ABSTRACT

SHAEFFER, GREGORY LEE. Evaluation of Basic Zinc Chloride as a Zinc Source for Cattle. (Under the direction of Dr. Jerry W. Spears.)

Two trials were conducted to evaluate basic zinc chloride as a zinc source for cattle. In Experiment 1, 120 Angus cross steers were used to determine the effects of zinc level and zinc source on performance and carcass characteristics. Treatments consisted of 1) control (no supplemental zinc), 2) 30 ppm supplemental zinc from zinc sulfate, 3) 90 ppm supplemental zinc from zinc sulfate, 4) 30 ppm supplemental zinc from zinc chloride, and 5) 90 ppm supplemental zinc from zinc chloride. Carcass data was obtained from a USDA grader 48 h after slaughter. Zinc supplementation regardless of source, lowered gain and gain:feed ($P < 0.05$) in the growing phase. During the growing phase steers supplemented with 30 ppm from zinc sulfate gained faster ($P<0.05$) than those fed 30 ppm of zinc from basic zinc chloride. In the finishing phase zinc supplementation, regardless of level or source, did not affect gain, feed intake or gain:feed. All carcass characteristics were similar for all treatments except for marbling, which was slightly higher ($P<0.05$) in steers supplemented with 90 ppm compared to those fed 30 ppm of zinc. Liver zinc was also significantly ($P < 0.05$) higher in steers fed 90 ppm verses 30 ppm of zinc. Plasma zinc concentrations were higher for steers supplemented with 30 ppm zinc from basic zinc chloride on day 28 and for steers supplemented with 90 ppm zinc from basic zinc chloride on day 84 of the growing phase and day 56 of the finishing phase compared to those fed similar concentrations of zinc from zinc sulfate. Steers supplemented with 90 ppm of zinc also had higher plasma zinc concentrations

than those supplemented with 30 ppm of zinc on day 84 of the growing phase and day 56 of the finishing phase. Ruminal soluble zinc concentrations were increased by zinc supplementation of the control diet and were higher in steers supplemented with 90 ppm compared to 30 ppm. Steers supplemented with 90 ppm of zinc from zinc sulfate had higher ruminal soluble zinc concentrations than steers fed 90 ppm from basic zinc chloride. Plasma alkaline phosphatase activity was not affected by zinc during the growing phase, but was higher in steers supplemented with 90 ppm zinc from basic zinc chloride compared to those fed a similar concentration of zinc sulfate on day 56 of the finishing phase. Results indicate that zinc sulfate and basic zinc chloride produce similar performance when fed to growing and finishing steers.

In Experiment 2, 16 steers were randomly separated into two groups. Four Angus steers and 4 Simmental steers were in each group and fed a diet low in zinc (25 ppm) for 14 days. The 2 groups were then supplemented with 25 ppm zinc from either zinc sulfate or basic zinc chloride. After 4 days of consumption of supplemental zinc, steers were placed in stainless steel metabolism crates and total fecal and urine were collected for a 5 day period. Steers were fed to minimize orts and refusals were weighed and analyzed. Plasma zinc concentrations were higher for steers supplemented with basic zinc chloride. Steers supplemented with basic zinc chloride also had higher apparent zinc absorption and retention than those supplemented with zinc sulfate. Results indicate that zinc from basic zinc chloride was more bioavailable than from zinc sulfate.

EVALUATON OF BASIC ZINC CHLORIDE AS A ZINC SOURCE FOR CATTLE

By

GREGORY LEE SHAEFFER

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APPROVED BY:

_____	_____
Dr. Vivek Fellner	Dr. Matthew H. Poore

_____
Chair of Advisory Committee
Dr. Jerry W. Spears

DEDICATION

This thesis is dedicated to my wife who gave me continuous love and support throughout the education process. Her enormous sacrifices through this period of our lives showed me the great dedication she has for our marriage and myself. She gave up living near her family and having a job she loved and moved 500 miles away to a place where we knew no one, and she had no job. Looking back it was worth it now to have a new house, both having jobs we like and having the opportunity to meet some remarkable friends.

I also dedicate this to my grandparents and parents who gave me the financial support, materials, and assets to get involved in 4-H and FFA. These programs got me involved in agriculture and helped me get into the career I am in today.

BIOGRAPHY

Gregory Lee Shaeffer was born in Columbus, Ohio to John Shaeffer Jr. and Pauline Shaeffer. He was raised with an older sister (Shelley) and a younger brother (Michael) in the small community of Amanda, Ohio. He grew up on his grandparent's farm and was heavily involved in 4-H. Once in high school he became extremely active in the Future Farmers of America and received his State FFA Degree from the State of Ohio. After graduating from Amanda Clearcreek High School he attended Berea College in Berea, Kentucky. Berea College is a work-study school, meaning that tuition was paid but working for the college a minimum of ten hours per week was required. The summer of 1998, he married his wife, April, who was also a student at Berea College. By his senior year, he was a student farm manager working more than thirty hours per week on all aspects of the farm. His hard work and dedication to the college farm earned him Student Employee of the Year 2000 from the State of Kentucky. In May 2000, he graduated from Berea College as a member of Delta Tau Alpha, an agriculture honor society, with his degree in Agriculture and Natural Resources. Upon graduation he became a full time staff member with the college as an assistant farm manager until July 2000. In July, he and his wife moved to Raleigh, North Carolina to pursue his Master of Science in Animal Science under the direction of Dr. Jerry Spears at North Carolina State University. In January 2002, he became a part time student finishing his degree as well as a full time employee with the university as a research technician at the Butner Beef Cattle Field Lab with University Field Labs.

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Table of Contents

LIST OF TABLES

Page

Chapter 1

Literature Review

Introduction

Zinc is a shiny bluish-white, soft metal with an atomic number of 30 and an atomic weight of 65.37. The metal is found naturally in ores mainly in the sulfide form. A scientist named Raulin discovered the first evidence that zinc was essential in life. In 1869 he showed the fungus Aspergillus niger required it. In 1877 Lechartier and Bellamy also provided evidence that zinc had a biological function (O'Dell, 1983). Fifty years passed before additional information about zinc was released, when Sommer and Lipman (1926) reported that zinc was required for higher plants. Zinc research with animals was not published until 1934, when Todd and co-workers reported that zinc was required for growth by the rat. Day and Skidmore (1947) later demonstrated the need for zinc in the mouse as cited in Lamand, 1984; McDowell, 2003.

Research indicating the importance of zinc in a domesticated animal species did not appear in the literature until 1955, when Tucker and Salmon reported that zinc supplementation prevented a condition called parakeratosis in swine (O'Dell, 1983). Two years later zinc was found to prevent skin lesions and poor feathering in chicks (O'Dell 1983). In the ruminant, the idea of zinc deficiency was slower to surface. In the early 1960's, Legg and Sears (1960) and Grashuis (1963), in Holland, linked parakeratosis in cattle to zinc deficiency. Also in 1960 by feeding a purified diet low in zinc, Miller and Miller were able to

produce clinical zinc deficiency signs in calves (Chesters, 1983). After their research, zinc deficiency in cattle was later reported under field conditions by three groups of researchers (Lamand, 1984).

Function of Zinc

Zinc is found in all body organs and is needed for normal hair, skin, feather, and bone growth. Zinc is linked to wound healing where zinc is involved in the synthesis of collagen in the bone and skin. Injuries from zinc deficient animals take longer to heal. Zinc plays a role in immunity, fertility, and nearly 200 zinc metalloenzymes. These metalloenzymes include carbonic anhydrase, which is required to transfer carbon dioxide in the red blood cells, carboxypeptidase (a digestive enzyme), and alkaline phosphatase. The largest number of zinc enzymes belong to the hydrolases, class III. Some zinc metalloenzymes are found among several species but each enzyme catalyzing a specific reaction is a unique protein for each species. Alkaline phosphatase has been found in 11 species, aminopeptidase in 17 species and neutral protease in 25 species (O'Dell, 1983). It is thought that zinc enzymes are so critical that in a deficiency situation zinc metalloenzyme are the last proteins to lose the zinc ion. Zinc is also involved in DNA and RNA metabolism. Duplication of genes and cell multiplication requires zinc in DNA polymerase and transcription regulation involves zinc-containing histones (Underwood and Suttle, 1999). Zinc is also involved in vitamin A metabolism. Alcohol dehydrogenase, a zinc metalloenzyme, is responsible for the interconversion of vitamin A alcohol

(retinol) to vitamin A (retinal) which is a process required for normal vision (McDowell, 2003).

Sources of Zinc

Pastures vary widely in zinc and range from 7 to 100 ppm with a mean of 36 ppm on a dry matter basis (Underwood and Suttle, 1999). Forage zinc is affected by soil type and condition as well as maturity, and species. Basic igneous rocks are rich in zinc, sandstone is poor and calcareous rocks are variable. Peat is a soil amendment but if it is formed over acidic rocks, it will be poor in zinc (Lamand, 1984). Soils that are flooded or poorly drained will have lower zinc content because of the zinc being soluble in the excess water. Other soil factors that affect forage zinc concentrations include fertilization with excess nitrogen and phosphorus, soil pH rising above 6.4, and the degree of compaction (Lamand, 1984, McDowell, 2003). Forages differ by species with legumes being higher in zinc than grasses. Cereal grains usually contain 20 to 30 ppm zinc while plant protein sources can provide 50 to 70 ppm zinc (NRC, 1996).

