) speculated that low serum zinc concentrations during stress may be a mechanism to activate phagocytic cells that are inhibited at normal zinc concentrations. Zinc supplementation of stressed animals, however, does not improve their growth status (Chesters and Will, 1981; Kornegay et al., 1993). Chesters and Will (1981) supplemented endotoxin stressed pigs with zinc and found no

improvement in the growth status of the animals. Kornegay et al. (1993) reported that weanling pigs stressed with restricted floor space showed no improvement in performance with increased dietary zinc supplementation.

Reproduction

Zinc is distributed throughout the primary and secondary sex organs of the male. The major function of zinc in male reproductive organs appears to be in testosterone production and the production and maintenance of viable sperm. Abbasi et al. (1980) found that during zinc deficiency circulating testosterone levels decrease. Additionally, Lei et al. (1976) found that in zinc deficient rats, testosterone levels in Leydig cells are depleted. Furthermore, Saito et al. (1967) reported that zinc is necessary in the semen of rats and dogs to maintain sperm viability and motility.

In the female, the zinc content of the reproductive organs (uterus, ovaries and cervix) is low compared with other body tissues (Hambidge et al., 1986). However, zinc has been shown to influence every stage of the female reproductive cycle. Zinc deficient rats have abnormal estrous cycles and remain anestrus (Swenerton and Hurley, 1968). Swenerton and Hurley (1968) found that supplementation of the rats with zinc resulted in a normal cycle. During gestation, zinc deficient rats were found to

mobilize only 1% of their bone zinc (Hurley and Swenerton, 1971). This amount of zinc mobilization from bone was insufficient to prevent teratogenic effects on the fetuses. Furthermore, the liver zinc concentrations of the zinc deficient pregnant rats were not different from control pregnant rats. Hurley and Swenerton's study clearly demonstrates the inability of pregnant rats to mobilize tissue zinc stores for normal fetal development. Moreover, the largest effect of zinc deficiency on gestation is severe abnormal development of the fetuses ranging from neuromuscular malformation to malformations of the eye (Hurley, 1981). In pigs, litter size is not affected by dietary zinc concentration, but the number of animals born alive and their survivability are affected by dietary zinc concentration (Hill et al., 1983; Kalinowski and Chavez, 1984). However, Pond and Jones (1964) reported no improvement in litter size or subsequent growth of pigs from gilts fed several different levels of zinc. Hedges et al. (1976) reported no improvement in sow performance through five parities when different levels of zinc were fed.

Structure

Like many other functions of zinc, the exact role of zinc in bone is not known. Zinc is thought to play a role in the formation of bone matrix because zinc represents a large percentage of the composition of matrix (Hambidge et

al., 1986). During bone formation, zinc appears to influence the activity of the epiphyseal growth plate; a deficiency can reduce the activity of the osteocytes (Hambidge et al., 1986). Oner et al. (1984) reported a strong correlation between tibial epiphyseal widths and bone zinc concentrations. They found that rats with wider epiphyseal plates had higher femur zinc concentrations and higher serum zinc concentrations.

The normal calcification of bone in rats also appears to be dependant on a sufficient supply of zinc (Becker and Hoekstra, 1966). In fact, they found that by inducing increased bone calcification with vitamin D supplementation, more zinc was deposited in bone.

The sensitivity of bone zinc concentrations to dietary Zn levels led Flanagan (1984) to report that bone zinc content of rats is a good indicator of overall body zinc status. However, another study (Berg and Kollmer, 1987) found that bone zinc content is a poor indicator of zinc status due to maintenance of bone zinc content during periods of altered zinc intake.

Vision

Zinc is also an integral component of vision as a part of retinol dehydrogenase in the rods of the eye. Retinol dehydrogenase functions to convert retinol to retinal which is necessary to form rhodopsin for night vision (Morrison et

al., 1978). An indicator of zinc deficiency in humans is night blindness.

Body stores

The highest concentration of zinc in the body is in the eye (Hambidge et al., 1986). However, the eye only represents a small proportion of total body zinc. Bones and muscles contain approximately 80% of body zinc, with muscles containing the largest proportion of zinc in the body of humans (Jackson, 1989). Highly oxidative red skeletal muscles contain higher concentrations of zinc than do glycolytic white skeletal muscles (Cassens et al. 1967). Hambidge et al. (1986) reported that blood, hair, bone, testis and liver are sensitive to feeding different levels of zinc, whereas brain, lung, muscle and heart were insensitive to dietary levels. Berg and Kollmer (1987), however, found that bone stores of zinc do not drop appreciably during zinc deficiency in rats. In chickens, liver, kidneys, muscle and bone have all been reported to be sensitive indicators of zinc status (Henry et al., 1987). Rabbits fed a zinc deficient diet had decreased zinc concentrations in bone, fur, liver, and testis (Bentley and Grubb, 1991). They found, however, that skeletal muscle and thymus zinc concentrations were not affected by dietary zinc concentrations in rabbits.

Most of the zinc in blood is bound in erythrocytes to carbonic anhydrase. Only 10 to 20% of zinc in blood is in the plasma. Approximately two thirds of the zinc in plasma is bound to albumin (Prasad and Oberleas, 1970). When comparing the sensitivity of zinc concentrations in plasma versus serum, Hambidge et al. (1986) found no differences in sensitivity. In fact, zinc concentrations in the plasma were not different from the zinc concentrations of the serum.

The consumption of food has been reported to be responsible for diurnal variation in plasma zinc concentrations in women (Goodall et al., 1987). Liptrap et. al. (1970) reported no differences in serum zinc concentrations between growing barrows and gilts fed diets with several concentrations of zinc for 4 wk. Ullrey et al. (1967) reported serum zinc concentrations at birth of .60 mg/L. Serum zinc concentrations then rose during the first two weeks after birth to approximately 1.0 mg/L. At 3 to 5 wk of age, the serum zinc concentrations dropped to .55 mg/L and then recovered to a range of .85 to 1.0 mg/L. After 5 wk of age, the serum zinc concentrations fluctuated within this range through 5 mo. of age. The reason for the drop in serum zinc concentrations at 3 wk of age is not known. Interestingly, Liptrap et al. (1970) reported a close relationship between serum zinc concentrations and growth.

Because of this strong relationship, Liptrap et al. (1970) concluded that serum zinc is a good indicator of overall zinc status.

The zinc concentration of milk has been reported to decrease as lactation progresses in rats (Reiss et al., 1990). Interestingly, the decrease in zinc concentrations in milk corresponds to a decrease in zinc turnover rates in the whole body; zinc turnover rates decrease as an animal gets older (Reis et al., 1990). It appears that animals become more efficient in regulating mineral homeostasis as they age. This may be one reason mineral requirements decline with age.

Requirements

As mentioned above, dietary requirements for zinc decline as pigs age (NRC, 1988). Using casein diets, Shanklin et al. (1968) found that the zinc requirement for baby pigs was between 14 and 20 mg Zn/kg of diet. Levels at or below 14 mg Zn/kg of diet resulted in reduced performance and low serum zinc concentrations and low serum alkaline phosphatase activity compared with pigs receiving higher levels. Using autoclaved-spray-dried egg white as the protein source, Hankins et al. (1985) reported the zinc requirement of baby pigs to be above 19 mg Zn/kg of diet. Feeding 19 mg Zn/kg of diet in a phytate free diet resulted in parakeratotic lesions in the esophagus.

The NRC (1988) recommends 50 mg Zn/kg of diet for growing and finishing swine. Using growth performance as an indicator, Liptrap et al. (1970) reported a higher zinc requirement for growing gilts and boars compared to barrows.

The recommended zinc requirement for gestating and lactating females and boars is 50 mg/kg (NRC, 1988). Hedges et al., (1976) reported no differences in sow performance through five parities by feeding either 33 or 83 mg/kg. Thus, the suggested requirement appears to provide a margin of safety.

Zinc homeostasis

According to Wastney et al. (1987), there are five sites in the body that regulate zinc homeostasis. These include intestinal absorption, urinary excretion, release by muscle, secretion into the gut, and exchange with red blood cells. By controlling the uptake and secretion of zinc, the intestinal cell is thought to be the primary regulator of zinc homeostasis. During periods of high dietary zinc intake, zinc in the intestinal cell is moved into the circulatory system and is also secreted into the intestinal lumen. During low dietary zinc intake, the amount of zinc secreted into the lumen is decreased (Lonnerdal, 1989). Zinc homeostasis is also controlled by the excretion of zinc by the kidney and storage of zinc in various tissues such as muscle, liver and bone.

Deficiency

The primary characteristics of zinc deficiency are reduced feed intake and growth. Animals consuming a diet deficient in zinc will reduce feed intake and growth will be slowed before body stores are depleted (Wada and King, 1986). The secondary characteristics of zinc deficiency include parakeratosis and hair loss. Some of the other symptoms of zinc deficiency include abnormal bone growth, arthritis of the joints, impaired reproduction, night blindness and diarrhea (Hambidge et al., 1986).

Many of the deficiency signs of zinc are thought to be related to the function of zinc in protein synthesis. Additionally, the reduction in feed intake of zinc deficient animals is thought to contribute to the symptoms of zinc deficiency. In other words, it is difficult to determine if many of the symptoms of zinc deficiency are a direct effect of reduced zinc intake or an effect of reduced feed intake.

Zinc deficiency also creates a destabilization of membranes as reported by Paterson and Bettger (1985). Rats fed a zinc deficient diet had a significant reduction in the stability of erythrocyte membranes.

Tissues in pigs that were found to be sensitive to zinc deficiency included liver, kidney, rib bone and pancreas (Prasad et al., 1971). Oxidative red muscles have been found to be more sensitive to zinc deficiency than

glycolytic white muscles of rats (O'Leary et al., 1979). Additionally, the RNA content of the liver, kidney, rib bone and pancreas of zinc deficient pigs were lower than pair fed control pigs (Prasad et al., 1971).

The gastrointestinal tract is also altered during zinc deficiency in rats (Southon et al., 1984). There is a significant decrease in absorptive area in the small intestine of zinc-deficient rats which is characterized by reduced villi height and reduced villi density (Southon et al., 1984).

Development of all male sex organs and spermatogenesiss can be negatively affected by zinc deficiency (Hambidge et al., 1986). Additionally, gestation, lactation, and all other phases of the female reproductive cycle can be adversely affected by zinc deprivation (Hurley and Swenerton, 1966).

Toxicity

Generally, pigs have a very high tolerance for zinc. Pigs can effectively eliminate zinc from the body through the feces and urine. Tolerance has been reported for weanling pigs fed levels as high as 5,000 ppm (Hahn and Baker, 1993). Brink et al. (1959) reported toxic effects when weanling pigs were fed 2,000 to 8,000 ppm of zinc as zinc carbonate. The toxicity signs they reported were depressed growth and severe arthritis at 2,000 ppm and

above. Gilts (30 kg of BW) fed 5,000 ppm of zinc as ZnO for 20 wk showed no signs of toxicity (Hill and Miller, 1983).

In rats, feeding high levels of zinc resulted in anemia characterized by a reduction in hemoglobin levels (Cox and Harris, 1960). Magee and Matrone (1960) further reported that feeding rats high levels of zinc interfered with iron and copper metabolism. They were unable to show an interference with copper or iron absorption, only an increase in the excretion of these minerals. Consequently, a zinc toxicity in pigs may manifest itself by altering other mineral metabolism without showing any specific zinc related toxicity problems. Hill et al. (1983) did report lowered copper and iron concentrations in the liver when 5,000 ppm of zinc were fed for 20 weeks.

Absorption

The exact mechanism for zinc absorption is unknown. However, much is known about the characteristics of zinc absorption.

General

Absorption of minerals in newborn animals is known to be almost three-fold higher than in adult animals (Sullivan et al., 1984). This increased absorption is probably necessary for the rapid growth of newborns. Similarly, zinc absorption is increased during late gestation and during lactation in rats (Davies and Williams, 1977). This appears

to be a specific zinc effect because in the same study lysine absorption was not increased.

Site of absorption

Results from many studies conflict regarding the primary site of zinc absorption in the intestinal tract (Antonson et al., 1979; Davies, 1980). Davies found that fasted rats have the greatest absorption of zinc from the duodenum (60%) with the ileum and jejunum accounting for 30 and 10% respectively. Using non-fasted rats, Antonson et al. (1979) found that the ileum was responsible for 60% of zinc absorption with the duodenum and jejunum accounting equally for the remainder of the zinc absorption. Using complete intact intestinal tracts of pigs, Swinkels (1992) reported that the jejunum and ileum were the primary sites of zinc absorption. To the contrary, segments of the large intestine have also been identified as having the capacity to absorb significant amounts of zinc (Wapnir et al., 1985; Partridge, 1978). But despite these discrepancies, which could be attributed to experimental designs, most agree that the upper small intestine has the greatest capability for zinc absorption (Lonnerdal, 1989). Furthermore, because all the segments of the G.I. tract exhibit the potential to absorb zinc, the amount of zinc absorbed by each segment may be dependant on factors such as dietary composition and zinc source.

Mechanism of absorption

Two mechanisms of zinc transport appear to exist. The first is postulated to be a saturable carrier mediated mechanism which is stimulated to increase zinc absorption during zinc deficiency situations (Hoadley et al., 1987). Kennedy and Lonnerdal (1988) reported a saturable uptake of zinc from brush border membrane vesicles of rats. Similarly, Blakeborough (1987) described a saturable zinc uptake process in the small intestine of the pig that was consistent with a carrier mediated process. He also reported that the absorption of zinc at the pig brush border membrane was pH dependant. A range of pH between 7 and 8 was found to be necessary for zinc uptake.

The second mechanism is simple diffusion (Steel and Cousins, 1985). This mechanism has been reported by several researchers in several species (Steel and Cousins, 1985; Blakeborough, 1987). The extent to which simple diffusion influences absorption has not been fully elucidated.

Interactive influences of absorption

Despite the fact that the exact mechanism for zinc absorption remains unknown, much is known about factors that influence the absorption of zinc. These factors include the zinc status of the animal, the zinc source, the age of the animal, and dietary factors. The list of negative influences on the absorption of zinc include: high dietary

calcium, fiber, low dietary protein, phytate and high iron. Factors identified as positive influences on zinc absorption include high dietary protein, lysine, cysteine, glycine, low dietary iron, and lactose. Conflicting results have been reported regarding the influence of histidine, EDTA, citrate, picolinate and copper; each of these has been shown to have a negative, positive and/or no effect on zinc absorption (Cousins, 1985).

At relatively low doses of dietary zinc, the saturable mechanism of zinc absorption appears to control zinc uptake. But at higher doses, this mechanism appears to be overridden (Coppen and Davis, 1987; Cousins, 1985)). Cousins (1985) speculated that at high dietary zinc concentrations, the intestinal membrane may allow zinc to pass more freely into the intestinal cell by diffusion. Therefore, the body regulates zinc absorption to a certain level of dietary intake, above which the body must excrete sufficient quantities of zinc to avoid toxicity problems.

Serum zinc carriers such as albumin apparently do not influence the absorption of zinc from the intestine. Jackson et al. (1981) reported that saturation of serum albumin with zinc by intraperitoneal injection of high zinc concentrations does not reduce the absorption of zinc.

Calcium. High levels of dietary calcium have been shown to interfere with the uptake of zinc in the intestine

(Heth and Hoekstra, 1965). Newland et al. (1958) showed that a high calcium diet fed to weanling pigs can alter zinc metabolism by reducing zinc stores. Berr et al. (1961) reported decreased zinc absorption in pigs fed excess calcium. Similarly, Heth et al. (1966) reported that calcium decreased zinc absorption in rats fed a semipurified diet. The high dietary calcium also reduced the liver and kidney stores of zinc in the rats.

Iron. Iron can also interfere with zinc absorption (Momcilovic and Kello, 1979). In rats, high dietary iron concentrations have been found to reduce zinc absorption, but adequate dietary iron concentrations were not found to affect zinc absorption (Fairweather-Tait and Southon, 1988). Apparently, the wider the iron to zinc ratio in the diet the more magnified the reduction in zinc absorption becomes. In humans, dietary iron to zinc ratios of 1:1 and 2.5:1 were insufficient to reduce zinc absorption, but 25:1 greatly reduced zinc absorption (Sandstrom et al., 1985).

Phytate. Phytate is another inhibitor of zinc uptake in the intestine. Zinc bound to phytate molecules forms a complex that renders the zinc unavailable for absorption. Oberleas et al. (1962) reported a reduction in zinc availability in growing pigs fed .5 or .7% phytic acid added to a soybean meal or casein basal diet respectively. Supplementation of these pigs with 100 ppm of zinc returned

performance to a level similar to controls. Furthermore, using tube-fed zinc deficient rats, Flanagan (1984) found that the addition of sodium phytate to the diet increased the amount of zinc excreted in the feces. He speculated that phytate reduced the reabsorption of endogenously secreted zinc.

Protein. Dietary protein content has also been shown to affect zinc status of animals. In rats and swine, a low dietary protein content has led to decreased serum or plasma zinc concentrations (Filteau and Woodward, 1982; Pond and Yen, 1984; Pond et al., 1985). Supplementation of these animals with zinc did not increase serum or plasma zinc concentrations. These studies suggest that a sufficient amount of protein is necessary for zinc absorption.

Other dietary components have also been found to negatively affect zinc uptake. Citrate and picolinate have also been reported to reduce zinc absorption by rat brush-border-membranes (Menard and Cousins, 1983). Similarly, Turnbull et al. (1990) found that citrate and picolinate reduced zinc uptake by pig intestinal brush border membrane.

Availability

In simple terms, availability refers to the amount of a nutrient that can be absorbed and utilized by an animal. As previously discussed, the availability of a mineral source will depend on several factors, including the mineral status

of the animal, dietary components and the health of the animal. Increased concern over environmental contamination by animal waste has resulted in attempts to identify mineral sources that are highly available. As a result, many new sources of zinc for animals have been developed which are proposed to be highly availability to animals.

Sources. Inorganic salts have been used to supplement livestock diets for decades. Recently, interest in sources that might be more available to animals has led to the development of organic sources of zinc. These organic sources, known as complexes or chelates, are compounds of metals such as zinc, copper, iron and manganese combined with amino acids, proteins or carbohydrates.

Inorganic. Traditional sources of zinc for pigs are mainly inorganic salts. Sources of zinc commonly fed to pigs include ZnO, $ZnSO_4$, $ZnCl_2$ and $ZnCO_3$. The availability of these salts vary. Using evereted sacs of rat duodenum and ileum, Seal and Heaton (1983) found that $ZnSO_4$ was more available than either $ZnCl_2$ or $Zn(PO_4)_2$. Using growth performance as an indicator of availability, Edwards (1959) found that ZnO was more available to broilers than $ZnSO_4$. He also reported no difference in availability between $ZnCO_3$ and ZnO or $ZnSO_4$. Again using body weight gain as an indicator of zinc availability, Roberson and Schaible (1960) reported no differences in the availability of ZnO, $ZnCO_3$,

and $ZnSO_4$ when fed at several concentrations to broilers. Miller et al. (1981) found in weanling pigs that zinc from metallic zinc dust was approximately 30% more available than zinc from ZnO. Based on tibia zinc concentrations, Wedekind and Baker (1990) reported in chicks that the bioavailability of zinc from feed grade ZnO was only 44.1% of that of feed grade $ZnSO_4$ and 61% as available based on weight gain.

Chelates and complexes. A metal complex is defined as a central metal atom with ligands, at least one of which has a free electron pair. Proteins, amino acids and carbohydrates typically function as the ligands. A chelate is defined as a special form of complex in which two or more atoms of a ligand donate their electron pairs to the metal to form a ring structure.

Chelates are postulated to increase the availability of minerals by either protecting the mineral from binding to non-absorbable ingredients such as phytates, making the mineral available for absorption, or by aiding in the actual absorption of the mineral. An increase in the absorption of the mineral is speculated to be the result of co-transport of the zinc with the chelating agent or by presenting the zinc to the absorptive site in a more available form.

Recent interest in chelated minerals for animal nutrition has been stimulated by the discovery that low molecular weight ligands can influence the absorption of

zinc. Using intestinal perfusion studies in rats, Wapnir and Steil (1986) showed that amino acids added to a zinc solution significantly increased the absorption of zinc in the jejunum and ileum. Tryptophan, histidine, and proline all increased zinc absorption in rats to levels above those seen in the control rats which were perfused with a ligand-free zinc solution. These amino acids formed tightly bound complexes with zinc which led to the speculation that the zinc and ligands formed chelates before absorption. Thus, the prospect that chelated minerals may be more available than other sources has focused recent investigations on the availability of these new mineral sources. Fan et al. (1982) speculated that an in vitro increase in zinc uptake from a zinc-hisitidine complex was due to a reduction in zinc binding to large ligands that may inhibit zinc from freely being absorbed.

Contrary to the above findings, Dahmer et al. (1972) reported no increase in the availability of zinc when histidine was fed in a corn-soybean meal diet to weanling pigs. These researchers found that an increase in histidine up to 2% of the diet could not improve performance to levels similar to pigs fed various levels of zinc supplementation. Additionally, the increases in serum zinc, serum alkaline phosphatase activity, bone zinc and liver zinc seen with the addition of zinc to the diet could not be obtained with the

addition of histidine to the diet. Moreover, no apparent increased zinc availability was found with the addition of histidine.

Availability of complexes

Laboratory animals. Using an everted gut procedure in rats, Hill et al. (1987) found no differences in zinc absorption between Zn-methionine, Zn-lysine and ZnCl. Also using an everted gut technique in rats, Seal and Heaton (1983) reported an increase in zinc uptake in the presence of histidine and dipicolinic acid. The prescence of cysteine, tryptophan, and glutamic acid had no effect on zinc absorption (Seal and Heaton, 1983). Evans and Johnson, (1980a) reported that zinc from lactating rats fed zinc picolinate is transfered to nursing pups in greater amounts than zinc from lactating rats fed zinc acetate. Furthermore, Evans and Johnson (1980b) reported that supplemental dietary picolinic acid increased the absorption of zinc in rats compared with rats that were not supplemented with picolinic acid. On the contrary, Roth and Kirchgessner (1985) found no differences in absorption or utilization of zinc from zinc picolinic, zinc citrate or zinc sulfate in rats fed diets with different protein sources. Furthermore, Hempe and Cousins (1989) reported no difference in the availability of zinc from Zn-methionine compared with $ZnSO_4$ when fed to rats.

Swine. Hahn and Baker (1993) reported higher plasma zinc concentrations for weanling pigs fed a Zn-lysine complex compared with $ZnSO_4$ or ZnO. In another trial, they reported higher plasma zinc concentrations for weanling pigs fed either $ZnSO_4$ or Zn-methionine compared with pigs fed ZnO. Interestingly, the pigs fed the ZnO in this trial had higher ADG than pigs fed either the $ZnSO_4$ or Zn-methionine. Hill et al. (1986) reported no differences in performance or serum zinc concentrations in pigs fed either $ZnSO_4$, Zn-methionine or Zn-methionine and picolinic acid. The zinc sources in the study by Hill et al. (1986) were fed at 9 or 12 mg Zn/kg of a corn-soybean meal basal diet. No differences between sources for performance or serum zinc concentration were reported during a 112 d growing/finishing period. Additionally, no differences in metatarsal zinc concentrations were reported for the different sources after the finishing phase. Similarly, Kornegay and Thomas (1975) found no improvement in the performance of growing pigs fed zinc-methionine compared with pigs fed $ZnSO_4$ with supplemental methionine. Using zinc depleted growing pigs, Wedekind and Lewis (1993) found $ZnSO_4$ to be more available than ZnO, Zn-lysine or Zn-methionine when bone zinc concentrations were used as an indicator. Similarly, Swinkels (1992) did not report an increased availability of

a Zn-amino acid complex when compared with $ZnSO_4$ for zinc depleted weanling pigs.

Poultry. Using tibia zinc concentration as an indicator of availability, Aoyagi and Baker (1993) reported no difference in the availability of zinc from Zn-lysine when compared with $ZnSO_4$ when fed to broilers. In another broiler study using tibia zinc concentration, Wedekind et al. (1992) reported that Zn-methionine was 117% and 177% available compared with $ZnSO_4$ when fed in a purified or semipurified diet, respectively. Zinc oxide was found to be only 61% as available as $ZnSO_4$. Pimentel et al. (1991) reported that the availability of zinc in Zn-methionine was not different from that of ZnO when fed to chickens.

Ruminants. Zinc bioavailability from Zn-methionine and ZnO were not found to be different in heifers using growth rate, plasma zinc and plasma alkaline phosphatase activity as indicators of zinc status (Spears, 1989). Also, the apparent absorption of zinc in lambs was found to be similar for Zn-methionine and ZnO (Spears, 1989). Rojas et al. (1994) found no differences in tissue zinc storage in cattle fed Zn-methionine, $ZnSO_4$ or ZnO. Greene et al. (1988) reported an increase in quality grades for steers fed zinc-methionine compared to steers fed ZnO. These workers also reported an increase in external fat and kidney, pelvic and heart fat in steers fed the zinc-methionine compared to ZnO

fed steers. No differences in performance were reported between the steers fed the different zinc sources.

Feeding high levels of zinc

Researchers in Ireland (Kavanagh, 1992) reported that feeding 3,000 mg Zn/kg of diet as ZnO to weanling pigs reduced postweaning diarrhea and resulted in a corresponding increase in performance. Similarly, Poulsen (1992) reported a reduction in scouring for pigs two weeks after weaning when 2,500 mg Zn/kg of diet as ZnO was fed. Also, Poulsen (1992) reported an increase in performance for pigs receiving 2,500 or 4,000 mg Zn/kg of diet as ZnO. Hahn and Baker (1993) found that weanling pigs fed 3,000 or 5,000 mg Zn/kg of diet as ZnO had higher ADG than pigs fed 125 mgZn /kg of diet. They also found that pigs fed 3,000 mg Zn/kg of diet as $ZnSO_4$ had higher ADG than pigs fed the basal diet containing 125 mg Zn/kg of diet.

A proposed mode of action for increased performance by feeding high levels of zinc is through the reduction of scouring. Zinc deficiency has been reported to be a cause of diarrhea (Golden and Golden, 1985). In in vitro cultures, high levels of $ZnSO_4$ have been found to reduce the growth of microbes known to cause diarrhea (Duhamel et al., 1993). Fryer et al. (1992) found no performance benefit to supplementing weanling pig diets with 3,000 mg Zn/kg of diet as ZnO. In their study, feed intake, ADG and scouring were

not improved by zinc additions to the diets. However, Fryer et al. (1992) reported no scouring for pigs on any of the treatments. Consequently, if the mode of action is through reduced scouring, Fryer et al. (1992) would not have been able to see any performance benefits without a scouring incidence. The study by Fryer et al. (1992) further supports the hypothesis that the mode of action for performance improvement appears to be mediated through a reduction in scouring.

Summary

Zinc has been shown to play a role in many metabolic functions. The unique relationship between zinc status, growth and feed intake is of primary importance to livestock producers. Although the exact mechanisms of how zinc affects growth has not been elucidated, a reduction in DNA and protein synthesis appear to be influential in the growth depression seen in zinc deficient animals. The site and mechanism of zinc absorption also have not yet been fully identified. Most researchers agree that the small intestine plays the major role in a facilitated zinc absorption mechanism. The availability of zinc from organic complexes versus inorganic sources remains a topic of debate. The practice of feeding high levels of zinc to weanling pigs to improve performance has been shown to be equally effective and ineffective in the literature. Moreover, there remains

much to be learned about the mechanisms by which zinc affects the functions of the body.

Literature cited

Abbasi, A. A., A. S. Prasad, P. Rabbani, and E. DuMouchelle. 1980. Experimental zinc deficiency in man: effect on testicular function. J. Lab. Clin. Med. 96:544-550.

