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THE EFFECTS OF TRACE MINERAL SUPPLEMENTATION ON PERFORMANCE,
HEALTH, AND CARCASS QUALITY OF AT-RISK MINERAL DEFICIENT

FEEDLOT CATTLE

by

Tevan J. Brady

A Thesis proposal submitted in partial fulfillment
of the requirement for the degree

of

MASTER OF SCIENCE

in

Animal Nutrition

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2021

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ABSTRACT

The Effects of Trace Mineral Supplementation on Performance, and Carcass Quality of
At-Risk Mineral Deficient Feedlot Cattle

by

Tevan J. Brady, Master of Science
Utah State University, 2021

Major Professor: Kara Thornton-Kurth, Ph.D.
Department: Animal, Dairy, & Veterinary Sciences

Disease is the main cause of morbidity and mortality in feedlot cattle. Trace minerals are crucial in the immune response to disease and are important to the health and performance of stressed feedlot cattle. As such, mineral deficient cattle that enter a feedlot have higher morbidity and mortality rates and lower feedlot performance. The objective of this research was to determine best practices that feedlot producers can employ when receiving mineral deficient cattle. Forty steers were stratified by initial body weight and mineral status then assigned to one of four treatment groups: no mineral supplementation (CON; n = 10), oral supplementation of minerals provided at levels similar to NRC requirements (AM; n = 10), oral supplementation of minerals provided at levels above NRC requirements (HM; n = 10), or a MultiMin® injection at labeled dose (MM; n = 10). All steers received the same ration in pens equipped with GrowSafe® bunks. Standing liver biopsies were collected on d 0, 5, 10, 20, 30 and 40 and analyzed for mineral content. Cortisol levels were measured to determine the animal's level of stress. Average daily gain (ADG), dry matter intake (DMI) and feed efficiency (FE) were recorded. After the trial, steers were harvested at a commercial facility and carcass data

was obtained. There were no differences ($P > 0.05$) between treatments in weight or overall ADG. When analyzed over time, the HM treatment had an effect ($P < 0.001$) on liver copper and selenium concentrations, but not on liver manganese and zinc concentrations. Additionally, when DMI was analyzed over time, effects ($P < 0.05$) of time, treatment, and treatment*time were observed, and effects ($P < 0.05$) of time and treatment*time were observed on FE. Treatment had no effect ($P > 0.05$) on carcass or ultrasound data. No effects ($P > 0.05$) were seen on BHV or BPIV3 antibody titers. Steers in the HM treatment had an increased ($P < 0.05$) number of bunk visits/feed bouts, and time duration at each bunk visit/feed bout, and had decreased ($P < 0.05$) consumption per bunk visit/feed bout, and time with their head down per bunk visit/feed bout when compared to steers receiving the other three treatments. These data indicate that feeding varying mineral levels can alter performance and health of an animal, but more research is needed to determine the effects of specific minerals on an animal's performance and health.

(99 Pages)

PUBLIC ABSTRACT

The Effects of Trace Mineral Supplementation on Performance, and Carcass
Quality of At-Risk Mineral Deficient Feedlot Cattle

Tevan J. Brady

Morbidity in feedlot cattle due to disease is a common economical loss for feedlot producers. Utah is not typically considered a ‘feedlot state’, but there are several producers in the southern part of the state that specialize in receiving at-risk cattle. These cattle are at-risk because they are coming from areas known to be mineral deficient. Areas such as the western US, are known to be deficient in several trace minerals important in immune response. Therefore, it is critical that producers have an adequate plan to decrease the negative effects that this has on economic viability of their beef operations, especially for those feedlots specializing in receiving at-risk cattle. In this study yearling Black Angus steers received one of four treatments: no mineral supplementation, oral supplementation of minerals provided at levels similar to NRC requirements, oral supplementation of minerals provided at levels above NRC requirements, or a MultiMin® injection at labeled dose. Liver and blood samples were collected to measure mineral status and cortisol levels. Backfat and ribeye area measurements were also recorded. Weight of the steers were recorded every two weeks to determine average daily gain and feed:gain ratio. At the end of the study, steers were harvested at a local harvesting plant and carcass traits were obtained and recorded. There were no differences in weight gain, overall average daily gain, dry matter intake, or feed:gain ratio between the different treatment groups. Steers that were fed the HM had

increased liver copper and selenium concentrations, but there was no effect observed on liver manganese and zinc concentrations. Carcass quality and antibody titers did not differ between the different treatments. These data indicate that different mineral supplementation strategies have a diverse effect on feedlot performance and liver mineral concentrations over time, and additional research is needed to better understand these differences.

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CHAPTER I

INTRODUCTION

The main source of morbidity (sickness) and mortality (death loss) of feedlot cattle is disease (Smith, 1998). It has been estimated that feedlots naturally incur anywhere between 5 and 15% morbidity (Edward, 1996). However, as high as 44% morbidity have been reported by some operations (Snowder, 2006). Management of morbidity and mortality is critical to maintaining productivity of a feedlot operation. Although mortality is a main concern for producers, morbidity can have a greater economic impact than mortality. When all costs that are associated with morbidity, such as medication, labor, premature culling, and reduced performance of the animal are considered it results in less of an economic return for producers (Smith, 1998).

Bovine respiratory disease (BRD) is a common and costly disease of feedlot cattle (NAHMS, 2000a). Majority of all deaths caused by BRD happen briefly after arriving at a feedlot, normally within the first 45 d (Loneragan et al., 2001). In order to treat BRD in at-risk, stressed newly received feedlot cattle the use of prophylactic antibiotics is the most common practice. The use of these antibiotics helps producers ameliorate the effects of morbidity and mortality (Duff and Galyean, 2007). However, with the increase in antibiotic resistant bacteria, this has led to social and scientific concern that the overuse and misuse of human prescribed antibiotics in conjunction with the increased and wide-spread use of sub-therapeutic doses of antibiotic in agriculture may play a role in this trend (Sarmah et al., 2006, Smith et al., 2002). The three main concerns that consumers have with modern food production systems are food safety, protection of the environment and animal welfare (de Passillé and Rushen, 2005). To many consumers, these three

aspects are linked; it is believed that improved methods to raise animals will simultaneously result in better and safer food, reduced environmental concern and improved animal welfare (Fraser, 2001).

On January 1, 2017 the Veterinary Feed Directive (VFD) was implemented to help reduce the use of antibiotics in agriculture. The VFD was developed by the Food and Drug Administration (FDA) to help with three criteria which include: lower antibiotic resistance, reduce over used antibiotics and control antibiotics in the human food supply (Griffin, 2016a). In order for a producer to feed certain antibiotics, a VFD must be obtained from a veterinarian. A VFD can be used for three different things: prevention of disease, treatment of disease, and control of disease (Griffin, 2016a). However, the VFD only applies to antibiotics that are used in feed, it does not affect all feed additives such as ionophores, coccidia and other parasitic/insect control drugs (Griffin, 2016b). The VFD does not apply to antibiotics used by injection, tablet, bolus or water (Griffin 2016b). Prior to implementation of the VFD, provision of sub-clinical levels of antibiotics in the feed was commonly used for at-risk feedlot cattle as a prophylactic prevention. However, with the implementation of the VFD, it is more difficult for producers to utilize this tool and new avenues must be discovered to mitigate the effects of feeding at-risk feedlot cattle, which will allow us to improve animal welfare and health of feedlot animals.

Trace minerals are vital in immune response and are particularly important to both the performance and health of stressed feeder cattle (Underwood and Suttle, 1999). Trace mineral deficiencies can result in a reduction in forage intake, decreased reproductive efficiency, decreased immune function, limited daily gains and feed conversion and can

also compromise enzyme function (Paterson and Engle, 2005). The western US is known to be deficient in several trace minerals, especially in copper (Cu), zinc (Zn), selenium (Se), and manganese (Mn).

Although the western US is not your typical feedlot state, there are many smaller feedlots in the area that specialize in receiving at-risk cattle. As of March 1, 2020, at-risk cattle contributed to 20% of the 1.65 million head in feedlots across the US (USDA NASS, 2020). In the state of Utah producers report that the percent of at-risk cattle is higher than the national average due to having feedlots that specialize in at-risk cattle. Animals considered at-risk have an increase susceptibility to disease. Some of these feedlot cattle are at-risk because they have: 1) a poor mineral status, 2) recently been weaned with no vaccine history, 3) comingled with other animals from different sale barns or ranches, 4) been transported for an extended period of time or 5) come from a ranch with poor management practices.

Common practices in the Intermountain West include ranging cattle on often unfarmable land for long periods of time. This makes it difficult or impossible in some scenarios, to provide mineral supplementation. This makes it very important for producers to know and understand the limitations of the feedstuffs in this area and what they can do to mitigate negative side-effects that may be associated with grazing cattle in mineral deficient areas.

Another factor that is causing the nutrient composition of feeds to become more variable is the changing climate (Cohn et al., 2017). Cattle that graze on pastures typically don't receive their mineral requirements based on a diet composed of primarily forages (Underwood, 1981; McDowell, 1992). Cattle on pastures can receive a certain

level of minerals from water and soil ingestion; however, forage consumption is the main dietary source of minerals. Of the mineral elements in soils, only a fraction is taken up by plants (McDowell, 1996). Although the quantity of minerals that is consumed from these sources might not supply an adequate amount to the animal unless some type of supplementation is added (McDowell, 1996). There are many factors that can contribute to the uptake of trace minerals in plants such as pH of soil, maturity, yield, species, management, and climate (Reid and Horvath, 1980; McDowell, 1985). The levels of Cu, Iron (Fe), Se, Cobalt (Co), Zn, and Molybdenum (Mo) have a tendency to decline as a plant matures (Reid and Horvath, 1980).