A number of supplemental zinc sources are also commercially available including both inorganic and organic sources. Current feed grade inorganic zinc sources include zinc sulfate and zinc oxide. Basic zinc chloride has also been evaluated in chicks and pigs and will likely become available commercially in the future (Cao et al., 2000a, Mavromichalis et al., 2001). Organic zinc sources include zinc complexes such as zinc methionine, zinc lysine, zinc glycine, and zinc proteinates formed from chelation of zinc with amino acids and or partially

hydrolyzed proteins, zinc amino acid chelates and zinc polysaccharide complexes (Spears, 1996).

Zinc Deficiency

Zinc deficiency is a concern in the nutritional aspects of food animal production. Deficiencies severely affect growth, health, and well being of animals and even marginal zinc deficiencies can lead to significant economic losses. All body systems and organs, that have high rates of cell division and protein synthesis, are affected by a deficiency. Deficiency signs include loss of appetite, reduction in growth rate, bone abnormalities, diarrhea, excessive salivation and skin lesions usually around the mouth, nose, neck, and eyes. The skin in some species will get rough and thicken, a condition referred to as parakeratosis. Other signs of zinc deficiency include atrophy of the thymus and progressive testicular atrophy, which is connected to the failure of spermatogenesis (Chesters, 1983). Miller et al. (1965) fed severely zinc deficient diets to nine calves. Four of the nine calves died from secondary conditions complicating the already greatly weakened condition of the deficient animals. Knowing that skin usually had high concentrations of zinc, they did a skin biopsy procedure. The deficient calves had slower growth of new skin as well as the development of parakeratosis. The deficient calves, after several months, had scars and abnormal tissues on the area of biopsy as well as gray hair covering the new skin. With this observation they concluded that zinc had an important role in skin metabolism (Miller et al., 1965). Serum alkaline phosphatase was also notably

lower in the deficient calves until the calves were fed a recovery diet, which reversed the low serum values.

Genetics and Zinc Deficiency

There are several genetic disorders that cause zinc deficiencies. One such disorder is found in mice, called lethal milk, which is a genetic defect that is recessively inherited by the dam (Chesters, 1983). Offspring from these dams die after nursing while the offspring nursing a normal dam survive. All the signs from what was called lethal milk mirrored zinc deficiency and milk tested from the affected dams was 34% lower in zinc than milk from normal dams (Chesters, 1983). Although the milk of these mice were lower in zinc the plasma and carcass zinc values were similar to mice not affected by lethal milk. Researchers increased survival and weight gain of the offspring by the supplementation of zinc glycine.

The most important genetic disorder from an animal production standpoint is a genetic defect of zinc metabolism called the lethal trait A46 or Adema disease. Between the years of 1951 and 1961, a problem emerged in a Scottish herd of cattle. In this herd, 4% of the calves born within this 10 year period had a disease that was put into remission with zinc supplementation (Chesters, 1983). These calves were normal at birth but began to produce excessive saliva at 2-4 weeks of age. This was followed by hair loss around the mouth, eyes, and anus, erosions within the mouth and skin lesions on the legs, abdomen and thorax. The skin lesions became dry and crusty which then shed. These symptoms were

also equally distributed between sexes. Through extensive research, a genetic association was determined from one common genetic line of bulls, which was imported from Holland. This was the start of hereditary zinc deficiency syndrome, also known as Lethal Trait A 46, Adema disease, Hereditary Thymic Hypoplasia, or Hereditary Parakeratosis. Cases were initially observed in Scotland and then other cases appeared in Italy (1969) and later in the Federal Republic of Germany and Holland. However, it was not until 1970 that the disorder was recognized. Andresen et al. (1970) were the first to publish information about the lethal trait. At the time they wrote their article, 45 lethal or semi-lethal factors had been compiled by Stormont (1958) and Fechheimer et al. (1968) (Andresen et al., 1970). These factors are various hereditary issues, which cause individuals to die before they have the ability to reproduce. In 1970, Gronborg-Pedersen was also studying the same syndrome in cattle. Clinical symptoms were described and it was speculated that the traits might be inherited (Andresen et al., 1970). The syndrome is associated with the Black Pied Danish cattle of Friesian descent. All affected calves were sired by the same bull, Hornshoj Adema S 8181, from which the syndrome's first name was applied: "Morbus Ademae". This name also applied to the Friesian bull Adema 21. Andresen et al. (1970) did not want to use "Adema" in designating the disease because the trait was genetically recessive. The Adema bulls were not the only cause; the dams had to be genetically recessive as well for the trait to be expressed. The second reason for not using "Adema" as the syndrome title was because several non-carriers from other countries had the Adema name and

could have been thought of as carriers. So with the lack of a suitable descriptive name, the researchers proposed the code designation A 46 (Andresen et al., 1970).

In the study performed by Andresen et al. (1970), calves expressing lethal trait A 46 appeared normal at birth and up to 1 month of age. At 6-8 weeks of age, development of external signs occurred, which was followed by death or euthanasia by 1.5 to 4 months of age, because of the pronounced development of the condition (Andresen et al., 1970). Basse found a diminutive thymus in all affected calves subjected to an autopsy (Andresen et al., 1970). A chromosome analysis on infected calves, which were not in the study, possessed a normal diploid chromosome number and no abnormalities were found. A sex ratio was also considered. Statistically, the ratio did not differ from a 1:1 ratio. While controlled mating prevented calves from expressing the trait, Andresen et al. (1970) concluded that the lethal trait was a result of autosomal recessive inheritance.

By 1975, the syndrome was described as an autosomal recessive genetic disorder with a basic defect that caused an inability to effectively absorb zinc from the intestinal tract. This genetic defect is mostly associated with the Adema bloodline from Holland. Further research indicated a Dutch A.I. bull named Adema 21 van de Woulhoeve N.R.S. 26781, which was born in 1946 was sire to many affected calves (Kroneman et al., 1975). It is from this bull's name that the disorder was referred to as Adema disease. This bull was a fourth generation descendant of another bull, which was found to be the probable source of the

original mutation. The bull was called Egbert N.R.S. 13110, and was born in 1932 (Kroneman et al., 1975). Kroneman observed in his research that the calves responded well to an oral zinc sulfate solution or other zinc salts. Kroneman gave the oral zinc solution at a dose of 0.21 mg zinc per kilogram of body weight. Kroneman et al. (1975) noticed that clinical signs of zinc deficiency tended to appear in calves approximately 2 weeks after zinc levels in serum decreased and stayed at low levels. Calves that seemed to be infected were given an oral zinc treatment. Treatment caused all symptoms to disappear until treatments stopped. When stopping the oral zinc treatment, the symptoms began to reappear. Kroneman concluded a simple recessive gene caused the hereditary disorder when the deficiency could not be controlled in the affected animals.

This syndrome also parallels a human problem called Acrodermatitis enteropathica, which has been treated with halogenated oxychinolines since 1953 (Flagstad, 1977). The hypothesis put forth by Portnoy & Molokhia (1974) and Moynahan (1974) suggested that oxychinolines might transport zinc or make zinc available for transport across the intestinal barrier (Flagstad, 1977). At this time, there was no research to support the hypothesis, but Neldner and Hambridge (1975) did observe a slight increase in plasma zinc concentrations after treatment with oxychinolines (Flagstad, 1977). Flagstad found that this hypothesis was indeed correct. Period one of this study involved a calf with Adema disease. The calf was given joklokinol, a potent metal binding agent that chelates zinc, as an oral dose of 4 grams per day. Normal plasma zinc levels,

rapid resolution of skin lesions and full regrowth of hair occurred within 30 days of treatment. Flagstad also had a second period in his research that involved Adema calves receiving oral doses of 10 grams per day zinc oxide. This treatment resulted in faster improvements and extremely high zinc plasma levels. It was also concluded from Flagstad's research that Adema disease in cattle and the human form, Acrodermatitis enteropathica, were identical.

Kroneman et al. (1977) and Brummerstedt et al. (1977) demonstrated that supplemental zinc given prior to the terminal stage of Adema disease resulted in complete remission and reversal of the involution of the thymus (Chesters, 1983). The doses of zinc needed to treat the animals with the trait were 200 times higher than those needed by normal animals, simply because the lethal trait animals were unable to absorb zinc efficiently. This strongly suggested the loss of a transport mechanism for zinc in the intestinal mucosa. This idea was strengthened when Flagstad (1977) administered an oxychinoline supplement to a calf with the genetic mutation. The skin condition was alleviated without additional zinc supplementation and fractional absorption of a tracer dose of ^{65}Zinc given orally was increased as well (Chesters, 1983).