Antonson, D.L., A. J. Barak, J. A. Vanderhool. 1979. Determination of the site of zinc absorption in rat small intestine. J. Nutr. 109:142-147.

Aoyagi, S. and D. H. Baker. 1993. Nutritional evaluation of copper-lysine and zinc-lysine complexes for chicks. Poul. Sci. 72:165-171.

Beach, R. S., M. E. Gershwin, R. K. Makishima, and L. S. Hurley. 1980. Impaired immunologic ontogeny in postnatal zinc deprivation. J. Nutr. 110:805-815.

Becker, W. M. and W. G. Hoekstra. 1966. Effect of vitamin D on 65-zinc absorption, distribution and turnover in rats. J. Nutr. 90:301-309.

Bentley, P. J. and B. R. Grubb. 1991. Effects of a zinc-deficient diet on tissue zinc concentrations in rabbits. J. Anim. Sci. 69:4876-4882.

Berg, D. and W. E. Kollmer. 1987. The influence of zinc deficiency on the storage of zinc in bone. In: L.S. Hurley, C. L. Keen, B. Lonnerdal, and R.B. Rucker (Eds.) Trace Elements in Man and Animals 6. Plenum Press. New York. pp.455-457.

Berr, R. K., M. C. Bell, R. B. Grainger, and R. G. Buescher. 1961. Influence of dietary calcium and zinc on calcium-45, phosphorus-32 and zinc-65 metabolism in swine. J. Anim. Sci. 20:433-437.

Blakeborough, P. 1987. The intestinal transport of zinc studied using brush-border-membrane vesicles from the piglet. Br. J. Nutr. 57:45-55.

Brink, M. F., D. E. Becker, S. W. Terrill and A. H. Jensen. 1959. Zinc toxicity in the weanling pig. J. Anim. Sci. 18:836-842.

Carlomagno, M. A., C. L. Mintzer, C. L. Tetzlaff and D. N. McMurray. 1985. Differential effect of protein and zinc deficiencies on lymphokine acivity in BCG-vacinated guinea pigs. Nutr. Res. 5:959-968.

Cassens, R. G., W. G. Hoekstra, E. C. Faltin, and E. J. Briskey. 1967. Zinc content and subcellular distribution in red vs. shite porcine skeletal muscle. Am. J. Physiol. 212: 688-692.

Chandra, R. K. 1985. Trace element regulation of immunity and infection. J. Amer. Coll. Nutr. 4:5.

Chandra, R. K. and B. Au. 1980. Single nutrient deficiency and cell-mediated immune responses: 1. zinc. Am. J. Clin. Nutr. 33:736-738.

Chen, S. Y. 1986. Autoradiographic study of cell proliferation in acanthotic buccal epithelium of Zn-deficient rabbits. Arch. Oral Biol. 31:535-539.

Chesters, J.K. 1989. Biochemistry of zinc in cell division and tissue growth. In: C. F. Mills (Ed.) Zinc in Human Biology. Springer-Verlag. London, Great Britain. pp. 109-116.

Chesters, J.K. and J. Quarterman. 1970. Effects of zinc deficiency on food intake and feeding patterns of rats. Br. J. Nutr. 24:1061.

Chesters, J. K. and M. Will. 1981. Measurement of zinc flux through plsama in normal and endotoxin-stressed pigs and the effects of Zn supplementation. Br. J. Nutr. 46:119-130.

Chvapil, M. 1976. Effect of zinc on cells and biomembranes. Med. Clin. North Am. 60:799-812.

Coppen, D. E. and N. T. Davies. 1987. Studies on the effects of dietary zinc dose on ^{65}Zn absorption in vivo and on the effects of Zn status on ^{65}Zn absorption and body loss in young rats. Br. J. Nutr. 57:35-44.

Cousins, R. J. 1985. Absorption, transport and hepatic metabolism of copper and zinc: special reference to metallothionein and ceruloplasmin. Physiol. Rev. 65:238-309.

Cox, D. H. and D. L. Harris. 1960. Effect of excess dietary zinc on iron and copper in the rat. J. Nutr. 70:514-520

Cunningham-Rundles, C, S. Cunningham-Rundles, T. Iwata, G. Incefy, J. A. Garogalo, C. Menendez-Botet, V. Lewis, J.J. Twomey and R. A. Good. 1981. Zinc deficiency, depressed thymic hormones, and T lymphocyte dysfunction in patients with hypogammaglobulinemia. Clin. Immunol. Immunopathol. 21:387-396.

Dahmer, E. J., B. W. Coleman, R. H. Grumner, and W. G. Hoekstra. 1972. Alleviation of parakeratosis in zinc deficient swine by high levels of dietary histidine. J. Anim. Sci. 35:1181-1189.

Davies, N.T. 1980. Studies on the absorption of zinc by rat intestine. Br. J. Nutr. 43:189-203.

Davies, N. T. and R. B. Williams. 1977. The effect of pregnancy and lactation on the absorption of zinc and lysine by the rat duodenum in situ. Br. J. Nutr. 38:417-423.

De Wys, W. 1972. Inhibition of a spectrum of animal tumors by dietary Zn deficiency. J. Natl. Cancer Inst. 48:375-381.

Duhamel, G. E., D. P. Dupont, and M. P. Carlson. 1993. Potential application of feedgrade zinc to control of swine dysentery. Univ. of Nebraska Res. Rep. pp. 10-12.

Edwards, H. M. 1959. The availability to chicks of zinc in various compounds and ores. J. Nutr. 69:306-308.

Evans, G. W. and E. C. Johnson. 1980a. Zinc concentration of liver and kidneys from rat pups nursing dams fed supplemental zinc dipicolinate or zinc acetate. J. Nutr. 2121-2124.

Evans, G. W. and E. C. Johnson. 1980b. Growth stimulating effect of picolinic acid added to rat diets. Proc. Soc. Exp. Biol. Med. 165:457-461.

Fairweather-Tait, S. J. and S. Southon. 1988. Studies of the iron:zinc interatctions in adult rats and the effect of iron fortafication of two commercial infant weaning products on iron and zinc status of weanling rats. J. Nutr. 119:599-606.

Fan, S., S. A. Burton and R. V. Petersen. 1982. Bioavailability of zinc: effect of animo acid

chelation. In: Chelated Mineral Nutrition in Plants, Animals and Man, D. Ashmead (Ed.) pp. 137-151.

Filteau, S. M. and B. Woodward. 1982. The effect of severe protein deficiency on serum zinc concentrations of mice fed a requirement level of a very high level of dietary zinc. J. Nutr. 112:1974-1977.

Flanagan, P. R. 1984. A model to produce pure zinc deficiency in rats and its use to demonstrate that dietary phytate increases the excretion of enogenous zinc. J. Nutr. 114:493-502.

Fraker, P. J. , M. E. Gershwin, R. A. Good, and A. Prasad. 1986. Interrelationships between zinc and immune function. Fed. Proc. 45:1474.

Fryer, A., E. R. Miller, P. K. Ku, and D. E. Ullrey. 1992. Effect of elevated dietary zinc on growth performance of weanling swine. Mich. State. Res. Rep. pp. 128-132.

Georgievskii, V.I. 1982. The physiological role of microelements. In: V.I. Georgievskii, B.N. Annenkov, and V.I. Samokhin (Eds.) Mineral Nutrition of Animals. Butterworths. Boston.

Golden, B. E. 1989. Zinc in cell division and tissue growth: physiological aspects. In: C. F. Mills (Ed.) Zinc in Human Biology. pp. 119-128. Springer-Verlag. London, Great Britain.

Golden, B. E. and M. H. N. Golden. 1985. Zinc, sodium, and potassium losses in the diarrheas of malnutrition and zinc deficiency. In: Trace Elements in Man and Animals, C. F. Mill, I. Bremner, and J. K Chesters (Eds.). Commonwealth Agricultural Bureaux. Farnham Royal, United Kingdom.

Goodall, M. J., K. M. Hambridge, C. Stall, J Pritts, and E. E. Nelson. 1987. Daily variations in plasma zinc in normal adult women. In: L.S. Hurley, C. L. Keen, B. Lonnerdal, and R.B. Rucker (Eds.) Trace Elements in Man and Animals 6. pp.491-492. Plenum Press. New York.

Greene, L. W., D. K. Lunt, F. M. Byers, N. K. Chirase, C. E. Richmond, R. E. Knutson, and G. T. Schelling. 1988. Performance and carcass quality of steers supplemented with zinc oxide or zinc methionine. J. Anim. Sci. 66:1818-1823.

Grey, P. C. and I. E. Dreosti. 1972. Deoxyribonucleic acid and protein metabolism in zinc-deficient rats. J. Comp. Path. 82:223-228.

Guigliano, R. and D. J. Millward. 1984. Growth and zinc homeostasis in the severely Zn-deficient rat. Br. J. Nutr. 52:545-560.

Hahn, J. D. and D. H. Baker. 1993. Pharmacologic zinc levels for weanling pigs: growth and plasma zinc responses. J. Anim. Sci. 71(Suppl. 1):66.

Hambidge, K. M., C. E. Casey, and N. F. Krebs. 1986. Zinc. In: W. Mertz (Ed.) Trace Elements in Human and Animal Nutrition. Academic Press, Inc., Orlando, FL pp. 1-137.

Hankins, C. C., T. L. Veum, and P. G. Reeves. 1985. Effects of autoclaved-spray-dried egg white as the sole source of dietary protein on zinc requirement and performance of the baby pig. Nutr. Rept. Intr. 31:1057-1070.

Hedges, J. D., E. T. Kornegay, and H. R. Thomas. 1976. Comparison of dietary zinc levels for reproducing sows and the effect of dietary zinc and calcium on the subsequent performance of their progeny. J. Anim. Sci. 43:453-463.

Hempe, J. M. and R. J. Cousins. 1989. Effect of EDTA and zinc-methionine complex on zinc absorption by rat intestine. J. Nutr. 119:1179-1187.

Henry,P. R., C. B. Ammerman, and R. D. Miles. 1987. Effect of dietary zinc on tissue mineral concentration as a measure of zinc bioavalability in chicks. Nutr. Rep. Int. 35:15-23.

Heth, D. A., W. M. Becker, and W. G. Hoekstra. 1966. Effect of calcium, phosphorus and zinc on zinc-65 absorption and turnover in rats fed semipurified diets. J. Nutr. 88:331-337.

Heth, D. A. and W. G. Hoekstra. 1965. Zinc-65 absorption and turnover in rats. J. Nutr. 85:367-374.

Hill, D. A., E. R. Peo, Jr., and A. J. Lewis. 1987. Influence of picolinic acid on the uptake of ^{65}Zinc-amino acid complexes by the everted rat gut. J. Anim. Sci. 65:173-178.

Hill, D. A., E. R. Peo, Jr., A. J. Lewis and J.D. Crenshaw. 1986. Zinc-amino acid complexes for swine. J. Anim. Sci. 63:121-130.

Hill, G. M. and E. R. Miller. 1983. Effect of dietary zinc levels on the growth and development of the gilt. J. Anim. Sci. 57:106-113.

Hill, G. M., E. R. Miller and H. D. Stowe. 1983. Effect of dietary zinc levels on health and productivity of gilts and sows through two parities. J. Anim. Sci. 57:114-122.

Hoadley, J. E., A. S. Leinart, and R. J. Cousins. 1987. Kinetic analysis of zinc uptake and serosal transfer by vacularly perfused rat intestine. Am. J. Physiol. 252:G825-G831.

Hsu, J. M., K. M. Kim, and W. L. Anthony. 1974. Biochemical and electron microscopic studies of rat skin during Zn deficiency. Adv. Exp. Med. Biol. 48:347-388.

Hughes, B.O. and W.A. Dewar. 1971. A specific appetite for zinc in zinc-depleted domestic fowls. Poult. Sci. 12: 255-258.

Hurley, L. S. 1981. Teratogenic aspects of manganese, zinc, and copper nutrition. Physiol. Rev. 61:249-295.

Hurley, L. S., and H. Swenerton. 1966. Congenital malformations resulting from zinc deficiency in rats. Proc. Soc. Exp. Biol. Med. 123-692-697.

Hurley, L. S., and H. Swenerton. 1971. Lack of mobilization of bone and liver zinc under teragenic conditions of zinc deficiency in rats. J. Nutr. 101:597-604.

Jackson, M.J. 1989. Physiology of zinc: general aspects. In: C. F. Mills (Ed.) Zinc in Human Biology. pp. 1-11. Springer-Verlag. London, Great Britain.

Jackson, M. J., D. A. Jones, and R. H. T. Edwards. 1981. Zinc absorption in the rat. Br. J. Nutr. 46:15-27.

Kalinowski, J. and E. R. Chavez. 1984. Effect of low dietary zinc during late gestation and early lactation on the sow and neonatal piglets. Can. J. Anim. Sci. 22:749.

Kavanagh, N. T. 1992. The effect of feed supplemented with zinc oxide on the performance of recently weaned pigs. Proceedings: International Pig Veterinary Meetings 1992. p. 616.

Kennedy, M. L., and B. Lonnerdal. 1988. Zinc uptake by brush border membrane vesicles from pre-weanling rat small intestine. In: Trace Elements in Man and Animals 6. L. S. Hurley, C. L. Keen, B. Lonnerdal, and R. B. Rucker (Eds.) Plenum Press. New York. pp. 599-600.

King, J. C. 1990. Assement of zinc status. J. Nutr. 120:1474.

Kirchgessner, M., and H. P. Roth. 1985. Depenency of serum growth hormone (GH) levels on zinc supply in rats. In: C.F. Mills, I. Bremner, and J. K. Chesters (Eds.) Trace Elements in Man and Animals 5. pp.62-65. Commonwealth Agricultural Bureaux, London.

Klasing, K. C. 1984. Effect of inflammatory agents and interleukin I on iron and zinc metabolism. Am. J. Physiol. 247:R901.

Kornegay, E. T., J. B. Meldrum, and W. R. Chickering. 1993. Influence of floor space allowance and dietary selenium and zinc on growth performance, clinical pathology measurements and liver enzymes, and adrenal weights of weanling pigs. J. Anim. Sci. 71:3185-3198.

Kornegay, E. T. and H. R. Thomas. 1975. Zinc-proteinate supplement studied. Hog Farm Management:50-51.

Lei, K. Y., A. Abbasi, and A. S. Prasad. 1976. Function of pituitary-gonadal axis in zinc-deficient rats. Am. J. Physiol. 230:1730-1732.

Liptrap, D. O., E. R. Miller, D. E. Ullrey, D. L. Whitenack, B. L. Schoepke, and R. W. Luecke. 1970. Sex influence on the zinc requirement of developing swine. J. Anim. Sci. 30:736-741.

Lonnerdal, B. 1989. Intestinal absorption of zinc. In: Zinc in Human Biology, C. F. Mills (Ed.) Springer-Verlag. London. pp.33-52.

Magee, A. C. and G. Matrone. 1960. Studies on growth, copper metabolism, and iron metabolism of rats fed high levels of zinc. 72:233-242.

Manard, M. P., and R. J. Cousins. 1983. Effect of citrate, glutathione and picolinate on zinc transport by brush border membrane vesicles form rat intestine. J. Nutr. 113:1653-1656.

Miller, E. R., P. K. Ku, J. P. Hitchcock, and W. T. Magee. 1981. Availability of zinc from metallic zinc dust for youn swine. J. Anim. Sci. 52:312-315.

Momcilovic, B. and D. Kello. 1979. Fortification of milk with zinc and iron. Nutr. Rep. Int. 20:429-436.

Morley, J. E., J. Gordon, and J. M. Hershman. 1980. Zinc deficiency, chronic starvation, and hypothalamic-pituitary-thyroid function. Am. J. Clin. Nutr. 33:1767-1770.

Morrison, S. A., R. M. Russell, E. A. Carney, and E. V. Oaks. 1978. Zinc deficiency: a cause of abnormal dark adoptation in cirrhotics. Am. J. Clin. Nutr. 31:276-281.

Newland, H. W., D. E. Ullrey, J. A. Hoefer, and R. W. Luecke. 1958. The relationship of dietary calcium to zinc metabolism in pigs. J. Anim. Sci. 17:886-892.

N.R.C. 1988. Nutrient Requirements of Swine. National Academy Press. Washington D.C.

Oberleas, D., M. E. Muhrer, and B. L. O'Dell. 1962. Effects of phytic acid on zinc availability and parakeratosis in swine. J. Anim. Sci. 21:57-61.

O'Leary, M. J., C. J. McClain, and P. V. J. Hegarty. 1979. Effect of zinc deficiency on the weight, cellularity and zinc concentration of different skeletal muscles in the post-weanling rat. Br. J. Nutr. 42:487-495.

Oner, G., B. Bhaumick, and R. M. Bala. 1984. Effect of zinc deficiency on serum somatomedin levels and skeletal growth in young rats. Endocrinology 114:1860-1863.

Partridge, I. G. 1978. Studies on digestion and absorption in the intestine of growing pigs. 4. Effects of dietary cellulose and sodium levels on mineral absorption. Br. J. Nutr. 39:539-545.

Paterson, P. G. and W. J. Bettger. 1985. Effect of dietary zinc intake on the stability of the rat erythrocyte membrane. In: C.F. Mills, I. Bremner, and J. K. Chesters (Eds.) Trace Elements in Man and Animals 5. pp.79-83. Commonwealth Agricultural Bureaux, London.

Pfeiffer, C. J., O. Bulbena, J. V. Esplugues, G. Escolar, C. Navarro, and J. Esplugues. 1987. Anti-ulcer and membrane stabilizing actions of zinc acexamate. Arch. Inter. Pharm. Ther. 285:148-156.

Pfeiffer, C. J. and C. H. Cho. 1980. Modulating effect by zinc on hepatic lysosomal fragility induced by surface-active agents. Chem. Path. Pharm. 27:587-597.

Pimentel, J.L., M. E. Cook, and J. L. Greger. 1991. Bioavailability of zinc methionine for chicks. Poult. Sci. 70:1637-1639.

Pond, W. G. and J. R. Jones. 1964. Effect of level of zinc in high calcium diets on pigs from weaning through on reproductive cycle and on subsequent growth of their offspring. J. Anim. Sci. 23:1057-1060.

Pond, W. G., and J. Yen. 1984. Effect of protein deficiency on growth and plasma zinc concentration in genetically lean and obese swine. J. Anim. Sci. 59:710-724.

Pond, W. G., J. T. Yen, and L. H. Yen. 1985. Effect of dietary protein and zinc levels on weight gain and plasma traits in weanling pigs. Nutr. Rep. Int. 31:253-264.

Poulsen, H. D. 1992. Zinc oxide for weaned pigs. Eleventh annual Prince feed ingredient conference. Dublin. Appendix 1.

Prasad, A. S., D. O. Oberleas, E. R. Miller, and R. W. Luecke. 1971. Biochemical effects of zinc difficiency: changes in activites of zinc-dependent enzymes and ribonucleic acid and deoxyribonucleic acid content of tissues. J. Lab. Clin. Med. 77:145-152.

Prasad, A. S. and D. Oberleas. 1970. Binding of zinc to amino acids and serum proteins in vitro. J. Lab. Clin. Med. 76:416-425.

Prasad, A. S. and D. Oberleas. 1974. Thymidine kinase activity and incorporation of thymidine into DNA in zinc-deficient tissue. J. Lab. Clin. Med. 83:634.

Reis, B. L., C. L. Keen, B. Lonnerdal, and L. S. Hurley. 1990. Longitudinal changes in the mineral composition of mouse milk and the relationship to zinc metabolism of the suckling neonate. J. Nutr. 121:687-699.

Roberson, R. J. and P. J. Schaible. 1960. The availability to the chick of zinc as the sulfate, oxide or carbonate. Poult. Sci. 39:835-837.

Rojas, L. X., L. R. McDowell, R. J. Cousins, F. G. Martin, N. S. Wilkinson, A. B. Johnson, and C. A. Njeru. 1994. Relative bioavailability of zinc methionine and two inorganic zinc sources fed to cattle. J. Anim. Sci. 72(Suppl. 1)95.

Roth, H. P. and M. Kirchgessner. 1985. Utilization of zinc from picolinic or citric acid complexes in relation to dietary protein source in rats. J. Nutr. 115:1641-1649.

Saito, S. L. Zeitz, I. M. Bush, R. Lee, and W. F. Whitmore. 1967. Zinc content of spermatozoa from various levels of canine and rat reproductive tracts. Am. J. Physiol. 213:749-752.

Sandstrom, B., L. Davidsson, A. Cederblad, and B. Lonnerdal. 1985. Oral iron, dietary ligands and zinc absorption. J. Nutr. 115:411-414.

Seal, C. J. and F. W. Heaton. 1983. Chemical factors affecting the intestinal absorption of zinc in vitro and in vivo. Br. J. Nutr. 50:317-324.

Shanklin, S. H., E. R. Miller, D. E. Ullrey, J. A. Hoefer, and R. W. Luecke. 1968. Zinc requirement of baby pigs on casein diets. J. Nutr. 96:101-108.

Southon, S., J. M. Gee, C. E. Bayliss, G. M. Wyatt, N. Horn, and I. T. Johnson. 1986. Intestinal microflora, morphology and enzyme activity in zinc-deficient and Zn-supplemented rats. Br. J. Nutr. 55:603-611.

Southon, S., J. M. Gee, and I. T. Johnson. 1984. Hexose transport and mucosal morphology in the small intestine of the zinc-deficient rat. Br. J. Nutr. 52:371-380.

Southon, S., G. Livesey, J. M. Gee, and I. T. Johnson. 1985. Intestinal cellular proliferation and protein synthesis in zinc-deficient rats. Br. J. Nutr. 53:594-603.

Spears, J. W. 1989. Zinc methionine for ruminants: relative bioavailability of zinc in lambs and effects of growth and performance of growing heifers. J. Anim. Sci. 67:835-843.

Steel, L. and R. J. Cousins. 1985. Kinetics of zinc absorption by luminally and vascularly perfused rat intestine. Am. J. Physiol. 248:G46-G53.

Sullivan, M. F., B. M. Miller, and J. C. Goebel. 1984. Gastrointestinal absorption of metals (^{51}Cr, ^{65}Zn, ^{95m}Tc, ^{109}Cd, ^{113}Sn, ^{147}Pm, and ^{238}Pu) by rats and swine. Envir. Res. 35:439-453.

Swenerton, H. and L. S. Hurley. 1968. Severe zinc deficiencies in male and female rats. J. Nutr. 95:8-18.

Swinkels, J. W. G. M. 1992. Availability of zinc from an amino acid chelate in Zn depleted pigs. Ph.D. Dissertation. Virginia Polytechnic Institute and State University, Blacksburg.

Todd, W. R., C. A. Elvehjem, and E.B. Hart. 1934. Zinc in the nurition of the rat. Am. J. Physiol. 107:146-156.

Tucker, H. F., and W. D. Salmon. 1955. Parakeratosis or zinc deficiency disease in the pig. Proc. Soc. Exp. Biol. Med. 88:613.

Turnbull, A. J., P. Blakeborough, and R. P. H. Thompson. 1990. The effects of dietary ligands on zinc uptake at the porcine intestinal brush-border membrane. Br. J. Nutr. 64:733-741.

Ullrey, D. E., E. R. Miller, B. E. Brent, B. L. Bradley, and J. A. Hoefer. 1967. Swine hematology from birth to maturity IV. Serum calcium, magnesium, sodium, potassium, copper, zinc and inorganic phosphorus. J. Anim. Sci. 26:1024-1029.

Wada, L. and J.C. King. 1986. Effect of low zinc intake on basal metabolic rate thyroid hormone and protein utilization in adult men. J. Nutr. 116:1045.

Wapnir, R. A., J. A. Garcia-Aranda, D. E. K. Mevorach and F. Lifshitz. 1985. Differential absorption of zinc and

low-molecular weight ligands in the rat gut in protein-energy malnutrition. J. Nutr. 115:900.

Wapnir, R. A. and L. Steil. 1986. Zinc intestinal absorption in rats: specificity of amino acids as ligands. J. Nutr. 116:2171-2179.

Wastney, M. E., R. L. Aamodt, and R. I. Henkin. 1987. Identification of five sites of regulation of human Zn metabolism. In: L.S. Hurley, C. L. Keen, B. Lonnerdal, and R.B. Rucker (Eds.) Trace Elements in Man and Animals 6. pp.423-424. Plenum Press. New York.

Wedekind, K. J. and D. H. Baker. 1990. Zinc bioavailability in feed-grade sources of zinc. J. Anim. Sci. 68:684-689.

Wedekind, K. J., A. E. Hortin, and D. H. Baker. 1992. Methodology for assessing zinc bioavailability: efficacy estimates for zinc-methionine, zinc sulfate, and zinc oxide. J. Anim. Sci. 70:178-187.

Wedekind, K. J. and A. J. Lewis. 1993. Assessing zinc bioavailability with pigs fed corn-soybean meal diets. University of Nebraska Research Report. pp. 24-25

Westmoreland, N. 1971. Connective tissue alteration in Zn deficiency. Fed. Proc. Fed. Soc. Exp. Bio. Med. 30:1001-1010.

Williams, R. J. P. 1989. An introduction to the biochemistry of zinc. In: C. F. Mills (Ed.) Zinc in Human Biology. pp. 15-31. Springer-Verlag, London, Great Britain.

Williams, R. B. and J. K. Chesters. 1970. The effects of early zinc deficiency on DNA and protein synthesis in the rat. Br. J. Nutr. 24:1053-1069.

Chapter III

Objectives

The objectives of this research were two-fold. The first objective was to investigate the growth performance effects of supplementing weanling pigs with pharmacological levels of zinc. The second objective was to evaluate the availability of zinc from several sources for weanling pigs. The specific objectives of these primary objectives are outlined below.

I. Zinc supplementation of weanling pigs

- A. Attempt to produce a growth stimulating response by supplementing weanling pigs with pharmacological levels of zinc
  - 1. Zinc supplementation by injection
    - a. Examine types of injection
    - b. Examine sources of zinc to inject
    - c. Determine amount to inject
    - d. Determine when to inject
    - e. Examine factors that may be responsible for the performance boost
      - 1). Serum blood concentrations
      - 2). Scouring
      - 3). Stress
  - 2. Zinc supplementation by feeding
    - a. Determine source of zinc to feed
    - b. Determine level of zinc to feed
    - c. Determine optimum age to feed the zinc

II. Compare the availability of several zinc sources to weanling pigs

A. Determine tissue storage patterns and absorption of Zn, Cu, and Fe in weanling pigs fed ZnO, $ZnSO_4$, Zn-lysine or Zn-methionine at concentrations of 3,000, 2,000, and 1,000 ppm.

B. Compare the absorption and storage of Zn in weanling pigs fed feed grade $ZnSO_4$ or reagent grade $ZnSO_4$

C. Compare the availability of zinc in Zn-lysine and $ZnSO_4$ when fed in a low lysine diet and a lysine adequate diet

D. Compare the availability of zinc in ZnO, $ZnSO_4$, Zn-lysine and Zn-methionine when fed to zinc deficient weanling pigs

Chapter IV

Effectiveness of zinc acetate injection in alleviating postweaning lag in pigs

ABSTRACT

Four trials were conducted using crossbred pigs (n=308) to determine the effects of Zn acetate injection near weaning on serum Zn concentrations and performance. In Trial 1, 78 pigs (BW of 5.1 kg and age of 18 d) were injected i.m. 3 d before weaning with either 3 mg of Zn from Zn acetate (in saline) per kg of BW or saline. Serum Zn concentrations were higher ($P < .05$) for Zn injected pigs at 1 wk postweaning than controls. In Trial 2, 96 pigs (BW of 7.2 kg and age of 26 d) were used to compare time of i.p. injection of Zn (weaning or 12 h postweaning) and amount of Zn injected (0, 3 or 4 mg of Zn/ kg of BW). Serum Zn concentrations were higher ($P <.01$) for Zn injected pigs 1 d after injections and remained higher than controls until after wk 2. In Trial 3, pigs (n=30, BW of 6.9 kg and 24 d of age) that were injected with 3 mg of Zn/kg BW i.p. 3 d postweaning had lower ($P < .05$) ADG and ADFI and lower ($P < .01$) gain/feed from d 4 to d 7 postweaning than controls. Zinc injected pigs had higher ($P < .05$) serum Zn concentrations at d 7. In Trial 4, 104 pigs (BW of 6.4 kg) were used in a 2 x 2 x 2 factorial arrangement of

treatments: 1) regrouped 3 d before weaning or not regrouped, 2) Zn injections (3 mg i.p.) or placebo, and 3) time of injection, 2 d before or 1 d after weaning. All pigs were weaned at 23 d of age. Pigs injected with Zn at either time had higher serum Zn concentrations at wk 1 ($P < .01$) than controls, but performance was similar. Regrouping decreased ($P < .05$) preweaning ADG, but had no effect on serum Zn concentrations. Moreover, the injection of Zn three days before to three days after weaning did not prevent a decrease in performance at weaning although serum Zn concentrations were increased.