Therefore, it's vital that managerial practices be implemented to address the seasonal and regional changes in forages, allowing ranchers to produce healthy, profitable cattle (Duff and Galyean, 2007). Receiving cattle that have a negative mineral status will not be as responsive to vaccinations as cattle that have an adequate mineral status. Thus, they have an increased risk of morbidity/mortality which will result in an economic loss for the producers. The objective of this research was to determine the effects of providing natural, trace mineral supplementation to increase cattle health and possibly eliminate the need to use prophylactic antibiotics and develop safe receiving protocols that can be followed to improve welfare, health, and performance of feedlot animals.

CHAPTER II

LITERATURE REVIEW

Feedlot Performance

Lightweight stressed calves will have a reduction in feed intake in the first two weeks after arrival to a feed yard (Hutcheson and Cole, 1986). Bovine respiratory disease (BRD) has been known to decrease feed intake and efficiency of animals (Hutcheson and Cole, 1986). One way that BRD can reduce feed intake is by creating an inflammatory response, which causes proinflammatory cytokines to be released and can decrease feed intake by more than 50% (Bosi and Trevisi, 2006). It has been reported that as much as \$750 million annually has been lost in the industry due to BRD (Chirase and Greene, 2001). Feedlot cattle that have been treated once for BRD have an economic return of \$40.64 less, those that receive treatments twice bring \$58.35 less, and those feedlot cattle treated three or more times return \$291.93 less than cattle that are not treated for BRD (Fulton et al., 2002). Steers that have received treatment have been reported to have a 4% decrease in average daily gain (ADG), 2.6% loss in hot carcass weight (HCW) and a 1.7% reduction in body weight (BW) (Gardner et al., 1999). Bateman et al. (1990) conveyed in a study that calves who had been treated for BRD had a 0.6 kg lower ADG than those not treated.

Holland et al. (2010) examined the effects of BRD during a 63 d preconditioning period using 360 heifers. During the preconditioning period there was a decrease in ADG as the number of vaccinations increased (Holland, et al., 2010). However, in the finishing phase there was not a difference in ADG between the different treatments (Holland et al., 2010). Dry matter intake (DMI) also was decreased as the number of vaccinations during

the preconditioning phase increased (Holland et al., 2010). When the cattle were fed to the same endpoint, cattle that were treated for BRD were able to regain what they had lost in the preconditioning phase, except those that were labeled as chronically ill (Holland et al., 2010).

Schneider et al., (2009) examined the effects of BRD on 5,976 animals located in multiple Midwest feedlots (Schneider et al., 2009). Schneider reported that BRD when recorded on treatment record or had pulmonary lesions at slaughter resulted in a reduction of ADG, lighter HCW, less internal fat, and had a decrease in marbling score than steers that remained healthy through the feedlot (Schneider et al., 2009). Hot carcass weight and marbling score of individuals showed a significant effect with reductions of 8.16 kg and 0.13, respectively, in treated cattle (Schneider et al., 2009). It is clear that there is a decrease in performance from calves that are treated for BRD. In addition to decreased performance, BRD can also have a negative effect on carcass quality and retail yield (Gifford et al., 2012).

Carcass Quality

Current research shows that even though some diseased animals can be fed similarly to healthy animals, carcass weights and marbling scores are still reduced when compared to their healthy counter-parts (Maxwell et al., 2018). There is evidence that respiratory disease can influence multiple carcass traits such as carcass weight, marbling and subcutaneous fat cover (McNeil et al., 1996). In a study by Gardner et al. (1999) it was found that 33% of the steers in the study had lung lesions symptomatic of BRD. These steers with lung lesions had a lighter HCW, lower dressing percent, less internal fat and received a lower marbling score (Gardner et al., 1999). They also reported that they

had smaller rib eye area and not as much external fat than those steers that did not have lung lesions (Gardner et al., 1999). Even though research shows that diseased animals can be fed similarly, they will not have the same carcass quality when compared to those animals that are healthy.

Roeber et al., 2001 researched the variation in economic returns from calves that were purchased from producers that took part in some type of a “preconditioning program.” Preconditioning programs serve to help animals go from a range setting to a feedlot setting. They accomplish this by introducing a bunk where the animals eat and feed small amounts of concentrates, so they are accustomed to eating grains. It was found that those producers whose cattle came from a preconditioning program had significantly improved feedlot performance than those that did not come from a preconditioning program. Cattle that were preconditioned had significantly higher ADG, had lower mortality and morbidity rates, and were more efficient in the feedlot setting (Roeber et al., 2001). By preconditioning cattle this allows them to be located in a more accessible area, making it easier for producers to deliver trace minerals to them. Supplementing trace minerals results in animals with an improved mineral status, which results in improved immune responses and performance in the feedlot.

Garcia et al., 2010 looked at the effects of BRD on carcass quality and meat quality traits in two different herds. This group saw cattle that demonstrated signs of BRD had a 5 kg lighter HCW and had between 10 – 15% less 12th rib fat thickness, which in turn resulted in a decrease in USDA yield grade (Garcia et al., 2010). Schneider et al., 2009 reported that as the number of treatments for BRD increased, there was more variation of HCW, subcutaneous fat cover, and marbling score (Schneider et al., 2009).

This same group found that cattle treated for BRD had a reduction of 0.58 cm² for longissimus muscle area, and a 0.76 mm reduction in fat cover (Schneider et al., 2009). Results from this study also showed that greater than 71% that were never treated graded choice or higher whereas cattle that were treated once, twice, or three times graded choice or better 57%, 55% and 52% respectively of the time (Schneider et al., 2009). This research provides further evidence that feedlot cattle that have suffered from disease produce a less than desirable quality grade than those that are healthy.

Respiratory morbidity reduces performance of beef steers which effects their ability to deposit fat and effects their carcass weight and longissimus muscle area (Gardner et al., 1999). Research shows that fat deposition by cattle is mostly affected, yet there is still a decrease in growth and reduced DMI that can occur as demonstrated by a low HCW (Gardner et al., 1999). It is apparent that BRD can have negative effects on carcass quality. McNeill et al. (1996) detected that in his evaluation of more than 7,000 cattle, 39% were never treated for BRD and they graded USDA Choice or better while only 27% of those treated for BRD were graded USDA choice or better. The results from several different studies demonstrate that cattle affected by BRD will have a decrease in multiple carcass traits such as: smaller ribeye area, lighter HCW and a lower marbling score. Implementation of a preconditioning program allows producers to supplement minerals to the animals prior to the feedlot, which has been found to increase immune system response, feedlot performance and carcass quality (Garcia et al., 2010).

Immunity to Potential Virus

Bovine respiratory disease (BRD) is a common issue affecting health of cattle herds (Gulliksen et al., 2009). It has been estimated that feedlots can naturally incur

anywhere between 5 and 15% morbidity. However, as high as 44% morbidity have been reported in some operations (Snowder, 2006). The spread of BRD infections among young animals is extremely quick. About 91% of calves will be infected with BRD within the first 27 d after arrival to a feedlot (Buhman et al., 2000). Bovine respiratory disease is a complex of diseases which are categorized by different types of infections. Each type has its different causes, signs and implications. Viral causes for BRD include: Bovine Herpes Virus 1 (BHV-1), Bovine Parainfluenza Virus 3 (BPIV3), Bovine Viral Syncytial Virus (BRSV), Bovine Viral Diarrhea Virus (BVDV), and Bovine Coronavirus (BCV). Bacterial causes for BRD include *Mannheimia haemolytica*, *Pasturella multocida*, *Haemophilus somnus* and *Mycoplasma* (Ellis, 2001). Shipping and processing calves at feedlots can increase the cause of stress. This can have a major impact on immune function, predisposing them to developing BRD. Preparing an animal's immune system before experiencing stressful events with vaccines against viral and bacterial agents involved in BRD is a critical piece to maintaining an animal's health for cow-calf, stocker, and feedlot operators (Taylor et al., 2010). The immune system can be divided into two parts, innate and acquired immunity. Special sensors that can recognize self and non-self factors that are expressed during infection are used to induce an immune response. Components that contribute to the innate and adaptive immune response will act together in mediating a defense against infections (Biron, 2016). The innate system is the first line of defense against pathogens and therefore plays a critical role in the early recognition and triggering of a proinflammatory response to the invading pathogens (Meylan, 2006).

Sentinel cells such as macrophages, dendritic cells and mast cells come equipped with a diverse number of pattern recognizing receptors that will recognize pattern associated molecular patterns (PAMP's) located on invading pathogens (Marshall et al., 2018). Once a sentinel cell has recognized a PAMP, this will trigger a cascade eventually resulting in the release of cytokines such as: tumour necrosis factor alpha, interleukin-1, and interleukin 6 (Mogensen, 2009). Cytokines are short lived proteins that are critical to cell recruitment and to local inflammation which is essential for the clearance of many pathogens (Biron, 2016). Other characteristics of cytokines include pleiotropism, the ability to act on multiple cells types, and they can not only function locally but also can act systemically (McDonald and Levy, 2013). These cytokines recruit neutrophils which are the first cell type to arrive at the site of damaged tissue. Activation of neutrophils leads to a respiratory burst and the release of granules to control bacterial growth (Biron, 2016). Neutrophilic granules contain certain enzyme pathways that assist in the elimination of pathogenic microbes (Marshall et al., 2018). Macrophages are long-lived cells that have phagocytic properties and can sustain prolonged phagocytic activity. They are also involved in antigen presentation to T cells (Marshall et al., 2018). Macrophages and other antigen-presenting cells (APC) such as dendritic cells also display antigens to T cells in order to activate them (Ott, 2019).