Machen et al. (1996) reported that diarrhea developed within 3 days after plasma zinc levels dropped below 0.5 ppm in calves with Adema disease. The diarrhea was dark green and watery with a sweet metallic odor. Calves became lethargic and depressed as well as developing a clear nasal discharge. Skin lesions developed on the muzzle from patches of dry scaly skin within 24 hours. However, one unique aspect to this study was the calves developed a diminished

suckling ability. They had problems curving their tongues around the nipple of the bottle and also excessive salivation was observed. Once zinc treatment began, the suckling problem as well as the drooling ceased within 24 hours (Machen et al., 1996). These results suggested that zinc deficiency caused an impairment of the glossal motor skills. Machen discussed the two pathways via which zinc is absorbed. The first pathway, transporter-dependent saturable system, is effective at low concentrations of luminal zinc. The second pathway, the nonsaturable system, involves passive diffusion of zinc. In the affected cattle, large doses of zinc allowed proper uptake of zinc, which indicated the nonsaturable component of zinc transport was what was functioning in hereditary zinc deficiency (Machen et al., 1996). Current research is looking at a cysteine rich intestinal protein. The cysteine rich intestinal protein appears to be involved in the transporter-dependent pathway of zinc absorption (Machen et al., 1996).

Zinc Toxicity

Excess zinc can cause as many problems in the body as a deficiency. The first signs of zinc toxicity include reduced feed intake, reduced weight gain, anemia, and bone resorption. As the animal receives higher levels of zinc or toxic levels for longer periods of time one will see diarrhea, lameness, internal hemorrhaging, and even death (Kincaid et al., 1997). Kincaid et al. (1997) completed research with 40 calves using 4 levels of zinc in a calf starter. With his research it was confirmed that the calf can consume up to 300 mg/kg of zinc in complexes with amino acids without issues of toxicity (Kincaid et al., 1997).

This research agrees with the National Research Council, which states the maximum tolerance level of zinc is 500 mg/kg diet. Decreased weight gain was reported in calves that were fed 900 mg/kg zinc for a 12 week period (NRC, 1996).

Zinc Metabolism

Zinc is absorbed according to need by the rat and ruminants (Underwood and Suttle, 1999). Zinc is absorbed throughout the small intestine of monogastric animals, but may be absorbed by the rumen better than the small intestine in sheep (McDowell, 2003). Zinc is transferred from the lumen into the mucosal cell. This process is regulated by metallothionine. Zinc absorption is reduced by high concentrations of copper, cadmium, calcium, phosphorus, chromium, phytate, and fiber (Underwood and Suttle, 1999).

Approximately two-thirds of the plasma zinc is loosely bound to albumin (Underwood and Suttle, 1999). Tissues can readily take up zinc that is attached to albumin. The remaining portion of the plasma zinc is tightly bound to high molecular weight proteins (McDowell, 2003). The liver extracts about 30-40% of the zinc that enters the bloodstream and then the liver releases the zinc back into the bloodstream. Zinc uptake by the central nervous system is slow but is bound for long periods. Zinc in bones and hair is not readily available for mobilization to other organs. The most turnover and accumulation of zinc occurs in the pancreas, liver, kidneys, and spleen. Movement of zinc into and out of cells occurs by transport proteins. These proteins protect the cells from zinc toxicity,

obtain zinc from the environment, and maintain supplies of zinc. Intracellular zinc is found mainly in the cytosol bound to proteins (60-80%), with only 10 -20% in the nuclear fraction, and small amounts in the mitochondrial and microsomal fractions (McDowell, 2003).

Zinc is stored in organs such as liver, kidney, and pancreas. Minor stores can be found in bone, muscle and skin (Rojas et al., 1995). There are limited stores of zinc that can be mobilized during zinc deficiency. Metallothionein, which is concentrated in the liver, kidney, pancreas, and intestine is the major form in which zinc can be stored (McDowell, 2003). Zinc can also be redistributed from pools in muscle and bone during deficiency, which can prolong the time before a clinical deficiency occurs (Underwood and Suttle, 1999). Zinc absorbed in excess of requirements is eliminated primarily via endogenous fecal excretion with minor amounts excreted in the urine. Endogenous zinc that is excreted in the feces originates from pancreatic, gastrointestinal, and biliary secretions as well as loss of mucosal cells.

Zinc Bioavailability

Bioavailability has been widely studied across species lines using a wide range of diets including purified diets, deficient in zinc, as well as diets meeting the National Research Council recommendations. The majority of these studies compared organic sources with the traditional inorganic source, zinc sulfate. Less research has been published comparing bioavailability of zinc from inorganic sources in ruminants

Wedekind et al. (1990) found that the typical source of zinc being used at the time in most poultry and swine rations, zinc oxide, was not as available as feed grade zinc sulfate. In this study they fed male chicks 3 levels of supplemental zinc from each of the 2 sources. The basal diet contained 13 ppm zinc and they supplemented either 0, 7.5, or 15 ppm zinc from each source. Conclusions of the study indicated the bioavailability of zinc oxide, using tibia zinc regressed on supplemental zinc intake, was only 44.1% when compared to zinc sulfate set at 100%. Also when weight gain was regressed on supplemental zinc intake, it was found that zinc oxide was only 61.2% as bioavailable as zinc sulfate (Wedekind et al., 1990). Wedekind et al. (1992) used 3 different diets to assess the bioavailability of zinc methionine using male chicks. They used a purified amino acid diet (1 ppm zinc), a semi-purified diet (13 ppm zinc), and a complex corn-soybean diet (45 ppm zinc). The trials were conducted with each zinc source supplemented at 0, 7.5, and 15 ppm zinc. They found a linear response to weight gain and tibia zinc for both zinc methionine and zinc sulfate. Plasma zinc concentrations did not increase for zinc methionine but an increase in the zinc sulfate treatment was seen (Wedekind et al., 1992). Bioavailability of zinc was determined to be 117% for zinc methionine in a purified diet and 177% in a soy isolate diet when compared to zinc sulfate. It was theorized that complexes and chelates are able to compete with phytic acid for zinc binding capacity. They postulated that zinc chelate-complex forms soluble complexes with zinc, which makes it available to the animal, while inorganic zinc sources may complex with phytate, which makes it unavailable. They concluded that

bioavailability estimates for zinc are dependent on the diets used and they also suggested that the metabolism for zinc methionine is different from that of inorganic zinc sulfate in poultry (Wedekind et al., 1992).

In swine relative bioavailability of different zinc sources differed from results obtained in poultry. Wedekind et al. (1994) adjusted 240 weanling pigs to a diet (42 ppm zinc) which depleted zinc stores in the body. At the end of this stage approximately 5% of the pigs developed signs of zinc deficiency. One hundred ninety two animals from this group were then allotted to 6 treatment diets all containing adequate nutrients with the exception of zinc. Zinc sulfate was used to develop a standard curve using 3 levels, 0, 7.5, and 15 ppm. This curve was then used with the 3 other groups of pigs fed zinc oxide (15 ppm supplemental zinc), zinc lysine (7.5 ppm supplemental zinc), and zinc methionine (7.5 ppm supplemental zinc). Results of this trial indicated that in swine zinc sulfate was the most bioavailable (100%) followed by zinc methionine (60.4%), zinc oxide (66.7%), and then zinc lysine (37.5%) as the least bioavailable using metacarpal zinc concentrations and setting zinc sulfate at 100% (Wedekind et al., 1994).

Rojas et al. (1995) using wether lambs obtained even different results in regard to zinc bioavailability than the research completed with poultry and swine. They used 40 crossbred wether lambs and 5 different treatments. The basal diet contained 16-20 ppm zinc and zinc was supplemented from zinc sulfate, zinc oxide, zinc methionine, and zinc lysine to supply 360 mg per day of zinc to the basal diet. Feed intake was restricted to 1000 grams per lamb per day. Results

showed mean serum zinc concentrations were different on day 49 when zinc concentrations increased for the controls by .20 µg/ml, while zinc lysine, zinc oxide, and zinc sulfate increased by .74, .83, and .62 µg/ml, respectively but zinc methionine was not different at .52 µg/ml. On day 55 zinc lysine was also significantly higher ($P < 0.05$) than the other treatments. Rojas et al. (1995) suggested that zinc sulfate and zinc lysine may be more bioavailable than zinc oxide but zinc methionine is not different than zinc sulfate in lambs. The data also suggested that zinc lysine was more bioavailable based on higher accumulations of zinc in the liver, kidney, and pancreas than the other zinc sources (Rojas et al., 1995).

Once again with a different species a different source was found to be superior in bioavailability. Cao et al. (2000b) also worked with wether lambs using zinc sulfate as a comparison with complexes and chelated products. Forty-two crossbred wether lambs were assigned to 1 of 7 treatments. Zinc sulfate was supplemented at 0, 700, 1400, or 2100 ppm added zinc and zinc from zinc proteinate, zinc amino acid chelate, or zinc methionine was supplemented at 1400 ppm (Cao et al., 2000b). The lambs were fed a corn-soybean meal-cottonseed hull diet (58 ppm zinc) at a restricted intake level of 1 kg per day as fed. Cao et al. (2000b) used multiple linear regression slope ratios of liver, kidney, and pancreas zinc, and liver metallothionine to compare relative bioavailability of the zinc sources to zinc sulfate. Bioavailability values relative to zinc sulfate were 130, 110, and 113% for zinc proteinate, zinc amino acid chelate and zinc methionine, respectively (Cao et al., 2000b). Cao et al. (2000b)

concluded that with the exception of the zinc proteinate, the other sources had similar bioavailablity as zinc sulfate.