Key Words: Pigs, Weaning, Serum, Zinc, Growth, Performance.

Introduction

The poor performance exhibited by pigs immediately after weaning which is characterized by low feed intake and suboptimal daily gains (Ogunbameru et al., 1992) has been speculated to be the result of the stress of weaning (Funderburke and Seerley, 1990). Removal of pigs from the sow and regrouping them are thought to be major contributors to the stress of weaning. Stress has also been shown to cause a decline in serum Zn concentrations (Chesters and Will, 1981). Furthermore, due to the role of Zn in growth, animals with suboptimal body Zn were shown to grow more slowly (Chesters and Quarterman, 1970; King, 1990). Consequently, the possibility exists that there may be a

direct relationship between Zn status, stress and performance.

Because serum zinc is known to decline at weaning (Ullrey et al., 1968), and because zinc plays a key role in regulating feed intake and growth (Grummt et al., 1986), this research was directed at determining the influence of supplemental zinc on postweaning performance. Trials were conducted with Zn treatments designed to maintain serum zinc concentrations at weaning to investigate a possible beneficial response in performance. Additionally, an attempt was made to further characterize the relationship between zinc supplementation and the stress that may result from regrouping at weaning.

Materials and Methods

Crossbred pigs (n=308) were utilized in four trials to examine the effects of Zn injections near weaning on performance. In Trial 1, 78 pigs (BW of 5.1 kg) were blocked by weight, gender and litter, and randomly assigned to one of two treatments. The treatments were Zn injection (i.m.) with 3 mg of Zn/kg BW as Zn acetate (28.1 mg of Zn acetate/ml saline) or an equivalent volume of saline. The injections were given in the semitendinosus muscle at an average of 18 d of age. Pigs were weaned 3 d after the injections and were housed four pigs per pen in plastic coated welded wire pens (1.2 m x 1.2 m) in environmentally

regulated nurseries. The initial nursery temperature was 31°C, and was reduced 2°C per week. Pigs were weighed prior to injection, at weaning, and at d 2, 7, 14, and 21 postweaning. Blood was collected via jugular venipuncture prior to injection, at weaning, d 7, 14, and 21. Feed was removed 30 min prior to bleeding to minimize the effect of any recent feed consumption on serum Zn concentrations. Blood was analyzed for serum Zn concentration and alkaline phosphatase activity. Pen feed consumption was determined on each of the weigh days. All pigs were fed the same diet (Table 1) and were allowed ad libitum access to feed and water.

In Trial 2, 96 crossbred pigs were weaned at an average age of 26 d and an average weight of 7.2 kg, blocked by weight, gender, and litter, and then randomly assigned to a 2 x 3 factorial arrangement of treatments. At weaning or 12 h after weaning, pigs were injected i.p. with 0, 3 or 4 mg of Zn/kg of BW as Zn acetate. The concentration of Zn was the same as that used in Trial 1. Control pigs were injected with saline (equal volume to pigs receiving the 3 mg injection). Pigs were housed two/pen (.6 m x 1.2 m) and allowed ad libitum access to feed (Table 1) and water. Environmental conditions were as in Trial 1. Pigs were weighed and blood samples were taken via jugular

Table 1. Diet composition[a]

Item	Percent
Corn	57.47
Soybean meal (44% CP)	24.95
Dried whey	15.00
Dicalcium phosphate	1.15
Limestone	.80
Vitamin premix[b]	.25
Lysine[c]	.18
Antibacterial (Mecadox 10)	.10
Mineral premix[d]	.05
Selenium premix[e]	.05

[a]Diet calculated to contain 18% protein, 1.15% lysine, .8% Ca and .65% P.
[b]Supplied per kilogram of diet: 4,400 IU of vitamin A, 440 IU of vitamin D, 11 IU of vitamin E, 489.4 mg of choline, 4.4 mg of riboflavin, 22 mg of d-pantothenic acid, 22 mg of niacin, .44 mg of biotin, .022 mg of B_{12}, and 1.1 mg of vitamin K as menadione dimethylprimidinol bisulfite.
[c]L-lysine (78.8%), monohydrochloride, Archer Daniels Midlands Co. Decatur, IL.
[d]Supplied per kilogram of diet: 105 mg of Zn, 123 mg of Fe, 42 mg of Mn, 12 mg of Cu, and 2 mg of I.
[e]Supplied .3 mg of Se per kilogram of diet.

venipuncture at weaning, and at d 1, 4, and 7 postweaning. Feed was removed 30 min prior to bleeding. Scouring was evaluated daily on a pen basis using a scale of 1 to 5 with 2 being a normal feces and 5 a liquid defecation (Sweet et al., 1990).

In Trial 3, 30 crossbred pigs with an average wt of 6.9 kg and an average age of 24 d were assigned at weaning (three pigs/pen) by litter, gender and weight to one of two treatments: either a 3 mg/kg BW i.p. injection of Zn as zinc acetate (same concentration as in Trial 1) or a saline placebo. Each treatment was given 4 d postweaning. Environmental conditions, diet, and feeding were as in the previous trials. Blood samples were taken using a jugular venipuncture at weaning, and at d 4, 7, 14, 21, and 28 postweaning.

In Trial 4, 104 crossbred pigs with an average weight of 6.4 kg and an average age of 20 d were assigned by sire, gender and litter to a 2 x 2 x 2 factorial arrangement of treatments. Treatments were: 1) regrouping or no regrouping at 20 d of age, 2) Zn (3 mg/kg BW) or saline i.p. injection, and 3) time of injection at either d 21 or d 24 of age. The number of pigs on each sow was not changed by regrouping. All pigs were weaned 3 d after treatments (average age of 23 d) and placed by treatment in pens with either two pigs per pen (.6 m x 1.2 m) or five pigs per pen

(1.2 m x 1.2 m). When possible, pigs from the same sow were not penned together at weaning in order to maximize the potential for stress. Environmental conditions were the same as in Trial 1. Blood was collected by jugular venipuncture at 20 d of age, weaning, and 1, 2, and 3 wk postweaning. Feed was removed 30 min prior to bleeding. Pigs were also weighed and pen feed consumption was determined at each of the above times. Pigs were allowed ad libitum access to feed (Table 1) and water for the duration of the trial.

Blood samples. All blood samples were refrigerated at 3°C for 24 h and then centrifuged for 10 min at 625 x g. Serum was collected and serum Zn concentrations were then determined using flame atomic absorption spectrophotometry on a Perkin Elmer 5100 (Norwalk, CT). Alkaline phosphatase activity was determined (Sigma Procedure 5100, Norwalk, CT) using an Autoanalyzer (Centrifichem 500, Union Carbide, New York, NY) and a computer controlled vertical photometer (Titertek Multiskan MCC/340, Flow Laboratories, McLean, VA).

Statistical analysis. Each trial was analyzed using the General Linear Models procedure of SAS (1988) with pen as the experimental unit for the performance and scouring data and pig as the experimental unit for the serum data. Models included treatment and replicate effects. Each trial was also analyzed using the multiple analysis of variance

procedure of SAS to determine the partial correlations between performance and serum zinc concentrations. In Trial 3, d 4 weight was used as a covariate because the pigs assigned to each treatment gained differently prior to injection. In Trial 4, all interactions between regrouping, Zn treatment and injection time were also analyzed using the all possible comparison procedure of SAS (1988).

In Trial 2, three pigs died. The first pig, which had received a saline injection, died on d 3 and was diagnosed with mulberry heart disease. The second pig, which had received 3 mg of zinc at weaning, died on d 3 and was diagnosed with gastroenteritis and meningioencephalitis, possibly due to infection at the time of injection. The third pig, which had received 3 mg of zinc 12 h after weaning, died on d 4 and was diagnosed with polyserositis and septicemia. Each of the diagnosis were performed within 24 h after death at the necropsy lab at the Virginia-Maryland Regional College of Veterinary Medicine. All data for these pigs were discarded. One pig also died in Trial 4 due to tracheal puncture during bleeding at weaning. The data for this pig were also discarded.

Results and discussion

In Trial 1, serum Zn concentrations were not influenced at weaning by a preweaning i.m. injection of Zn, but they were higher ($P < .05$) in the zinc treated pigs 1 wk after

Table 2. Serum Zn concentrations and alkaline phosphatase activity (ALP) of weanling pigs injected with zinc acetate 3 d prior to weaning. Trial 1.

Item	Treatment[a]		SEM
	Control	Zinc[b]	
Serum Zn, mg /L			
Preweaning[c]	.79	.83	.02
Weaning	.62	.62	.01
Week 1[d]	.43	.47	.01
Week 2[e]	.53	.57	.02
Week 3	.64	.65	.03
Serum ALP[f], U/L			
Preweaning[c]	263	248	8
Weaning[d]	231	255	6
Week 1	155	160	12
Week 2	172	177	8
Week 3	154	153	4

[a] Thirty-six pigs per treatment mean.
[b] Zinc acetate injected i.m. (3 mg/kg BW) 3 d prior to weaning.
[c,d,e] Zinc injection given prior to bleeding.
[f] Treatment main effect ($P < .05$ and .10 respectively).
Due to preweaning differences, preweaning ALP was used as a covariate.

weaning and tended to be higher ($P < .10$) after wk 2 compared with saline treated pigs (Table 2). Serum alkaline phosphatase activity was higher ($P < .02$) in zinc treated pigs compared with control pigs at weaning, but they were not higher thereafter. There were no differences between treatments for ADG, ADFI, and gain to feed ratio. Mean values (SEM) across treatments and days were: .23 kg/d (.01) for ADG; .42 kg/d (.01) for ADFI; .55 kg/kg (.01) for gain to feed. Furthermore, there were no significant correlations between serum Zn concentration and performance ($r = .22$). The lack of a positive growth response to Zn injection may be attributed to the decline in the serum zinc concentrations after weaning. All pigs in this trial experienced a drop in serum Zn concentrations similar to that seen by Ullrey et al. (1968) and Swinkels (1992). It was the goal of this trial to prevent the decline in serum Zn concentrations and to determine the resulting effect on performance. Consequently, because the serum Zn concentrations declined similarly in both groups, an observable response in performance may not have been possible. The timing, source and concentration of the Zn injected may have contributed to the failure of the injection to maintain serum Zn concentrations.

In Trial 2, pigs that received the Zn injection at weaning or 12 h postweaning had higher ($P < .01$) serum Zn

concentrations than pigs given the saline placebo, at d 1 and 3 d postweaning, and the concentrations remained higher ($P < .01$) through wk 2 (Table 3). Average daily gain, ADFI and gain/feed ratios were not affected by either level of Zn injection. Mean values (SEM) across treatments and days were: .28 kg/d (.02) for ADG; .92 kg/d (.05) for ADFI; and .30 kg/kg (.03) for gain:feed. Additionally, there were no significant correlations between serum Zn concentrations and performance ($r = .12$).

The serum Zn concentrations of the control pigs in this trial did not decline from preweaning concentrations until after d 3 postweaning. In a previous trial in our laboratory (unpublished data), it was shown that the i.p. injection of Zn at 3 mg/kg BW or 4 mg/kg BW would only elevate serum Zn concentrations for about 3 days (Figure 1). Therefore, the injection of Zn in this trial did not correspond to the time period in which the serum Zn concentration was declining. Consequently, the injection of Zn to maintain serum Zn concentrations may have been administered too early to be effective.

Scour indexes were not different across treatments, but a low incidence of scouring was observed for all treatments with an average scour index of 2 for each treatment. Therefore, a lack of scouring may have prevented the

Table 3. Serum Zn concentrations of pigs injected with Zn acetate at weaning or 12 h postweaning. Trial 2.

Time[a]	Treatment						
	0 h			12 h			
Zn,[b] mg/kg BW	0	3	4	0	3	4	SEM
Initial	.60	.61	.64	.65	.63	.62	.03
Day 1[c]	.67	.72	.74	.68	.78	.81	.03
Day 3[c]	.60	.67	.70	.59	.67	.68	.02
Week 1[c]	.53	.64	.68	.50	.66	.68	.02
Week 2[c]	.38	.44	.47	.34	.49	.46	.03
Week 3	.27	.35	.33	.34	.35	.32	.02
Week 4	.39	.37	.39	.35	.41	.37	.02

[a]Injection given postweaning. Serum Zn values expressed as mg/L. There were 14 pigs per treatment mean.
[b]Pigs were given 3 or 4 mg of Zn per kg BW i.p. in the form of zinc acetate, or saline in an equivalent volume to the 3 mg Zn injection.
[c]Treatment main effect ($P < .01$).

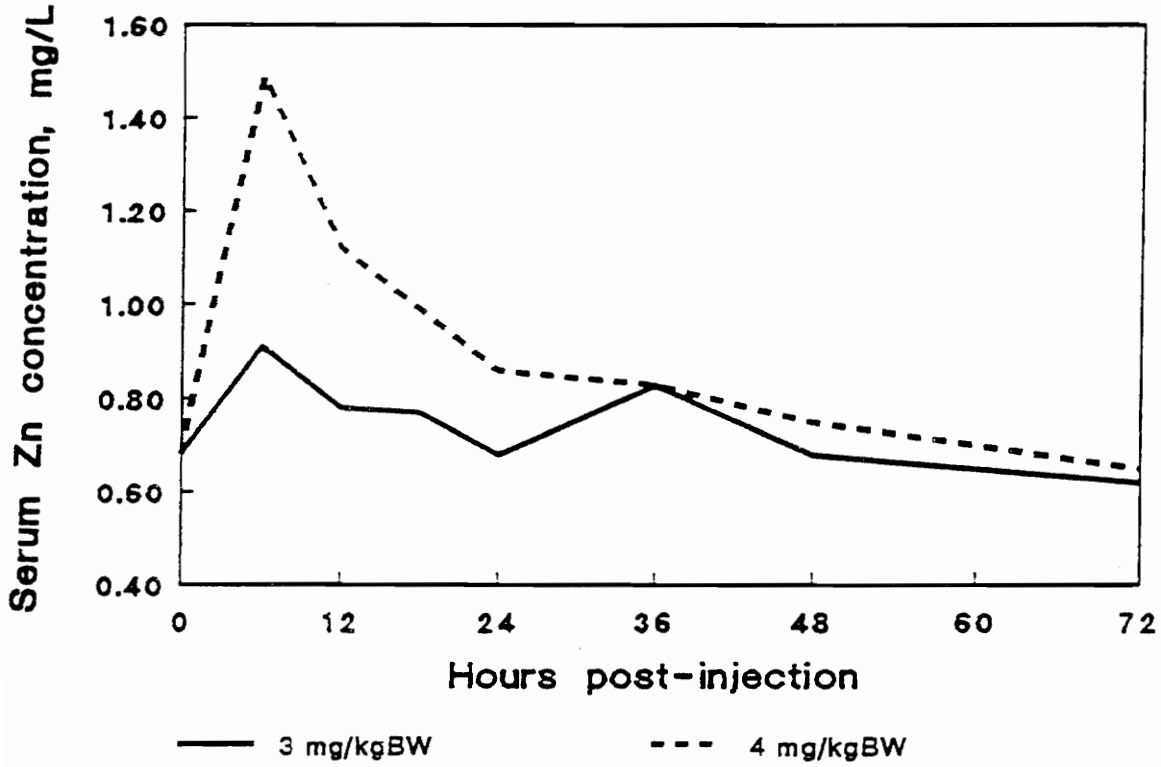


Figure 1. Serum zinc concentrations of crossbred pigs (65 d old ± 3 d, average BW of 22.5 kg) injected with either 3 or 4 mg/kg BW of Zn as Zn acetate (six pigs per treatment mean). Pooled SEM = .04.

detection of a lowered incidence of scouring with the injection of zinc as was hypothesized.

In Trial 3, pigs given the zinc injection had higher ($P < .05$) serum Zn concentrations compared with control pigs on d 7 (Table 4). Zinc injected pigs did not show the drop in serum Zn concentrations that was observed for the saline treated pigs on d 7. However, serum Zn concentrations did not drop as dramatically in this trial as in Trial 1, which may have been due to the size or age differences of the pigs between trials. The pigs in this trial were older and larger (24 d and 6.9 kg) than pigs in Trial 1 (18 d and 5.1 kg). Additionally, Ullrey et al.(1968) and Swinkels (1992) reported a decline in serum Zn concentration in pigs 21 d old. Consequently, the pigs in this trial may have been too old to display the magnitude of decline that was reported by Ullrey et al. (1968) and Swinkels (1992). Because the decline of serum Zn was not of the magnitude as seen previously, a positive response to the supplementation of Zn may not have been possible.

Additionally, pigs injected with zinc had decreased ($P < .05$) ADG and ADFI for d4- d 7 compared with pigs given the saline placebo (Table 5). This trend, although not statistically significant, continued throughout the remainder of the trial. Correspondingly, gain/feed was

Table 4. Serum Zn concentrations of weanling pigs injected with zinc acetate on d 3 postweaning. Trial 3.

	Treatment[a]		
Time	Saline	Zinc	SEM
Initial[b]	.72	.68	.03
Day 4	.72	.65	.03
Day 7[c]	.63	.77	.03
Week 2[c]	.65	.84	.04
Week 3[c]	.74	.85	.02
Week 4	.77	.89	.04

[a]Serum Zn concentrations are expressed as mg/L. There were 15 pigs per treatment mean.
[b]Zinc (3 mg per kg of BW) injected i.p. on d 4 in the form of zinc acetate.
[c]Treatment main effect ($P < .05$).

Table 5. Performance of weanling pigs injected with zinc acetate on d 3 postweaning. Trial 3.

Item	Treatment		SEM
	Saline	Zinc[a]	
Body wt[b], kg			
Initial	6.9	7.0	.02
Final	14.1	12.5	.55
ADG[b], kg			
Day 4	.04	.03	.01
Days 4 to 7[c]	.12	.01	.02
Week 1 to 4	.26	.20	.02
ADFI[b], kg			
Day 4	.12	.13	.01
Days 4 to 7[c]	.19	.12	.01
Week 1 to 4	.44	.35	.03
Gain/feed[b]			
Day 4	.22	.16	.07
Days 4 to 7[d]	.72	.02	.07
Week 1 to 4	.59	.56	.02

[a]Zinc (3 mg per kg of BW) injected i.p. on d 4 in the form of zinc acetate.
[b]There were 15 pigs per treatment mean, d 4 weight was used as a covariate.
[c,d]Treatment main effect ($P < .05$ and .01 respectively).

Table 6. Serum Zn concentrations of weanling pigs regrouped or not regrouped and injected with zinc acetate at one of two times. Trial 4.

	Treatment[a]								
	Not regrouped				Regrouped				
Zn, mg/kg BW	0		3		0		3		
Day	21	24	21	24	21	24	21	24	SEM
Initial[b]	.91	.97	.92	.91	.91	.80	.82	.83	.04
Weaning[c]	.70	.74	.82	.71	.67	.64	.67	.60	.02
Week 1[c]	.53	.60	.67	.67	.54	.55	.67	.67	.03
Week 2[d]	.63	.66	.64	.76	.58	.76	.68	.71	.04
Week 3	.61	.62	.65	.58	.59	.61	.60	.58	.04

[a]Twelve pigs per treatment means for not regrouped pigs and 14 pigs per treatment mean for regrouped pigs except 13 pigs injected with Zn on d 21 and regrouped (one died). [b]Serum Zn concentrations are expressed as mg/L.
[c,d]Treatment effect ($P < .01$ and .10 respectively).

lower ($P < .01$) for the d4-7 period for pigs injected with zinc compared with control pigs.

In Trial 4, serum Zn concentrations were higher ($P < .01$) at weaning for pigs that were not regrouped (Table 6). These results agree with those of Chesters and Will (1981) who reported a decline in plasma Zn concentrations in stressed pigs. Pigs that were regrouped also had lower ($P < .05$) preweaning ADG (Table 7). Although zinc treatment had no effect on postweaning performance, the partial correlation analysis showed that serum Zn concentrations at weaning and week 1 were positively correlated ($P < .05$) with ADG and ADFI through week 3 ($r = .62$). To the contrary, no significant correlations were observed in the three previous trials. However, in agreement with the findings of Trials 1, 2, and 3, no performance benefit was seen with the supplementation of zinc. It is apparent that regrouping 3 d prior to weaning did not afford pigs an advantage over non-regrouped pigs, as postweaning performance was not improved. Additionally, time of injection had no effect. There were also no interactions for performance among regrouping, zinc treatment or time of injection. Several possibilities exist for the lack of response to the treatments in this trial. First, there may be no benefit to these treatments. Second, the type and timing of the administration of Zn may not be the optimum dosing technique. Evidence of oral

Table 7. Performance of pigs regrouped or not regrouped before weaning and injected with zinc acetate at one of two times. Trial 4.

	Treatment[a]								
	Not regrouped				Regrouped				
Zn, mg/kg BW	0		3		0		3		
Day	21	24	21	24	21	24	21	24	SEM
Body weight, kg									
Initial	6.5	6.4	6.2	6.4	6.4	6.4	6.3	6.0	.3
Final	13.0	12.6	12.3	13.4	12.9	12.5	12.1	12.0	1.0
ADG, kg									
Preweaning[b]	.26	.28	.21	.26	.22	.18	.21	.18	.05
D 1 to d 7	.06	.02	.08	.08	.10	.07	.05	.06	.02
D -3 to d 21[c]	.27	.26	.26	.29	.27	.26	.24	.25	.03
ADFI, kg									
D 1 to d 7	.18	.17	.18	.20	.20	.18	.16	.17	.01
D -3 to d 21	.53	.47	.45	.50	.48	.46	.44	.44	.04
G/F									
D 1 to d 7	.34	.12	.42	.38	.49	.37	.32	.34	.08
D -3 to d 21	.53	.55	.58	.57	.58	.58	.57	.58	.04

[a]Twelve pigs per treatment means for not regrouped pigs and 14 pigs per treatment mean for regrouped pigs except 13 pigs injected with Zn on d 21 and regrouped (one died).
[b]Zinc x group effect ($P < .05$)
[c]Three days preweaning = day -3.

administration of supplemental Zn having a positive response on performance supports the second possibility (Kavanagh, 1992). Some swelling that was evident at the site of i.p. injection may have been indicative of peritoneal trauma which may have influenced the results. However, further investigations are needed to determine if this is the case.

The changes in serum Zn concentrations of the control pigs of each of the trials are illustrated in Figure 2. It appears that the earlier the pigs are weaned the larger the magnitude of the decline in serum Zn concentration one week after weaning (Trials 1 and 4 vs. Trials 2 and 3). Serum Zn concentrations appear to increase at approximately 28 d of age. Interestingly, the performance of the pigs in the trials followed the pattern of the serum Zn concentration; as serum Zn was declining, BW remained relatively unchanged and after d 28 the pigs showed increases in body weights (Figure 2). This is the relationship that was the premise of this research. The treatments imposed in these trials, however, did not significantly alter the serum Zn concentration with a concurrent increase in performance as hypothesized.

Implications

The results of these trials illustrate that the injection of zinc acetate to provide pigs with additional Zn near weaning did not have a positive effect on performance.

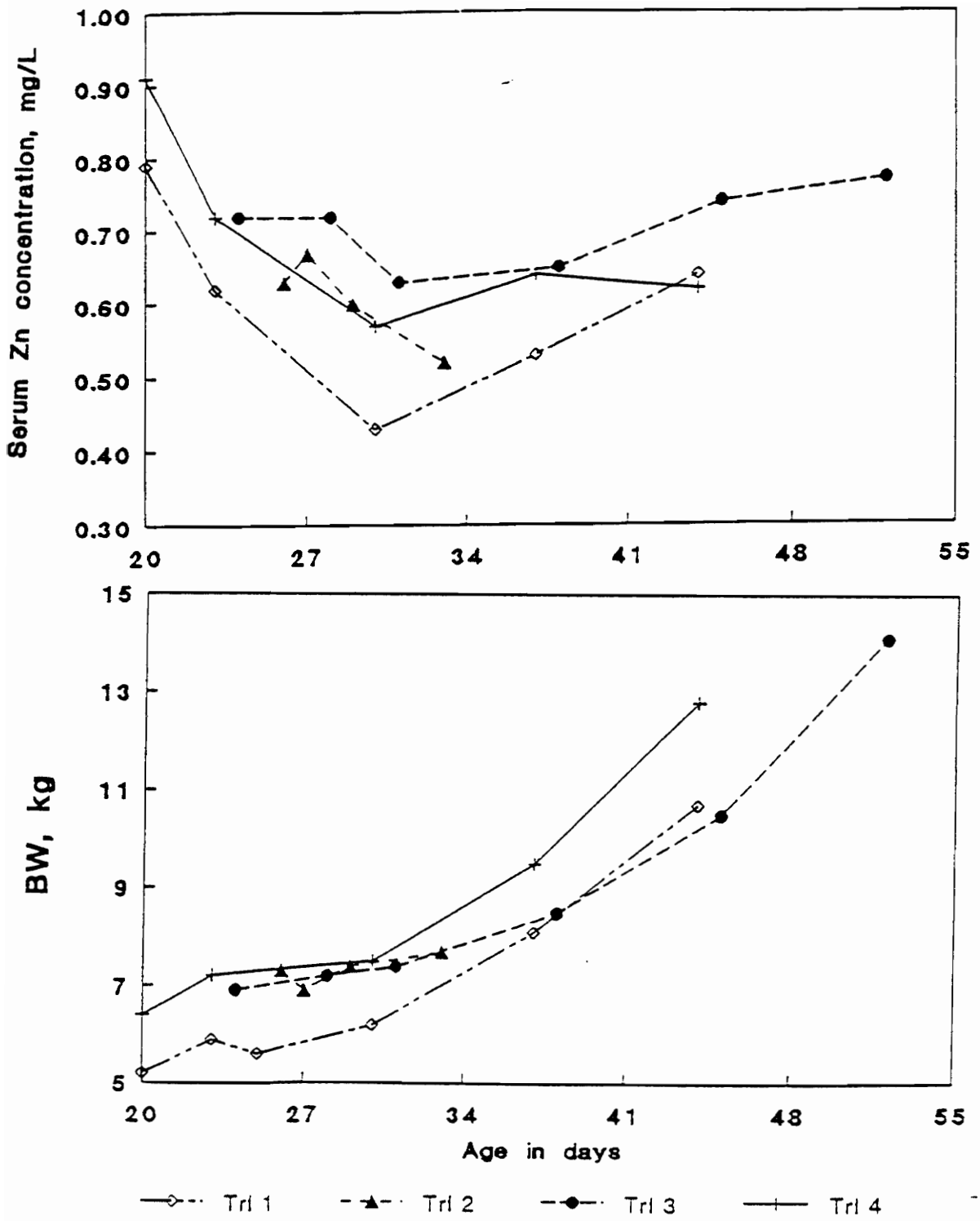


Figure 2. Serum Zn concentrations (pooled SEM = .03) and BW (pooled SEM = .49) of control pigs from Trials 1, 2, 3, and 4 versus age of the pigs.