The adaptive immune system is responsible for the elimination of pathogens several days after exposure to a pathogen and in the generation of immunological memory (Mogensen, 2009). Adaptive immunity is a group of specific second-line defense responses that occur days to weeks after exposure to microbial antigens during the innate immune response (Snyder, 2017). Lymphocytes are the cells responsible for

mounting an adaptive immune response. Adaptive immunity can be broken down into two subgroups, humoral and cell-mediated immunity (Abbas et al., 1991). B lymphocytes are what mediate the humoral system. These B cells turn into anti-body producing cells such as plasma and memory cells in order to provide protection against extracellular microbial infections by responding to antigens (Johnson et al., 1998). B cells contain B cell receptors (BCR) which can be membrane bound immunoglobulins or soluble immunoglobulins (antibodies) that bind to foreign pathogens. B cell receptors are formed through somatic recombination which creates a vast selection of sequences therefore increasing an individual's ability to recognize and respond to a wide range of antigens giving the animal more protection (Hoehn et al., 2016). B cell receptors consist of two large heavy chains and two smaller light chain molecules. These heavy and light chains are held together by di-sulfide bonds. One result of diversity in the BCR is a result of the random arrangements of heavy chain V, D, and J genes, and light chain V and J genes. Light chains can also be classified as either kappa or lambda light chains; this is known as combinatorial diversity (Collins and Watson, 2018). Another result of diversity of B cells is due to junctional diversity. This is accomplished by the extent of which random nucleotides are either inserted or deleted between joining genes (Jackson et al., 2013).

In the cell-mediated system, T cells are responsible for providing a defense against intracellular pathogens (Johnson et al., 1998). T cells can take many forms. Two forms are T helper cells (TH1, TH2) or Cytotoxic T Lymphocytes (CTL). T cells have two basic methods for protecting an individual from pathogens: 1) Cytotoxic T cells, 2) T cells that secrete cytokines (Roth and Perino, 1998). Cytotoxic T Lymphocytes can recognize antigens that are presented on MHC-I proteins on APC's. When activated,

CTL's will kill virally infected cells (Andersen et al., 2006). Cytotoxic T Lymphocytes (CTL's) can either bind to a target cell and release cytotoxic granules (perforin and granzymes) to kill the target cell or the CTL activates death receptors (CD95 and CD95L) which bind to the cell and trigger apoptosis through the caspase cascade (Tizard, 2012). T cells that can secrete cytokines are TH1 and TH2 cells. T helper 1 cells secrete cytokines such as interferon 2 and interferon alpha which help to increase cytolytic activity of CTL's, macrophages, neutrophils, and natural killer cells. T cells are also important in the activation of antibody producing B cells (Roth and Perino, 1998). T helper 2 cells are responsible for secreting cytokines like interleukin 4 and interleukin 10 and help stimulate B cells to produce certain antibodies (Tizard, 2012). Therefore, the production of normal antibody response involves the help of T lymphocytes (Roth and Perino, 1998).

For years it has been known that stress can have negative impacts on the health of an individual (Webster, 2008). There is an abundance of research that explains that there is an increase in disease susceptibility when immunity has been impaired due to environmental, physiological, or physical stress (Kelly, 1985; Kelly, 1980). A normal reaction the body has against stress is to release the hormone adrenocorticotrophic (ACTH) from the pituitary gland. This hormone acts on the adrenal cortex to synthesize and release cortisol (Roth, 1985). Increased levels of cortisol in the blood due to an animal experiencing some type of stress has been shown to have negative effects on the immune system (Webster et al, 2008). Some of these effects include decreased lymphocyte proliferation, reduction in number and activity of NK cells, decreased antibody response from B cells to viral infection, and reactivation of prior viral pathogens an animal was exposed to (Webster et al, 2008).

Stress can be placed into two categories depending on the duration that the stress is applied. Acute stress comes from exposure to stress for a short amount of time. This type of stress is thought to actually act as a priming mechanism helping the body prepare for a possible invasion of pathogens (Hughes et al., 2014). The second category is chronic stress. This results from an animal being exposed to stress for long periods of time. When this occurs, it switches the body from preparing to suppressing the immune system (Carroll and Forsberg, 2007). One way the immune system gets suppressed is by the continual release of glucocorticoids. Effector cells (such as B and T cells) can be stimulated by glucocorticoids, but with chronic stress they are constantly being prepared for an invasion by pathogens (Carroll and Forsberg, 2007). With the constant stimulation these effector cells can become desensitized and lose the ability to respond to invading pathogens. Also, with increased concentrations of glucocorticoids pre B and pre T cells can increase participation in cell death through apoptosis (Fraker and King, 2004).

Cattle going to feedlots experience a number of stressful events such as vaccinations, weaning, social mixing, shipping, and long periods without feed or water. Past studies have shown that stress-induced glucocorticoid concentrations due to transportation to feedlots result in an increase in the number of animals with shipping fever (Mormede et al., 1982; Mackenzie et al., 1997). With stress causing a suppression in the immune system, this doesn't allow the body to maintain a homeostatic balance which is needed for maximum livestock production, where growth is a priority (Hughes et al., 2014). This can result in large economical losses to the producers, it has been estimated that \$750 million have been lost due to BRD (Chirase and Greene, 2001). One

way that an animal can overcome infection may be influenced by the animal's mineral nutrition (Orr et al., 1990).

Minerals

It is well established that both vitamin and mineral deficiencies can have a severe impact on productivity of livestock animals (Galyean et al., 1999). The general physiological functions of trace minerals are to promote normal tissue growth, homeostasis, enzyme function, and cell regulation. Paterson and Engle (2005) and McDowell (1996) have suggested that there are five factors that have an impact on the amount of trace minerals required by cattle, and include: animal age, stage of production (growth, lactation or gestation), breed, stress and other mineral antagonists. It is imperative that trace elements are maintained within a small concentration in the body. If proper trace mineral homeostasis is maintained, optimum growth, health, and productivity of domestic livestock can be ensured (Underwood and Suttle, 1999). Immune cells require an adequate supply of trace elements to support their function and structure of metalloproteins that participate in processes such as energy production and protection against reactive oxygen compounds (Failla, 2003).

The western US is known to be deficient in many minerals that are essential for the immune system. Several trace minerals, such as Zn, Cu, and Se, are known to have positive effects on the immune system when provided at adequate levels (Galyean et al., 1999). Fletcher et al. (1988) found that Zn, Fe, Cu, Mn and Se are important trace minerals for normal immune function and disease resistance. If one or more of these trace minerals is deficient in the animal's diet, the immune status of the animal could be compromised (Suttle and Jones, 1989; Spears, 1991). Therefore, it is of utmost

importance that the mineral status be maintained to ensure that there are enough mineral stores available to maximize animal production and health when they are exposed to disease or become stressed (Tomlinson et al., 2008).

Copper

The recommended concentration of Cu by NRC for beef cattle is 10 mg/kg (National Academies of Sciences, 2016). However, industry professionals have reported feeding on average 20 mg/kg of Cu in receiving/finishing diets of feedlot cattle (Samuelson et al., 2016). Copper is present in and essential for the activity of multiple enzymes (tyrosinase, converts tyrosine to melanin for hair pigmentation), cofactors and reactive proteins (Suttle, 2010). Sulfur (S) and Mo can interact and bind with Cu and form a copper thiomolybdate complex (Cu-TM). This suggests that when Cu is combined in a thiomolybdate complex, it becomes unavailable for absorption and the Cu that is not bound to these complexes is readily available to be absorbed into the blood stream, which increases blood Cu values (Dick et al., 1975). Copper is also active in the production of neutrophils and affects phagocyte killing ability (Linder and Hazegh-Azam, 1996). Animals have important biological functions that depend on Cu such as erythropoiesis, protection from oxidants, heart development, and development of the central nervous system (CNS) (Underwood and Suttle, 1999).

The immune system can also be greatly impacted by Cu status of an animal. The concentration of Cu is decreased in the spleen, liver, thymus and lung by Cu deficiency, and this suggests that Cu-deficient animals carry a higher risk for infection than non-deficient animals (Stabel et al., 1993). Copper helps give energy to the immune system

by activating cytochrome-c-oxidase which is found in the mitochondrial electron-transport chain (Failla, 2003).

The enzyme ceruloplasmin is the major Cu carrying protein in the blood and has been shown to demonstrate anti-inflammatory activity (Healy and Tipton, 2007). Ceruloplasmin is an α -glycoprotein that is composed of a single polypeptide chain of 1046 amino acids and can bind up to 95% of Cu circulating in the body (Healy and Tipton, 2007). Cattle have the highest risk for developing Cu poisoning prior to weaning (Shand and Lewis, 1957). Cattle can also succumb to acute copper poisoning when they are given an excess amount of Cu through injections (Mylrea and Byrne, 1974). A few clinical signs that occur with Cu deprivation are anemia, neonatal ataxia, loss of hair/fleece color and skeletal abnormalities such as osteoporosis and widening of epiphyses (Underwood and Suttle, 1999). These studies clearly demonstrate that Cu is involved in the immune system, which impacts performance and health of feedlot cattle, making it imperative that supplementation practices are determined in order to ensure that the cattle are healthy and have the best performance possible.

Manganese

The Mn requirement for growing cattle is listed at 20 mg/kg by the NRC (National Academies of Sciences, 2016). It's not uncommon for industry professionals to add 50 mg/kg of Mn in receiving/finishing diets of feedlot cattle (Samuelson et al., 2016). Research has shown that inadequate amounts of Mn in the body can impair immunity and CNS function (Hurley, 1981). It can also cause decreased skeletal and soft tissue growth, weak and abnormally shaped bones, and reproductive disorders (Suttle, 2010). Similarly, Cu, Zn, and Mn play a role in the removal of superoxide radicals (Tomlinson et al.,

2008). Manganese also plays a role in other cellular pathways such as lipid and carbohydrate metabolism (Underwood and Suttle, 1999).

Manganese is required for the function of glycosyltransferases, which synthesizes mucopolysaccharides in cartilage, to allow for proper bone formation (Suttle, 2010). Much like other trace minerals, Mn functions as an enzyme activator and also helps form enzymes (McDowell, 2003). Manganese can also activate multiple hydrolases, kinases, transferases and decarboxylases (McDowell, 2003). It has also been hypothesized that Mn is involved in cholesterol synthesis (Davis et al., 1990), CNS function (Hurley and Keen, 1987), and steroid hormone production and carbohydrate metabolism (McDowell, 1992).