Two groups have studied the bioavailability of zinc in cattle. Spears (1989) used 36 Hereford-Simmental heifers fed a corn silage based diet (23.1 ppm zinc) supplemented with 25 ppm zinc from either zinc oxide or zinc methionine. There was not a source effect on plasma zinc or alkaline phosphatase. Rojas et al. (1996) compared zinc methionine, zinc sulfate, and zinc oxide supplementing 360 mg of zinc per head per day. They found no differences among zinc sources when using 32 yearling Limousin and Angus crossbred heifers. These cattle were fed ad-libitum bermuda grass hay (19.6 ppm zinc) and 1.8 kg of corn based concentrate containing the treatment. The control diet in this trial ranged from 20-26 ppm zinc (Rojas et al., 1996). This trial concluded that at adequate levels of zinc, bioavailability of the zinc source may not be as important in cattle, but at lower zinc levels sources could be a factor (Rojas et al., 1996).

A number of studies have determined the effects of various sources of zinc on performance and carcass characteristics of finishing cattle. Spears and Kegley (2002) compared the traditional zinc oxide to organic sources of zinc proteinate. Research was conducted using 60 Angus and Angus-Hereford cross steers. Four treatments, which included a control (24 ppm zinc), zinc oxide, zinc proteinate-A, and zinc proteinate-B, were evaluated in this study. Zinc sources were supplemented to the control diet at 25 ppm of zinc. Zinc supplementation, no matter the source, was found to increase average daily gain during the

growing phase and performance tended to be higher during the finishing phase for steers fed the proteinate form verses zinc oxide. Hot carcass weight and dressing percentages were significantly higher for steers in the proteinate treatments than those in the control and zinc oxide groups. Quality grade, yield grade, marbling, and backfat was found to be increased by zinc supplementation but no differences among zinc sources were found (Spears and Kegley, 2002). Malcolm-Callis et al. (2000) used 84 Brangus and Angus sired steers fed steam flaked corn-based diet containing 30 ppm supplemental zinc from 1 of 3 dietary sources. The zinc sources included traditional inorganic zinc sulfate, organic amino acid complex and a zinc polysaccharide complex. Final diets analyzed 81.75 ppm zinc, 66.25 ppm zinc, and 62.75 ppm zinc, respectively. Average daily gain, gain to feed ratio, or dry matter intake was not affected by zinc source although kidney, pelvic and heart fat was higher for the zinc sulfate treatment verses the two complex sources. Fat thickness was greater in the zinc amino acid complex and the zinc polysaccharide treatments verses the zinc sulfate treatment (Malcolm-Callis et al., 2000). Another study was conducted with Red Holstein bulls comparing zinc proteinate, zinc polysaccharide, and zinc oxide (Kessler et al., 2003). These bulls were fed corn and grass silage along with a concentrate and mineral and vitamin mix. The control diet contained 35 ppm of zinc and zinc sources were added to supply an additional 10 ppm of zinc. Zinc source did not significantly affect dry matter intake, growth rate, feed conversion, zinc status, or carcass and meat quality (Kessler et al., 2003).

Spears et al. (1991), Gaylean et al. (1995), and Nunnery et al. (1996) reported no effect of zinc methionine on average daily gain of cattle. Greene et al. (1988) used a control diet (81 ppm zinc) and then supplemented 360 mg zinc per day from either zinc methionine or zinc oxide. They found that zinc methionine increased quality grade, marbling score, kidney, pelvic and heart fat, as well as external fat when compared to zinc oxide. The quality grade response to zinc methionine may allow marketing at an earlier age or increase marbling in cattle that typically do not grade well (Greene et al., 1988). Huerta et al. (2002) also found some positive carcass correlation with zinc treatments. Their diets consisted of a control diet (64 ppm zinc) and 200 ppm supplemental zinc from the 2 sources. They found that zinc methionine and zinc sulfate lowered maturity scores, and that quality grades were significantly higher in finishing heifers supplemented with zinc methionine but these results were not seen with finishing steers (Huerta et al., 2002). Martin et al. (1987) could not replicate the responses to zinc methionine shown by Green et al. (1988). They supplemented zinc at 37 ppm. The research found no differences in gain, intake, or feed efficiency. Also there was no increase of kidney, heart, and pelvic fat, ribeye area or percentage of carcasses grading choice. Huerta et al. (2002) took the research with zinc methionine one step further and evaluated the interaction of zinc with growth implants. Their diets consisted of a control diet (64 ppm zinc) and 200 ppm supplemental zinc from either zinc methionine or zinc sulfate. Average daily gain from heifers that were not given implants and being fed zinc methionine was 26% higher than the group that was given a growth implant and

fed zinc methionine. The opposite affect was seen when zinc sulfate was used as the treatment. Implanted heifers had a higher weight gain than non-implanted heifers when either no additional zinc or zinc sulfate was used (Huerta et al., 2002). Research completed with steers also indicated that growth implants increased gain in animals supplemented with zinc sulfate but not in those supplemented with zinc methionine.

Zinc Chloride

Research evaluating the bioavailability of zinc from basic zinc chloride has recently been published. Cao et al. (2000a) found that bioavailability of zinc from basic zinc chloride was at least equal to reagent grade zinc sulfate when fed in excess of dietary requirements. It is known that the sulfate forms, which are acid salts, are highly water soluble allowing free radical formation. This reaction has potential to lead to the breakdown of vitamins and degradation of fats and oils leading to a decrease in nutrient value of the feed (Batal et al., 2001). Basic zinc chloride may have advantages as a source of supplemental zinc because it is insoluble in water yet completely soluble in 0.4%hydrochloric acid and 2% citric acid (Cao et al., 2000a). Batal et al. (2001) compared bioavailability of basic zinc chloride and zinc sulfate in chicks fed diets deficient in zinc. Baby chicks were fed either zinc sulfate or zinc chloride at 0, 5.81, and 10.81 ppm zinc to a basal diet containing 13.5 ppm zinc. There was a significant weight gain from the supplementation of both sources but it turned out that the zinc sulfate and zinc chloride did not differ in bioavailability. Tetrabasic zinc chloride may still be a

better source in poultry diet because of the insolubility in water, which reduces the oxidation of other nutrients (Batal et al., 2001).

Mavromichalis et al. (2001) took the tetrabasic zinc chloride research one step further and looked at the pharmacological aspect with nursery pigs. Research was completed with zinc oxide as the comparison to tetrabasic zinc chloride using 150 weaned pigs getting diets with no antimicrobial agents included. Pigs were fed 0, 1500, or 3000 ppm zinc from either zinc oxide or tetrabasic zinc chloride. It was concluded that the tetrabasic zinc chloride at 1500 ppm zinc was equal to 3000 ppm zinc from zinc oxide. At that level there was a significant increase in weight gain and feed efficiency, as well as a significant increase in feed efficiency for the tetrabasic zinc chloride (Mavromichalis et al., 2001). A second trial was conducted, this time with the antimicrobial agent carbadox, using 180 weaned pigs. Six treatments were assessed: no supplemental zinc, 3000 ppm from zinc oxide, or 750, 1500, 2250, or 3000 ppm from tetrabasic zinc chloride. The addition of 3000 ppm of zinc from zinc oxide and 1500 ppm or higher from tetrabasic zinc chloride resulted in a significant increase in weight gain and gain to feed ratio compared to the control pigs (Mavromichalis et al., 2001). These studies indicate that basic zinc chloride is a highly bioavailable source of zinc for nonruminants. Currently there is no published literature using ruminant animals as a model to compare zinc chloride to other sources of zinc. Research is needed to determine the bioavailability of zinc from basic zinc chloride in ruminants.

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Chapter 2

Effect of Zinc Source (Zinc Sulfate vs. Basic Zinc Chloride) and Level on Performance, and Carcass Characteristics in Growing and Finishing Steers[1]

G. L. Shaeffer, J. W. Spears[2], and K. E. Lloyd

Department of Animal Science and Interdepartmental Nutrition Program

North Carolina State University

Raleigh, NC 27695-7621

[1]Use of trade names in this publication does not imply endorsement by the North Carolina Agricultural Research Service nor criticism of similar ones not mentioned.

[2]Correspondence: Campus Box 7621; North Carolina State University, Raleigh, NC 27695-7621; (Phone: 919-515-4008; Fax 919-515-4463; Email: Jerry_Spears@ncsu.edu).