It remains to be determined whether the injection of zinc in another form or under different conditions can be beneficial to weanling pigs. Furthermore, because the decline in serum Zn concentrations of pigs at weaning was not prevented in these trials, the relationship of this decline to postweaning performance remains unclear.

Literature cited

Chesters, J.K. and J. Quarterman. 1970. Effects of zinc deficiency on food intake and feeding patterns of rats. Br. J. Nutr. 24:1061.

Chesters, J. K., and M. Will. 1981. Measurement of zinc flux through plasma in normal and endotoxin-stressed pigs and the effects of Zn supplementation during stress. Br. J. Nutr. 46:119.

Funderburke, D. W., and R. W. Seerley. 1990. The effects of postweaning stressors on pig weight change, blood, liver, and digestive tract characteristics. J. Anim. Sci. 68:155.

Grummt, F., C. Weinmann-Dorsch, J. Schneider-Schaulies, and A. Lux. 1986. Zinc as a second messenger of mitogenic induction. Exp. Cell. Res. 163:191.

Kavanagh, N. T. 1992. The effect of feed supplemented with zinc oxide on the performance of recently weaned pigs. Proceedings of the International Pig Veterinary Society, 12th Congress. The Hague, Netherlands. II:616.

King, J.C. 1990. Assessment of zinc status. J. Nutr. 120:1474.

Ogunbameru, B. O., E. T. Kornegay, and C. M. Wood. 1992. Effect of evening or morning weaning and immediate or delayed feeding on postweaning performance of weanling pigs. J. Anim. Sci. 70:196.

SAS. 1988. SAS User's Guide: Statistics. SAS Inst. Inc., Cary NC.

Sweet, L. A., E. T. Kornegay, and M.D. Lindemann. 1990. The effects of dietary luprosil NC on the growth performance and scouring index of weanling pigs. Agribiol. Res. 43:271.

Swinkels, J. W. G. M. 1992. Availability of zinc from an amino acid chelate in Zn depleted pigs. Ph.D. Dissertation. Virginia Polytechnic Institute and State University, Blacksburg.

Ullrey, D. E., E. R. Miller, B. E. Brent, B. L. Bradley, and J. A. Hoefer. 1968. Swine hematology from birth to maturity IV. Serum calcium, magnesium, sodium, potassium, copper, zinc and inorganic phosphorus. J. Anim. Sci. 26:1024.

Chapter V

Performance and tissue zinc concentrations of weanling pigs fed pharmacological concentrations of zinc from ZnO, $ZnSO_4$, Zn-methionine and Zn-lysine

ABSTRACT

Five trials were conducted to examine the performance and tissue mineral deposition of pigs fed high concentrations of zinc from ZnO, Zn-methionine, Zn-lysine and $ZnSO_4$. In Trial 1, pigs (n=80, 28 d, 7.5 kg) were assigned to dietary treatments of 3,000 mg Zn/kg of diet as ZnO, Zn-methionine, Zn-lysine or $ZnSO_4$ or a control diet with 105 mg Zn/kg of diet for 2 wk. Pigs fed the ZnO diet had the highest ADFI ($P < .01$) during wk 1. Serum, liver, and rib Zn concentrations were higher ($P < .01$) for pigs fed the high Zn levels compared with controls. In Trial 2, pigs (n=80, 26 d, 7.1 kg) were fed diets containing 2,000 mg Zn/kg of diet from the same sources as in Trial 1. Feeding high Zn depressed performance compared with controls ($P < .01$). Pigs fed the high Zn had higher ($P < .01$) serum, liver, kidney, and rib Zn concentrations than controls. In Trial 3, pigs (n=70, 23 d, 5.3 kg) were assigned to treatments with 1,000 mg Zn/kg of diet from the same sources

as in Trial 1. There were no differences in ADG or ADFI. Serum and liver Zn concentrations were lower ($P < .01$) for pigs fed the control diet compared with the high Zn diets; zinc sources did not differ. In Trials 4 and 5 (n=72, 25 d, 7.1 kg), the data were pooled. Pigs were fed diets containing 3,000 mg Zn/kg of diet from feed grade $ZnSO_4$, reagent grade $ZnSO_4$, or feed grade ZnO for 4 wk. During wk 2, pigs fed the ZnO diet had higher ($P < .05$) ADG and G/F than pigs fed the $ZnSO_4$ diets. Serum, liver and kidney Zn concentrations were lower for pigs fed the ZnO after wk 2 than pigs fed the $ZnSO_4$. The results of these trials indicate no performance benefit to the feeding of high levels of zinc after weaning. Additionally, ZnO is less available than $ZnSO_4$, Zn-lysine or Zn-methionine which appear to be equally available.

Key words: Pigs, zinc, availability, complexes, serum, tissues.

Introduction

Feeding therapeutic levels of Zn to weanling pigs was shown to increase performance and reduce scouring immediately after weaning (Kavanagh, 1992; Poulsen, 1992). In these studies, a beneficial response was reported using 2,500 to 3,000 mg Zn/kg of diet as ZnO or $ZnSO_4$. However, other researchers have reported no benefits with addition of high concentrations of Zn to weanling pig diets (Fryer et

al., 1992). These conflicting results illustrate a need to further evaluate the effects of feeding high concentrations of Zn to weanling pigs.

Interest in using mineral complexes as mineral sources for swine has increased because of potential improvements in availability compared with inorganic sources. Hahn and Baker (1993) suggested a higher availability of Zn, based on higher plasma Zn concentrations for weanling pigs fed a Zn-lysine complex compared with pigs fed $ZnSO_4$ or ZnO. However, Wedekind and Lewis (1993) found that $ZnSO_4$ was more available to pigs than Zn-lysine or Zn-methionine.

The objectives of this study were to investigate the practice of feeding high concentrations of Zn to pigs to improve postweaning performance, and to compare the availability of several sources of Zn for swine when fed at high concentrations.

Materials and Methods

Five trials were conducted using 302 crossbred weanling pigs. The procedures were similar in Trials 1, 2, and 3, and in Trials 4 and 5.

Trials 1, 2, and 3. Each trial contained a control diet with 105 mg Zn/kg of diet and four test diets each using one of the following Zn sources: feed grade ZnO (Bullard Feed Co., Bremen, IN), reagent grade $ZnSO_4$ (#Z76-3, Fisher Scientific, Fairlawn, NJ), Zn-lysine, or Zn

methionine (ZinPro Corp. Inc., Edina, MN). Chromium oxide (#C333-3, Fisher Scientific Co., Fairlawn, NJ) was added to all of the diets at the rate of .05% for the indirect determination of apparent Zn, Cu, and Fe absorption (Dellaert et al., 1990). In Trial 1, 80 crossbred pigs with an average initial age of 28 ± 3 d and weight of 7.5 kg were fed either the control diet or a diet containing one of the four Zn sources at a level to supply 3,000 mg of Zn per kg of diet. In Trial 2, 80 crossbred pigs with an average initial age of 26 ± 6 d and weight of 7.1 kg were fed experimental diets that were calculated to supply 2,000 mg of Zn from each of the test sources. In Trial 3, 70 crossbred pigs with an average initial age of 23 ± 3 d and weight of 5.3 kg were fed either the control diet or a diet with one of the Zn sources at a level to supply 1,000 mg of Zn per kg of diet.

In each of the trials, pigs were randomly assigned to treatments from outcome groups based on weight, gender and litter. Pigs were placed two (1 barrow and 1 gilt) per pen (.6 m x 1.2 m) for the 2 wk of each trial. The pens had plastic welded wire floors and were located in totally enclosed environmentally controlled rooms. Room temperatures were adjusted according to the recommendations of Harp and Huhnke (1992). In each of the trials, pigs were given ad libitum access to a standard corn-soybean meal

basal diet with 15% added dried whey. The diets met or exceeded NRC (1988) estimated recommendations for all nutrients (Table 1). Experimental Zn concentrations were achieved by replacing an appropriate amount of corn with one of the Zn sources. All of the diets contained equivalent concentrations of lysine and methionine. Pigs were given ad libitum access to nipple waters throughout the trials. The care and treatment of the pigs followed published guidelines (Consortium, 1988).

In trial 1, scouring was scored per pen of pigs on a scale of one to five as described by Sweet et al. (1991). A score of two was considered a normal feces, 5 was considered severe scouring, and a score of 1 was a dry stool. Scour scores were not taken in Trials 2 and 3 due to an observed lack of scouring.

Trials 4 and 5. In Trials 4 and 5, 72 crossbred pigs were used to compare the storage and absorption of Zn from feed grade ZnO (Bullard Feed Co., Bremen, IN), reagent grade $ZnSO_4$ (#Z76-3, Fisher Scientific, Fairlawn, NJ), and feed grade $ZnSO_4$ (Zinc Nacional, S.A. Monterrey, N.L. Mexico) and to compare the collection of tissue data after 2 wk on treatment diets versus tissue collection after 4 wk on treatment diets. Both trials were performed using the same protocol. In each trial, 36 pigs (Trial 4: ave age 26 $\pm$ 2

Table 1. Diet compositions (%)[a]

Item	control diet
Corn	49.9
Soybean meal (44)	31.0
Whey	15.0
Dicalcium phosphate	1.15
Limestone	.80
Methionine	.60
Lysine	.55
Vitamin premix[b]	.25
Cr_2O_3-starch[c]	.20
Antibacterial[d]	.10
Mineral premix[e]	.05
Selenium premix[f]	.05

[a]Diets calculated to contain 20% protein, .8% Ca, .65% P, 1.66% lysine and .88% methionine.
[b]Supplied per kilogram of diet: 4,400 IU of vitamin A, 440 IU of vitamin D, 11 IU of vitamin E, 4.4 mg of riboflavin, 22 mg of d-pantothenic acid, 22 mg of niacin and 489.4 mg choline.
[c]Contained 3 g corn starch : 1 g Cr_2O_3.
[d]Mecadox 10, 2.2% carbadox, Animal Health Co. NY.[e]Supplied per kilogram of diet: 105 mg of Zn, 123 mg of Fe, 42 mg of Mn, 12 mg of Cu, and 2 mg of I.[f]Supplied .3mg of Se per kilogram of diet.

d, ave BW 7.9 kg; Trial 5: ave age 23 ± 3 d, ave BW 6.2 kg) were randomly assigned to one of the three treatments from outcome groups based on weight, gender and litter. Diets were similar to the diets in Trials 1, 2, and 3 and were calculated to provide 3,000 mg Zn per kg of diet from each of the Zn sources. Chromium oxide was again added to the diets at .05% for the indirect determination of apparent Zn, Cu, and Fe absorption. For the first 2 wk of the trials, the diets contained 15% dried whey and were calculated to contain 20% CP. For the second 2 wk, the diets were calculated to contain 18% CP with no added whey. The treatment of the animals was the same as for Trials 1, 2, and 3 except that the pigs were placed three (2 barrows and 1 gilt) to a pen (1.2 m x 1.2 m) for the first 2 wk and then 2 (1 barrow and 1 gilt) per pen (1.2 m x 1.2 m) for the second 2 wk.

All trials. In all the trials, pigs were weighed and bled via jugular venipuncture at the initiation of each trial and then weekly. To avoid postprandial fluctuations in serum Zn concentrations, feed was removed from all pigs 30 min prior to bleeding (Goodall et al., 1987). Blood samples were stored for 24 h at 4° C and then centrifuged (700 x g) for 10 min. Serum was then removed and serum Zn concentrations were determined using atomic absorption spectophotometry (Perkin Elmer 5100, Norwalk, CT). Serum

iron concentrations were also determined in Trial 1. The procedure for iron determination was the same as for the serum Zn determination.

At the beginning of wk 2 (and wk 4 for Trials 4 and 5) random grab samples of feces (approximately 10 g) were collected at 0700 and 1900 h from each pen for 1 wk. After collection, the feces were dried at 60° C and ground to pass a 1 mm screen. The feces were then analyzed for Zn, Fe, Cu, and Cr concentration using flame atomic absorption spectophotometry. Absorption of Zn, iron and copper was then calculated using the indirect method (Dellaert et al., 1990).

At the conclusion of all trials (wk 2 for Trials 1, 2, and 3 and wk 4 for Trials 4 and 5), the barrow in each pen was killed by electro-immobilization and exsanguination. At the end of wk 2 for Trials 4 and 5, one of the two barrows in each pen was randomly selected and killed. Livers, kidneys, and 10th left rib were removed for later mineral analysis. In Trial 1, the left external carpi radialis muscles were also removed for Zn analysis. Livers and kidneys were homogenized and analyzed for Zn, Fe, and Cu content. Ribs were dried for 48 h at 60° C and hand ground using a mortar and pestal before digestion and Zn determination. Muscles were freeze-dried before digestion and Zn determination. Tissue, feed and fecal samples were

prepared for mineral analysis using nitric/perchloric acid wet digestion (AOAC, 1990). Flame atomic absorption spectophotometry (Perkin Elmer 5100, Norwalk, CT) was used for mineral determinations.

Data from each trial were analyzed using the GLM procedure of SAS (1992) with treatment and replicate in the model and pen of pigs as the experimental unit for the performance and scouring data, and pig as the experimental unit for the serum and organ analysis. A contrast statement was used to compare all Zn sources to the control. Differences between Zn sources were determined by paired t-tests.

For Trials 4 and 5, the data were pooled because no significant trial x treatment interactions were found. The models contained treatment, replicate, trial and trial x treatment interactions. Differences between Zn sources were determined using paired t-tests.

Results

Trial 1. The analyzed Zn concentrations of the diets for Trial 1 were 111 mg/kg of diet for the control diet, 2,902 mg/kg for the ZnO diet, 2,856 mg/kg for the Zn-methionine diet, 2,760 mg/kg for the Zn-lysine diet and 3,293 mg/kg for the $ZnSO_4$ diet.

There were no performance differences between pigs that were fed the high Zn diets and pigs that were fed the

control diet. Within zinc sources, pigs fed the ZnO diet had a higher ($P < .05$) ADG during wk 1 than pigs fed the Zn-methionine ($P < .01$) or the Zn-lysine ($P < .05$; Table 2). For the overall ADG, pigs fed the ZnO had higher ($P < .05$) ADG than pigs fed the Zn-methionine diet. The ZnO fed pigs also had a higher ($P < .01$) ADFI during wk 1 than the pigs fed any of the other treatment diets.

Control pigs were the only pigs to scour during the trial. The treatment effect for scouring was significant for d 12 and d 13 ($P < .01$). Scouring in general was minimal; each of the control pens were observed scouring for only 2 d during the trial.

Each week after the initiation of the trial, serum Zn concentrations for the control pigs were lower ($P < .01$) than for the pigs fed the high Zn diets (Table 3). The serum Zn concentrations of pigs fed the Zn-methionine were lower than those of pigs fed the $ZnSO_4$ after wk 1. Serum Fe concentrations were not different between treatments at any of the bleedings and were relatively constant (1.8 mg/L) after a rise of approximately .7 mg/L during the first week.

Feeding 3,000 mg Zn/kg of diet led to higher ($P < .01$) hepatic and kidney Zn concentrations compared with control pigs (Table 4). Pigs fed the $ZnSO_4$ diet had higher ($P < .05$) Zn concentrations in the liver compared with pigs fed

Table 2. Performance of weanling pigs fed diets with high concentrations of zinc from different sources (Trials 1 and 2)[a]

	Treatments				
	Control	ZnO	ZnMet	ZnLys	$ZnSO_4$	SEM
Trial 1 (3,000 mg Zn/kg of diet)						
ADG, kg						
Week 1	.02	.04[df]	-.03[e]	-.02[g]	.01	.02
Week 2	.20	.27	.17	.25	.20	.03
Wk 1-2	.11	.15[d]	.07[e]	.12	.09	.02
ADFI, kg						
Week 1	.22	.26[d]	.15[e]	.18[e]	.18[e]	.02
Week 2	.61	.79	.64	.62	.58	.06
Wk 1-2	.42	.53	.39	.40	.38	.04
G/F						
Week 1	.05	.11	.01	.01	.06	.13
Week 2	.32	.35	.27	.41	.34	.17
Wk 1-2	.25	.29	.18	.29	.24	.13
Trial 2 (2,000 mg Zn/kg of diet)						
ADG, kg						
Week 1[b]	.06	.01[d]	-.01[d]	-.01[d]	.07[e]	.02
Week 2[c]	.24	.30	.26	.31	.29	.02
Wk 1-2	.15	.15	.13[d]	.15	.18[e]	.01
ADFI, kg						
Week 1[b]	.19	.14	.11[d]	.13[f]	.17[eg]	.01
Week 2	.44	.43[f]	.43[f]	.45	.51[g]	.03
Wk 1-2	.31	.29[f]	.27[f]	.29[f]	.34[g]	.02
G/F						
Week 1	.30	.07	.01[f]	.01[f]	.42[g]	.14
Week 2	.55	.70	.61	.72[f]	.57[g]	.05
Wk 1-2	.49	.53	.47	.54	.54	.03

[a]Means are for 16 pigs per treatment. Initial BW was 7.5, and 7.1 kg respectively for Trials 1, and 2.

[b,c]Contrast high zinc diets were different from the control diet ($P < .01$ and .05 respectively).

[d-g]Using paired t-tests between high zinc diets, means with different superscripts differ [d,e] ($P < .01$), [f,g] ($P < .05$).

Table 3. Serum zinc concentrations (mg/L) of weanling pigs fed diets with high concentrations of zinc from different sources[a]

	Treatments					
	Control	ZnO	ZnMet	ZnLys	$ZnSO_4$	SEM
Trial 1 (3,000 mg Zn/kg of diet)						
Initial	.58	.53	.58	.56	.57	.02
Week 1[b]	.48	.65	.58[c]	.72	.73[d]	.04
Week 2[b]	.49	.97	1.04	1.06	1.12	.10
Trial 2 (2,000 mg Zn/kg of diet)						
Initial	.72	.64	.67	.71	.71	.03
Week 1[b]	.59	.65[e]	.69[e]	.74[e]	.94[f]	.03
Week 2[b]	.65	.95[e]	1.31[f]	1.16[g]	1.49[fh]	.07
Trial 3 (1,000 mg Zn/kg of diet)						
Initial	.82	.83	.86	.89	.82	.03
Week 1[b]	.61	.67	.75	.69	.71	.03
Week 2[b]	.65	.78	.80	.78	.82	.03

[a]Means are for 16 pigs per treatment in Trials 1 and 2, and 14 pigs per treatment mean in Trial 3.
[b]Contrast: high Zn diets were higher than the contol diet ($P < .01$).
[c-h]Using paired t-tests between the high Zn diets, means with different superscripts differ [c,h]($P < .05$), [e,f]($P < .01$), [g,h]($P < .01$).

Table 4. Tissue Zn concentrations[a] of weanling pigs fed diets with high concentrations of zinc from different sources[b]

		Treatments				
	Control	ZnO	ZnMet	ZnLys	$ZnSO_4$	SEM
Trial 1 (3,000 mg/kg of Zn added)						
Livers[c]	155	1,224[d]	1,067[d]	1,721[e]	1,883[e]	191
Kidneys[c]	109	185	168	203	235	25
Rib bones[cf]	131	193	199	212	219	15
Muscles[gh]	119	97	113	99	105	6
Trial 2 (2,000 mg/kg of Zn added)						
Livers[c]	160	347[i]	747[d]	636[d]	1,007[ej]	73
Kidneys[c]	103	129[di]	157[ei]	143[i]	196[j]	8
Rib bones[cf]	178	211[d]	236	214[d]	245[e]	9
Trial 3 (1,000 mg/kg of Zn added)						
Livers[h]	115	153	136	135	149	13
Kidneys	127	127	130	120	120	6
Rib bones[f]	168	176	184	168	187	11

[a]All tissue mineral concentrations reported as mg/kg of DM.
[b]Means are for 8 pigs per treatment for both Trials 1 and 2, 7 pigs per treatment mean for Trial 3.
[c]Contrast: high zinc diets are different from the control diet ($P < .01$).
[d,e]Using paired t-tests between the high Zn diets, means with different superscripts differ ($P < .05$)
[f]Left 10th rib bone.
[g]Left external carpi radialis muscle.
[h]Contrast: high zinc diets are different from the control diet ($P < .05$).
[i,j]Using paired t-tests between the high Zn diets, means with different superscripts differ ($P < .01$).

the ZnO or the Zn-methionine. Pigs fed the Zn-lysine diet also had a higher ($P < .05$) hepatic Zn concentration than pigs fed the Zn-methionine. There were no differences in kidney Zn concentrations among the pigs fed the high Zn diets. Liver Fe and Cu concentrations were not different across treatments (407 mg/kg of DM, SEM 71; and 49 mg/kg of DM, SEM 12, respectively). Feeding the high concentration of Zn reduced ($P < .05$) the Fe concentrations of the kidney (136 vs. 165 mg/kg DM), and increased ($P < .01$) the Cu concentrations of the kidneys compared with controls (43 vs 20 mg/kg DM).

Pigs fed the Zn-lysine and the $ZnSO_4$ diets had higher ($P < .01$ and .05, respectively) Cu concentrations in the kidney than pigs fed the Zn-methionine and ZnO diets (55 and 52 vs. 32 and 34 mg/kg DM, respectively). Although the muscle Zn concentrations were lower ($P < .05$) for pigs fed the high Zn diets than for control pigs this difference does not appear to be biologically significant. As with the livers and kidneys, pigs fed the 3,000 mg Zn/kg of diet had higher ($P < .01$) rib Zn concentrations than control pigs. There were no differences in muscle or rib bone Zn concentrations among the pigs fed the high Zn diets. Liver and kidney weights relative to BW were not different across treatments (2.5%, .1 SEM, and .51%, .02 SEM, respectively).

Trial 2. The analyzed Zn concentrations of the diets for Trial 2 were 105 mg/kg for the control diet, 1,996 mg/kg for the ZnO diet, 2,247 mg/kg for the Zn-methionine diet, 2,061 mg/kg for the Zn-lysine diet and 2,454 mg/kg for the $ZnSO_4$ diet.

During the first week, pigs fed the control diet had higher ($P < .01$) ADG and ADFI than pigs fed the high Zn diets (Table 2). During the second week, ADG for pigs fed the control diet was lower ($P < .05$) than for pigs fed the treatment diets. Gain to feed ratios were not different between control pigs and treatment pigs during any week or over both weeks. During the first week, pigs fed the $ZnSO_4$ diet had higher ADG ($P < .01$), and gain to feed ratios ($P < .05$) than pigs fed the other diets with 2,000 mg Zn/kg of diet added. Feed intake for pigs fed the $ZnSO_4$ diet was higher than for pigs fed the Zn-methionine ($P < .01$) or the Zn-lysine ($P < .05$) diets. However, the ADG, feed intake and feed efficiency of pigs fed the $ZnSO_4$ were similar to the performance levels of the control pigs during the first week.

During the second week, feed intake remained higher ($P < .05$) for pigs fed the $ZnSO_4$ diet compared with the Zn-methionine fed pigs. The $ZnSO_4$ fed pigs also consumed more ($P < .05$) than pigs fed the ZnO diet during the second week.

Overall, the pigs fed the $ZnSO_4$ diet ate more ($P < .05$) than pigs fed the other high Zn diets.

For the overall trial, the ADG of the pigs fed the $ZnSO_4$ diet was only larger ($P < .01$) than that of pigs fed the Zn-methionine diet. The pigs fed the Zn-lysine diets converted feed into body weight more efficiently ($P < .05$) during wk 2 than pigs fed the $ZnSO_4$ diet. However, overall feed efficiency was not different across treatments.

As in Trial 1, pigs fed the control diet had lower ($P < .01$) serum Zn concentrations after wk 1 and wk 2 than pigs fed the high Zn diets (Table 3). Also after the first week, serum Zn concentrations were higher ($P < .01$) for pigs fed the $ZnSO_4$ diet than for pigs fed the other high Zn diets. After wk 2, pigs fed the $ZnSO_4$ diet had higher ($P < .01$) serum Zn concentrations than pigs fed the Zn-lysine or the ZnO diets. Pigs fed the Zn-methionine diet also had higher ($P < .01$) serum Zn concentrations after wk 2 than pigs fed the ZnO diet.

Pigs that were fed the high Zn diets had higher ($P < .01$) hepatic, kidney and rib bone Zn concentrations than pigs fed the control diet (Table 4). The livers of the pigs fed the control diet were heavier relative to BW than pigs fed the high Zn diets (2.9 vs. 2.6%). Pigs fed the $ZnSO_4$ diet had a higher Zn concentration in the liver than pigs fed the ZnO ($P < .01$), Zn-lysine ($P < .01$) or the Zn-

methionine ($P < .05$) diets. Similarly, pigs fed the $ZnSO_4$ diet had higher ($P < .01$) Zn concentrations in the kidneys compared with pigs fed the other high Zn diets. The pigs fed the Zn-methionine diet also had higher ($P < .05$) kidney Zn concentrations compared with pigs fed the ZnO diet. The rib bones of the $ZnSO_4$ fed pigs also had higher ($P < .05$) Zn concentrations than the pigs fed the ZnO or the Zn-lysine diets.

As in Trial 1, the Cu concentrations of the kidneys of pigs fed the high Zn diets were higher ($P < .01$) than in pigs fed the control diet (46 vs. 21 mg/kg DM). There were no differences in liver Fe (393 mg/kg DM, SEM 59), kidney Fe (120 mg/kg DM, SEM 11) and liver Cu (80 mg/kg DM, SEM 7) concentrations. The weights of the kidneys relative to BW were not different across treatments (.49%, SEM .02).

Trial 3. The analyzed Zn concentrations of the diets for Trial 2 were 99 mg/kg for the control diet, 939 mg/kg for the ZnO diet, 1,014 mg/kg for the Zn-methionine diet, 973 mg/kg for the Zn-lysine diet and 906 mg/kg for the $ZnSO_4$ diet.

Average daily gain and ADFI were not different between controls and pigs fed the high Zn diets and there were also no differences among pigs fed the high zinc diets (Table 5). Pigs fed the Zn-lysine diet had higher ($P < .05$) gain to feed ratios than pigs fed the ZnO diet during the first

Table 5. Performance of weanling pigs fed diets with high concentrations of zinc from different sources (Trial 3)[a]

	Control	Treatments				SEM
		ZnO	ZnMet	ZnLys	$ZnSO_4$	
Trial 3 (1,000 mg/kg of Zn added)						
ADG, kg						
Week 1	.06	.03	.04	.06	.04	.01
Week 2	.24	.24	.24	.27	.26	.01
Wk 1-2	.15	.13	.14	.16	.15	.01
ADFI, kg						
Week 1	.15	.14	.13	.14	.14	.01
Week 2	.38	.36	.37	.38	.34	.02
Wk 1-2	.26	.25	.25	.26	.24	.01
G/F						
Week 1	.39	.12[b]	.28	.37[c]	.28	.08
Week 2	.62	.65[b]	.64[b]	.70	.76[c]	.03
Wk 1-2	.56	.52[d]	.55[bh]	.61[i]	.63[ec]	.02

[a]Means are for 14 pigs per treatment. Initial BW was 5.3 kg.
[b-g]Using paired t-tests between high zinc diets, means with different superscripts differ [b,c]($P < .05$), [d,e]($P < .01$) and [f,g]($P < .05$).

week. However, the gain to feed ratios of the Zn-lysine pigs were similar to controls. During the second week, pigs fed the high Zn diets had higher ($P < .05$) gain to feed ratios than pigs fed the control diet. Pigs fed the $ZnSO_4$.diet had higher ($P < .05$) G/F than pigs fed either the ZnO diet or the Zn-methionine diet. Overall, the pigs fed the $ZnSO_4$ had higher ($P < .05$) gain to feed ratios than pigs fed the ZnO ($P < .01$) or the Zn-methionine ($P < .05$) diet. The pigs fed the Zn-lysine diet also had higher G/F than pigs fed the ZnO diet.