When looking at Mn status in beef cattle, it's often difficult and requires more than one criterion to diagnose a deficiency in Mn (McDowell, 2003). In beef production, Mn has more influence than what was previously realized (Corah and Arthington, 1993). The liver can effectively remove Mn from blood serum, then it gets excreted from the liver through the bile (Kincaid, 1999). This research demonstrates that Mn is important for many biological functions in the body. These functions can help an animal perform better in the feedlot setting and also help improve the health of the animal during exposure to stress.

Zinc

The Zn requirement for growing cattle is listed at 30 mg/kg by the NRC (National Academies of Sciences, 2016). Feedlot consultants have been known to add on average 100 mg/kg of Zn in diets of feedlot cattle (Samuelson et al., 2016). Zinc has been found to be vital for growth and development of all organisms (Cousins and King, 2004). It has

a role in catalytic, structural, and regulatory functions (Cousins and King, 2004). There are over one thousand known proteins that are associated with Zn (Maret, 2002). Animals have a limited capacity for storing Zn in a form that is readily available; typically, Zn stores are quite small (Underwood and Suttle, 1999).

Zinc deficiencies in the early stages in ruminants leads to a reduction of feed intake, growth rate and feed efficiency (McDowell, 2003). As the deficiency goes longer and worsens, the signs include listlessness, excessive salivation, swollen feet with lesions, parakeratotic lesions on the legs, neck, head and around the nostrils, failure of wounds to heal and alopecia (NRC, 1996). Zinc retention becomes negative when an animal experiences stress caused by either feed or water deprivation and ACTH injections (Galyean et al., 1999). Zinc is also important for normal growth rates, water and cation balance, skin and wound healing and vitamin A metabolism (McDowell, 2003)

The immune system is a highly proliferating cell system and relies heavily on the availability of Zn (McDowell, 2003). Zinc can influence T cell development and function, modulation of cellular functions and cytokine production from B and T cells. It also has large effects on the thymus, lymph nodes and tonsils. Zinc is a part of antioxidant defense, where it functions both extracellularly and intracellularly (Suttle, 2010). Research has shown that Zn plays a large role in the immune system, it is a part of nearly one thousand proteins and impacts feed intake and growth rate, making it an important mineral for cattle to receive to optimize health and performance.

Selenium

The Se requirement for growing cattle is listed at 0.1 mg/kg by the NRC (National Academies of Sciences, 2016). Feedlots have recently reported adding Se at a level of 0.3 mg/kg to their feedlot diets (Samuelson et al., 2016). The role of Se in the immune system remains largely based around the selenoprotein glutathione peroxidase. Glutathione peroxidase is an enzyme that inactivates oxygen radicals such as hydrogen peroxide (Tomlinson et al., 2008). These peroxides could injure the cell where they are produced (Hoekstra, 1975). Reffett et al. (1988) reported calves with Se deficiency had lower serum IgM concentrations and anti-Infectious Bovine Rhinotracheitis Virus (IBVR) titers when they were challenged with IBRV.

Selenium also helps with the formation and the activity of NK, helper T and cytotoxic T cells (Petrie et al., 1989). Vitamin E is a factor that can influence the consumption of dietary Se. However, the antioxidant functions of Se and Vitamin E are interdependent from each other (NRC, 1996). Selenium is involved in the metabolism of thyroid hormones. Therefore, selenium deficient diet causes triiodothyronine (T3) to decrease and tetraiodothyronine (T4) to increase (Thompson et al., 1995). This effects growths rates since T3 is an active form of T4, which is involved in growth mechanisms (Beckett et al., 1989).

With cattle not receiving sufficient Se, immune responsiveness can be reduced before the appearance of any clinical signs (Boyne and Arthur, 1979). A deficiency in Se has been shown to change the immune response to various infectious agents (Reffett et al., 1988). Deficiencies in Se may result in malfunction of thyroid metabolism, which can cause a drop in disease resistance, reduce growth rate, and change phagocytic response (Zust et al., 1996). With these affects cause by a deficiency in Se, it is important that

cattle receive an adequate amount to ensure that they are growing and have their immune system ready to fight off any pathogens that they might encounter leading up to entering the feedlot.

These data demonstrate that minerals can alter many biological functions such as: immune system, growth, and development. Feedlot cattle experience two stressful events at the same time: weaning and transportation. With these added measures of stress, cattle are more susceptible to morbidity and/or mortality caused by diseases such as BRD. With BRD causing large economical losses, and decreased performance in the feedlot setting it is important that we minimize the stress seen by the animals beforehand and prepare the body to help fight off any pathogens causing BRD. Literature indicates that provision of minerals that are essential for the immune system, such as Cu, Se, Zn, and Mn, will improve the animal's ability to stimulate an immune response and help fight off pathogens that can result in increases occurrences of morbidity and/or mortality. However, there are currently no best practices to alleviate the health concerns associated with receiving at-risk cattle into a feedlot. As such, more research is needed to determine which mineral supplementation practices can impact performance and health of mineral deficient cattle (Galyean et al., 1999).

Hypothesis

Providing either a multi-min injection or oral mineral supplementation at higher levels than NRC requires will result in improved cattle health, feedlot performance and carcass quality of at-risk mineral deficient calves.

Objectives

The objectives of this study are to determine how different mineral supplementation strategies impact feedlot performance, concentration of minerals in the liver, immunity to potential virus exposure and carcass quality in mineral deficient receiving cattle.

CHAPTER III

MATERIALS & METHODS

Animals

All animal experiments were conducted following procedures approved by IACUC protocol #10045 at Utah State University. A total of 40 yearling angus influenced steers from the Utah State University herd were used over the 110 d trial period. Steers were stratified by initial body weight and liver mineral concentration and then placed in one of four treatments. The treatments in this study included: no mineral supplementation (Con, n=10), one injection with Multimin® 90 (Multimin USA, Fort Collins CO) (MM, n=10), oral supplementation of minerals provided at levels similar to NRC requirements (AM; n = 10), oral supplementation of minerals provided at levels above NRC requirements (HM; n = 10). Upon commencement of the project, steers were loaded into a semi stock trailer and transported locally for five hours to emulate travel stress. After arriving back at the Utah State University feedlot, the steers were then processed and inoculated with a Merck Vision 8 vaccine (Merck, Kenilworth, NJ). They were fed a typical background ration, followed by a series of step up rations until the final feedlot ration was reached. Steers were housed under a 200 ft x 75 ft covered, open-sided shed and were fed twice a day at 0600 hours and 1600 hours. The pens were equipped with two GrowSafe bunks per pen and steers were housed in four different pens, one per treatment. The mineral supplement used in the current study was obtained from a local producer who served as a producer cooperator in this project. We chose to use this mineral to ensure that we were using a mineral supplement that was used by local producers. Animals received each treatment and were provided the same basic feedlot

ration in addition to their respective treatments for the first 40-d of the trial. After that period, they continued to receive the same basic feedlot ration but all animals received the adequate mineral treatment (AM). Steers were then treated this way through the remainder of the feedlot trial.

Feedlot Performance

Steer weights were obtained using a scale (True-Test GR3000, College Station, Texas) on d 0, 14, 28, 41, 56, 70, 84, and 110 of the trial. Body weights were recorded along with daily intake (DI) obtained from the GrowSafe system. These were used to calculate average daily gain (ADG) and feed efficiency (FE). Ultrasound measurements were also taken by the same trained ultrasound technician using an Exago Portable ultrasound (Exago, Bedford Hills, New York) Backfat thickness, and ribeye area were collected on d 5, 20, and 40, and every 28 d after the initial 40 d treatment period using an ExaGo 1509EX25 Version 1.12 portable ultrasound machine. Backfat was measured using ImageJ software using images in eFilm 4.0.3. Ribeye area was measured using a GTCO CalComp Peripherals DrawingBoard VI 12" x 12" Digitizer Model DB6 1212.

Feeding Behavior

Feeding behaviors were analyzed based on data that was collected from the GrowSafe units and following previously described procedures (McGee et al., 2014). Behaviors that were analyzed were categorized by bunk visit (BV) data and feed bout (FB) data. Parameters for bunk visit data will be: bunk visit (BV: reading of a single animal eid tag when entering at a bunk whether it consumed feed or not), average bunk visit duration (AVEBVDUR: average length of time that an animal had its head in a feed bunk consuming feed or not), average bunk visit consumption (AVEBVCONS: average

amount that was consumed every BV), average bunk visit head down (AVEBVHD: average time an animal spent with its head down in the bunk for each BV). Feed bout data parameters that were analyzed were: feed bouts (FB: reading of a single animal eid tag when entering a bunk and consumed at least 10 g of feed), average feed bout duration (AVEFBDUR: average amount of time animal was present with its head in a bunk while consuming more than 10 g of feed), average feed bout consumption (AVEFBCONS: measurement of the average amount of feed consumed during each FB), and average feed bout head down (AVEFBHD: average time that an animal's head was down while it consumed feed during a BV).

Collection of Carcass Data

Once all animals reached an average of 7 mm of backfat as measured by ultrasound (ExaGo), they were then harvested at a commercial processing facility (Hyrum, UT). Commercial carcass data was obtained from the harvesting facility and included: hot carcass weight, marbling score, ribeye area, ribeye fat thickness, USDA yield grade, and USDA quality grade.

Blood Collection and Analysis

Blood samples from the steers was collected on d 0, 5, 10, 20, 30, and 40 after initiation of the trial via jugular venipuncture using red top tubes. Blood samples were then placed into portable coolers and transported back to the laboratory where they were centrifuged at 1,000 x g for 15 mins. Serum was also collected, aliquoted and stored at -20°C for further analysis.