Abstract

One hundred twenty Angus and Angus cross steers averaging 251 kg initially, were used to determine the effects of zinc level and zinc source on performance and carcass characteristics of growing and finishing steers. Steers were blocked by weight and origin and then randomly assigned to treatments. Treatments consisted of 1) control (no supplemental zinc), 2) 30 ppm supplemental zinc from zinc sulfate, 3) 90 ppm supplemental zinc from zinc sulfate, 4) 30 ppm supplemental zinc from zinc chloride, and 5) 90 ppm supplemental zinc from zinc chloride. Steers were housed in pens of 5 animals. All treatments consisted of five replicate pens with the exception of the control, which contained four replicate pens. Steers were fed a corn silage-based diet during the 85 d growing phase and a high corn diet during the finishing phase. Steers remained on their dietary treatment throughout the growing and finishing phases. Plasma zinc concentrations, plasma alkaline phosphatase activities, blood urea nitrogen concentration were determined at d 28 and 84 of the growing phase and on d 56 of the finishing phase. Ruminal soluble zinc was also measured once during the growing and finishing phase. Steers were slaughtered (equal number per treatment) and liver samples were obtained after receiving the finishing diets for 90, 103, 110, or 117 days. Carcass data was obtained by a USDA grader 48 h after slaughter. Zinc supplementation regardless of source lowered gain and gain:feed ($P < 0.05$) in the growing phase. During the growing phase steers supplemented with 30 ppm from zinc sulfate gained faster ($P<0.05$) than those fed 30 ppm of zinc from basic zinc chloride. In the finishing phase

zinc supplementation, regardless of level or source, did not affect gain, feed intake or gain:feed. All carcass characteristics were similar for all treatments except for marbling, which was slightly higher ($P<0.05$) in steers supplemented with 90 ppm compared to those fed 30 ppm of zinc. Liver zinc was also significantly ($P < 0.05$) higher in steers fed 90 ppm verses 30 ppm of zinc. Plasma zinc concentrations were higher for steers supplemented with 30 ppm zinc from basic zinc chloride on day 28 and for steers supplemented with 90 ppm zinc from basic zinc chloride on day 84 of the growing phase and day 56 of the finishing phase compared to those fed similar concentrations of zinc from zinc sulfate. Steers supplemented with 90 ppm of zinc also had higher plasma zinc concentrations than those supplemented with 30 ppm of zinc on day 84 of the growing phase and day 56 of the finishing phase. Ruminal soluble zinc concentrations were increased by zinc supplementation of the control diet and were higher in steers supplemented with 90 ppm compared to 30 ppm. Steers supplemented with 90 ppm of zinc from zinc sulfate had higher ruminal soluble zinc concentrations than steers fed 90 ppm from basic zinc chloride. Plasma alkaline phosphatase activity was not affected by zinc during the growing phase but was higher in steers supplemented with 90 ppm zinc from basic zinc chloride compared to those fed a similar concentration of zinc sulfate on day 56 of the finishing phase. Results indicate that zinc sulfate and basic zinc chloride produce similar performance when fed to growing and finishing steers.

Key Words: Zinc, Performance, Zinc Chloride, Carcass Quality

Introduction

Studies in poultry have indicated that the bioavailability of zinc from basic zinc chloride is similar to reagent-grade zinc sulfate (Cao et al., 2000; Batal et al., 2001). Basic zinc chloride has not been evaluated as a zinc source for ruminants. Because of its low water solubility (Cao et al., 2000), basic zinc chloride would be expected to be less soluble in the rumen than zinc sulfate. A low solubility in the rumen may reduce interactions among zinc and other compounds in the rumen and therefore, increase zinc absorption from the small intestine.

Most previous studies in growing and finishing cattle have indicated no response in performance to zinc supplementation of diets containing NRC (1996) recommended concentrations of 30 ppm zinc. However, Galyean (1996) surveyed feedlot nutrition consultants and found that a higher percentage of consultants supplemented zinc concentrations well above NRC recommendations. Reasons given by nutritionist for using high supplemented zinc concentrations were improved carcass quality and more rapid growth rate by current types of cattle (Galyean, 1996). Limited research suggests that zinc concentrations above NRC requirements may alter carcass quality (Malcolm-Callis et al., 2000). The present study was conducted to determine the effect of zinc level (30 vs. 90 ppm supplemented) and source (zinc sulfate vs. basic zinc chloride) on performance and carcass characteristics of growing-finishing steers.

Materials and Methods

The study was conducted at the Butner Beef Cattle Field Laboratory, under the direction of University Field Laboratories at North Carolina State University. Cattle used in this trial were either produced by the University cowherd or purchased from local North Carolina graded feeder calf sales. Several weeks prior to the start of the trial cattle were vaccinated for IBR, BVD type I and II, PI3, and BRSV using Titanium 5 (Diamond Animal Health, Des Moines, IA) and for Clostridium chauvoei (Blackleg), septicum (Malignant edema), novyi (Black disease), sordellii and perfringens types C and D (enterotoxemia) using Vision 7 (Intervet Inc., Millsboro, DE). Cattle were also dewormed using Cydectin Pour-on (Fort Dodge, Fort Dodge, IA). Basic zinc chloride was supplied by Micronutrients, A Division of Heritage Technologies, LLC (Indianapolis, IN) and the zinc sulfate originated from Eastern Minerals (Henderson, NC). Care, handling, and sampling of the animals used in the trial was approved by the North Carolina State University Institutional Animal Care and Use Committee.

One hundred twenty Angus and Angus cross steers averaging 251 kg initially, were blocked by weight and origin and randomly assigned to treatments. Treatments consisted of 1) control (no supplemental zinc), 2) 30 ppm supplemental zinc from zinc sulfate, 3) 90 ppm supplemental zinc from zinc sulfate, 4) 30 ppm supplemental zinc from zinc chloride, and 5) 90 ppm supplemental zinc from zinc chloride. Zinc sulfate was analyzed 35.5% zinc

while zinc chloride analyzed 61.7% zinc. Steers remained on their dietary treatment throughout the growing and finishing phases.

Steers were housed 5 steers per pen in one covered, open sided barn with 24 pens, 12 pens per side and a slatted concrete floor with a flush system designed by Hog Slat. Each treatment consisted of 5 replicate pens of 5 steers with the exception of the control treatment, which had 4 replicate pens. Steers were fed ad-libitum from a concrete bunk feeder once per day in the morning and had access to automatic waterers. During the 85 d growing phase, steers were fed a corn silage-based diet (silage crude protein average 6.43%) supplemented with protein, minerals, and vitamins (Table 1). The control diet analyzed 38 ppm of zinc. At the end of the growing phase, a 9 d transition feeding schedule allowed for the switch to a high concentrate finishing diet with all animals remaining on their same dietary treatment they had received during the growing phase. Composition of the finishing diet is shown in Table 2. All steers were implanted with Synovex-Plus at the beginning of the finishing phase. Growing and finishing diets were formulated to meet or exceed all nutrient requirements for medium-framed steers with the exception of zinc (NRC, 1996). Fifteen steers were harvested after 90 d on the finishing diet, 30 steers after 103 d, 35 steers after 110 d, and 40 steers after 117 d on the finishing diet. Equal number of steers per rep and treatment were slaughtered on each date chosen by weight. Steers were slaughtered at a commercial abattoir where a liver sample and hot carcass weight was obtained the day of harvest. Other carcass data was acquired 48 hours after slaughter by a USDA grader.

Body weights were taken prior to feeding on two consecutive days at the start and end of the trial. Interim weights were taken every 28 days throughout the growing and finishing phases. Blood samples were taken at day 28 and 84 of the growing phase and day 56 of the finishing phase to determine plasma zinc, blood urea nitrogen, and alkaline phosphatase activity. Samples were obtained from the same 2 steers per pen on all sample days 2 hours post feeding. Blood samples were collected via jugular venipuncture in heparinized vacuum tubes designed for trace mineral analysis (Becton Dickenson, Rutherford, NJ) and plasma was obtained following centrifugation. Plasma zinc was measured by atomic absorption spectroscopy (AA-6701F, Shimadzu Scientific Instruments, Kyoto, Japan). Alkaline phosphate was assayed within 24 hours after centrifuging the blood using a commercial kit obtained from Sigma Diagnostics. Absorbance readings were taken using a Bausch and Lomb Spectronic 1001 (Bausch and Lomb, Rochester, NY) at 405 nm. Blood urea nitrogen was also analyzed using a Sigma Diagnostics kit. Absorbance readings were measured at 530nm.

Ruminal fluid was obtained via stomach tube from two steers per pen on day 85 of the growing phase and day 109 of the finishing phase. Ruminal fluid was collected 2 hours post feeding in sealed bottles until returned to the laboratory. Samples were centrifuged at 28,000X g for 30 minutes and then the supernatant was removed for zinc analysis. A sample of strained ruminal fluid was acidified with 25% meta-phosphoric acid, centrifuged and supernatant fluid was used for VFA analysis. Ruminal VFA were measured by gas liquid

chromatography (Model 3380, Varian, Walnut Creek, CA) using a nukol fused silica column, 30 m X 0.25 mm X 0.25 µm.

Statistical Analysis

Data were analyzed statistically by least squares analysis of variance using the GLM procedure of SAS (1990). Differences among treatments were determined using single degree of freedom contrasts. Contrasts made were: 1) control vs. all zinc supplemented treatments, 2) 30 vs. 90 ppm of supplemental zinc, 3) basic zinc chloride vs. zinc sulfate when supplemented at 30 ppm of zinc, and 4) basic zinc chloride vs. zinc sulfate when supplemented at 90 ppm of zinc. Repeated measures were also run on blood urea nitrogen, alkaline phosphatase, and plasma zinc. Significance was declared at $P < 0.10$.

Results and Discussion

During the 85 d growing phase, gain and gain:feed was slightly higher ($P < 0.05$) for controls compared with steers supplemented with zinc (Table 3). The control diet analyzed higher in zinc at 38 ppm than the NRC recommendation of 30 ppm. However, the higher gain in control verses the zinc supplemented steers was not expected. In contrast to the present study zinc supplementation (25 ppm) of a corn silage-based diet containing 33 ppm zinc increased gain in steers (Spears and Kegley, 2002). However, in growing heifers zinc addition to a corn silage-based diet containing 23.8 ppm of zinc did not affect performance over a 126-d study (Spears, 1989). Feed efficiency was not affected by zinc source, although dry matter intake and gain were slightly lower in steers supplemented with 30 ppm zinc from zinc chloride than in animals given a similar

level of zinc sulfate. The decreased gain in cattle supplemented with 30 ppm zinc from basic zinc chloride verses zinc sulfate is difficult to explain because performance of cattle fed 90 ppm supplemental zinc was similar for the two zinc sources.