As in Trials 1 and 2, pigs fed the high Zn diets had higher ($P < .01$) serum Zn concentrations after wk 1 and 2 than pigs fed the control diet (Table 3). There were no differences in serum Zn concentrations between pigs fed the high Zn diets.

The liver Zn concentrations of pigs fed the high Zn diets were higher ($P < .05$) than those for pigs fed the control diet (Table 5). The liver Cu concentrations of the $ZnSO_4$ fed pigs were higher than those of pigs fed either the Zn-methionine ($P < .01$) or the Zn-lysine ($P < .05$) diets (73 vs. 38 and 47 mg/kg DM, respectively). There were no differences in the liver and kidney weights relative to BW across treatments (2.9%, SEM .1 and .59%, SEM .03, respectively). Also, there were no differences in liver Fe (630 mg/kg DM, SEM 81) concentrations across treatments.

Kidney Zn (Table 5), Fe (239 mg/kg DM, SEM 27), and Cu (31 mg/kg DM, SEM 4) concentrations were not different between treatments. Rib bone Zn concentrations were also unaffected by the treatments (Table 5).

Trials 4 and 5. The average analyzed Zn concentrations of the diets were 2,870 mg/kg for the reagent grade $ZnSO_4$ diets, 2,877 mg/kg for the ZnO diets, and 2,810 mg/kg for the feed grade $ZnSO_4$ diet.

Pigs fed the ZnO diet had higher ($P < .05$) ADG during wk 2 compared with pigs fed the reagent grade $ZnSO_4$ ($P < .05$) and the feed grade $ZnSO_4$ ($P < .01$) diets (Table 6). During wk 4, pigs fed the reagent grade $ZnSO_4$ had lower ($P < .05$) ADG than pigs fed the other two diets. The overall ADG showed that the pigs fed the reagent grade $ZnSO_4$ grew slower ($P < .05$) than the pigs fed the ZnO diet. Feed intake was not different across treatments throughout the trial (overall ADFI, .86 kg/d, SEM .06). The feed efficiency during wk 2 for the pigs fed the ZnO diet was higher ($P < .01$) than that of the pigs fed either of the $ZnSO_4$ sources. As with ADG, the pigs fed the reagent grade $ZnSO_4$ had lower G/F for wk 4 than pigs fed the ZnO ($P < .05$) or the feed grade $ZnSO_4$ ($P < .01$). This result carried through to the overall feed efficiency, in which the pigs fed the reagent grade $ZnSO_4$ had the lowest G/F ($P < .01$).

Table 6. Performance, serum Zn concentrations and tissue Zn concentrations of weanling pigs fed diets with 3,000 mg/kg of zinc added from three inorganic sources (Trials 4 + 5)[a]

	Treatments			
	ZnO feed grade	ZnSO4 reagent grade	$ZnSO_4$ feed grade	SEM
ADG, kg				
Week 1	.06	.05	.07	.01
Week 2	.41[bd]	.35[c]	.33[e]	.02
Week 3	.29	.31	.32	.05
Week 4	.53[b]	.46[c]	.58[b]	.03
Wk 1-2	.23	.20	.20	.01
Wk 3-4	.27	.23	.29	.02
Wk 1-4	.26[b]	.21[c]	.25	.01
G/F				
Week 1	.22	.23	.35	.09
Week 2	.74[d]	.63[e]	.67[e]	.02
Week 3	.36	.37	.44	.04
Week 4	.28[b]	.20[cd]	.31[e]	.03
Wk 1-2	.64[b]	.53[c]	.59	.02
Wk 3-4	.32[bd]	.26[c]	.36[be]	.01
Wk 1-4	.37[d]	.30[e]	.39[d]	.01
Serum Zn, mg/L				
Initial	.73	.76	.72	.02
Week 1	.90	1.04	1.04	.06
Week 2	1.62[d]	2.26[e]	1.93	.12
Week 3	2.18[d]	2.97[e]	2.91[e]	.13
Week 4	2.20[d]	2.98[e]	2.99[e]	.11
Tissue Zn concentrations, mg/kg DM				
Week 2				
Liver	910[b]	1,168	1,312[c]	135
Kidney	185[b]	259[c]	258[c]	23
Rib bone	242	273	267	13
Week 4				
Liver	1,569	1,592	1,344	176
Kidney	455	518	454	61
Rib bone	345	375	350	27

[a]The data for Trial 4 and 5 were pooled. Means are for 24 pigs per treatment for weeks 1 and 2 and 16 pigs per treatment for weeks 3 and 4. Initial and final body weights were 7.2 and 13.8 kg respectively.

[b,c] and [d,e]Using paired t-tests between the high Zn diets, means with different superscripts differ ($P < .05$ and .01 respectively).

Serum Zn concentrations of pigs fed the ZnO diet were lower ($P < .01$) after 2 wk than those of pigs fed the reagent grade $ZnSO_4$ diet but were not different from pigs fed the feed grade $ZnSO_4$ diet (Table 6). However, after 3 wk, the pigs fed the ZnO diets had lower ($P < .01$) serum Zn concentrations than pigs fed either of the $ZnSO_4$ sources. This pattern carried through wk 4 ($P < .01$). The serum Zn concentrations of pigs fed the $ZnSO_4$ diets did not differ between sources throughout the trials.

At the end of wk 2, the liver Zn concentrations of the pigs fed the ZnO were lower ($P < .05$) than pigs fed the feed grade $ZnSO_4$ (Table 6); intermediate values were observed for pigs fed the reagent grade $ZnSO_4$. There were no differences in liver Fe (439 mg/kg DM, SEM 89), and Cu (69 mg/kg DM, SEM 12) concentrations. Pigs fed the ZnO diet had lower ($P < .05$) kidney Zn concentrations than pigs fed either $ZnSO_4$ source. There were no differences in kidney Fe concentrations (128 mg/kg DM, SEM 14). Kidney Cu concentrations of pigs fed the ZnO were lower ($P < .05$) than the concentrations of pigs fed the feed grade $ZnSO_4$ diet (53 vs. 80 mg/kg DM). There were no differences in rib bone Zn concentrations between Zn sources.

At the conclusion of the trials, there were no differences in hepatic Zn (Table 6), Fe (424 mg/L, SEM 55), and Cu (26 mg/kg DM, SEM 4) concentrations or kidney Zn

(Table 6), Fe (170 mg/kg DM, SEM 10) and Cu (116 mg/kg DM, SEM 12). Similarly, there were no differences in rib bone Zn concentrations among the treatments (Table 6). The liver and kidney weights relative to BW were not different across treatments (2.9 %, SEM .09 and .57%, SEM .04, respectively).

The calculated digestibilities of Zn, copper, and iron were inconclusive. Many calculated values were negative. The standards run with the feces and feed samples indicate a very low laboratory error rate (less than 5%). Thus, the apparent absorption data were not used for comparison of availability of the sources.

Discussion

The results of these trials indicate no performance benefit to feeding high levels of Zn after weaning. In Trials 1 and 4 + 5 the ZnO fed pigs had a performance advantage for the first 2 wk after weaning when compared with pigs fed the other sources at 3,000 mg Zn/kg of diet, but showed no improvement over the controls. In Trials 2 and 3, pigs fed the high concentrations of Zn performed at or below the levels of control pigs. These results are similar to others who have reported no performance-enhancing effects with the feeding of high levels of zinc (Fryer et al., 1992).

Although scouring was measured only in Trial 1, where the control pigs were the only pigs to scour, our data

indicates that the feeding of high concentrations of Zn may reduce scouring in weanling pigs. In fact, the failure of pigs to scour in our trials may have inhibited the positive growth response seen by others (Poulsen, 1992). If the mode of action for feeding high concentrations is mediated through a reduction in scouring as speculated by Poulsen (1992), this may explain why only a limited performance response was seen in our trials. Fryer et al. (1992), who found no performance improvement in response to the addition of high Zn to weanling pig diets, also reported no scouring in their study.

The data collected to evaluate the availability of the Zn sources does not indicate a higher availability for a particular Zn source. Feeding the high concentrations of Zn in Trials 1, 2 and 3 elevated serum Zn concentrations above the levels of pigs fed the control diets, but differences between pigs fed the various Zn sources were limited. The higher serum Zn concentrations for pigs fed the $ZnSO_4$ diets in Trial 2 may be real despite the higher concentration of Zn in that diet. Expressed as an increase in serum Zn per mg of added Zn above the control diet Zn concentration (Kornegay, 1972), the pigs fed ZnO had only 44% the increase in serum Zn concentration compared with the pigs fed $ZnSO_4$ (Table 7). Similarly, the Zn-lysine fed pigs and the Zn-methionine fed pigs had 75% and 86% of the increase in serum

Table 7. Relative availability of Zn from different Zn sources compared with $ZnSO_4$[a]

	Zinc Sources			
	ZnO	ZnMet	ZnLys	$ZnSO_4$
Trial 1 (3,000 mg Zn/kg of diet)				
Serum (wk 2)	86	101	109	100
Livers	70	61	110	100
Kidneys	68	53	88	100
Rib bones[b]	79	89	111	100
Trial 2 (2,000 mg Zn/kg of diet)				
Serum (wk 2)	44	86	75	100
Livers	28	94	94	100
Kidneys	28	58	43	100
Rib bones[b]	59	93	66	100
Trials 4 + 5[c]	**ZnO**	**$ZnSO_4$ (feed)**	**$ZnSO_4$ (reagent)**	
Serum (wk 2)	84	117	100	
Serum (wk 4)	74	100	100	
Livers (wk 2)	78	112	100	
Kidneys (wk 2)	71	100	100	
Rib bones[b] (wk2)	89	98	100	

[a]Values were calculated by subtracting the Zn concentration of the control diet from each of the sources. Similarly, the tissue Zn concentration of the control diet was subtracted from the tissue Zn concentration from each source. The tissue Zn concentration was then divided by the concentration of Zn in the respective diet. These values were then expressed as a percentage of the values for $ZnSO_4$.
[b]Left 10th rib bone.
[c]Pooled data. Dietary Zn levels were calculated to be 3,000 mg/kg of diet. Values are expressed as percentages of the tissue Zn concentration of pigs fed reagent grade $ZnSO_4$.

Zn per mg of added Zn in the diet compared with the pigs fed the $ZnSO_4$ diet. In Trials 4 + 5, the serum data after 2 wk indicates a 84% availability of the ZnO compared with the reagent grade $ZnSO_4$. This suggests a higher availability of $ZnSO_4$ over the ZnO and possibly the other sources, but because our diets in Trial 2 were found to have different Zn concentrations and because there were no differences at the other dietary Zn concentrations our serum data only suggests a higher availability of $ZnSO_4$ over the ZnO.

As with the serum data, the feeding of 3,000 and 2,000 mg of Zn per kg of diet resulted in an increased level of Zn accumulation in the livers, kidneys and rib bones compared with control pigs. These results are in agreement with Ansari et al. (1976), who reported increases in liver, heart and tibia Zn concentrations when rats were fed 2,400 and 3,600 mg Zn/kg of diet. Feeding the 1,000 mg Zn/kg of diet for 2 wk only increased the liver Zn concentrations above the control level; kidney and rib Zn concentrations were not elevated above the concentrations of the control pigs. Similarly, Ansari et al. (1976) reported that feeding rats 1,200 mg Zn/kg of diet did not increase kidney or tibia Zn concentrations above the levels of control rats fed 38 mg Zn/kg of diet.

Similar to the serum data, the results for the tissue data do not strongly indicate a higher availability for any

particular Zn source. After adjusting the data on a tissue Zn deposition per mg Zn above the control (Table 7), there appeared to be some differences between the Zn sources. In Trial 1, pigs fed the Zn-lysine appeared to store higher levels of Zn in livers and ribs compared with pigs fed $ZnSO_4$; pigs fed ZnO or Zn-methionine stored less Zn in the liver and ribs. In Trial 2, in which pigs were fed 2,000 mg Zn/kg of diet instead of 3,000 mg Zn/kg as in Trial 1, Zn stored in the liver appeared to be slightly lower for pigs fed Zn-lysine and Zn-methionine compared with pigs fed $ZnSO_4$; pigs fed ZnO had the lowest concentrations of Zn stored. Kidney and rib Zn concentrations seemed to follow a similar trend but were more variable. Collectively, the serum and tissue results show that the ZnO was less available than the other sources. The Zn-methionine and Zn-lysine appear to be slightly less available or equally available when compared with the $ZnSO_4$. Using bone and plasma zinc concentrations as zinc availability indicators Wedekind and Lewis (1993) also found the relative availability of ZnO lower than $ZnSO_4$. Wedekind and Lewis (1993) also reported a lower availability of Zn-lysine and Zn-methionine compared with the availability of $ZnSO_4$.

There was an increase in the storage of copper in the kidneys of pigs fed 3,000 and 2,000 mg Zn/kg of diet compared with control pigs. These results support the work

of Cousins and Lee-Ambrose (1992), who reported that increased dietary Zn induced metallothionein synthesis primarily in the kidney. Therefore, because metallothionein has a higher affinity for copper than for Zn, increased dietary Zn may result in increased copper accumulation in the kidney, as seen here.

The iron concentrations of the livers and kidneys were consistently unaffected by the dietary Zn concentrations. These results differ from the results of Hill et al. (1983), who reported decreased iron storage in sows fed high Zn concentrations. The age or reproductive state of the sows versus the pigs in our trials may have resulted in the difference between the results.

The results of Trials 4 + 5, show no differences in the availability of feed grade $ZnSO_4$ and reagent grade $ZnSO_4$. Serum, liver, kidney and rib bone Zn concentrations were not different between the sources. Either source should be considered adequate for availability comparisons involving a $ZnSO_4$ source for pigs.

Additionally, the results of Trials 4 + 5 which compared the accumulation of tissue minerals after feeding high Zn for 2 wk versus after feeding for 4 wk, indicate that the 2 wk data appears to more accurately reflect the previously established relative availability of ZnO and $ZnSO_4$. Wedekind and Baker (1990) reported that the

availability of Zn from ZnO was approximately 60% of that from $ZnSO_4$ when fed to broilers. Our 2 wk results show a 79% availability of Zn from ZnO compared to $ZnSO_4$, whereas the 4 wk data shows no difference in availability. This discrepancy may be related to an increased absorption rate of Zn at the younger age. As the pigs got older the absorption rate may have declined or reached a saturation point, resulting in similar concentrations of Zn being deposited in the tissues. However, because the serum data show higher Zn concentrations when the $ZnSO_4$ was fed compared to when the ZnO was fed at both 2 wk and 4 wk, a more likely explanation is that the older pigs became more efficient at eliminating excess Zn. Therefore, at 4 wk the tissues may not have been accumulating Zn at the same proportion as at 2 wk. Moreover, it appears that a 2 wk feeding period is sufficient to discriminate differences in tissue mineral accumulations in pigs and that serum Zn concentrations appear to remain indicative of availability for at least 4 wk.

Among the tissues used as availability indicators, the liver appears to be the most sensitive and most consistent relative to Zn status. Hepatic Zn concentrations were consistently elevated when the high Zn levels were fed at 1,000, 2,000 and 3,000 mg/kg of diet. Additionally, the trends of Zn accumulation between the sources appeared to be

magnified to the largest extent in the liver. Based on the tissue accumulation of Zn in Trial 3 (1,000 mg Zn/kg of diet), the rib bones and kidneys appeared to be less sensitive indicators of Zn status than the liver. These results are in agreement with the results of Berg and Kollmer (1987), who found bone Zn concentrations of rats to be insensitive to dietary Zn status.

Contrary to the findings of Henry et al. (1987) with broilers, muscle tissue appears to be a poor indicator of Zn status. Feeding 3,000 mg Zn/kg of diet for 2 wk lowered the muscle Zn concentration below the concentration of the control pigs fed 105 mg Zn kg of diet. Similar results have not been previously reported. The magnitude of the differences in this trial indicate that there is not a large degree of difference in the muscle Zn concentrations. Consequently, although a statistical difference existed, it does not appear that there is a biologically significant difference. Others have reported an insensitivity of muscle to high dietary Zn concentrations (Ansari et al., 1976).

The results of the estimations of the apparent digestibility of Zn using the indirect indicator method were inconclusive. The variability of the values and the large number of negative calculated values suggest that the indicator method described by Dellaert et al. (1990) for use with macrominerals is not a useful method for determining

the apparent digestibilities of microminerals. Furthermore, the high concentrations of Zn fed in these trials would have led to very low percentages of absorbed Zn making the sensitivity of the indirect method lower. Even with an acceptable experimental error, these percentages in many cases would be negative, as obtained in these trials. Therefore, our results indicate that the indirect method of determining mineral absorption as described by Dellaert et al. (1990) is insensitive to differences in micromineral availability when the minerals are fed at high concentrations.

Implications

The results of these trials show no performance benefits for the inclusion of high concentrations of Zn to weanling pig diets in the absence of scouring. Furthermore, there appears to be little difference in the availability of Zn from $ZnSO_4$, Zn-methionine and Zn-lysine when comparing serum Zn concentrations and tissue Zn accumulations resulting from feeding high dietary Zn concentrations. The availability of ZnO appears to be lower than that of $ZnSO_4$, Zn-lysine and Zn-methionine. Feeding high concentrations of Zn does not appear to be an effective method of evaluating Zn availability in swine as it is in poultry.

Literature cited

Ansari, M. S., W. J. Miller, M. W. Neathery, J. W. Lassiter, R. P. Gentry, and R. L. Kincaid. 1976. Zinc metabolism and homeostasis in rats fed a wide range of high dietary zinc levels. Proc. Soc. Exp. Biol. Med. 152:192-194.

AOAC. 1990. Official methods of analysis. 15th ed. Association of Official Analytical Chemists, Arlington, VA.

Aoyagi, S. and D. H. Baker. 1993. Nutritional evaluation of copper-lysine and zinc-lysine complexes for chicks. Poult. Sci. 72:165-171.

Berg, D. and W. E. Kollmer. 1987. The influence of zinc deficiency on the storage of zinc in bone. In: L. S. Hurley, C. L. Keen, B. Lonnerdal, and R. B. Rucker (Eds.) Trace elements in Man and Animals 6. Plenum Press. New York. pp 455-457.

Consortium. 1988. Guide for the Care and Use of Agricultural Animals in Agricultural Research and Teaching. Consortium for Developing a Guide for the Care and Use of Agricultural Animals in Agricultural Research and Teaching, Champaign, IL.

Cousins, R. J. and L. M. Lee-Ambrose. 1992. Nuclear zinc uptake and interactions and metallothionein gene expression are influenced by dietary zinc in rats. J. Nutr. 122:56-64.

Dellaert, B. M., G. F. V. Van der Peet, A. W. Jongbloed, and S. Beers. 1990. A comparison of different techniques to assess the biological availability of feed phosphates in pig feeding. Netherlands J. Agri. Sci. 38:555-566.

Fryer, A., E. R. Miller, P.K. Ku, and D.E. Ullrey. 1992. Effect of elevated dietary zinc on growth performance of weanling swine. Mich. State Res. Rep. pp. 128-132.

Goodall, M. J., K. M. Hambridge, C. Stall, J. Pritts, and E. E. Nelson. 1987. Daily variations in plasma zinc in normal adult women. In: L. S. Hurley, C. L. Keen, B. Lonnerdal, and R.B. Rucker (Eds.) Trace Elements in Man and Animals 6. Plenum Press, New York. pp. 491-492.

Hahn, J. D. and D. H. Baker. 1993. Pharmacologic zinc levels for weanling pigs: growth and plasma zinc responses. J. Anim. Sci. 71(Suppl. 1):66.

Harp, S. L., and R. L. Huhnke. 1992. Supplemental heat for swine. In: Pork Industry Handbook, #57. Purdue Univ., West Lafayette. IN.

Henry, P. R., C. B. Ammerman, and R. D. Miles. 1987. Effect of dietary zinc on tissue mineral concentration as a measure of zinc bioavailability in chicks. Nutr. Rep. Int. 35:15-23.

Hill, D. A., E. R. Miller, and H. D. Stowe. 1983. Effect of dietary zinc levels on health and productivity of gilts and sows through two parities. J. Anim. Sci. 57:114-122.

Kavanagh, N. T. 1992. The effect of feed supplemented with zinc oxide on the performance of recently weaned pigs. Proceedings: International Pig Veterinary Meetings. 1992. p. 616.

Kornegay, E. T. 1972. Availability of iron contained in defluorinated phosphate. J. Anim. Sci. 34:569-572.

NRC. 1988. Nutrient requirements of swine (9th Ed.). National Academy Press, Washington DC.

Poulsen, H. D. 1992. Zinc oxide for weaned pigs. Eleventh annual Prince feed ingredient conference. Dublin. Ireland. Appendix 1.

SAS. 1990. SAS/STAT User's Guide: Statistics (Release 6.04 Ed.). SAS Inst. Inc., Cary, NC.

Sweet, L. A., E. T. Kornegay, and M. D. Lindemann. 1991. The effects of dietary luprosil on the growth performance and scouring index of weanling pigs. Agribiol. Res. 43:271-282.

Wedekind, K. J. and D.H. Baker. 1990. Zinc bioavailability in feed-grade sources of zinc. J. Anim. Sci. 68:684-689.

Wedekind, K. J. and A. J. Lewis. 1993. Assessing zinc bioavailability with pigs fed corn-soybean meal diets. Nebraska Res. Rep. p. 24-25.

Chapter VI

A comparison of zinc availability from a Zn-lysine complex and $ZnSO_4$ forweanling pigs

Abstract

Three trials (n=272) were conducted to compare the availability of Zn from $ZnSO_4$ and Zn-lysine for weanling pigs fed lysine deficient and adequate diets. The first two trials (n=128, ave age 27 d, ave BW 7.7 kg) consisted of four dietary treatments. Two treatments contained 2,000 mg Zn/kg of diet from $ZnSO_4$ or Zn-lysine added to a basal corn-soybean meal diet that contained .65% lysine. The other two treatments were the same as the first two plus .45% lysine as L-lysine. The Zn-lysine complex added .45% lysine. Pigs fed the diets with Zn-lysine had higher ($P < .01$) ADG and G/F than pigs fed the diets with $ZnSO_4$. After 4 wk, serum and hepatic Zn concentrations were higher ($P < .01$) for pigs fed the Zn-lysine than for pigs fed $ZnSO_4$. Kidney and rib bone Zn concentrations showed no Zn source effects. In Trial 3, 144 crossbred pigs (ave age 28 d, BW 7.7 kg) were assigned to one of eight treatments. Three dietary lysine levels (.80%, .95% and 1.10%) were fed with either $ZnSO_4$ or Zn-lysine at a level to supply 100 mg/kg of Zn. The two additional diets contained either the high or low lysine level with no added Zn. The low lysine level reduced ADG

and G/F ($P < .01$) compared with pigs fed the other two lysine levels. There were no differences in tissue or serum Zn concentrations between Zn sources, but pigs fed diets without supplemental Zn had reduced serum and liver Zn concentrations ($P < .01$). Overall, there appears to be no difference in Zn availability between $ZnSO_4$ and Zn-lysine for weanling pigs.
Key words: Pigs, zinc, lysine, availability, serum, salt.

Introduction

Recent interest in mineral chelates and complexes for animal nutrition has been stimulated by the discovery that low molecular weight ligands can increase the absorption of minerals. Wapnir and Stiel (1986) reported that adding amino acids to a Zn solution increased the absorption of Zn in rats. The amino acids were thought to bind tightly with the Zn and form complexes before absorption. Mineral complexes are postulated to increase the availability of a mineral by either protecting the mineral from binding to non-absorbable ingredients or by aiding in the transport of the mineral across the brush border membrane.

The results of research on the availability of mineral complexes for swine have been mixed. Hahn and Baker (1993) reported higher plasma Zn concentrations in pigs fed a Zn-lysine complex compared with pigs fed $ZnSO_4$ or ZnO. On the other hand, Wedekind and Lewis (1993) found that Zn from

$ZnSO_4$ was more available than Zn-lysine or Zn-methionine when bone Zn concentrations were used as an indicator of Zn availability.

The objectives of this research were to compare the availability of Zn from $ZnSO_4$ and Zn-lysine for weanling pigs fed lysine deficient and lysine adequate diets.

Materials and methods

Three trials were performed to evaluate the availability of Zn from a Zn-lysine complex compared with $ZnSO_4$ for pigs fed lysine deficient and adequate diets. Trials 1 and 2 had the same protocols except for the number of pigs in each trial. In Trial 1, 48 crossbred pigs (average age of 26 $\pm$ 2 d and BW of 7.1 kg) were used and in Trial 2, 80 crossbred pigs (average age of 28 $\pm$ 2 d and BW of 8.2 kg) were used. In Trial 3, 144 crossbred pigs (average age of 28 d and BW of 7.7 kg) were used to further evaluate the availability of Zn for pigs when $ZnSO_4$ or Zn-lysine were added to supply 100 mg Zn/kg of diet in diets ranging in lysine concentration from .80% to 1.10%.

In Trials 1 and 2, pigs were randomly assigned to four dietary treatments from outcome groups based on weight, gender, and litter. Diet compositions are shown in Table 1. The basal diets were calculated to contain 18% CP and .65% lysine which is approximately 60% of the recommended lysine

Table 1. Diet compositions for Trials 1 and 2 (%)

Item	Diet[a]			
	I	II	III	IV
Corn	71.38	70.30	71.83	70.75
Soybean meal (44)	14.30	14.30	14.30	14.30
Corn gluten meal (61)	10.00	10.00	10.00	10.00
Dicalcium phosphate	1.66	1.66	1.66	1.66
Limestone	.74	.74	.74	.74
Vitamin premix[b]	.25	.25	.25	.25
Cr_2O_3	.20	.20	.20	.20
Antimicrobial[c]	.10	.10	.10	.10
Mineral premix[d]	.05	.05	.05	.05
Selenium premix	.05	.05	.05	.05
Lysine[e]	.45	.45	---	---
$ZnSO_4$	.82	---	.82	---
Zn-lysine[f]	---	1.90	--	..1.90.

[a]Diets calculated to contain 18% protein, .8% Ca, .65% P, and 2,000 ppm of Zn. Diets I and IV contained 1.10% lysine Diet II contained 1.55% lysine and Diet III contained .65% lysine.

[b]Supplied per kilogram of diet: 4,400 IU of vitamin A, 440 IU of vitamin D, 11 IU of vitamin E, 4.4 mg of riboflavin, 22 mg of d-pantothenic acid, 22 mg of niacin and 489.4 mg choline.

[c]Mecadox 10, 2.2% carbadox, Animal Health Co. NY.

[d]Supplied per kilogram of diet: 105 mg of Zn, 123 mg of Fe, 42 mg of Mn, 12 mg of Cu, and 2 mg of I.

[e]Crystalline lysine (78% L-lysine).

[f]LyZin 100 contained, 10% Zn and 18% lysine. Supplied by ZinPro Corp. Inc., Edina, MN.

requirement (NRC, 1988). Dietary treatments were: 1) basal (.65% lysine) plus 2,000 mg Zn/kg of diet as $ZnSO_4$ (#Z76-3, Fisher Scientific, Fairlawn, NJ), 2) basal (.65% lysine) plus 2,000 mg Zn/kg of diet as Zn-lysine (ZinPro Corp. Inc., Edina, MN), 3) basal (1.1% lysine), plus 2,000 mg Zn/kg of diet as $ZnSO_4$, and 4) basal (1.1% lysine) plus 2,000 mg Zn/kg of diet as Zn-lysine. Diet 2 contained a total of 1.1% lysine which included the lysine in the Zn-lysine complex. Diet 4 contained a total of 1.55% lysine which included lysine in Zn-lysine. All the diets were calculated to meet or exceed the estimated requirements for all other nutrients except sodium (.02% for the $ZnSO_4$ diets and .035% for the Zn-lysine diets) and chlorine (.04% for the $ZnSO_4$ diets and .046% for the Zn-lysine diets).