Serum Cortisol

Serum was used to evaluate cortisol by ELISA assay MBS2557040 (MyBioSource, San Diego, CA) to determine the level of stress that the animals experienced during transportation. Serum was diluted 10-fold using distilled water. A Standard working solution was added to the first two columns of a 96-well plate. Samples were then loaded into 2 other wells side by side on the plate. After all cells were loaded 50 μ L of biotinylated detection Ab working solution was added then covered with a plate covered and placed in an incubator for 45 min at 37°C. Then each well was decanted and washed with 350 μ L of wash solution. Each plate was washed three times. Then 100 μ L of HRP conjugate was added to each well and placed back in the incubator for 30 min at 37°C. After incubation the plate was washed 5 times with 350 μ L of wash solution. Next 90 μ L of substrate reagent was placed in each well and covered and placed back in the incubator for 15 min at 37°C. After final incubation 50 μ L of stop solution was added to each well and were then placed into a Biotek all-in-one microplate reader using Gen 5 2.0 software (Biotek Instruments, Winooski, VT) set to 450 nm to determine optical density. An average of the duplicate readings was then taken and plotted on a four-parameter logistic curve. Then the numbers plotted on the curve were then multiplied by 10 and reported as ng/mL of cortisol in the blood.

Immunity to Potential Virus

Serum was also used to evaluate virus-specific antibody titers by virus neutralization assays to determine: onset of immunity, duration of immunity, and max antibody response. Viruses that were used to test the blood serum were: BHV, and BPIV3, (Tuncer and Yeşilbağ, 2015). Fifteen μ l of serum was added to 225 μ l of media and loaded into the top row of a 96 well plate. 120 μ l of media was then be added to the

other seven rows. Then, an eight series dilution happened by taking 120 μ l from the first row and adding it to the second row. Then, 120 μ l from the second row was added to the third row until all eight rows contained 120 μ l of diluted serum. 120 μ l from the final row was taken out and disregarded. Finally, 120 μ l of a pre-dilution of the interested virus was added to all eight rows in the 96 well plate except for in the control column. One row is used for the control to make sure the cells are growing properly in the plate. Four wells in one column are used as a virus control to make sure that the virus is affecting the cells correctly. The other four wells in the same column are used for the back-titer which checked the dilutions. The plates were then placed into an incubator on a shaker where 5% CO₂ at 37 °C is applied and set for an hour (Tuncer and Yeşilbağ, 2015). For each serum sample, two-parallel columns and 5 rows in a 96-well plate that contained Madin Darby Bovine Kidney (MDBK) cells were used (Tuncer and Yeşilbağ, 2015). Each row received 100 μ l of the mixture of antibody, virus, and media. One column also consisted of a back titer, virus control, and also has a control column. The test results were then scored with an inverted light microscope after five d of incubation. The highest serum dilution with a positive result was recorded as the antibody titer for the tested virus (Tuncer and Yeşilbağ, 2015). The log₂ of each antibody titer was then calculated and reported as the titer response to the vaccine.

Mineral Liver Analysis

Liver samples were taken at the same time points as the blood. Liver samples were collected with a liver biopsy kit performed by USUs clinical veterinarians. After the six collections have been completed, liver samples were then sent to the Iowa State University Veterinary Diagnostic Lab and analyzed for Calcium (Ca), Cadmium (Cd),

Co, Chromium (Cr), Cu, Fe, Magnesium (Mg), Mn, Mo, Sodium (Na), Phosphorous (P), Potassium (K), Se, and Zn using Inductively coupled plasma mass spectrometry (ICP-MS, Analytik Jena Inc. Woburn, MA, USA) in CRI mode with hydrogen as the skimmer gas. Standards for elemental analyses were obtained from Inorganic Ventures (Christiansburg, VA) while digestion vessels, trace mineral grade nitric acid and hydrochloric acid were obtained from Fisher Scientific (Pittsburgh, PA). Following thawing at room temperature, samples were then processed and analyzed following the established SOP on a wet weight basis. Briefly, samples were digested in 70% nitric acid using a microwave digester. 0.5 g samples were weighed into a 50 mL digestion tubes and 10 mL of 70% nitric acid was added. After digestion, all samples were diluted to 25 mL using 1% nitric acid with 0.5% hydrochloric acid. An additional 1:10 dilution using 1% nitric acid is made and then analyzed by ICP-MS. For quality control, Bismuth, Scandium, Indium, Lithium, Yttrium, and Terbium were used as internal standards for the ICP-MS.

Statistical Analysis

The PROC MIXED procedure of SAS version 9.4 (SAS Institute, Inc., Cary, NC) was used to estimate the main effects of treatment on feedlot performance, animal behavior, carcass data, immunity to a potential virus, and mineral liver status. Steers were blocked by initial body weight and initial liver mineral status of Cu, Zn, Se and Mn as determined by analysis of a liver biopsy. The pen and individual steer were used as random effects in the model. Repeated measures analyses were used to determine the effects of treatment, time, and treatment*time for body weight, DI, FE, mineral status,

and immune response. A $P < 0.05$ was considered statistically significant and a $P < 0.10$ was considered a trend in the data.

CHAPTER IV

RESULTS

Serum Cortisol

After a 5 h transit, cortisol was measured in the serum of all animals. As expected, there was no difference ($P > 0.05$) in cortisol levels assessed on d 0 of the trial between animals that received different mineral treatments, table 4. This data indicates even though there was no difference between the treatments the steers utilized in this study did experience a moderate amount of stress following 5 h of travel.

Feedlot Performance

Weight gain over the 110 d feeding period was not altered by treatment ($P = 0.12$) or treatment*time ($P = 0.99$), Figure 1. However, there was an effect of time ($P < 0.0001$) on weight gain demonstrating that weights increased over time, Figure 1. These data indicate that the weight of the steers increased over the 110 d finishing period, but there was no effect of treatment or treatment*time, indicating that the different mineral supplementation strategies implemented at receiving had no effect of weight gain. Average daily gain over the 110 d feeding period was not altered by treatment ($P > 0.05$), treatment*time ($P > 0.05$), or time ($P > 0.05$), Table 4. Steers that received the HM treatment had an increased ($P < 0.05$) ADG compared to those receiving the MM treatments during d 56 - 69 of the feed trial. These data indicate that different mineral supplementation strategies at receiving does not have an effect on overall ADG during the finishing period, but ADG was increased in animals receiving the HM treatment during specific period of the feedlot phase. Dry matter intake over the 110 d feeding

period was affected by treatment ($P = 0.03$), treatment*time ($P < 0.0001$), and time ($P = 0.05$), Figure 2. Steers receiving the HM treatment had increased ($P < 0.05$) DMI compared to those receiving the Con and AM treatments when analyzed with repeated measures. These data indicate that different mineral supplementation strategies at receiving impacts DMI during the finishing period. Feed efficiency over the 110 d feeding period was not affected by treatment ($P > 0.05$), Figure 3. However, there was an effect of time ($P < 0.0001$), and treatment*time ($P = 0.01$) on gain:feed. These data indicate that the gain:feed of the steers changes over time while steers are in the feedlot.

Ultrasound Measurements During the Feedlot Period

There was an effect of treatment ($P = 0.0001$) and time ($P < 0.0001$) on backfat thickness when assessed by ultrasound during the feedlot period, Figure 4A. However, there was no difference in treatment*time ($P = 0.99$), Figure 4A. Animals receiving the HM and Con treatments had increased ($P < 0.05$) backfat thickness when compared to those animals receiving the AM and MM treatments, Figure 4A. Additionally, in the initial backfat thickness measurement on d 5, steers receiving the Con treatment had a larger ($P < 0.05$) backfat thickness when compared to animals receiving the MM treatment, Figure 4A. There were no differences between steers receiving different in another of the other time points analyzed. These data indicate that the backfat thickness of the steers increased over the 68 d finishing period and animals receiving the HM and Con treatments had increased accretion of backfat when compared to the other two treatments. There was an effect of treatment ($P = 0.0003$) and time ($P < 0.0001$) on ribeye area measured via ultrasound during the feedlot period, Figure 4B. However, there was no effect ($P > 0.05$) of treatment*time, Figure 4B. Steers that received the MM treatment

had a decreased ($P < 0.05$) ribeye size compared to the other three treatments, Figure 4B. However, the initial ribeye area measurements on d 5 indicated that MM had the smallest ribeye area, Figure 4B. These data indicate that the ribeye area of the steers increased over the finishing period and that steers receiving the MM treatment had the smallest ribeye size when compared to steers receiving the other treatments, but it is important to note that there was a difference from the initial measurement. Carcass data was collected by a commercial facility at the time of harvest. There was no difference ($P > 0.05$) in marbling score, ribeye area, backfat thickness, yield grade, or marbling:backfat ratio between steers receiving the different mineral treatments, Table 6. There was a tendency ($P = 0.08$) for steers receiving the HM treatment to have an increased hot carcass weight when compared to steers receiving the MM treatment, Table 6. These data indicate that different mineral supplementation strategies at receiving does not have an effect on carcass characteristics of the steers at time of harvest.

Hepatic Mineral Concentrations

There was an effect of treatment ($P < 0.0001$), time ($P < 0.0001$), and treatment*time ($P < 0.0023$) on hepatic Co concentration, Figure 5. Steers that received either the AM or HM treatment had increased ($P < 0.05$) liver Co when compared to the Con and MM treated steers, Figure 5. These data indicate that providing oral mineral supplementation is the best way to maintain liver Co concentrations in steers.