The control finishing diet was lower in zinc compared to the growing diet (25 vs. 38 ppm), but zinc level or source did not affect performance (Table 3). A trial with a control compared to zinc supplementation from zinc oxide or zinc proteinate found a tendency for an increase in average daily gain and gain:feed when finishing steers were fed the proteinate form while gain and feed efficiency were similar for the oxide and control treatments (Spears and Kegley, 2002). Perry et al. (1968) reported in two of four trials, improved average daily gain in steers that were receiving finishing diets 18 to 29 ppm zinc. Pond and Oltjen (1988) feeding diets containing 24 ppm zinc and Nunnery et al. (1996) feeding 30 ppm zinc were unable to detect an increase in gain or efficiency when supplementing zinc as cited by Spears and Kegley, 2002. Improvements in gain were also not seen in studies in which the negative control diet was over the NRC (1996) recommendation before additional zinc supplementation (Greene et al., 1988; Malcolm-Callis et al., 2000).

Carcass characteristics are presented in Table 7. Steers supplemented with 90 ppm of zinc had slightly higher marbling scores than those supplemented with 30 ppm of zinc. Zinc level or source did not affect other carcass measurements. Zinc sulfate supplemented at 20, 100, or 200 ppm had no effect on hot carcass weight, dressing percentage, kidney, pelvic, and heart fat, or

marbling score in finishing cattle (Malcolm-Callis et al., 2000). However, steers supplemented with 100 ppm zinc had higher yield grade and fat thickness than those receiving 20 or 200 ppm supplemental zinc (Malcolm-Callis et al., 2000). Spears and Kegley (2002) found that the addition of 25 ppm of zinc to a control finishing diet containing 26 ppm zinc increased quality grade, yield grade, marbling, and backfat in steers.

Plasma zinc concentrations tended to be higher in steers supplemented with basic zinc chloride compared with those given zinc sulfate at all sampling times (Table 4). On day 28 of the growing phase, plasma zinc differed significantly ($P < 0.05$) among steers supplemented with basic zinc chloride and zinc sulfate when 30 ppm of zinc was added. Steers supplemented with 90 ppm of zinc from basic zinc chloride had higher ($P < 0.10$) plasma zinc concentrations than those supplemented with 90 ppm from zinc sulfate on day 84 of the growing phase. Plasma zinc was also higher ($P < 0.05$) in steers supplemented with 90 ppm from basic zinc chloride compared with those fed zinc sulfate on day 56 of the finishing phase. The higher plasma zinc concentrations in steers supplemented with basic zinc chloride are consistent with the higher zinc absorption observed for basic zinc chloride compared to zinc sulfate in growing steers (Shaeffer, unpublished). Regardless of zinc source, steers supplemented with 90 ppm zinc had higher ($P < 0.05$) plasma zinc concentrations than those fed 30 ppm during the growing (day 84) and finishing phases. Control steers had lower ($P < 0.05$) plasma zinc concentrations than steers receiving zinc supplementation during the finishing phase. In contrast Spears and Kegley

(2002) reported that the addition of 25 ppm of supplemental zinc to a control diet containing 26 ppm zinc did not affect plasma zinc in finishing steers.

Ruminal soluble zinc concentrations were increased by zinc supplementation to the control diet (Table 4). Steers fed basic zinc chloride had lower ruminal soluble zinc concentrations than steers fed zinc sulfate when zinc was added at 90 ppm. These findings suggest that basic zinc chloride is less soluble in the ruminal environment. This is also supported by research of Cao et al. (2000) indicating basic zinc chloride was insoluble in water but completely soluble in 0.4% HCL, 2% citric acid, and neutral ammonium citrate.

Liver zinc concentration was not affected by zinc source (Table 4). Steers receiving 90 ppm of supplemental zinc had higher liver zinc concentrations than steers supplemented with 30 ppm of zinc ($P < 0.05$). However, addition of 30 ppm of supplemental zinc did not increase liver zinc concentrations above those observed in controls. The higher liver zinc in steers supplemented with 90 ppm zinc is consistent with other studies (Wright and Spears, 2004; Kincaid et al., 1997) indicating that fairly high dietary zinc concentrations will elevate liver zinc concentrations.

Total ruminal volatile fatty acid concentration was lower ($P < 0.01$) when steers were supplemented with 90 ppm zinc sulfate verses 90 ppm zinc chloride during the growing phase (Table 5). Propionate molar proportion was higher ($P < 0.01$) in steers supplemented with 90 ppm verses 30 ppm of zinc. Steers supplemented with basic zinc chloride at 30 or 90 ppm of zinc also had higher molar proportions of propionate than steers fed similar concentrations of zinc

from zinc sulfate. Acetate:propionate ratio was lower ($P < 0.01$) in steers supplemented with 90 verses 30 ppm of zinc and tended ($P<0.01$) to be lower in steers fed basic zinc chloride when zinc was added at 90 ppm. During the finishing phase only total volatile fatty acid concentration was affected. Zinc sulfate at 30 ppm increased the total VFA concentrations when compared to the basic zinc chloride at the same level ($P < 0.05$).

Zinc level or source did not affect plasma alkaline phosphatase activity during the growing phase (Table 6). Zinc supplementation of the control diet increased ($P < 0.10$) plasma alkaline phosphatase activity during the finishing phase. Alkaline phosphatase is a zinc dependent enzyme, and these results suggest that a lack of dietary zinc limited activity of this enzyme during the finishing phase. Steers supplemented with 90 ppm of zinc from basic zinc chloride also had higher ($P<0.05$) plasma alkaline phosphatase activities than steers given 90 ppm of zinc from zinc sulfate. Previous research indicated that zinc supplementation to a corn silage-based control diet increased activity of alkaline phosphatase in heifers (Spears and Samsell, 1986). Alkaline phosphatase was not affected by supplemental zinc in lambs fed a forage diet but alkaline phosphatase was increased by zinc supplementation in lambs fed a concentrate based diet (Kegley and Spears, 1992).

Blood urea nitrogen levels were affected during the growing phase but no differences were seen during the finishing phase (Table 6). On day 28 of the growing phase steers supplemented with 90 ppm zinc sulfate had higher levels than those supplemented with 90 ppm from basic zinc chloride. At the day 84

sampling date in the growing phase there was a tendency for zinc supplementation to lower blood urea nitrogen levels ($P < 0.10$) and steers supplemented with 90 ppm of zinc sulfate had higher ($P < 0.05$) blood urea nitrogen concentrations than those fed 90 ppm zinc from basic zinc chloride.

Results from the present study suggest that supplementation of zinc from either zinc sulfate or basic zinc chloride results in similar performance of growing and finishing cattle. Marbling was the only carcass characteristic affected by dietary zinc. Marbling scores were higher in steers fed a level of 90 ppm zinc compared to 30 ppm but this affect was not observed as a difference in zinc source. Zinc source differences were only observed in plasma zinc concentrations on day 84 of the growing phase and day 56 of the finishing phase. Steers supplemented with basic zinc chloride had higher plasma zinc concentrations than those fed zinc sulfate when zinc was supplemented at 90 ppm. Basic zinc chloride at 90 ppm also increased plasma alkaline phosphatase activity compared to zinc sulfate. Steers fed 90 ppm of zinc from basic zinc chloride had a lower ruminal soluble zinc concentrations than those fed 90 ppm from zinc sulfate.

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Table 1. Ingredient Composition of Growing Diet

Ingredient	Percent DM
Corn silage	88.00
Soybean meal	9.06
Urea	1.06
Corn	0.36
Calcium sulfate	0.83
Calcium carbonate	0.48
Salt	0.20
Vit A, D & E[a]	0.01
Trace mineral mix[b]	0.01

[a]Provided per kg of diet: 2,643 IU of vitamin A, 881 IU of vitamin D_3, and 4.4 IU of Vitamin E.

[b]Provided per kg of diet: 20.0 mg Mn as $MnSO_4$, 10.0 mg Cu as$CuSO_4$, 0.5 mg I as $Ca(IO_3)_2 \cdot H_2O$, 0.1 mg Co as $CoCO_3$, and 0.1 mg Se as Na_2SeO_3.