The treatments for Trial 3 are shown in Table 2. The diets were calculated to contain 20% CP and meet the NRC (1988) estimated requirements for all nutrients except for Zn and lysine. The experiment was designed as a 2 x 3 factorial arrangement of treatments with two Zn sources ($ZnSO_4$ and Zn-lysine to provide 100 mg Zn/kg of diet) and three lysine levels (.80%, .95% and 1.10%). Two additional control diets were added to the trial; these treatments were the low or high lysine level with no supplemental Zn. The pigs were randomly assigned to treatments in the same manner as in Trials 1 and 2.

Table 2. Composition of Diets for Trial 3

	Diets							
Zn, mg/kg	0	100	100	100	100	0	100	100
Lysine, %	.80	.80	.80	.95	.95	1.10	1.10	1.10
Ground corn (8.5)	66.74	66.74	66.74	66.74	66.74	66.74	66.74	66.74
Soybean meal (44%)	19.86	19.86	19.86	19.86	19.86	19.86	19.86	19.86
Corn gluten meal (62%)	9.00	9.00	9.00	9.00	9.00	9.00	9.00	9.00
Defluorinated phosphate	1.60	1.60	1.60	1.60	1.60	1.60	1.60	1.60
Limestone	.57	.57	.57	.57	.57	.57	.57	.57
Vitamin premix[b]	.25	.25	.25	.25	.25	.25	.25	.25
Special T.M. premix[c]	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Antimicrobial[d]	.10	.10	.10	.10	.10	.10	.10	.10
Salt	.40	.40	.40	.40	.40	.40	.40	.40
Lysine[e]	.018	.018	-	.210	.192	.402	.402	.384
$ZnSO_4$[f]	-	.028	-	.028	-	-	.028	-
Zn-Lysine[g]	-	-	.10	-	.10	-		.10
Starch	.466	.438	.384	.246	.192	.082	.054	-

[a]Calculated to contain 20% crude protein, .80% Ca and .65% tP, and .25% phytate P.
[b]Supplied per kilogram of diet: 4,400 IU of vitamin A, 440 IU of vitamin D, 11 IU of vitamin E, 4.4 mg of riboflavin, 22 mg of d-pantothenic acid, 22 mg of niacin and 489.4 mg choline.
[c]Trace mineral premix contained (g/kg). $FeSO_4 \cdot 7H_2O$, .25; $CuSO_4 \cdot 5H_2O$, .024; Mn $SO_4 \cdot H_2O$, .012; KIO_3, .00024; Na_2SeO_3, .00066; Dextrose, 9.7131. This mixture supplied (mg/kg): Fe, 50; Cu, 6; Mn, 4; I, .14; Se, .3.
[d]Mecadox10 (10 g/lb), 2.2% carbadox, Animal Health Co. NY.
[e]Crystalline lysine (78% L-lysine).
[f]Feed grade zinc sulfate (36% Zn).
[g]LyZin 100 contained 10% Zn and 18% Lysine. Supplied by ZinPro Corp, Inc., Edina, MN.

After the treatments were assigned, pigs were placed two (1 barrow and 1 gilt) per pen (.6 m x 1.2 m) in Trials 1 and 2 and three (at least 1 barrow) to a pen (.6 m x 1.2 m) in Trial 3. The pigs were allowed ad libitum access to the diets and to nipple waterers for 4 wk of the trials. The pens had plastic welded wire floors and were located in environmentally controlled rooms. Room temperatures were adjusted according to the recommendations of Harp and Huhnke (1992). The care and treatment of the pigs followed published guidelines (Consortium, 1988).

In Trials 1 and 2, pigs were weighed and bled via jugular venipuncture at the initiation of the trial and then weekly. In Trial 3, pigs were weighed weekly and bled initially and then every 2 wk. To avoid postprandial fluctuations in serum Zn concentrations, feed was removed from all pigs 30 min prior to bleeding. Blood samples were stored for 24 h at 4° C and then centrifuged (700 x g) for 10 min. Serum was then removed and serum Zn concentrations were determined using atomic absorption spectophotometry (Perkin Elmer 5100, Norwalk, CT).

At the conclusion of each trial, the barrow from each pen in Trials 1 and 2 and a randomly selected barrow from each pen in Trial 3 were killed by electo-immobilization and exsanguination for the collection and subsequent determination of mineral concentrations of the left 10th rib

bones, livers, and kidneys. Extraneous tissue was removed from the livers and kidneys before they were homogenized and analyzed for Zn, iron and copper content. After removal of extraneous tissue, ribs were only analyzed for Zn concentration. Ribs were dried for 48 h at 60° C before digestion and Zn determination. All mineral analysis were done using nitric/perchloric acid wet digestion (AOAC, 1990) and flame atomic absorption spectophotometry (Perkin Elmer 5100, Norwalk, CT).

In Trials 1 and 2, the data were pooled because no trial x treatment interactions were found. The data were then analyzed using the GLM procedure of SAS (1990). The models included treatment and replicate effects with the pig as the experimental unit for the serum and organ data and pen of pigs as the experimental unit for the performance data. Because treatment differences in ADFI during wk 4 may have influenced the amount of Zn absorbed, ADFI during wk 4 was used as a covariate for the serum Zn analysis at wk 4 and for the analysis of the organ tissue data. Orthogonal contrasts were used to determine Zn, lysine and Zn x lysine interactions.

In Trial 3, the total data set was analyzed using the GLM procedure of SAS (1990). The two diets without supplemental Zn were then dropped from the data set and the data were analyzed as a 2 x 3 factorial with replicate, Zn,

lysine and the Zn x lysine interaction effects in the model. Orthogonal contrasts were used to compare the Zn deficient control diets to the diets with similar lysine levels. Orthogonal contrasts were also used to compare specific diets with different lysine levels.

Results

Trials 1 and 2. The average analyzed Zn concentrations of the diets for Trials 1 and 2 were 1,925 mg of Zn per kilogram of diet; variation between diets was less than 51 mg/kg of Zn.

The main effects of Zn-source and lysine level were significant. Pigs fed the Zn-lysine diets had higher ($P < .01$) ADG and gain to feed ratios than pigs fed the $ZnSO_4$ (Table 3). Average daily feed intake during wk 4 was higher ($P < .01$) for pigs fed the Zn-lysine diets compared with pigs fed the $ZnSO_4$ diets (.67 vs. .59 kg, respectively). However, overall feed intake was not different between pigs fed the different Zn sources. Pigs fed the .65% lysine diet had higher ($P < .05$) ADG than pigs fed the 1.10% lysine diets. The Zn x lysine interaction effect showed that pigs fed the low lysine basal diet and the Zn-lysine grew faster ($P < .01$) than pigs fed the Zn-lysine with the 1.10% lysine diet. Similarly, the overall interaction effect for feed efficiency showed that pigs fed the Zn-lysine with the low lysine basal diet had higher ($P < .01$) gain to feed ratios

Table 3. Performance and tissue zinc concentrations of weanling pigs fed $ZnSO_4$ or Zn-lysine in a marginally lysine deficient diet or a lysine adequate diet. Trials 1 and 2[a]

	Treatments				
Basal diet lysine %	1.10		.65[b]		
Zn source	$ZnSO_4$	Zn-lysine	$ZnSO_4$	Zn-lysine	
Total lysine, %	1.10	1.55	.65	1.10	SEM
ADG, kg					
Wk 1-4[ceg]	.13	.15	.12	.18	.01
ADFI, kg					
Wk 1-4	.38	.38	.39	.42	.01
Gain/feed					
Wk 1-4[cg]	.33	.40	.31	.44	.01
Serum Zn, mg/L					
Initial	.78	.75	.77	.72	.03
Week 1	.91	.82	.88	.83	.04
Week 2[d]	1.22	1.11	1.26	1.17	.05
Week 3[f]	1.36	1.34	1.48	1.45	.04
Week 4[ch]	1.33	1.51	1.43	1.57	.15
Liver Zn,[ch]	700	838	523	808	77
Kidney, Zn[h]	239	217	251	232	14
Rib, Zn[eh]	230	237	253	247	9

[a]Performance and serum means are for 32 pigs per treatment and organ means are for 16 pigs per treatment.
[b]Prior to the addition of the Zn sources.
[c,d]Zinc effect ($P < .01$ and .05 respectively).
[e,f]Lysine effect ($P < .05$ and .01 respectively).
[g]Zinc x lysine effect ($P < .01$).
[h]Tissue Zn concentrations are in mg/kg of DM. Means are adjusted using ADFI during week 4 as a covariate.

than pigs fed the 1.10% lysine diet supplemented with the Zn-lysine.

The serum Zn concentrations were higher ($P < .05$) after wk 4 for pigs fed the Zn-lysine diets compared with pigs fed the $ZnSO_4$ diet (Table 3). The serum Zn concentrations of pigs fed the low lysine basal diets were higher ($P < .01$) than the serum Zn concentrations of pigs fed the 1.10% lysine diets.

The Zn concentrations of the livers of pigs fed the Zn-lysine diets were higher ($P < .01$) than those of pigs fed the $ZnSO_4$ diets (Table 3). Lysine levels had no effect on the Zn concentration of the livers. The Zn sources and lysine levels had no effect on the iron and copper concentrations of the livers (459 mg/kg of DM, SEM 38 and 31 mg/kg of DM, SEM 4, respectively).

The Zn concentrations of the rib bones were higher ($P < .05$) for pigs fed the low lysine diets compared with the pigs fed the 1.10% lysine diets (Table 3). Zinc source had no effect on the Zn concentrations of the ribs.

The Zn concentration of the kidneys were not different between the sources of Zn and levels of dietary lysine (Table 3). The kidneys of pigs fed the Zn-lysine were lighter relative to BW than those of pigs fed the $ZnSO_4$ (.51 vs. .55%). The copper concentrations of the kidneys of pigs fed the $ZnSO_4$ were higher ($P < .05$) than the levels of the

pigs fed the Zn-lysine (75 vs. 65 mg/kg of DM). The Fe concentrations of the kidneys were not different across treatments (144 mg/kg of DM, SEM 6).

Trial 3. The performance effects in Trial 3 were due to the low lysine diets. Pigs fed the low lysine diets with supplemental Zn had lower ($P < .01$) overall ADG and gain to feed ratios than pigs fed the diets with the two higher levels of lysine (Table 4). Feeding the low Zn levels did not reduce performance compared with pigs fed the diets supplemented with 100 mg Zn/kg of diet. There were no treatment effects on feed intake.

The pigs receiving the diets with no supplemental Zn had lower ($P < .01$) serum Zn concentrations at 2 wk and 4 wk compared with the diets with added Zn and similar lysine concentrations (Table 4). Also, the serum Zn concentrations of pigs fed the diets with .80% lysine and supplemental Zn were lower ($P < .01$) at wk 4 than those of pigs fed the diets with the higher lysine levels with supplemental Zn. There were no serum Zn concentration differences between the Zn sources.

Pigs fed the diets without Zn supplementation had lower ($P < .01$) liver, kidney, and rib Zn concentrations than pigs fed similar lysine levels with Zn supplementation. There were no Zn source effects on the Zn concentrations of the

Table 4. Performance and tissue zinc concentrations of weanling pigs fed diets with several levels of lysine and different Zn sources. Trial 3[a]

	Treatments							
		$ZnSO_4$	Zn-Lys	$ZnSO_4$	Zn-Lys		$ZnSO_4$	Zn-Lys	
Zn, mg/kg	0	100	100	100	100	0	100	100	
Lysine, %	.80	.80	.80	.95	.95	1.10	1.10	1.10	SEM
ADG, kg wk 1-4[b]	.24	.22	.21	.28	.28	.28	.26	.27	.02
ADFI, kg wk 1-4	.47	.45	.41	.45	.48	.46	.42	.44	.03
Gain/feed wk 1-4[b]	.50	.50	.51	.62	.59	.62	.61	.62	.02
Serum Zn, mg/L									
Initial	.47	.49	.45	.47	.48	.45	.47	.46	.02
Week 2[d]	.36	.61	.64	.62	.66	.31	.60	.62	.03
Week 4[bd]	.38	.67	.61	.73	.75	.30	.73	.77	.04
Tissue Zn concentrations (mg/kg DM)									
Liver[d]	100	160	142	143	151	92	147	167	13
Kidney[cd]	95	125	118	113	114	92	109	112	5
Rib[d]	107	179	172	158	176	89	162	160	7

[a]Peformance and serum means are for 18 pigs per treatment. Tissue means are for 6 pigs per treatement. Initial BW 7.7 kg, final BW 14.9 kg.

[b,c]Contrast: .80% lysine diets with added Zn differ from .95 and 1.1% lysine diets with zinc ($P < .01$ and .05 respectively).

[d]Contrast: low zinc diets differ from diets with adequate zinc and similar lysine levels ($P < .01$).

livers. Similarly, there were no Zn source effects on the Zn concentrations of the rib bones.

Feeding the diets with .80% lysine supplemented with Zn resulted in higher ($P < .05$) kidney Zn concentrations than feeding the diets with the higher levels of lysine with Zn supplementation. Similarly, kidney Cu concentrations were higher ($P < .01$) in pigs fed the .80% lysine diet with Zn compared with pigs fed the diets with higher lysine and Zn supplementation (39 vs. 32 mg/kg of DM).

The Zn x lysine interaction effect showed a higher copper concentration in the livers of pigs fed $ZnSO_4$ with the two low levels of lysine (30 vs. 23 mg/kg DM) compared with pigs fed the Zn-lysine ($P < .01$). But, $ZnSO_4$ fed pigs had lower liver copper concentrations (24 vs. 39 mg/kg of DM) when the high lysine diets were fed compared with pigs fed the Zn-lysine diets ($P < .01$). The pigs fed the low Zn diets also had lower ($P < .01$) kidney copper concentrations than pigs fed the diets with similar lysine levels and Zn supplementation (low lysine: 22 vs. 39 mg/kg DM and high lysine: 16 vs. 32 mg/kg DM). Liver and kidney iron concentrations were not affected by the treatments (406 mg/kg DM, SEM 52 and 118 mg/kg DM respectively).

Discussion

The performance of the pigs in Trials 1 and 2 were lower than from other trials where 2,000 mg Zn/kg of diet

were fed to weanling pigs (Chapter V). This can be attributed to the deficient level of salt in the diets used in this study. The performance seen here is similar to the performance reported by Kornegay et al. (1991) who fed low salt diets. Honeyfield and Froseth (1985) fed 4 wk old pigs similar levels of Na and Cl and reported almost identical performance levels to those seen in our study. The sodium in the Zn-lysine may have contributed to the higher performance seen by the pigs fed the Zn-lysine diets.

The reduction in performance of the pigs fed the low lysine diets in Trial 3 is consistent with the results of other research that found .80% lysine insufficient for supporting maximal growth (Asche et al., 1985).

It does not appear that the low salt levels of the diets in Trials 1 and 2 affected the absorption of Zn. The serum and tissue Zn concentrations of pigs in these trials are higher than the the concentrations of pigs fed the same Zn sources at 2,000 mg of Zn per kg of diet in Chapter V. The feeding of the high Zn in this trial for 4 wk compared with 2 wk in the previous trial may account for the higher tissue concentrations here. If the low salt diet were to have affected Zn uptake, then tissue Zn concentrations would probably have been closer to the levels of the 2 wk trial or perhaps even lower. Additionally, Partridge (1987) reported no effects on Zn absorption in pigs fed a variety of salt

levels. Similarly, Kornegay et al. (1991) reported that bone mineralization in growing pigs was not affected by feeding Na deficient diets.

In the present study, the feeding of 2,000 mg/kg of Zn for 4 wk produced differences in tissue Zn concentrations, whereas the feeding of 3,000 mg/kg of Zn for 4 wk in Chapter V (Trials 4 and 5) did not produce differences in tissue Zn concentrations between pigs fed different Zn sources. This may be a result of feeding a lower dietary Zn concentration here (2,000 vs. 3,000 mg Zn/kg) or perhaps the low lysine diets in conjunction with the lower performance may have resulted in the accumulation of Zn to the point were differences were obtained in this trial.

The liver, kidney and rib Zn concentrations of pigs fed the diets with supplemental Zn in Trial 3 are similar to the concentrations of pigs fed control diets in Chapter V (livers: 152 vs. 143 mg/kg DM, kidneys 115 vs. 113 mg/kg DM, ribs: 168 vs. 159 mg/kg DM, present study and Chapter V, respectively).

Reduced tissue Zn concentrations for pigs fed the diets with the low levels of Zn agrees with reports by Hill et al. (1986) and Swinkels (1992) who showed reduced Zn concentrations in the serum, livers, kidneys and bones of pigs fed Zn deficient diets.

The higher concentration of Zn in the serum and livers of pigs fed the Zn-lysine diets in Trials 1 and 2 suggest a higher availability of the Zn from Zn-lysine compared with $ZnSO_4$. Hahn and Baker (1993) also reported higher plasma Zn concentrations in pigs fed Zn-lysine compared with pigs fed $ZnSO_4$. However, in Trial 3 there were no differences in serum or tissue Zn concentrations between Zn sources. Similarly, feeding 2,000 mg of Zn per kg of diet in Chapter V did not result in differences between Zn sources. This discrepancy may be due to several things. First, Trials 1 and 2 were designed to afford the Zn-lysine pigs an advantage in Zn absorption. The initial hypothesis was that in a lysine deficient situation the pigs fed the Zn-lysine would accumulate more Zn because of the increase in lysine demand. Our results clearly show an increase in Zn absorption in pigs fed the Zn-lysine diet with the reduced lysine basal diet compared with the pigs fed $ZnSO_4$ with the low lysine basal diet. However, the pigs fed the Zn-lysine with the low lysine did not have higher hepatic Zn concentrations than pigs fed either Zn source with the 1.10% lysine diet. Therefore, the difference in Zn accumulation can not be separated from the differences in lysine content of the diets. All of the pigs receiving the adequate lysine levels had similar Zn concentrations. The Zn source effect seen in these trials appears to be due primarily to the

reduced Zn concentrations of pigs fed the combination of low lysine and $ZnSO_4$, rather than an independent increased availability of Zn from Zn-lysine.

Secondly, although there is no evidence to suggest that the low salt levels of the diets in these trials influenced the Zn status of the pigs, a salt effect can not be ruled out. To the authors knowledge, no one has investigated the specific relationship between Zn absorption and dietary salt concentration.

Because there were no differences between the pigs fed the .65% lysine diet with the Zn-lysine and the pigs fed the Zn-lysine with the 1.10% lysine, it appears that the Zn in Zn-lysine is not transported in conjunction with the lysine. If this were the case, the increased demand for lysine should have led to an increase in Zn-lysine absorption in the pigs fed the low lysine diet compared with the pigs fed the Zn-lysine and 1.10% lysine. If the lysine and Zn were transported intact, the Zn-lysine fed pigs would have had a higher accumulation of Zn than the pigs fed the $ZnSO_4$ with the adequate lysine. However, the pigs fed these two diets had similar Zn concentrations in the liver suggesting that the Zn in Zn-lysine is not co-transported with the lysine.

Interestingly, reducing the dietary lysine content from 1.10% to .80% in Trial 3 did not reduce the accumulation of liver Zn concentrations as in Trials 1 and 2 where the

lysine level was reduced from 1.10% to .65%. The liver Zn concentrations from Trial 3 showed no differences in Zn accumulation between the pigs fed the different lysine levels whereas, in Trials 1 and 2, feeding .65% lysine with $ZnSO_4$ resulted in a lowered Zn accumulation compared with pigs fed 1.10% lysine. This discrepancy may be due to the extremely low lysine level of the .65% lysine diet compare to all the other diets.

The tissue mineral concentrations in Trial 3 also show no difference in the availability of the two Zn sources. These results disagree with the results of Wedekind and Lewis (1993) who reported a higher availability Zn from $ZnSO_4$ compared with Zn-lysine. Additionally, Hahn and Baker (1993) reported a higher availability of Zn from Zn-lysine compared with $ZnSO_4$. However, the results of these trials do agree with our previous findings (Chapter V) that showed no clear advantage in Zn availability of either source.

In agreement with the findings reported in Chapter V, the feeding of higher levels of Zn in Trial 3 elevated the kidney copper concentrations above those of the Zn deficient pigs. As previously described (Chapter V), the feeding of Zn may be stimulating metallothionein production and causing the accumulation of copper in the kidneys (Cousins and Lee-Ambrose, 1992).

Implications

These trials indicate no differences in the availability of Zn from Zn-lysine and $ZnSO_4$. Additionally, our research does not support the hypothesis that Zn from the organic complex Zn-lysine is more available due to a co-transport of the Zn with the lysine into the enterocyte. Therefore, the use of some of the organic Zn sources to reduce the amount of Zn in swine waste may be unwarranted.

Literature cited

AOAC. 1990. Official Methods of Analysis 15th ed. Association of Official Analytical Chemists. Arlington, VA.

Asche, G. L., A. J. Lewis, E. R. Peo, Jr. and J. D. Crenshaw. 1985. The nutritional value of normal and high lysine corns for weanling and growing swine when fed at four lysine levels. J. Anim. Sci. 60:1412-1428.

Consortium. 1988. Guide for the Care and Use of Agricultural Animals in Agricultural Research and Teaching. Consortium for Developing a Guide for the Care and Use of Agricultural Animals in Agricultural Research and Teaching, Champaign, IL.

Cousins, R. J. and L. M. Lee-Ambrose. 1992. Nuclear zinc uptake and interaction and metallothionein gene expression are influenced by dietary zinc in rats. J. Nutr. 122:56-64.

Hahn, J. D. and D. H. Baker. 1993. Pharmacological zinc levels for weanling pigs: growth and plasma zinc responses. J. Nutr. 116:2171-2179.

Harp, S. L. and R. L. Hunke. 1992. Supplemental heat for swine. In: Pork Industry Handbook, #57, Purdue Univ. West Lafayette, IN.

Hill, D. A., E. R. Peo, Jr., A. J. Lewis and J. D. Crenshaw. 1986. Zinc-amino acid complexes for swine. J. Anim. Sci. 63:121-130.

Honeyfield, D. C. and J. A. Froseth. 1985. Effects of dietary sodium and chloride on growth, efficiency of feed utilization, plasma electrolytes and plasma basic amino acids in young pigs. J. Nutr. 115:1366-1371.

Kornegay, E. T., M. D. Lindemann, and H. S. Bartlett. 1991. The influence of sodium supplementation of two phosphorus sources on performance and bone mineralization of growing-finishing swine evaluated at two geographical locations. Can. J. Anim. Sci. 71:537-547.

N.R.C. 1988. Nutrient Requirements of swine. National Academy Press. Washington D.C.

Partridge, I. G. 1978. Studies on digestion and absorption in the intestines of growing pigs. 4. Effects of dietary cellulose and sodium levels on mineral absorption. Br. J. Nutr. 39:539-546.

SAS. 1990. SAS/STAT Users Guide: Statistics (Release 6.04 Ed.) SAS Inst. Inc. Cary, NC.

Swinkels, J. W. G. M. 1992. Availability of zinc from an amino acid chelate in Zn depleted pigs. Ph.D. Dissertation. Virginia Polytechnic Institute and State University, Blacksburg.

Wapnir, R. A. and L. Steil. 1986. Zinc intestinal absorption in rats: specificity of amino acids as ligands. J. Nutr. 116:2171-2179.

Wedekind, K. J. and A. J. Lewis. 1993. Assessing zinc bioavailability with pigs fed corn-soybean meal diets. Univ. Nebraska Res. Rep. p. 24-25.

Chapter VII

A comparison of zinc availability from ZnO, Zn-methionine, Zn-lysine and $ZnSO_4$ when fed to zinc deficient pigs

Abstract

This research compared the availability of Zn-lysine, Zn-methionine, ZnO and $ZnSO_4$ in Zn deficient pigs. Forty-eight crossbred weanling pigs which had been fed a Zn deficient semi-purified diet (17 mg Zn/kg) for 21 d, were randomly assigned to semi-purified diets with one of the four Zn sources added to supply 30 mg Zn/kg of diet. There were no differences in ADG, ADFI or gain/feed during the 21 d depletion or 7 d repletion periods. After 3 and 7 d of Zn repletion, serum Zn concentrations were increased but were not different between Zn sources. Liver and kidney Zn concentrations determined at the end of 7 d were also not different between Zn sources; however, the Zn concentration of the 10th ribs from pigs fed the ZnO diet were lower ($P < .05$) than those for pigs fed the Zn-lysine or the $ZnSO_4$ diets. Moreover, during a 7 d repletion of Zn deficient pigs, the availability of Zn from Zn-lysine, Zn-methionine, ZnO and $ZnSO_4$ was similar, although a slightly lower Zn concentration was obtained for the 10th rib of pigs fed ZnO.

Key words: Pigs, Zn-deficiency, Availability, bones, serum.

Introduction

The use of organic complexed mineral sources in the livestock industry has increased. It is generally thought that these organic mineral sources are more available than traditional inorganic sources. Several researchers have reported an increased availability of organic sources of Zn compared with inorganic sources in swine and poultry (Wedekind et al., 1992; Aoyagi and Baker, 1993; Hahn and Baker, 1993). Others have not found an improvement in Zn availability when organic sources were fed compared with inorganic sources (Hill et al., 1986; Wedekind and Lewis, 1993). In light of these conflicting results, there is a need to further investigate the availability of organic mineral sources for swine.

The purpose of this research was to compare the availability of Zn from two zinc-amino acid complexes and two inorganic zinc sources when fed to Zn deficient pigs.

Materials and Methods

Crossbred pigs with an average initial weight of 8.4 kg were weaned at 28 $\pm$ 2 d of age and placed 2 or 3 per pen with littermates in stainless steel cages (.91 m x 1.2 m or .61 m x 1.5 m). The pigs were fed a semipurified marginally zinc deficient diet (Table 1). The diet was calculated to contain 20% CP and 17 mg Zn/kg of diet. This diet was

Table 1. Diet composition (%)[a]

Item	%
Corn starch[b]	33.66
Dextrose[c]	30.00
Isolated soy protein[d]	20.00
Corn oil	6.00
Cellulose[e]	3.00
Dynafos[f]	2.90
Mineral mix[g]	1.66
Dyna-K[f]	1.44
$CaCO_3$	.58
Vitamin mix[h]	.40
Dynamate[f]	.36

[a]Diet calculated to contain 20% CP, 1.15% lysine, .84% Ca, .69% P and 3,570 ME/kg.
[b]Cargill Inc., Minneapolis, MN.
[c]Cerelose dextrose 2001, Corn Products, Summit-Argo, IL.
[d]PP500E, Protein Technologies International, St. Louis, MO.
[e]Purified powder cellulose BH200, International Filler Co., North Tonawanda, NY.
[f]Dynafos contained 20% Ca (min), 18.5% P, and .185% F (max); Dynamate contained 22% S (min), 18% K, and 11% Mg; Dyna-K contained 96.5% KCl (min) and 50% K. Pitman-Moore Inc., Mundelein, IL.
[g]Provided the following amounts per kilogram of diet: 50 mg of Fe as $FeSO_4 \cdot 7H_2O$, 6 mg of Cu as $CuSO_4 \cdot 5H_2O$, 4 mg of Mn as $MnSO_4 \cdot H_2O$, 140 ug of I as KIO_3, 30 mg of Se as Na_2SeO_3, and 16,300 mg/kg of dextrose was used as a carrier.
[h]Provided the following amounts per kilogram of diet: 4,950 IU of vitamin A, 660 IU vitamin D_3, 33 IU vitamin E, 6 mg vitamin K, 3.3 mg riboflavin, 35.2 mg niacin, 19.8 mg pantothenic acid, .022 mg vitamin B_{12}, 1,184 mg choline, 2.6 mg thiamin, 3 mg vitamin B_6, .4 mg biotin, 2 mg folacin, 1,200 mg D,L-methionine.

reported by Swinkels (1992) to significantly deplete the Zn stores of young pigs after a 24 or 32 d feeding period. Pigs were fed approximately 75% of the estimated ad libitum intake in three equal feedings per day and were given ad libitum access to deionized water for 21 d. On d 21, 48 pigs were randomly assigned to treatments from outcome groups based on weight, gender and litter. The treatments included 30 mg Zn/kg of diet added as: 1) feed grade ZnO (Bullard Feed Co., Bremen, IN), 2) Zn-methionine (ZinPro Corp. Inc., Edina, MN), 3) Zn-lysine (ZinPro Corp. Inc., Edina, MN) or 4) reagent grade $ZnSO_4$ (#Z76-3, Fisher Scientific, Fairlawn, NJ). The Zn sources replaced the appropriate amount of corn starch in the zinc deficient diet. Pigs were placed two (1 barrow and 1 gilt) per pen in painted steel pens (.6 m x 1.2 m) with plastic coated welded wire floors and stainless steel feeders. Pigs were given ad libitum access to feed and nipple waters for 1 wk.