Hepatic Cu concentrations were affected by treatment ($P < 0.0001$), time ($P < 0.0001$), and treatment*time ($P < 0.0001$) Figure 6. Steers that received the HM treatment had increased ($P < 0.05$) liver Cu when compared to animals receiving the other three treatments, Figure 6. Furthermore, animals receiving AM and MM treatments had

increased ($P < 0.05$) liver Cu when compared to those receiving the Con treatments. These data indicate that providing HM levels of mineral supplementation results in the largest increase in liver Cu status, but animals receiving AM levels or MM also increase liver Cu status when compared to animals not receiving any mineral supplementation. There was not an effect of treatment ($P = 0.31$) or treatment*time ($P = 0.56$) on hepatic Mn concentrations, Figure 7. However, there was an effect of time ($P = 0.01$), Figure 7. These data indicate that even though steers received different mineral supplementation strategies, there was a decrease in liver Mn status from the initial sampling time, but treatment had no effect on this change. Hepatic Se concentrations were affected by treatment ($P < 0.001$) and treatment*time ($P < 0.001$), but there was no effect of time ($P > 0.05$), Figure 8. Steers that received the MM treatment had Se levels that were increased ($P < 0.05$) when compared to the other treatments at 5 and 10 d after initiation of treatment, Figure 8. At 20 d after treatment, there was no difference ($P > 0.05$) between steers receiving the MM and HM treatments, but animals receiving these treatments were increased ($P < 0.05$) when compared to those receiving the Con treatment, Figure 8. At 30 and 40 d post initiation of treatment, steers that received the HM treatment had increased ($P < 0.05$) liver Se when compared to the other three treatments, Figure 8. However, during this time frame, steers that received the MM or AM treatment still had increased ($P < 0.05$) liver Se compared to those receiving the Con treatment. These data indicate that providing MM at receiving results in a large initial increase in liver Se at 5 and 10 d after treatment when compared to the other treatments, but provision of oral minerals at HM levels results in increased liver Se at 30 and 40 d after initiation of the treatments when compared to steers receiving the other treatments.

There was not an effect of treatment ($P = 0.14$) or treatment*time ($P = 0.82$) on hepatic Zn concentrations, Figure 9. However, there was an effect of time ($P = 0.0001$), Figure 9. These data indicate that over time hepatic Zn increases 5 and 10 d after entering the feedlot and then it decreased on d 20, 30 and 40. However, levels of Zn in the liver were not altered by treatment.

Feeding Behavior

The number of bunk visits and feed bouts was affected by treatment ($P < 0.0001$), time ($P < 0.0001$), and treatment*time ($P < 0.0001$), Figure 10. As the feedlot trial progressed, the number of bunk visits and feed bouts decreased ($P < 0.05$), Figure 10. Steers that received the HM mineral treatment had an increase ($P < 0.05$) in number of bunk visits and feed bouts when compared to steers receiving the other three treatments, Figure 10. Specifically, steers receiving the HM treatment had significantly higher number of ($P < 0.05$) bunk visits and feed bouts from d 14-28 and d 29-41 of the feedlot trial. However, after this time, there was no difference ($P > 0.05$) in bunk visits between animals receiving the different treatments. These data indicate that providing oral mineral supplementation at HM increases the number of bunk visits and feed bouts early in the feedlot period, but there is no difference in bunk visits or feed bouts between the different treatments after 41 d in the feedlot. Average consumption at each bunk visit and feed bout were both affected by treatment ($P < 0.05$) and time ($P < 0.001$), but there was no effect of treatment*time ($P > 0.05$), Figure 11. As the feedlot trial progresses, animals consumed more ($P < 0.05$) during each feed bout and bunk visit, Figure 11. Steers that received the HM mineral treatment had decreased ($P < 0.05$) consumption at each bunk visit and feed bout when compared to steers receiving the other three treatments, Figure

11. These data indicate that mineral supplementation strategy at receiving has an effect on the amount consumed at each bunk visit and feed bout such that steers receiving HM consume less than the other three treatments at each bunk visit or feed bout. The average time that animals spent with their head down at each bunk visit and feed bout was affected by treatment ($P = 0.002$) and time ($P < 0.001$), however there was no effect of treatment*time ($P > 0.05$), Figure 12. Steers that received the HM treatment spent less ($P < 0.05$) time with their head during both bunk visits and feed bouts when compared to steers that received the other three treatments. Over time, the average time that steers spend with their head down at each bunk visit and feed bout decreased ($P < 0.05$) during the feedlot period. These data indicate that as time progresses, animals spend less time with their head down while at the feed bunk and mineral treatment effects the average time that animals spend with their head down while at the bunk. The average amount of time that each steer spent at the bunk during each bunk visit and feed bout was affected by treatment ($P < 0.01$) and time ($P < 0.001$), but there was no effect of treatment*time ($P > 0.05$), Figure 13. As time went on, the amount of time that animals spent during each bunk visit and feed bout decreased ($P < 0.05$), Figure 13. Steers that received the HM treatment spent more ($P < 0.05$) time during each bunk visit and feed bout from d 14-28 and 29-41 when compared to the other three treatments, Figure 13. There was no difference between animals receiving the different treatments after d 41 of the trial. Taken together, these data demonstrate that as time progresses animals spend less time, on average, during each feed bout and bunk visit and that in the beginning of the feedlot phase, animals that receive more mineral orally will spend more time at the bunk during each feed bout and bunk visit.

Virus Neutralization Assays

The average virus neutralization antibody titer for BPIV3 was affected by time ($P < 0.01$), Figure 14. However, there was no effect of treatment ($P > 0.05$) or treatment*time ($P > 0.05$), Figure 14. As time went on the amount of antibodies found increased ($P < 0.05$) up until d 10 and then decreased as the trial period progressed. These data indicate that even though steers received different mineral supplementation strategies, there was no difference in amount of antibodies to BPIV3 found in the blood of the steers. The average virus neutralization antibody titer for BHV was affected by time ($P < 0.01$), Figure 15. However, there was no effect of treatment ($P > 0.05$) or treatment*time ($P > 0.05$), Figure 15. As time went on the amount of antibodies found increased up until d 20 and then decreased after that time. These data indicate that even though steers received different mineral supplementation strategies, there was no difference in amount of antibodies to BHV found in the blood of the steers. None of the animals during the trial were treated for sickness indicating that there was no effect of treatment on development of sickness.

CHAPTER V

DISCUSSION

Trace minerals are essential in immune response, health, and performance of cattle (Galyean et al., 1999; Underwood and Suttle, 1999). Morbidity and mortality resulting from BRD in feedlot cattle continues to be one of the most substantial health problems facing the beef cattle industry (Duff and Galyean, 2007). Cattle that are mineral deficient are considered at-risk due to the effects on immune response, making them more susceptible to development of BRD and other diseases (McGill and Sacco, 2020). Bovine respiratory syndrome has been shown to result in a negative impact on economics (Duff and Galyean, 2007), health performance (Hutcheson and Cole, 1986), and immune response (Underwood and Suttle, 1999) of feedlot cattle. Improving mineral status of receiving cattle may help decrease occurrence of BRD. In the present study we investigated the effects of different TM supplementation strategies at receiving on performance, carcass quality, and immunity to a potential virus of mineral deficient feedlot cattle in order to determine best practices that producers can follow.

Cattle in the United States average 7 h transport time to the feedlot (Cernicchiaro et al., 2012). The cattle in the present study were transported 5 h and experienced a moderate amount of stress. Mean baseline cortisol measurements for beef cattle are typically less than 10 ng/mL (Mormède et al., 2007). Grandin, et al. (1997) reported that cortisol measurements >70 ng/mL could be a good indicator of a stressful event. Previous studies have shown that stress-induced increases in glucocorticoid concentrations due to transportation have increased number of animals with shipping fever and decreased feedlot performance (Mormede et al., 1982). Mormede (1982) reported levels changed

from 1.72 ng/mL prior to transport to 17.98 ng/mL one week after calves arrived at a fattening unit. In the current study the animals experienced a moderate amount of stress as cortisol levels averaged around 35 ng/mL following transportation. This level of stress is typical of what receiving cattle would experience during transportation to the feedlot.

In the present study, we found that feeding varying levels of minerals or providing an injectable TM supplement did not have an effect on BW, ADG or G:F of the steers when compared to the control steers. However, DMI was increased in the HM steers when compared to steers receiving the AM and CON treatments. Niedermayer et al. (2017) reported that there were no differences in BW, ADG, or G:F when administering an injectable TM supplement or a sterilized saline injection to certified natural angus steers. Previous research has demonstrated that varying levels of Mn (10, 20, 30, 120, or 240 mg of DM) in the diet had no effect on steer performance (Legleiter et al., 2005). Ahola et al. (2005) reported that growth responses from dietary TM supplementation or injection are more evident in cattle that are mildly or severely deficient (Ahola et al., 2005). The cattle utilized in the present study were only mildly deficient as evidenced by their starting liver mineral concentrations. Nunnery et al. (1996) reported steers that were supplemented with $ZnSO_4$ had increased gain compared to steers supplemented with zinc methionine, but feed efficiency and feed intake were unaffected by the different treatments. Spears (1995) indicated that supplementing zinc above recommended requirements did not have a significant impact on gain, intake, or feed efficiency of growing and finishing beef cattle. Gaylean et al. (1995) analyzed the effects of dietary Zn and supplemental Cu on performance and health during the receiving, growing, and finishing phase. Neither source nor concentration of Zn had an effect on performance

during the three phases (Galyean et al., 1995). Martin et al. (1987) also reported the effects of zinc methionine on performance and no differences in ADG, DMI or feed efficiency were observed (Martin et al., 1987). Several other studies also demonstrate that TM supplementation has no impact on feedlot performance (Greene, 1988; Rust and Schlegel, 1993; Malcolm, 2000). Engle and Spears (2000a) found that feedlot performance of sixty Angus and twenty-four Angus/Hereford cross steers was not affected by Cu levels during the growing phase, and feed intake and feed efficiency were reduced. Additionally, Rust and Schlegel (1993) reported that Zn supplementation in finishing cattle resulted in increased ADG. Another study found that as dietary Zn concentration increase, DMI decreases (Malcolm-Callis, 2000). Similar to the present study, Niedermayer et al. (2017) also reported seeing an increase in DMI with steers receiving an injectable TM during d 56 – 124 and overall DMI (Niedermayer et al., 2017). Neidermayer et al. (2017) hypothesized the increased DMI is due to supplementation of vitamin B₁₂ and its role in propionate production (Allen et al., 2009). In the current study, the increase in DMI could be related to the effects that oral supplementation of minerals may have on feeding behavior. Generally, the results of the present study match those in existing literature and demonstrate that animals that are experiencing a mild mineral deficiency do not exhibit increased feedlot performance with mineral supplementation when compared to those animals that do not receive TM supplementation. To date, there have been relatively few studies that have analyzed the effects of changing the level/method of all TM on feedlot performance. As such, more research needs to be completed to determine how providing different levels of TM

impacts feedlot performance when compared to animals that do not receive TM supplementation.