Table 2. Ingredient Composition of Finishing Diet

Ingredient	Percent DM
Corn	85.51
Soybean meal	5.88
Cottonseed hulls	5.00
Urea	0.74
Calcium sulfate	0.78
Calcium carbonate	1.27
Potassium Chloride	0.58
Salt	0.20
Vit A, D & E	0.01
Trace mineral mix	0.01
Monensin	0.02

Table 3. Effect of Zinc Level and Source on Performance of Growing and Finishing Steers

	Treatment					
	1	2	3	4	5	
Item	Control	$ZnSO_4$ 30ppm	$ZnSO_4$ 90ppm	ZnCl 30ppm	ZnCl 90ppm	SEM
Growing phase						
Initial wt, kg	252.5	250.2	252.4	251.2	250.2	0.8
Final wt, kg	371.2	364.5	364.2	360.0	363.2	4.9
Gain, kg/d[a.b]	1.39	1.33	1.30	1.27	1.31	0.02
DMI, kg/d[c]	8.17	8.17	7.99	7.76	8.13	0.15
Gain:feed[a]	0.170	0.163	0.163	0.162	0.163	0.003
Finishing phase						
Final wt, kg	480.7	486.4	487.0	479.6	485.7	5.1
Gain, kg/d	1.41	1.46	1.48	1.46	1.48	0.06
DMI, kg/d	7.58	7.84	7.73	7.69	7.73	0.68
Gain:feed	0.186	0.186	0.193	0.190	0.191	0.009

[a]Control vs. zinc supplemented treatments($P < 0.05$).
[b]30 ppm $ZnSO_4$ vs. 30 ppm ZnCl ($P < 0.05$).
[c]30 ppm $ZnSO_4$ vs. 30 ppm ZnCl ($P < 0.10$).

Table 4. Effect of Zinc Level and Source on Plasma, Ruminal Soluble Zinc, Liver Zinc Concentrations of Steers

	Treatment					
	1	2	3	4	5	
Item	Control	$ZnSO_4$ 30ppm	$ZnSO_4$ 90ppm	ZnCl 30ppm	ZnCl 90ppm	SEM
Plasma zinc, mg/L						
Growing phase						
Day 28[a]	0.87	0.85	0.94	1.04	1.01	0.06
Day 84[b,c]	1.00	0.95	1.02	1.02	1.12	0.04
Finishing phase						
Day 56 [b,d,e]	0.79	0.82	0.96	0.94	1.13	0.05
Ruminal soluble zinc, mg/L						
Growing phase [b,c,f]	0.43	0.52	0.96	0.46	0.70	0.10
Finishing phase [d,e]	0.29	0.47	0.75	0.44	0.41	0.08
Liver zinc, mg/kg DM [b]	136.3	131.0	162.2	135.2	156.2	9.7

[a]30 ppm $ZnSO_4$ vs. 30 ppm ZnCl ($P< 0.05$).
[b]30 ppm vs. 90 ppm Zn ($P< 0.05$).
[c]90 ppm $ZnSO_4$ vs. 90 ppm ZnCl ($P< 0.10$).
[d]Control vs. Zinc supplemented treatments ($P< 0.05$).
[e]90 ppm $ZnSO_4$ vs. 90 ppm ZnCl ($P< 0.05$).
[f]Control vs. Zinc supplemented treatments ($P< 0.10$).

Table 5. Effect of Zinc Level and Source on Ruminal VFAs in Steers

	Treatment					
	Control	$ZnSO_4$ 30 ppm	$ZnSO_4$ 90 ppm	ZnCl 30 ppm	ZnCl 90 ppm	SEM
Growing phase						
Total VFA, mM[a]	88.2	78.0	62.0	82.5	90.1	5.2
Acetate, mol/100 mol	69.4	70.1	69.7	70.3	67.4	1.0
Propionate, mol/100 mol[b,c,d]	15.9	14.6	15.9	15.9	16.9	0.3
Acetate:propionate ratio[b,d]	4.39	4.82	4.44	4.54	4.04	0.16
Isobutyrate, mol/100 mol	0.87	0.74	0.48	0.59	0.75	0.13
Butyrate, mol/100 mol	10.8	11.5	11.3	10.7	12.1	0.5
Isovalerate, mol/100 mol	1.70	1.85	1.46	1.56	1.67	0.18
Valerate, mol/100 mol	1.37	1.24	1.15	1.04	1.18	0.20
Finishing phase						
Total VFA, mM[c]	131.9	136.4	124.9	101.1	106.3	10.0
Acetate, mol/100 mol	49.8	49.1	51.7	51.8	55.7	2.9
Propionate, mol/100 mol	35.3	41.5	40.0	37.6	35.5	3.3
Acetate:propionate	1.47	1.19	1.31	1.49	1.65	0.22
Isobutyrate, mol/100 mol	0.50	0.39	0.44	0.47	0.51	0.10
Butyrate, mol/100 mol	5.5	5.3	5.0	6.7	5.5	1.3
Isovalerate, mol/100 mol	0.69	0.51	0.63	0.65	0.83	0.11
Valerate, mol/100 mol	2.88	2.68	2.16	2.82	2.05	0.54

[a]90 ppm $ZnSO_4$ vs. 90 ppm ZnCl ($P < 0.01$).
[b]30 ppm vs. 90 ppm Zn ($P < 0.01$).
[c]30 ppm Zn SO_4 vs. 30 ppm Zn Cl ($P < 0.05$).
[d]90 ppm Zn SO_4 vs. 90 ppm ZnCl ($P < 0.10$).

Table 6. Effect of Zinc Level and Source on Plasma Alkaline Phosphatase Activity and Blood Urea Nitrogen in Steers

	Treatment					
	1	2	3	4	5	
Item	Control	$ZnSO_4$ 30ppm	$ZnSO_4$ 90ppm	ZnCl 30ppm	ZnCl 90ppm	SEM
Plasma Alkaline Phosphatase, U/L						
Growing phase						
Day 28	109.9	121.3	85.7	99.3	111.7	10.9
Day 84	89.1	109.2	91.8	99.8	105.9	10.9
Finishing phase						
Day 56 [a,b]	117.8	148.7	125.8	134.6	165.8	11.6
Blood urea nitrogen, mg/dL						
Growing phase						
Day 28 [c]	8.71	9.20	9.71	10.20	7.83	0.79
Day 84 [a,b]	15.49	13.85	14.97	12.27	11.02	1.11
Finishing phase						
Day 56	5.07	4.31	4.50	3.45	4.49	0.57

[a]Control vs. Zinc supplemented treatments ($P< 0.10$).
[b]90 ppm $ZnSO_4$ vs. 90 ppm ZnCl ($P< 0.05$).
[c]90 ppm $ZnSO_4$ vs. 90 ppm ZnCl ($P< 0.10$).

Table 7. Effect of Zinc Level and Source on Carcass Characteristics of Steers

	Treatment					
	1	2	3	4	5	
Item	Control	$ZnSO_4$ 30ppm	$ZnSO_4$ 90ppm	ZnCl 30ppm	ZnCl 90ppm	SEM
Hot carcass wt, kg	302.4	305.0	304.2	302.1	304.9	5.2
Dressing percentage	58.2	58.3	57.9	58.4	58.3	0.01
Marbling[a,c]	5.46	5.35	5.87	5.32	5.57	0.18
Quality grade[b]	16.7	16.8	17.3	17.0	17.1	0.33
Yield grade	2.38	2.47	2.43	2.45	2.41	0.11
Ribeye area, inches	12.1	12.3	12.4	12.6	12.4	0.27
KPH, %	2.43	2.49	2.58	2.56	2.56	0.08

[a]30 vs. 90 ($P< 0.05$).
[b]Select^{+} = 16; Choice^{-} = 17; Choice^{0} = 18.
[c]Slight = 4; Small = 5; Modest = 6.

Chapter 3

Bioavailability of Basic Zinc Chloride Relative to Zinc Sulfate in Steers[1]

G. L. Shaeffer, J. W. Spears[2], and K. E. Lloyd

Department of Animal Science and Interdepartmental Nutrition Program

North Carolina State University

Raleigh, NC 27695-7621

[1]Use of trade names in this publication does not imply endorsement by the North Carolina Agricultural Research Service nor criticism of similar ones not mentioned.

[2]Correspondence: Campus Box 7621; North Carolina State University, Raleigh, NC 27695-7621; (Phone: 919-515-4008; Fax 919-515-4463; Email: Jerry_Spears@ncsu.edu).

Introduction

Zinc is widely supplemented to ruminant diets. It is important that zinc be supplemented to diets in a form that is highly bioavailable to the animal. Research in chicks has indicated that bioavailability of zinc from basic zinc chloride is similar to reagent-grade zinc sulfate (Cao et al., 2000; Batal et al., 2001). Solubility in solution is one point at which zinc sulfate differs from basic zinc chloride. Basic zinc chloride is insoluble in water while zinc sulfate is soluble suggesting that basic zinc chloride would be poorly soluble in the rumen. A low solubility of zinc in the rumen may prevent interactions of zinc with other components in the rumen, and thus increase bioavailability of zinc. Basic zinc chloride has not been evaluated as a zinc source for ruminants. The present experiment was conducted to compare apparent absorption and retention of zinc from basic zinc chloride and zinc sulfate in growing steers.

Materials and Methods

This trial was conducted at the Butner Beef Cattle Field Laboratory, under the direction of University Field Laboratories North Carolina State University. The cowherd on the university research facility produced cattle used in this trial. Several weeks prior to the start of the trial cattle were vaccinated for IBR, BVD type I and II, PI3, and BRSV using Titanium 5 (Diamond Animal Health, Des Moines, IA) and for Clostridium chauvoei (Blackleg), septicum (Malignant edema), novyi (Black disease), sordellii and perfringens types C and D (enterotoxemia) using Vision 7 (Intervet Inc., Millsboro, DE). Cattle were also

dewormed using Cydectin Pour-on manufactured by Fort Dodge (Fort Dodge, IA). Basic zinc chloride was supplied by Micronutrients, A Division of Heritage Technologies, LLC (Indianapolis, IN) and the zinc sulfate originated from Eastern Minerals (Henderson, NC). Care, handling, and sampling of the animals used in the trial was approved by the North Carolina State University Institutional Animal Care and Use Committee.