Pigs were bled via jugular venipuncture at the begining of the depletion, on d 16 and d 21 of the depletion, and on d 3 and d 7 of the repletion. Blood samples were refrigerated at 4°C for 24 h and centrifuged (700 x g) for 10 min. Serum was then removed and Zn concentrations were determined using flame atomic absorption spectophotometry (Perkin Elmer 5100, Norwalk, CT). Pigs were weighed

initially, at d 21 of the depletion period and at the end of the 7 d repletion period.

At the end of 7 d repletion, the barrow from each pen was killed via electro-immobilization and exsanguination. Livers, kidneys and left 10th rib bones were removed for determination of mineral concentrations. Mineral analyses were performed using nitric/perchloric acid digestion (AOAC, 1990) followed by flame atomic absorption spectophotometry (Perkin Elmer 5100, Norwalk, CT).

The data were analyzed using the GLM procedure of SAS (1990). The models contained replicate and treatment effects. The experimental unit for the performance data was the pen of pigs, whereas, the individual pig was used as the experimental unit for the serum data and tissue data. Paired t-tests were used to determine differences between specific diets.

Results and discussion

The analyzed Zn concentrations were 12 mg/kg for the depletion diet, 43 mg/kg for the ZnO diet, 46 mg/kg for the Zn-methionine diet, 42 mg/kg for the Zn-lysine diet and 46 mg/kg for the $ZnSO_4$ diet.

Average daily gains, ADFI and gain/feed during the Zn depletion period and during the Zn repletion period were similar among all treatments (Table 2). The performance of pigs in this trial is similar to the performance of Zn

Table 2. Performance, serum Zn and tissue Zn concentrations of weanling pigs fed a Zn deficient diet followed by the same diet with 30 mg/kg of Zn added from different sources[a]

	Treatments				
	ZnO	ZnMet	ZnLys	$ZnSO_4$	SEM
Body Wt, kg					
Pre-deficiency	8.22	8.23	8.44	8.67	.14
Initial (d21)	9.78	9.79	9.73	9.82	.04
Week 1 (d28)	11.21	11.08	11.31	11.10	.19
ADG, kg					
Deficiency	.07	.07	.06	.05	.01
Repletion 7 d	.21	.18	.23	.18	.03
Overall	.11	.10	.10	.09	.01
ADFI, kg					
Repletion 7 d	.37	.42	.38	.35	.03
G/F					
Repletion 7 d	.56	.44	.58	.51	.05
Serum Zn, mg/L					
Initial	.79	.82	.75	.79	.03
Depletion d 16	.52	.51	.52	.55	.02
Depletion d 21	.35	.35	.40	.38	.02
Repletion d 3	.49	.51	.51	.47	.02
Repletion d 7	.48	.50	.49	.49	.02
Tissue Zn, mg/kg of DM					
Livers	136	133	181	151	17
Kidneys	107	100	110	104	4
Rib bones	243[b]	252	259[c]	263[c]	5

[a]Performance and serum means are for 12 pigs per treatment and tissue means are for 6 pigs per treatment.
[b,c]Using paired t-tests means with different superscripts are different ($P < .05$).

deficient pigs repleted with 15 or 45 mg/kg of Zn reported by Swinkels (1992). Swinkels (1992) also reported no differences between pigs repleted with $ZnSO_4$ or a Zn-amino acid chelate (Albion Laboratories, Cadco Inc., Des Moines, IA).

During the depletion period, serum Zn concentrations decreased (Table 2) to levels similar to those reported by Swinkels (1992); in both experiments serum Zn dropped to approximately .35 mg/L. The longer depletion period used by Swinkels (24 and 32 d) did not produce serum Zn concentrations lower than the concentrations obtained in our 21 d depletion period (.38 vs. .37 mg/L respectively).

In our study, the serum Zn concentrations increased from d 0 to d 3 of Zn repletion. Continued feeding of the repletion diets to d 7 did not result in a further increase in the serum Zn concentrations. The serum Zn concentration of .50 mg/L resulting from feeding 30 mg Zn/kg of diet for 7 d falls directly between the serum Zn concentrations obtained by Swinkels (1992) after feeding 15 and 45 mg Zn/kg of diet for 7 d (.40 and .60 mg/L, respectively).

As reported by Swinkels (1992), there were no differences in serum Zn concentrations between Zn sources. Similarly, Wedekind and Lewis (1993) reported no differences in plasma Zn concentrations when Zn deficient pigs were repleted with the same Zn sources as in this trial.

The Zn concentrations of the livers and kidneys were not different between treatments (Table 2). Swinkels (1992) also reported no difference in liver and kidney Zn concentrations in Zn depleted pigs fed either $ZnSO_4$ or a Zn-amino acid chelate. The liver and kidney Zn concentrations obtained in our trial are higher than those obtained by Swinkels (1992) after a 32 d Zn depletion followed by a 6 d repletion with 15 mg Zn/kg of diet (livers: 150 vs. 76 mg/kg of DM; kidneys: 105 vs. 95 mg/kg of DM, respectively). However, our liver and kidney Zn concentrations are lower than values obtained for Zn depleted pigs repleted with 45 mg Zn/kg of diet for 6 d (Swinkels, 1991; livers: 150 vs. 188 mg/kg of DM; kidneys: 105 vs. 147 mg/kg of DM, respectively).

The 10th rib Zn concentrations of the pigs fed the ZnO were lower ($P < .05$) than the values for pigs fed the $ZnSO_4$ or the Zn-lysine (Table 2). Intermediate values were observed for pigs fed Zn-methionine. Similarly, Wedekind and Lewis (1993) also reported a lower bone Zn concentration in metacarpal and vertebrae bones from Zn depleted pigs fed ZnO compared with pigs fed $ZnSO_4$. However, Wedekind and Lewis (1993) did not find higher bone Zn concentrations for pigs fed Zn-lysine compared with pigs fed ZnO.

Hepatic Fe and Cu concentrations were not different between treatments (536 mg/kg of DM, SEM 71, and 128.5 mg/kg

of DM, SEM 33, respectively). Kidney Fe and Cu concentrations were also not affected by the treatments (132 mg/kg of DM, SEM 6, and 27 mg/kg of DM, SEM 3, respectively). Liver and kidney weights relative to BW were also not different between treatments (2.4%, SEM .1, and .52%, SEM .3, respectively).

A possible reason for not finding differences in Zn concentrations between the Zn sources in the serum, livers, and kidneys may be the Zn content of the water during the repletion period. The water was analyzed to contain 16 mg Zn/L. If the water intake is assumed to be 2.5 to 3 times the feed intake, then the pigs would have been receiving 15.6 mg of Zn in the feed and 17.3 to 18.2 mg of Zn in the water. Therefore, the pigs were obtaining slightly more Zn from their water intake than their feed intake. The Zn concentration of the water in this trial was much higher than trials reported by Hill et al (1986) using Zn deficient pigs. Hill et al. (1986) used water with 1.1 mg/L of Zn in the water of Zn deficient pigs and found no differences in Zn availability between $ZnSO_4$ or Zn-methionine when fed at 9 or 12 mg Zn/kg.

The contribution of the zinc from the water was a known factor during the repletion period. One goal of this trial was to determine if differences between sources could be obtained using tap water during the repletion phase. From

our results, it appears that the zinc contribution of the water may have reduced the possibility of obtaining differences between the dietary Zn sources in the serum, livers, and kidneys. However, as mentioned previously, our results are similar to the results of Hill et al. (1986) who used water with a much lower Zn concentration.

On the other hand, the Zn concentration of the 10th rib showed differences between sources. The rib bone may be more responsive to the dietary Zn concentrations fed during the repletion of Zn deficient pigs; however, pigs in previous trials were not responsive when high dietary levels (100, 1,000, 2,000, and 3,000 mg Zn/kg) of Zn were fed (Chapters V and VI). Hill et al. (1986) and Wedekind and Lewis (1993) have suggested that bone is sensitive to dietary Zn sources, and is therefore a good indicator of Zn status when low levels of Zn are fed.

Implications

This trial does not support the hypothesis that Zn from Zn-lysine or Zn-methionine is more available to weanling pigs than traditional inorganic Zn sources. Zinc repletion of Zn deficient pigs with 30 mg Zn/kg of diet from Zn-lysine, Zn-methionine, $ZnSO_4$ or ZnO showed no differences in serum, liver or kidney Zn concentrations between sources. Based primarily on a slightly lower Zn concentration of rib

bones, the availability of Zn from ZnO appeared to be less than that of Zn from Zn-lysine and $ZnSO_4$.

Literature cited

AOAC. 1990. Official methods of analysis. 15th ed. Association of Official Analytical Chemists, Arlington, VA.

Aoyagi, S. and D.H. Baker. 1993. Nutritional evaluation of copper-lysine and zinc-lysine complexes for chicks. Poult. Sci. 72:165-171.

Harp, S. L., and R. L. Hunke. 1992. Supplemental heat for swine. In: Pork Industry Handbook, #57. Purdue Univ. West Lafayette. IN.

Hahn, J. D. and D. H. Baker. 1993. Pharmacologic zinc levels for weanling pigs: growth and plasma zinc responses. J. Anim. Sci. 71(Suppl. 1).

Hill, D. A., E. R. Peo, Jr., A. J. Lewis and J. D. Crenshaw. 1986. Zinc-amino acid complexes for swine. J. Anim. Sci. 63:121-130.

NRC. 1988. Nutrient requirements of swine (9th Ed.). National Academy Press, Washington DC.

SAS. 1990. SAS/STAT User's Guide: Statistics (Release 6.04 Ed.). SAS Inst. Inc., Cary, NC.

Wedekind, K. J. and A. J. Lewis. 1993. Assessing zinc bioavailability with pigs fed corn-soybean meal diets. Univ. Nebraska Res. Rep. p. 24-25.

Wedekind, K. J., A. E. Hortin, and D. H. Baker. 1992. Methodology for assessing zinc bioavailability: efficacy estimates for zinc-methionine, zinc sulfate, and zinc oxide. J. Anim. Sci. 70:178-187.

Chapter VIII

SUMMARY

Zinc supplementation

The primary objective of this research was to examine the supplementation of weanling pigs with high concentrations of Zn. The previously established relationship between growth performance and zinc status of an animal (Chesters and Quarterman, 1970) led us to examine whether weanling pigs are deficient in Zn immediately after weaning. Research has shown that serum Zn concentrations drop around weaning and that serum Zn is a good indicator of body Zn status (King, 1990). Additionally, the dietary supplementation of pigs after weaning with 3,000 mg/kg to 2,500 mg/kg has been reported to improve growth performance (Kavanagh, 1992; Poulsen, 1992). The mechanism involved in the improvement of growth is not known.

Injection trials. The initial trials were conducted to prevent the decline in serum Zn concentrations around weaning and thus improve postweaning performance. Several methods of injection (i.m. and i.p.) and multiple injection times (3 d prior to weaning, weaning, 12 h after weaning, 4 d after weaning) were unsuccessful for preventing the decline in serum Zn concentrations. An additional trial was

conducted to investigate the role of stress on serum Zn concentrations and performance. Previous research showed that serum Zn concentrations decline in pigs when stressed (Chesters and Will, 1981). No performance improvement were observed with the injection of Zn or by stressing pigs prior to weaning, but, the decline in serum Zn was prevented by Zn injection. Additionally, there was a positive correlation between serum Zn concentrations and performance, although performance was not improved above the level of controls by the injection of Zn.

Feeding Trials. In the next series of trials, the dietary Zn supplementation of weanling pigs was examined. Previous research had found improvements in scouring and performance when pigs were fed high concentrations of Zn (Kavanagh, 1992; Poulsen, 1992). In the first trial, 3,000 mg Zn/kg of diet was fed from four different Zn sources to weanling pigs for 2 wk immediately after weaning. There were no performance differences between pigs fed the high Zn diets and pigs fed a control diet containing 105 mg Zn/kg of diet. Scouring was minimal. If the mode of action for feeding high Zn concentrations and improving performance is mediated through scouring as speculated by Poulsen (1992), then the lack of scouring may have prevented the positive performance response reported by others (Kavanagh, 1992; Poulsen, 1992). The follow-up trials in which 2,000 and

1,000 mg Zn/kg of diet were fed also showed no improvements in performance above control levels.

Supplementation conclusions. The results of these trials indicate no improvement in the performance of weanling pigs supplemented with high concentrations of Zn by injection or by dietary supplementation. Additionally, preventing the drop in serum Zn concentrations after weaning does not result in a beneficial response in performance. Furthermore, it is beyond the scope of this research to determine whether feeding high concentrations of Zn during periods of severe scouring results in an improvement in performance.

Zinc Availability

The secondary objective of this research was to compare the availability of Zn from two inorganic and two organic Zn sources. Researchers have suggested an increased availability of Zn in organic zinc complexes compared with traditional inorganic sources (Hahn and Baker, 1993). However, other research has shown no difference in the availability of Zn from organic and inorganic sources (Hill et al., 1986; Wedekind and Lewis, 1993).

Feeding high levels. The approach used to investigate the availability of Zn in these trials was to feed high levels of Zn for a short period (2 wk) and compare the accumulation of Zn in tissues. Henry et al. (1987) reported

that feeding high Zn levels to chickens was an effective method of distinguishing differences in availability.

Feeding, 3,000, 2,000 and 1,000 mg of Zn from ZnO, Zn-methionine, Zn-lysine and $ZnSO_4$ showed that Zn from ZnO is less available than the other sources. Liver, kidney, and rib Zn concentrations were found to be lower than for those of pigs fed the other sources. Feeding 2,000 and 3,000 mg Zn/kg resulted in the largest tissue differences. Additionally, our results showed no difference in Zn availability from reagent grade $ZnSO_4$ and feed grade $ZnSO_4$ and that feeding 3,000 mg Zn/kg for 2 wk was sufficient to produce differences in tissue Zn accumulations between Zn sources.

Lysine x Zinc interaction. The role of lysine in the absorption of Zn from a Zn-lysine complex and from $ZnSO_4$ was investigated in the next trials. However, feeding diets ranging from a severely deficient lysine content (.65%) to an adequate lysine content (1.10%) showed little influence on Zn accumulation in tissues. Feeding pigs a diet containing .65% lysine lowered the amount of Zn deposited in the livers compared with pigs fed an adequate lysine level (1.10%). Because pigs fed the diet with .65% lysine and Zn-lysine did not show increased Zn accumulation in tissues compared with pigs fed $ZnSO_4$, this research does not support the speculation that Zn from Zn-lysine is co-transported

with the lysine component of the complex. Additionally, the trials showed no difference in serum, liver, kidney and 10th rib Zn concentrations between pigs fed $ZnSO_4$ or Zn-lysine. Therefore, there appears to be no difference in Zn availability between these two sources.

Zinc deficiency. Finally, feeding Zn deficient pigs diets supplying 30 mg Zn/kg of diet from ZnO, Zn-methionine, Zn-lysine or $ZnSO_4$ showed that 10th rib Zn concentrations were lower for pigs fed ZnO than for pigs fed the other Zn sources. There were no differences in serum, liver or kidney Zn concentrations between the different sources. Therefore, ZnO appeared to be less available than the other sources based on rib Zn concentration.

Overall conclusions

From the results of these trial, there appears to be no performance benefit to supplementing weanling pigs with high concentrations of Zn by feeding or injecting Zn. Additionally, Zn from ZnO is less available to weanling pigs than Zn from Zn-lysine, Zn-methionine or $ZnSO_4$. Furthermore, the availability of Zn from Zn-lysine, Zn-methionine and $ZnSO_4$ appears to be similar.

Literature cited

Chesters, J.K. and J. Quarterman. 1970. Effects of zinc deficiency on food intake and feeding patterns of rats. Br. J. Nutr. 24:1061.

Chesters, J. K. and M. Will. 1981. Measurement of zinc flux through plsama in normal and encotoxin-stressed

pigs and the effects of Zn supplementation. Br. J. Nutr. 46:119-130.

Hahn, J. D. and D. H. Baker. 1993. Pharmacologic zinc levels for weanling pigs: growth and plasma zinc responses. J. Anim. Sci. 71(Suppl. 1):66.

Henry,P. R., C. B. Ammerman, and R. D. Miles. 1987. Effect of dietary zinc on tissue mineral concentration as a measure of zinc bioavalability in chicks. Nutr. Rep. Int. 35:15-23.

Hill, D. A., E. R. Peo, Jr., A. J. Lewis and J.D. Crenshaw. 1986. Zinc-amino acid complexes for swine. J. Anim. Sci. 63:121-130.

Kavanagh, N. T. 1992. The effect of feed supplemented with zinc oxide on the performance of recently weaned pigs. Proceedings: International Pig Veterinary Meetings 1992. p. 616.

King, J. C. 1990. Assement of zinc status. J. Nutr. 120:1474.

Poulsen, H. D. 1992. Zinc oxide for weaned pigs. Eleventh annual Prince feed ingredient conference. Dublin. Appendix 1.

Wedekind, K. J. and A. J. Lewis. 1993. Assessing zinc bioavailability with pigs fed corn-soybean meal diets. University of Nebraska Research Report. pp. 24-25

CHAPTER IX

APPENDIX

Appendix Table 1. Performance of pigs injected with Zn acetate 3 d prior to weaning.

Item	Treatment[a]		SEM	P-value
	Control	Zinc[b]		
BW, kg				
Preweaning (3d)	5.2	5.2	.03	.52
Weaning	5.9	6.0	.04	.74
Post-weaning (2d)	5.6	5.6	.04	.35
Week 1	6.2	6.3	.10	.54
Week 2	8.1	8.2	.16	.63
Week 3	10.7	11.1	.19	.26
ADG, kg				
Preweaning	.26	.25	.01	.67
Post-weaning (2d)	-.19	-.17	.01	.20
Week 1 (d2-d7)	.12	.13	.02	.81
Week 2	.28	.28	.01	.88
Week 3	.37	.40	.02	.30
Weaning -wk 1	.03	.04	.01	.62
Weaning -wk 2	.16	.16	.01	.69
Weaning -wk 3	.23	.24	.01	.28
ADFI, kg				
Postweaning (2d)	.01	.02	.003	.16
Week 1 (d2-d7)	.20	.21	.02	.74
Week 2	.44	.46	.02	.46
Week 3	.67	.66	.02	.68
Weaning- wk 1	.15	.16	.01	.67
Weaning- wk 2	.16	.16	.01	.47
Weaning- wk 3	.42	.42	.01	.71

[a]Thirty-six pigs per treatment.
[b]Zinc acetate injected i.m. (3 mg/kg of BW) 3 d prior to weaning. Controls injected with an equal volume of saline.

Appendix Table 2. Serum Zn concentrations of pigs injected with one of two concentrations of Zn acetate.

	Treatment[a]		
	3 mg/kg	4 mg/kg	SEM
Body weight, kg	20.6	19.1	1.0
Serum Zn, mg/L			
Initial	.68	.69	.06
6 h[b]	.91	1.49	.14
12 h[b]	.78	1.12	.06
24 h[b]	.68	.86	.04
36 h	.83	.83	.03
48 h	.68	.75	.04
72 h	.62	.75	.04

[a]Six pigs per treatment mean.
[b]Treatments differ ($P < .01$).
[c]Treatments differ ($P < .05$).

Appendix Table 3. Body weights and ADG of pigs injected with Zn acetate at weaning or 12 h postweaning.

Time[a]	Treatment						
	0 h			12 h			
Zn,[b] mg/kg BW	0	3	4	0	3	4	SEM
Body wt, kg							
Initial	7.2	7.3	7.1	7.3	7.2	7.3	1.0
Day 1	6.9	7.0	6.8	6.9	6.9	7.0	1.0
Day 3	7.3	7.4	7.2	7.5	7.4	7.3	1.1
Week 1	7.6	7.7	7.6	7.9	7.8	7.7	1.2
Week 2	9.1	9.4	8.9	9.7	9.5	9.2	1.6
Week 3	11.5	12.1	11.7	12.5	12.4	11.6	2.6
Week 4	14.0	14.8	14.7	15.2	15.0	14.4	2.8
ADG, kg							
Day 1	-.27	-.31	-.35	-.39	-.27	-.34	.03
D 2 to d 3	.11	.14	.14	.19	.15	.11	.03
D 4 to d 7	.12	.11	.14	.16	.15	.11	.03
Week 2	.21	.24	.20	.25	.24	.22	.03
Week 3	.34	.40	.39	.40	.41	.35	.04
Week 4	.41	.46	.50	.46	.44	.47	.05
D 1 to d 7	.06	.06	.07	.10	.09	.06	.02
D 1 to wk 2	.13	.16	.14	.17	.17	.13	.02
D 1 to wk 3	.20	.24	.22	.25	.25	.20	.02
D 1 to wk 4	.25	.29	.28	.30	.29	.26	.02

[a]Injection given postweaning. There were 14 pigs per treatment mean.
[b]Pigs were given 3 or 4 mg of Zn per kg of BW i.p. in the form of Zn acetate or saline in an equivalent volume to the 3 mg Zn injection.

Appendix Table 4. Average daily feed intake and feed efficiency of pigs injected with Zn acetate at weaning or 12 h postweaning.

Time	Treatment[a]						
	0 h			12 h			
Zn, mg/kg BW	0	3	4	0	3	4	SEM
ADFI, kg							
Day 1	.01	.00	.02	.00	.00	.00	.01
D 2 to d 3	.22	.32	.26	.41	.35	.30	.02
D 4 to d 7	.40	.42	.43	.48	.48	.44	.03
Week 2	.73	.72	.68	.75	.77	.67	.04
Week 3	1.05	1.19	1.16	1.23	1.19	1.07	.08
Week 4	1.44	1.48	1.59	1.67	1.60	1.52	.11
D 1 to d 7	.27	.32	.31	.39	.36	.32	.02
D 1 to wk 2	.50	.52	.49	.57	.56	.49	.03
D 1 to wk 3	.67	.72	.58	.71	.88	.66	.09
D 1 to wk 4	.85	.91	.91	.98	.96	.87	.05
Gain/feed							
D 2 to d 3	.21	.41	.40	.43	.44	.22	.17
D 4 to d 7	.29	.26	.28	.36	.31	.20	.07
Week 2	.28	.33	.31	.33	.30	.32	.04
Week 3	.38	.34	.36	.33	.33	.32	.04
Week 4	.29	.32	.33	.27	.29	.31	.03
D 1 to d 7	.15	.18	.18	.23	.25	.07	.08
D 1 to wk 2	.25	.31	.27	.30	.29	.23	.04
D 1 to wk 3	.31	.32	.32	.31	.31	.28	.03
D 1 to wk 4	.31	.32	.32	.30	.30	.30	.03

[a]Injection given postweaning. There were 14 pigs per treatment mean. Pigs were given 3 or 4 mg of Zn per kg of BW i.p. in the form of Zn acetate or saline in an equivalent volume to the 3 mg Zn injection.

Appendix Table 5. Scour scores of pigs injected with Zn acetate at weaning or 12 h postweaning.

Time	Treatment[a]						
	0 h			12 h			
Zn	0	3	4	0	3	4	SEM
Day[b]							
1	2.3	2.1	2.0	2.5	2.1	2.1	.4
2	2.1	2.1	2.1	2.3	2.0	2.3	.5
3	2.4	2.0	2.3	2.3	2.4	2.4	.7
4	3.1	2.3	2.4	2.5	2.8	3.0	1.2
5	2.8	2.3	2.8	2.6	3.0	2.9	1.2
6	3.1	2.4	3.1	3.1	2.1	3.1	1.2
7	3.1	2.4	2.8	3.0	2.5	2.6	1.1
8	3.4	3.1	3.1	3.5	3.3	3.5	1.3
9	3.1	2.9	2.9	3.1	3.0	2.9	1.1
10	3.0	2.6	2.8	2.6	2.9	2.4	1.1
11	3.0	2.6	2.8	2.3	2.9	2.9	1.1
12	2.8	2.6	2.9	2.5	2.6	2.8	.8
13	3.0	2.3	2.5	2.6	2.8	2.5	1.1
14	3.3	2.5	2.9	2.8	3.6	2.9	1.1
15	3.1	2.5	2.6	2.9	3.4	3.1	1.0
16	2.5	2.8	3.0	2.4	3.4	2.8	1.0
17	2.5	2.6	2.6	2.1	2.8	2.4	.9
18	2.6	2.4	2.8	2.0	2.4	2.5	.8
19	2.1	2.3	2.8	2.1	2.3	2.1	.7
20	2.1	2.0	2.5	2.1	2.8	2.0	.2
21	2.0	2.1	2.3	2.1	2.6	2.1	.5

[a]Injection given postweaning. There were 14 pigs per treatment mean, 8 pens (2 pigs per pen) per treatment. Pigs were given 3 or 4 mg of Zn per kg of BW i.p. in the form of Zn acetate or saline in an equivalent volume to the 3 mg Zn injection.

[b]Scoring: 1 - dry feces
2 - normal feces
3 - slightly watery feces
4 - very watery feces
5 - extremely watery feces

Appendix Table 6. Performance of weanling pigs injected with 3 mg/kg of BW of Zn as Zn acetate on d 3 postweaning.

	Treatment[a]		
	Saline	Zinc	SEM
Body weight, kg			
Initial	6.9	7.0	.02
Day 4[b]	7.2	7.0	.02
Week 1[b]	7.4	7.1	.06
Week 2	8.5	7.8	.21
Week 3	10.5	9.5	.30
Week 4	14.1	12.5	.55
ADG, kg			
Day 4	.04	.03	.01
D 4 to d 7[b]	.12	.01	.02
Week 2	.16	.10	.03
Week 3	.29	.24	.05
Week 4	.50	.43	.05
Week 1-2	.11	.06	.01
Week 1-3	.17	.12	.01
Week 1-4	.26	.20	.02
ADFI, kg			
Day 4	.12	.13	.01
D 4 to d 7[b]	.19	.12	.01
Week 2	.28	.20	.03
Week 3	.50	.39	.04
Week 4	.82	.72	.07
Week 1-2	.21	.15	.02
Week 1-3	.31	.23	.02
Week 1-4	.44	.35	.03
Gain/feed			
Day 4	.22	.16	.07
D 4 to d 7[b]	.72	.02	.07
Week 2	.55	.54	.07
Week 3	.57	.59	.08
Week 4	.61	.60	.03
Week 1-2	.56	.42	.04
Week 1-3	.57	.52	.03
Week 1-4	.59	.56	.02

[a]Zinc injected i.p. on d 4 after weaning. Fifteen pigs per treatment mean.
[b]Treatment effect ($P < .05$). Weight at d 4 used as a covariate for all variables other than d 4 weight.

Appendix Table 7. Performance of weanling pigs regrouped or not regrouped 3 d prior to weaning and injected or not injected with 3 mg/kg BW of Zn at one of two times.