The results of the present study and others demonstrate that administering an injectable TM does initially increase liver Cu concentration but is only effective over a short period of time. However, providing TM supplementation at increased levels than are provided by the NRC results in improved liver Cu more long term. Prior research has found that provision of an injectable TM was an effective way to increase liver Cu concentrations within 15 d of the injection (Pogge et al., 2012). Several other studies have also demonstrated that injectable TM are an effective way to increase liver Cu concentrations (Daugherty et al., 2002; Kurz, 2004). Similar to the present study, Engle and Spears (2000a) reported that after d 56 of the growing phase and through the rest of the finishing phase, steers that received 20 or 40 mg of Cu/kg DM of Cu supplementation had increased concentrations than those not supplemented with Cu (Engle and Spears, 2000a). In the current research we can see that as Cu levels are fed closer to levels provided by industry professionals, there is an increase in liver Cu concentration. Additional research needs to be completed to better understand how different mineral supplementation strategies impact liver Cu and which strategy is the best for receiving cattle that are known to be mineral deficient.

In the present study steers that received a MM injection initially had increased liver Se concentrations when compared to steers in the other treatment groups. However, after 30 d in the feedlot, cattle receiving the HM treatment had increased liver Se when compared to the other treatments. Consistent with previous work, the steers that received an injectable TM had increased liver Se concentrations compared to the other treatments

shortly after receiving the injection (Pogge et al., 2012; Genter and Hansen, 2014).

Other studies have also found that although injectable TMs can be an effective method to increase liver mineral status, the effective period is less than 45 d (Genter and Hansen, 2014; Maas et al., 1994). In addition, Richards et al. (2011) reported that by providing 0.34 mg of Se/kg DM for 130 d resulted in increased liver concentrations of Se when compared to steers not receiving any added supplementation. The results of this study and previous studies demonstrate that administering an injectable TM does initially increase liver Se concentration but is only effective over a relatively short period of time.

However, providing oral TM supplementation at increased levels that are provided by the NRC results in improved liver Se more long term. In the current research we can see that when Se levels are fed closer to levels that are used by industry professionals, there is an increase in liver Se concentration. Additional research needs to be completed to better understand how different mineral supplementation strategies impact liver Se and which strategy is the best for receiving cattle that are known to be mineral deficient.

The results of the present study demonstrate that feeding varying levels of minerals does not affect liver Mn concentration. Underwood and Suttle (1999) reported that supplying a diet that is high in Mn results in only slight differences in liver Mn concentration (Underwood and Suttle, 1999). This could be due to the fact that the liver does not have a long-term space for storing Mn, which is why it can be difficult to accurately measure in cattle (Hidiroglou, 1979). Bentley and Phillips (1951) reported that feeding dairy cows different levels of Mn (40, 60, 100, 350, and 550 ppm) for more than three years did not affect liver Mn concentration. Prior research that was completed with sheep, has also reported that Mn concentrations were not affected by supplementation of

Mn (Watson et al., 1973; Masters et al., 1988). In contrast, several other studies have reported that liver Mn concentration increase as dietary Mn increases (Howes and Dyer, 1971; Ivan and Hidioglou, 1980). Additional research needs to be completed to better understand how different mineral supplementation strategies impact liver Mn and how producers should provide Mn to cattle that are mineral deficient.

The results of the present study demonstrate that feeding varying levels of minerals does not affect liver Zn concentration. Olson et al. (1999) reported that there was no difference in Zn concentrations in the liver from newly weaned calves supplemented with organic or inorganic minerals. Spierenburg et al. (1988) reported that Zn levels in cattle was not affected when cattle were located within a 20 km radius of at least one zinc refinery plant. Nunnery et al. (1998) analyzed steers supplemented with either a Zn sulfate or Zn methionine at varying concentrations (5, 35, 95, 215, and 455 mg Zn/kg) and found that neither amount nor source had no effect on Zn liver concentration in the animals. These findings are similar to that of Rojas et al. (1996), who found that providing 360 mg/d to cattle provided no difference in Zn liver concentrations. However, Ott et al. (1966) reported that Zn liver concentrations increased as levels of dietary Zn increased. Additional research needs to be completed to better understand how different mineral supplementation strategies impact liver Zn and which strategy is the best for receiving cattle that are known to be mineral deficient.

In the present study feeding varying levels of minerals or providing an injectable TM supplement did not impact carcass characteristics when compared to control steers. Similar to the present study, other studies have shown that feeding varying levels of minerals has no effect on carcass quality of feedlot steers (Caldera et al., 2017; Rhoads et

al., 2003). Although very few studies have looked at the impacts of providing varying levels of all TM, many studies have analyzed how provision of specific TM impact carcass quality. In a survey of feedlot consultants, it was found that feeding excess concentrations of Zn was a common practice as it is thought to improve carcass quality, and increase growth rates of cattle (Galyean, 1996). However, several studies have demonstrated that supplementing growing and finishing cattle zinc at varying levels had no impact on carcass characteristics (Pringle et al., 1973; Spears and Samsell, 1984). In a different study completed with finishing and growing cattle, supplementation of Zn resulted in an increase in marbling score and REA when compared to steers that were not supplemented (Spears and Kegley, 2002). Genther et al. (2014) reported that steers that were administered a TM injection had an increase in marbling score and quality grade, and a decrease in yield grade, KPH, and BF. Additionally, increasing supplemental Mn and Se in finishing steers had no impact on carcass characteristics, similar to the findings of the present study (Legleiter et al., 2005; Lawler et al., 2004). Providing supplemental Cu is thought to impact the metabolization of lipids, which has been linked to an increase in REA (Ward and Spears, 1997; Engle and Spears, 2000b; Engle et al., 2000). Malcolm-Callis et al. (2000) found that feeding Zn at varying levels to feedlot steers had no effect on HCW, dressing percent, longissimus muscle area, marbling or yield grade. It has also been observed that carcass traits were similar between feedlot steers fed varying amounts of supplemental Mn (Nunnery et al., 1996). Generally, the results of the present study match those in existing literature and demonstrate that providing varying levels of TM has no impact on carcass characteristics. However, more research needs to be completed on specific minerals at varying concentrations to better understand how TM

supplementation may impact carcass characteristics when compared to those animals that do not receive TM supplementation.

In the present study we found steers that were fed the HM treatment had an increase in feed bouts and bunk visits during the first half of the 110 d trial. When comparing the amount consumed at each feed bout/bunk visit, the HM treatment consumed the least when compared to the other treatment groups. They also had their head down the least amount of time when visiting the bunk at each feed bout/bunk visit when compared to the other treatments. The HM treatment also spent the longest time at the bunk during the first half of the feed trial. Taken together, these results demonstrate that providing TM supplementation at HM causes animals to go up to the feed bunk more often, but consume less feed each time that are there. One explanation for this result might be that during each feed bout/bunk visit during the first half of the trial animals were receiving more oral mineral supplementation in a powder form and this impacted their feeding behavior. To our knowledge, no previous research has analyzed how provision of TM impacts feeding behavior of feedlot cattle. More research needs to be completed on specific minerals at varying concentrations to better understand how TM supplementation may impact feeding behavior when compared to those animals that do not receive TM supplementation and which strategy is the best for receiving cattle that are known to be mineral deficient.

Many viruses can contribute to the development of BRD in feedlot animals such as BRSV, BVDV, BHV and BPIV3. The steers in the current study were not inoculated with any virus that contribute to the development of BRD. This could be why we did not see a significant difference in vaccine response. In the present study BHV and BPIV3

were assessed by analyzing virus neutralization antibodies. It was found that feeding varying levels of TM or providing an injectable TM supplement did not affect the antibody titer for BPIV3 or BHV. To our knowledge, no previous research has analyzed how provision of varying levels of TM impacts immunity to a potential virus. However, the TM Zn, Cu, and Se (and to some extent Mn and Co) are known to be involved in the immune response of cattle (Underwood, 1971; McDowell, 1992; Galyean et al., 1999; Spears, 2000; Goswami et al., 2005). Additionally, several studies have analyzed the effects of supplementing specific TM on measures of immunity. Chirase et al. (1991) found that crossbred steers that were supplemented with dietary Zn at 90 ppm had an enhanced recovery rate of infectious bovine rhinotracheitis (IBRV) stressed cattle. Galyean et al. (1995) reported a tendency for decreased morbidity in newly weaned calves that were supplemented with 70 mg Zn/kg DM than with a diet that contains 35 mg of Zn/kg DM. Copper deficiency has been reported to result in a decrease in humoral, cell-mediated and non-specific immune function (Stabel and Spears, 1989). Dorton et al. (2003) conducted an experiment with 48 purebred angus steers and fed varying levels of Cu (10 mg Cu/kg DM of Cu sulfate, 10 mg Cu/kg Dm from a Cu-amino acid group, 20 mg Cu/kg Dm from CuSO₄, and 20 mg Cu/kg DM from Availa Cu) and saw that antibody responses tended to be enhanced by Cu supplementation (either 10 or 20 mg Cu/kg DM when compared to the control). Reffett et al. (1988) observed that Se deficient calves that received 0.03 mg Se/kg DM had a reduced humoral immune response after being challenged with IBRV on d 0 and 35 of the 70 d period when compared to calves that received 0.02 mg Se/kg of DM. However, Dorton et al. (2006) reported that 375 steers were supplemented with 125 mg of Cu, 360 mg Zn, 200 mg of Mn, and 12.5 mg of

Co from one of two sources (sulfates or amino acid complexes) for 30 d of on-farm backgrounding and a 28 d feedlot receiving period and found no differences in morbidity or mortality rates. Ryan et al. (2015) also reported no effects of source of TM (Zn, Cu, and Mn from sulfates, organic amino acid complexes, or hydroxyl sources) on morbidity during the receiving period. Generally, the results of the present study match those in existing literature and demonstrate that feeding varying levels of TM or providing an injectable TM does not have an impact on overall cattle health. However, more research needs to be completed on specific minerals at varying concentrations and on other viruses that contribute to BRD to better understand how TM supplementation may impact immune response when compared to those animals that do not receive TM.