Eight Angus and eight Simmental steers averaging 371 kg were randomly separated into two groups of eight animals, four of each breed per group. Animals were housed in covered, concrete, slotted floor pens designed by the Hog Slat company and fed individually using electronically controlled feeders (American Calan, Northwood, NH). All steers were fed a diet (Table 1) without supplemental zinc for 14 d in an attempt to deplete the animals of zinc. Feed was analyzed by ingredient in duplicate because of the difficulty in sampling and repeatability. The basal diet analyzed 29.8 ppm and the cottonseed hulls 10.4 ppm of zinc. The complete diet contained approximately 25 ppm zinc. Steers were then randomly assigned to receive the low zinc diet supplemented with 25 ppm zinc from either zinc sulfate (analyzed 32.2%) or basic zinc chloride (analyzed 54.4%). The two groups of animals started the study 14 days apart. This time was needed for adequate clean up time of crates and facilities between collection period of the two groups, because of limited number of metabolism crates. After 4 days consuming the zinc supplemented diets, steers were placed in elevated stainless steel metabolism crates for a three-day adaptation followed by a five-day total urine and fecal collection. Feed intake was restricted when in

the metabolism crates to minimize orts. Intake was determined from the average intake from the three adaptation days and then adjusted daily. At the end of the collection period steers were returned to the group pens and were fed using electronic Calan gates for an additional 28 d. Body weights were taken prior to feeding at the start and end of the trial. Interim weights were taken on day 1 of zinc supplementation, and the day animals were placed in metabolism crates. Blood samples were taken on d 1 and at the end of the study for both groups to determine plasma zinc concentrations. Samples were obtained 2 h post feeding on all sample days and collected via jugular venipuncture in heparinized vacuum tube designed for trace mineral analysis (Becton Dickenson, Rutherford, NJ). Plasma was obtained following centrifugation and measured by atomic absorption spectroscopy (AA-6701F, Shimadzu Scientific Instruments, Kyoto, Japan).

Total feces and urine collection lasted for a total of five days in which both were thoroughly mixed and an aliquot was saved for analysis. Urine was preserved by the addition of 150 ml of 6N hydrochloric acid to the urine pan prior to the attachment to the metabolism crate daily. Urine pan was weighed daily at the same time and a 1.0% aliquot was composited and frozen over the five day collection period. Feces were collected in fecal pans, which were lined with plastic. Each day at the same time, collection began by the removal of the plastic and sample and a new piece of plastic lined the pan. Plastic was used to tare the scales and then each sample was weighed, mixed thoroughly and a 10% aliquot was retained and frozen daily for later subsampling from all five days.

Fecal samples were dried in an oven at 55°C, weighed and mixed frequently until no additional weight was lost, then composited over the 5 day period for each steer and subsampling to reduce quantity of feces per animal.

Feed samples were collected every time feed was mixed and a sample of the total mixed diet was obtained daily during the collection period. All orts during the collection period were taken in order to determine total zinc intake, excretion, and retention. Samples of feed, orts, and feces were ground in a Wiley mill to pass through a 2 mm screen. Feed, orts, fecal, and urine samples were prepared for zinc analysis by wet ashing using nitric acid and hydrogen peroxide in a microwave digester (Mars 5^{TM}, CEM Corp., Matthews, NC). Zinc was measured by atomic absorption spectroscopy (AA-6701F, Shimadzu Scientific Instruments, Kyoto, Japan).

Statistical Analysis

Data were analyzed statistically by GLM procedure of SAS using steer, treatment, group and a treatment x group interaction as the model (SAS 1990). Significance was declared at $P < 0.10$. Zinc intake was also used as a covariate because of the higher zinc intake for the basic zinc chloride group. Plasma initial values were also used as a covariate for the end of treatment plasma zinc values.

Results and Discussion

Weight gain was not affected by zinc source during the study. Plasma zinc concentrations were affected by treatment on the final day of the trial (Table

4). Basic zinc chloride fed steers had a higher (P<0.03) zinc concentration (1.19 ppm) than those animals receiving zinc sulfate (1.05 ppm). Group 2 steers also had higher plasma zinc concentrations than those in group 1 did but there was not a treatment x group interaction. When initial plasma zinc values were used as a covariate for final plasma zinc concentrations, steers supplemented with basic zinc chloride still had higher plasma zinc concentrations.

During the metabolism study dry matter intake was similar across treatments (Table 2). Zinc intake during the collection period was not significantly affected by treatment but tended to be slightly higher for steers supplemented with basic zinc chloride. Because zinc intake tended to be higher for the basic zinc chloride treatment, data was statistically analyzed with (Table 3) and without (Table 2) zinc being used as a covariate. Regardless of whether zinc intake was used as a covariate, apparent absorption of zinc, expressed as percent of zinc intake or mg/day, was higher (P < 0.01) for steers supplemented with basic zinc chloride. Total fecal excretion of zinc was lower for steers fed basic zinc chloride when zinc intake was used as a covariate (Table 3). Urinary excretion of zinc was low and not affected by zinc source. The amount of zinc retained was higher (P < 0.01) in steers supplemented with basic zinc chloride. These results indicate that basic zinc chloride was absorbed to a greater extent than feed grade zinc sulfate by growing steers. The greater absorption of zinc may relate to zinc in the basic zinc chloride form interacting to a lesser extent with other dietary components and products of ruminal fermentation in the ruminal environment.

Shaeffer et al. (unpublished) also looked at basic zinc chloride compared to zinc sulfate in finishing steers. Plasma zinc concentrations were similar. In the current study and the finishing study, plasma zinc concentrations tended to be higher in steers fed basic zinc chloride than those being fed zinc sulfate. Spears and Kegley (2002) fed a control diet containing 26 ppm zinc and supplemented 25 ppm zinc. They did not see an affect on plasma zinc concentrations. When zinc chloride is fed to chicks it was found that zinc sulfate and zinc chloride were equal in bioavailability as well (Cao et al., 2000; Batal et al., 2001). Through this current study and Shaeffer et al. (unpublished) using finishing steers, basic zinc chloride is equal in value to zinc sulfate when fed to steers. Once the basic zinc chloride is marketed and if the price is similar to zinc sulfate, basic zinc chloride could be an option for an additional zinc source in the cattle industry.

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Table 1. Composition of Basal Diet

Ingredient %	Percent DM
Corn	50.3
Cottonseed Hulls	40.0
Soybean Meal	4.0
Urea	1.5
Calcium Sulfate	0.4
White Salt	0.17
Monocalcium Phosphate	0.52
Calcium Carbonate	1.00
Vitamin Mix[a]	0.01
Trace Mineral Mix[b]	0.10
Zinc Treatment[c]	2.00

[a]Contained per kilogram of premix: vitamin A, 2643 IU; vitamin D_3, 881 IU;and Vitamin E, 4.4 IU.

[b]Provided per kilogram of supplement: 10 mg of Cu as $CuSO_4$, 20 of Mn as $MnSO_4$, 0.5 mg I as $Ca(IO_3)_2(H_2O)$, 0.1 mg Co as $CoCO_3$, and 0.1 mg Se as Na_2SeO_3.

[c]Finely ground corn as a carrier to provide 25ppm zinc from either zinc sulfate or zinc chloride

Table 2. Effect of Zinc Source on Zinc Excretion, Apparent Absorption and Retention in Steers

	Zinc Source			
Item	Sulfate	Basic Chloride	SE	P value
DM intake, kg/d	7.30	7.14	0.40	0.78
Zinc intake, mg/d	356.6	399.2	25.2	0.26
Fecal zinc, mg/d	320.8	321.0	23.2	0.99
Urine zinc, mg/d	1.15	1.21	0.14	0.77
Absorbed zinc, % of intake	9.9	19.8	1.4	0.01
Absorbed zinc, mg/d	35.8	78.3	5.0	0.01
Retained zinc, mg/d	34.7	77.0	5.1	0.01

Table 3. Effect of Zinc Source on Zinc Excretion, Apparent Absorption and Retention in Steers (zinc intake used as a covariant)

	Zinc Source			
Item	Sulfate	Basic Chloride	SE	P value
Fecal zinc, mg/d	341.9	303.5	4.8	0.01
Urine zinc, mg/d	1.16	1.20	0.14	0.87
Absorbed zinc, % of intake	9.7	19.9	1.4	0.01
Absorbed zinc, mg/d	38.0	76.4	4.7	0.01
Retained zinc, mg/d	36.9	75.2	4.8	0.01

Table 4. Effect of zinc source on weight gain and plasma zinc concentrations

Item	Zinc Source		SE	P value
	Sulfate	Basic Chloride		
Weight Gain, kg				
Start trt	397.5	404.0	23.48	0.67
Final	438.6	434.1	16.55	0.68
Plasma Zinc Concentrations, mg/L				
Initial	1.07	1.07	0.05	0.92
Final	1.05	1.19	0.05	0.03