	Treatment[a]								
	Not regrouped				Regrouped				
Zn, mg/kg BW	0		3		0		3		
Day	21	24	21	24	21	24	21	24	SEM
BW, kg									
Initial	6.5	6.4	6.2	6.4	6.4	6.4	6.3	6.0	.3
Weaning	7.3	7.3	6.8	7.2	7.0	6.9	6.9	6.5	.4
Week 1	7.7	7.4	7.3	7.7	7.7	7.4	7.3	7.0	.4
Week 2	9.6	9.5	9.2	9.3	9.6	9.4	9.1	9.0	.6
Week 3	13.0	12.6	12.3	13.4	12.9	12.5	12.1	12.0	1.0
ADG, kg									
Pre[b,c]	.26	.28	.21	.26	.22	.18	.21	.18	.05
Week 1	.06	.02	.08	.08	.10	.07	.05	.06	.02
Week 2	.27	.29	.27	.22	.28	.28	.25	.29	.04
Week 3	.49	.45	.44	.59	.47	.45	.44	.42	.06
Pre-wk 1	.12	.10	.11	.13	.13	.10	.10	.10	.02
Pre-wk 2	.18	.18	.18	.17	.19	.18	.16	.18	.02
Pre-wk 3	.27	.26	.26	.29	.27	.26	.24	.25	.03
ADFI, kg									
Week 1	.18	.17	.18	.20	.20	.18	.16	.17	.01
Week 2[d]	.48	.47	.46	.49	.48	.44	.44	.45	.04
Week 3	.93	.76	.72	.80	.78	.76	.71	.71	.08
Week 1-2	.33	.32	.32	.35	.34	.31	.30	.31	.03
Week 1-3	.53	.47	.45	.50	.48	.46	.44	.44	.04
Gain/feed									
Week 1	.34	.12	.42	.38	.49	.37	.32	.34	.08
Week 2	.57	.63	.60	.43	.58	.63	.58	.63	.07
Week 3	.55	.59	.61	.71	.61	.60	.62	.60	.06
Week 1-2	.51	.50	.55	.42	.55	.56	.51	.56	.05
Week 1-3	.53	.55	.58	.57	.58	.58	.57	.58	.04

[a]Twelve pigs per treatment mean for pigs that were not regrouped and 14 pigs per mean for regrouped pigs except 13 pigs injected with Zn on d 21 and regrouped (one died).
[b]Pre=preweaning.
[c,d]Zn x group effect ($P < .05$ and .10, respectively).

Appendix Table 8. Performance of weanling pigs fed diets with 3,000 mg Zn/kg of diet added from different sources[a]

	Treatments					
	Control	ZnO	ZnMet	ZnLys	$ZnSO_4$	SEM
Body Wt, kg						
Initial[b]	7.49	7.51	7.53	7.51	7.52	.07
Week 1	7.66	7.78	7.32	7.39	7.46	.14
Week 2	9.04	9.64	8.52	9.12	8.85	.30
Final[b]	9.62	10.35	8.81	9.75	9.38	.37
ADG						
Week 1[c]	.02	.04	-.03	-.02	.01	.02
Week 2[b]	.20	.27	.17	.25	.20	.03
Final 2 d	.29	.35	.15	.31	.27	.08
Wk 1-2[b]	.11	.15	.07	.12	.09	.02
Wk 1-final[d]	.13	.18	.08	.14	.12	.02
ADFI						
Week 1[c]	.22	.26	.15	.18	.18	.02
Week 2[b]	.61	.79	.64	.62	.58	.06
Final 2 d	.93	1.13	1.01	1.08	.87	.09
Wk 1-2[d]	.42	.53	.39	.40	.38	.04
Wk 1 -final[b]	.48	.60	.47	.48	.44	.04
G/F[e]						
Week 1[c]	.05(.09)	.11(.15)	-.57(.20)	-.15(.11)	-.15(.06)	.13
Week 2[c]	.32(.33)	.35(.34)	-.64(.27)	.41(.40)	.34(.34)	.17
Final 2 d[d]	.34(.31)	.34(.31)	-.18(.15)	.31(.29)	.32(.31)	.13
Wk 1-2[c]	.25(.26)	.29(.28)	-.43(.18)	.29(.30)	.24(.24)	.13
Wk 1-Final[c]	.27(.27)	.30(.30)	-.21(.17)	.29(.29)	.26(.27)	.10

[a]Means are for 16 pigs per treatment.
[b,c,d]Treatment effect ($P < .10$, .01 and .05 respectively).
[e]G/F values in parenthesis are calculated values not LSM's.

Appendix Table 9. Serum zinc and iron concentrations of weanling pigs fed 3,000 mg Zn /kg of diet from different sources[a]

	Treatments					
	Control	ZnO	ZnMet	ZnLys	$ZnSO_4$	SEM
Zn, ppm						
Initial	.58	.53	.58	.56	.57	.02
Week 1[b]	.48	.65	.58	.72	.73	.04
Week 2[b]	.49	.97	1.04	1.06	1.12	.10
Final[b]	.46	1.05	1.07	1.18	1.44	.12
Fe, ppm						
Initial	1.10	1.20	1.14	.85	1.10	.22
Week 1	1.43	1.73	1.89	1.62	1.92	.17
Week 2	1.94	1.69	1.70	1.85	1.66	.15
Final	1.76	1.79	1.86	1.76	1.71	.13

[a]Means are for 16 pigs per treatment.
[b]Contrast: control diet vs. high Zn diets ($P < .01$).

Appendix Table 10. Mineral concentrations of the livers, kidneys, muscles and bones of weanling pigs fed diets with 3,000 mg Zn/kg of diet from different sources[a]

	Treatments					
	Control	ZnO	ZnMet	ZnLys	$ZnSO_4$	SEM
Liver wt, %[bc]	2.9	2.6	2.5	2.4	2.3	.1
Kidney wt, %[b]	.50	.53	.53	.50	.48	.02
Livers (mg/kg of DM)						
Zn[d]	155	1224[e]	1067[eg]	1721[h]	1883[f]	191
Fe	492	318	411	409	405	71
Cu	44	39	52	58	50	12
Kidneys (mg/kg of DM)						
Zn[d]	109	186	168	203	235	25
Fe[c]	165	128	135	126	156	13
Cu[d]	20	34[ei]	32[ei]	55[j]	52[f]	5
Muscles[k] (mg/kg of DM)						
Zn[c]	119	97	113	99	105	6
Rib bones[l] (mg/kg of DM)						
Zn[d]	131[f]	193[fg]	199[g]	212[g]	219[g]	15

[a]Means are for 8 pigs per treatment.
[b]Weights are a percentage of live body wt.
[c,d]Contrast: control diet vs. high Zn diets ($P < .05$, and .01, respectively).
[e-j]Using paired t-tests means with different superscripts are different [e,f]($P < .05$), [g,h]($P < .05$), [i,j]($P < .01$).
[k]Left external carpi radialis muscle.
[l]Left 10th rib bone.

Appendix Table 11. Performance of weanling pigs fed diets with 2,000 mg Zn/kg of diet added from different sources[a]

	Treatments					
	Control	ZnO	ZnMet	ZnLys	$ZnSO_4$	SEM
Body Wt, kg						
Initial	7.07	7.09	7.04	7.16	7.12	.04
Week 1[b]	7.52	7.11[d]	7.00[d]	7.11[d]	7.61[e]	.11
Week 2	9.21	9.24	8.81[df]	9.30[g]	9.62[e]	.16
ADG						
Week 1[b]	.06	.01	-.01	-.01	.07	.02
Week 2[c]	.24	.30	.26	.31	.29	.02
Wk 1-2	.15	.15	.13[d]	.15	.18[e]	.01
ADFI						
Week 1[b]	.19	.14	.11[d]	.13[f]	.17[eg]	.01
Week 2	.44	.43[f]	.43[f]	.45	.51[g]	.03
Wk 1-2	.31	.29[f]	.27[f]	.29[f]	.34[g]	.02
G/F						
Week 1	.30	-.01[f]	-.11[f]	-.13[f]	.42[g]	.14
Week 2	.55	.70	.61	.72[f]	.57[g]	.05
Wk 1-2	.49	.53	.47	.54	.54	.03

[a]Means are for 16 pigs per treatment.
[b,c]Contrast: control diet vs. high Zn diets ($P < .01$ and .05, respectively).
[d-g]Using paired t-tests means with different superscripts are different [d,e]($P < .01$), and [f,g]($P < .05$).

Appendix Table 12. Serum, liver, kidney, and bone mineral concentrations of weanling pigs fed diets with 2,000 mg Zn/kg of diet from different sources[a]

	Treatments					
	Control	ZnO	ZnMet	ZnLys	$ZnSO_4$	SEM
Serum Zn, mg/l						
Initial	.72	.64	.67	.71	.71	.03
Week 1[b]	.59	.65[c]	.69[c]	.74[c]	.94[d]	.03
Week 2[b]	.65	.95[ce]	1.31[f]	1.16[c]	1.49[d]	.07
Liver wt, %[i]	2.9	2.7	2.6	2.5	2.6	.1
Kidney wt, %[i]	.48	.50	.47	.49	.50	.02
Livers (mg/kg of DM)						
Zn[b]	160	347[c]	747[g]	636[c]	1007[dh]	73
Fe	409	479	294	416	365	59
Cu	77	87	71[g]	92[h]	71[g]	7
Kidneys (mg/kg of DM)						
Zn[b]	103	129[cg]	157[ch]	143[c]	196[d]	8
Fe	117	135	114	121	111	11
Cu[b]	21	38	47	49	49	6
Rib bones[j] (mg/kg of DM)						
Zn[b]	178	211[g]	236	214[g]	245[h]	9

[a]Serum means are for 16 pigs per treatment, and tissue means are for 8 pigs per treatment.
[b]Contrast: control diet vs. high Zn diets ($P < .01$).
[c-h]Using paired t-tests means with different superscripts are different, [c,d]($P < .01$), [e,f]($P < .01$), and [g,h]($P < .05$).
[i]Weights are a percentage of live body wt.
[j]Left 10th rib bone.

Appendix Table 13. Performance of weanling pigs fed diets with 1,000 mg Zn/kg of diet added from different sources[a]

	Treatments					
	Control	ZnO	ZnMet	ZnLys	$ZnSO_4$	SEM
Body Wt, kg						
Initial	5.29	5.28	5.30	5.35	5.35	.06
Week 1	5.71	5.51	5.57	5.75	5.66	.10
Week 2	7.37	7.16	7.26	7.63	7.46	.16
ADG						
Week 1	.06	.03	.04	.06	.04	.01
Week 2	.24	.24	.24	.27	.26	.01
Wk 1-2	.15	.13	.14	.16	.15	.01
ADFI						
Week 1	.15	.14	.13	.14	.14	.01
Week 2	.38	.36	.37	.38	.34	.02
Wk 1-2	.26	.25	.25	.26	.24	.01
G/F						
Week 1	.39	.12[c]	.28	.37[d]	.28	.08
Week 2[b]	.62	.65[c]	.64[c]	.70	.76[d]	.03
Wk 1-2	.56	.52[ce]	.55[g]	.61[d]	.63[fh]	.02

[a]Means are for 14 pigs per treatment.
[b]Contrast: control diet vs. high Zn diets ($P < .05$).
[c-h]Using paired t-tests means with different superscripts are different [c,d]($P < .05$), [e,f]($P < .01$) and [g,h]($P < .05$).

Appendix Table 14. Serum, liver, kidney, and bone mineral concentrations of weanling pigs fed diets with 1,000 mg Zn/kg of diet from different sources[a]

	Treatments					
	Control	ZnO	ZnMet	ZnLys	$ZnSO_4$	SEM
Serum Zn, mg/l						
Initial	.82	.83	.86	.89	.82	.03
Week 1[b]	.61	.67	.75	.69	.71	.03
Week 2[b]	.65	.78	.80	.78	.82	.03
Liver wt, %[h]	3.1	2.9	3.0	2.8	2.8	.1
Kidney wt, %[h]	.56	.60	.60	.60	.59	.03
Livers (mg/kg of DM)						
Zn[c]	115	153	136	135	149	13
Fe	649	600	745	569	586	81
Cu	67	56	38[d]	47[f]	73[eg]	10
Kidneys (mg/kg of DM)						
Zn	127	127	130	120	120	6
Fe	246	264	231	226	227	27
Cu	26	35	37	29	29	4
Rib bones[i] (mg/kg of DM)						
Zn	168	176	184	168	187	11

[a]Serum means are for 16 pigs per treatment, and tissue means are for 8 pigs per treatment.
[b,c]Contrast: control diet vs. high Zn diets ($P < .01$ and .05, respectively).
[d-g]Using paired t-tests means with different superscripts are different, [d,e]($P < .01$), and [f,g]($P < .05$).
[h]Weights are a percentage of live body wt.
[i]Left 10th rib bone.

Appendix Table 15. Performance of weanling pigs fed three Zn sources to provide 3,000 mg Zn/kg of diet[a]

	Treatments			
	$ZnSO_4$ (reagent)	ZnO	$ZnSO_4$ (feed)	SEM
Body Wt, kg				
Initial	7.17	7.19	7.20	.39
Week 1	7.50	7.58	7.66	.42
Week 2	9.93	10.43	9.94	.53
Week 3	11.96	12.64	12.32	.71
Week 4	13.04	14.33	14.12	.72
ADG, kg				
Week 1	.05	.06	.07	.01
Week 2	.35[b]	.41[cd]	.33[e]	.02
Week 3	.31	.29	.32	.05
Week 4	.15[b]	.24[c]	.26[c]	.03
Wk 1-2	.20	.23	.20	.01
Wk 1-3	.23	.26	.25	.02
Wk 1-4	.21[b]	.26[c]	.25	.01
ADFI, kg				
Week 1	.19	.17	.18	.01
Week 2	.56	.55	.49	.03
Week 3	.90	.85	.75	.05
Week 4	.90	.85	.83	.06
Wk 1-2	.37	.36	.33	.02
Wk 1-3	.67	.64	.58	.04
Wk 1-4	.90	.85	.83	.06
Gain/feed				
Week 1	.23	.22	.35	.09
Week 2	.63[d]	.74[e]	.67[d]	.02
Week 3	.37	.36	.44	.04
Week 4	.20[bd]	.28[c]	.31[e]	.03
Wk 1-2	.53[b]	.64[c]	.59	.02
Wk 1-3	.35[bd]	.41[c]	.43[e]	.02
Wk 1-4	.30[d]	.37[e]	.39[e]	.01

[a]Means are for 24 pigs per treatment for weeks 1 and 2 and 16 pigs per treatment for weeks 3 and 4. These data are pooled from two trials.

[b-e]Using paired t-tests means with different superscripts are different [b,c]($P < .05$), [d,e]($P < .01$)

Appendix Table 16. Serum zinc concentrations of weanling pigs fed three Zn sources to provide 3,000 mg Zn/kg of diet[a]

	Treatments			
	$ZnSO_4$ (reagent)	ZnO	$ZnSO_4$ (feed)	SEM
Zn, mg/l				
Initial	.76	.73	.72	.02
Week 1	1.04	.90	1.04	.06
Week 2	2.26[b]	1.62[c]	1.93	.12
Week 3	2.97[b]	2.18[c]	2.91[b]	.13
Week 4	2.98[b]	2.20[c]	2.99[b]	.11

[a]Means are for 24 pigs per treatment for the first 2 weeks and 16 pigs per treatment for weeks 3 and 4. These data are pooled from two trials.

[b,c]Using paired t-tests means with different superscripts are different ($P < .01$).

Appendix Table 17. Mineral concentrations of the livers, kidneys, and rib bones of weanling pigs fed several Zn sources to provide 3,000 mg Zn/kg of diet[a]

	Treatments			
	$ZnSO_4$ (reagent)	ZnO	$ZnSO_4$ (feed)	SEM
Week 2				
Liver wt, %[b]	2.6	2.6	2.6	.13
Kidney wt, %[b]	.52	.50	.61	.04
Livers (mg/kg of DM)				
Zn	1,168	910[c]	1,312[d]	135
Fe	522	430	364	89
Cu	61	69	76	12
Kidneys (mg/kg of DM)				
Zn	259[c]	185[d]	258[c]	23
Fe	139	130	114	14
Cu	61	53[c]	80[d]	8
Rib bones[e] (mg/kg of DM)				
Zn	273	242	267	13
Week 4				
Liver wt, %[b]	2.7	3.1	2.8	.09
Kidney wt, %[b]	.54	.61	.57	.04
Livers (mg/kg of DM)				
Zn	1,592	1,569	1,344	176
Fe	494	370	410	55
Cu	23	26	29	4
Kidneys (mg/kg of DM)				
Zn	518	455	454	61
Fe	186	171	153	10
Cu	130	115	102	12
Rib bones[e] (mg/kg of DM)				
Zn	375	345	350	27

[a]Means are for 8 pigs per treatment. These data are pooled from two trials.
[b]Weights are a percentage of live body wt.
[c,d]Using paired t-tests means with different superscripts are different ($P < .05$).
[e]Left 10th rib bone.

Appendix Table 18. Performance of weanling pigs fed $ZnSO_4$ or Zn-lysine with a lysine deficient or lysine adequate diet[a]

	Treatments				
Basal lysine, %	1.10		.65		
Zn source	$ZnSO_4$	Zn-lysine	$ZnSO_4$	Zn-lysine	
Total lysine, %	1.10	1.55	.65	1.10	SEM
Body Wt, kg					
Initial	7.13	7.13	7.18	7.14	.15
Week 1	7.26	7.26	7.23	7.31	.12
Week 2	8.30	8.31	8.23	8.47	.14
Week 3[b]	9.47	9.64	9.34	10.04	.16
Week 4[bcf]	10.64	11.42	10.53	12.29	.19
ADG					
Week 1	.02	.02	.01	.02	.01
Week 2	.15	.15	.14	.17	.01
Week 3[b]	.17	.19	.16	.23	.01
Week 4[bdf]	.17	.25	.17	.32	.01
Wk 1-2	.08	.08	.08	.09	.01
Wk 1-3[be]	.11	.12	.10	.14	.01
Wk 1-4[bcf]	.13	.15	.12	.18	.01
ADFI, kg					
Week 1	.15	.15	.15	.17	.01
Week 2	.31	.28	.32	.30	.01
Week 3[c]	.49	.44	.50	.51	.02
Week 4[b]	.59	.65	.59	.69	.02
Wk 1-2	.23	.22	.24	.24	.01
Wk 1-3[c]	.31	.29	.32	.33	.01
Wk 1-4	.38	.38	.39	.42	.01
Gain/feed					
Week 1	.01	.01	.01	.02	.09
Week 2	.49	.53	.47	.54	.04
Week 3[b]	.35	.43	.32	.44	.02
Week 4[bc]	.28	.38	.29	.47	.02
Wk 1-2	.36	.38	.31	.39	.03
Wk 1-3[be]	.36	.41	.31	.42	.01
Wk 1-4[bf]	.33	.40	.31	.44	.01

[a]Means are for 32 pigs per treatment. These data are pooled data from two trials.
[b]Zn effect ($P < .01$).
[c,d]Lysine effect ($P < .05$ and .01 respectively).
[e,f]Zn*lysine effect ($P < .05$, and .01 respectively).

Appendix Table 19. Mineral concentrations of the livers, kidneys and rib bones of weanling pigs fed $ZnSO_4$ or Zn-lysine with a lysine deficient diet or a lysine adequate diet[a]

	Treatments				
Basal lysine, %	1.10		.65		
Zn source	$ZnSO_4$	Zn-lysine	$ZnSO_4$	Zn-lysine	
Total lysine, %	1.10	1.55	.65	1.10	SEM
Liver wt, %[b]	2.3	2.4	2.5	2.5	.05
Kidney wt, %[b]	.53	.50	.57	.52	.02
Livers (mg/kg of DM)					
Zn[c]	700	838	523	808	77
Fe	429	416	425	406	26
Cu	41	39	46	32	4
Kidneys (mg/kg of DM)					
Zn	239	217	251	232	14
Fe	151	139	138	137	6
Cu[d]	77	65	73	64	5
Rib bones[e] (mg/kg of DM)					
Zn[f]	230	237	253	247	9

[a]Means are for 16 pigs per treatment. Because of differences between treatments ADFI for wk 4 was used as a covariate in the analysis of this data to account for possible influence. These data are pooled data from two trials.
[b]Weights are a percentage of live body wt.
[c,d]Zn effect ($P < .01$ and .05, respectively).
[e]Left 10th rib bone.
[f]Lysine effect ($P < .05$).

Appendix Table 20. Performance of weanling pigs fed Zn-lysine or $ZnSO_4$ with diets containing different levels of lysine[a]

	Treatments								
Zn source		$ZnSO_4$	ZnLy	$ZnSO_4$	ZnLy		$ZnSO_4$	ZnLy	
Zn, ppm	0	100	100	100	100	0	100	100	
Lysine, %	.80	.80	.80	.95	.95	1.1	1.1	1.1	SEM
BW, kg									
Initial	7.7	7.8	7.7	7.7	7.7	7.8	7.7	7.7	.05
Week 1	8.8	8.5	8.6	8.6	9.0	8.7	8.6	8.7	.14
Week 2[b]	10.3	9.9	9.9	10.4	10.5	10.5	10.3	10.3	.19
Week 3	12.2	11.6	11.5	12.1	12.5	12.6	11.9	12.1	.36
Week 4[b]	14.4	14.1	13.6	15.5	15.7	15.7	14.9	15.3	.59
ADG, kg									
Week 1[c]	.15	.09	.12	.12	.18	.14	.13	.14	.02
Week 2[c]	.21	.21	.19	.25	.22	.25	.24	.23	.02
Week 3	.27	.24	.24	.25	.29	.30	.22	.26	.04
Week 4[b]	.31	.35	.30	.48	.45	.44	.43	.44	.05
Wk 1-2[b]	.18	.15	.15	.19	.20	.20	.19	.19	.01
Wk 1-3[c]	.21	.18	.18	.21	.23	.23	.20	.21	.02
Wk 1-4[b]	.24	.22	.21	.28	.28	.28	.26	.27	.02
ADFI, kg									
Week 1	.18	.15	.13	.15	.20	.15	.16	.15	.02
Week 2	.37	.32	.33	.34	.38	.35	.34	.32	.02
Week 3	.56	.51	.47	.52	.55	.54	.42	.52	.05
Week 4	.76	.80	.69	.78	.82	.78	.74	.75	.03
Wk 1-2	.28	.24	.23	.25	.29	.25	.25	.24	.02
Wk 1-3	.37	.33	.31	.34	.37	.35	.31	.33	.02
Wk 1-4	.47	.45	.41	.45	.48	.46	.42	.44	.03
G/F									
Week 1	.84	.66	.84	.82	1.13	.95	.84	1.01	.18
Week 2[c]	.58	.64	.58	.74	.60	.73	.71	.73	.04
Week 3	.49	.49	.48	.48	.51	.55	.52	.50	.06
Week 4[b]	.34	.44	.41	.61	.55	.57	.58	.60	.07
Wk 1-2[b]	.66	.63	.67	.76	.69	.79	.75	.80	.02
Wk 1-3[b]	.57	.57	.57	.62	.61	.66	.65	.64	.03
Wk 1-4[b]	.50	.50	.51	.62	.59	.62	.61	.62	.02

[a]Means are for 18 pigs per treatment.
[b,c]Contrast: .65% lysine diets with supplemental Zn vs. diets with .95% and 1.10% lysine and supplemental Zn ($P < .01$ and .05, respectively.

Appendix Table 21. Mineral concentrations of the livers, kidneys, and rib bones of weanling pigs fed Zn-lysine or $ZnSO_4$ with diets containing different levels of lysine[a]

	Treatments								
Zn source		$ZnSO_4$	ZnLy	$ZnSO_4$	ZnLy		$ZnSO_4$	ZnLy	
Zn, ppm	0	100	100	100	100	0	100	100	
Lysine, %	.80	.80	.80	.95	.95	1.1	1.1	1.1	SEM
Liver wt, %[bc]	3.0	3.4	3.1	3.2	3.1	2.9	3.1	3.2	.08
Kidney wt, %[b]	.55	.58	.53	.52	.52	.52	.50	.54	.03
Livers (mg/kg of DM)									
Zn[c]	100	160	142	143	151	92	147	167	13
Fe	493	372	505	367	369	310	431	402	52
Cu	26	33	24	26	21	34	24	39	4
Kidneys (mg/kg of DM)									
Zn[cd]	95	125	118	113	114	92	109	112	5
Fe[d]	116	114	144	106	113	114	123	117	8
Cu[ce]	22	41	37	32	30	16	30	34	3
Rib bones[f] (mg/kg of DM)									
Zn[c]	107	179	172	158	176	89	162	160	7

[a]Means are for 6 pigs per treatment.
[b]Weights are a percentage of live body wt.
[c]Contrast: no zinc diets vs. diets with supplemental zinc and the respective similar lysine levels ($P < .01$).
[d,e]Contrast: .65% lysine diet with zinc vs. diets with supplemental Zn and .95% and 1.10% lysine ($P < .05$ and .01, respectively).
[f]Left 10th rib bone.

Appendix Table 22. Mineral concentrations of the livers, kidneys, and rib bones of weanling pigs fed a Zn deficient diet followed by diets with 30 mg Zn/kg of diet from different sources[a]

	Treatments				
	ZnO	ZnMet	ZnLys	$ZnSO_4$	SEM
Liver wt, %[b]	2.5	2.4	2.3	2.3	.1
Kidney wt, %[b]	.51	.57	.51	.50	.03
Livers					
Zn, ppm	136	133	181	151	17
Fe, ppm	584	516	566	477	71
Cu, ppm	131	150	141	92	33
Kidneys					
Zn, ppm	107	100	110	104	4
Fe, ppm	133	121	133	139	6
Cu, ppm	30.7	27.6	28.9	22.4	2.9
Rib bones[c]					
Zn, ppm	243[d]	252	259[e]	263[e]	5

[a]Means are for 6 pigs per treatment.
[b]Weights are a percentage of live body wt.
[c]Left 10th rib bone.
[d,e]Using paired t-tests means with different superscripts are different ($P < .05$).

Vita

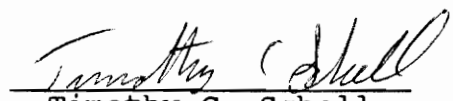
Timothy Charles Schell, son of Patricia Schell, was born June 27, 1964 in Nampa, Idaho. He graduated from Lafayette High School located in Williamsburg, Virginia in June of 1982. He recieved the Bachelor of Science degree in Animal and Dairy Science at Auburn University in June of 1986.

After graduation, he spent one year as a Senior Animal Technican in the Neurology Department at the University of California, San Francisco.

In August 1991, he completed a Master of Science degree in Animal Science at Virginia Polytechnic Institute and State University. Shortly thereafter, he began work toward a Doctorate degree in Animal and Poultry Sciences.

In 1993, he was elected as President of the Graduate Student Assembly and recieved the Outstanding Graduate Student Leader of 1993-94 Award at Virginia Tech.

He is an active member of the American Society of Animal Science, Omicron Kappa Delta leadership honorary and Delta Tau Delta Fraternity.



Timothy C. Schell

Published literature

Schell, T. C. and E. T. Kornegay. 1994. Effectiveness of zinc acetate injection in alleviating postweaning lag in pigs. J. Anim. Sci. 72(in press).

Schell, T. C. and E. T. Kornegay. 1994. Comparison of Zn availability from ZnO, Zn-lysine, Zn-methionine, and $ZnSO_4$ when fed at high concentrations to weanling pigs. J. Anim. Sci. 72(Suppl. 2):7.

Schell, T. C. and E. T. Kornegay. 1993. Effectiveness of zinc acetate injection of pigs before, at or after weaning on postweaning performance. J. Anim. Sci. 71(Suppl. 1):174.