CHAPTER VI

CONCLUSION

The objectives of this study are to determine how different mineral supplementation strategies impact feedlot performance, concentration of minerals in the liver, immunity to potential virus exposure and carcass quality in mineral deficient receiving cattle. Weight gains of steers did not differ between the different treatments. Results from the present study also indicate that ADG, DMI, and FE were unaffected by the varying levels of minerals supplemented. However, liver Cu and Se were commonly higher in steers fed the HM when compared to the other treatments. Liver Mn and Zn were unaffected by the different treatments. The data in the present study show that immune response to a potential virus was not affected. To our knowledge no previous research has looked into immune response to a potential virus with supplementing varying levels of TM. More research is needed to explore how TM effect immunity. Carcass quality was also not affected by feeding varying levels of minerals. By feeding increased levels of minerals, hepatic Cu and Se concentrations increased. However, more research is needed to determine the effects that varying levels have on cattle performance, health and carcass quality.

REFERENCES

- Abbas, A. K., A. H. Lichtman and J. S. Prober. 1991. Cellular and Molecular Immunology. W. B. Saunders Co., Philadelphia, PA.
- Ahola, J.K., Sharpe, L.R., Dorton, K.L., Burns, P.D., Stanton, T.L., Engle, T.E., 2005. Effects of Lifetime Copper, Zinc, and Manganese Supplementation and Source on Performance, Mineral Status, Immunity, and Carcass Characteristics of Feedlot Cattle. *The Professional Animal Scientist* 21, 305–317.
[https://doi.org/10.15232/S1080-7446\(15\)31222-5](https://doi.org/10.15232/S1080-7446(15)31222-5).
- Alberts, B., Johnson, A., Lewis, J., Raff, M., Roberts, K., Walter, P., 2002. Helper T Cells and Lymphocyte Activation. *Molecular Biology of the Cell*. 4th edition.
- Andersen, M.H., Schrama, D., Thor Straten, P., Becker, J.C., 2006. Cytotoxic T Cells. *Journal of Investigative Dermatology* 126, 32–41.
<https://doi.org/10.1038/sj.jid.5700001>.
- Behne, D. 1989. Selenium homeostasis. In: J. Neve and A. Favier (Ed.) *Selenium in Medicine and Biology*. Walter de Gruyter, Berlin. pp. 93-91
- Bentley, O.G., Phillips, P.H., 1951. The Effect of Low Manganese Rations upon Dairy Cattle. *Journal of Dairy Science* 34, 396–403.
- Berrie, R.A., Hallford, D.M., Galyean, M.L., Brundage, A.L., White, T.W., 1995. Effects of Zinc Source and Level on Performance, Carcass Characteristics, and Metabolic Hormone Concentrations of Growing and Finishing Lambs. *The Professional Animal Scientist* 11, 149–156. [https://doi.org/10.15232/S1080-7446\(15\)32579-1](https://doi.org/10.15232/S1080-7446(15)32579-1).

- Besedovsky, H., Rey, A. del, Sorkin, E., Dinarello, C.A., 1986. Immunoregulatory feedback between interleukin-1 and glucocorticoid hormones. *Science* 233, 652–654.
- Biron, C.A., 2016. Chapter 4 - Innate Immunity: Recognizing and Responding Foreign Invaders —No Training Needed, in: Katze, M.G., Korth, M.J., Law, G.L., Nathanson, N. (Eds.), *Viral Pathogenesis (Third Edition)*. Academic Press, Boston, pp. 41–55.
- Buhman, M.J., Perino, L.J., Galyean, M.L., Wittum, T.E., Montgomery, T.H., Swingle, R.S., 2000. Association between changes in eating and drinking behaviors and respiratory tract disease in newly arrived calves at a feedlot. *Am. J. Vet. Res.* 61, 1163–1168. <https://doi.org/10.2460/ajvr.2000.61.1163>.
- Caldera, E., Wagner, J.J., Sellins, K., Laudert, S.B., Spears, J.W., Archibeque, S.L., Engle, T.E., 2017. Effects of supplemental zinc, copper, and manganese concentration and source on performance and carcass characteristics of feedlot steers. *Prof Ani Sci* 33, 63–72. <https://doi.org/10.15232/pas.2016-01531>.
- Carroll, J.A., Forsberg, N.E., 2007. Influence of Stress and Nutrition on Cattle Immunity. *Veterinary Clinics of North America: Food Animal Practice, Topics in Nutritional Management of the Beef Cow and Calf* 23, 105-149. <https://doi.org/10.1016/j.cvfa.2007.01.003>

- Cernicchiaro, N., White, B.J., Renter, D.G., Babcock, A.H., Kelly, L., Slattery, R., 2012. Associations between the distance traveled from sale barns to commercial feedlots in the United States and overall performance, risk of respiratory disease, and cumulative mortality in feeder cattle during 1997 to 2009. *J Anim Sci* 90, 1929–1939. <https://doi.org/10.2527/jas.2011-4599>
- Cole, N. A., and D. P. Hutcheson. 1990. Influence of dietary protein concentrations on performance and nitrogen repletion in stressed calves. *J. Anim. Sci.* 68:3488-3497. <https://doi.org/10.2527/1990.68113488x>.
- Collins, A.M., Watson, C.T., 2018. Immunoglobulin Light Chain Gene Rearrangements, Receptor Editing and the Development of a Self-Tolerant Antibody Repertoire. *Front. Immunol.* 9. <https://doi.org/10.3389/fimmu.2018.02249>.
- Corah, L.R., and J. Arthington. 1993. Mineral nutrition- Identifying problems and solutions. Pages 100-119 in *Proceedings, Range Cow Beef Symposium XIII*, Cheyenne, WY.
- Cousins, R. J. and J. C. King. 2004. Zinc in Modern Nutrition in Health and Disease. M. E. Shils, A. C. Ross and R. J. Cousins, eds. Philadelphia: Leas and Febiger.
- Daugherty, S. R., G. E. Carstens, D. B. Herd, K. S. Barling, and R. D. Randel. 2002. Effects of prenatal and prebreeding TM/vitamin E injections on calf health and reproductive performance of beef cows. *Beef Cattle Research in Texas Report*. Dep. Anim. Sci., Texas A&M Univ., College Station.
- Davis, C. D., D. M. Ney, and J. L. Greger. 1990. Manganese, iron and lipid interactions in rats. *J. Nutr.* 120: 507-513. <https://doi.org/10.1093/jn/120.5.507>.

- Davy, J., Forero, L., Tucker, T., Mayo, C., Drake, D., Maas, J., Oltjen, J., 2016. Efficacy of selenium supplementation methods in California yearling beef cattle and resulting effect on weight gain. *California Agriculture* 70, 187–193.
<https://doi.org/10.3733/ca.2016a0016>
- Dick, A.T., Dewey, D.W., Gawthorne, J.M., 1975. Thiomolybdates and the copper–molybdenum–sulphur interaction in ruminant nutrition. *The Journal of Agricultural Science* 85, 567–568. <https://doi.org/10.1017/S0021859600062468>.
- Duff, G.C., Galyean, M.L., 2007. Recent advances in management of highly stressed newly received feedlot cattle. <https://doi.org/10.2527/jas.2006-501>.
- Edwards, A. (1996) Respiratory diseases of feedlot cattle in central USA. *Bovine Practitioner* 30, 5-10.
- Ellis, J. A. 2001. The immunology of the bovine respiratory disease complex. *Vet. Clin. Food Anim.* 17:2521-2527. [https://doi.org/10.1016/s0749-0720\(15\)30005-0](https://doi.org/10.1016/s0749-0720(15)30005-0).
- Engle, T.E., Spears, J.W., Xi, L., Edens, F.W., 2000b. Dietary copper effects on lipid metabolism and circulating catecholamine concentrations in finishing steers. *J. Anim. Sci.* 78, 2737–2744. <https://doi.org/10.2527/2000.78102737x>.
- Failla, M.L., 2003. Trace Elements and Host Defense: Recent Advances and Continuing Challenges. *J Nutr* 133, 1443S-1447S. <https://doi.org/10.1093/jn/133.5.1443s>.
- Fraker, P.J., King, L.E., 2004. Reprogramming of the Immune System During Zinc Deficiency. *Annual Review of Nutrition* 24, 277-298.
<https://doi.org/10.1146/annurev.nutr.24.012003.132454>.

- Fraser, D., 2001. The “new perception” of animal agriculture: legless cows, featherless chickens, and a need for genuine analysis. *J. Anim. Sci.* 79, 634–641.
<https://doi.org/10.2527/2001.793634x>.
- Fulton, R., Cook, B., L Step, D., Confer, A., T Saliki, J., Payton, M., Burge, L., Welsh, R., Shawn Blood, K., 2002. Evaluation of health status of calves and the impact on feedlot performance: Assessment of a retained ownership program for postweaning calves. *Canadian journal of veterinary research = Revue canadienne de recherche vétérinaire* 66, 173–80.
<https://www.ncbi.nlm.nih.gov/pubmed/12146889>.
- Galyean, M.L., Gunter, S.A., Berrie, R.A., Malcolm-Callis, K.J., Brazle, F.K., Essig, H.W., 1995. Effects of Zinc Source and Level and Added Copper Lysine in the Receiving Diet on Performance by Growing and Finishing Steers. *The Professional Animal Scientist* 11, 139–148. [https://doi.org/10.15232/S1080-7446\(15\)32578-X](https://doi.org/10.15232/S1080-7446(15)32578-X).
- Galyean, M. L. 1996. Disparity between requirements for zinc and current fortification levels in beef cattle finishing diets. In: *Proc. Southwest Nutrition and Management Conference*. pp 27–32. University of Arizona, Tucson.
- Galyean, M.L., Perino, L.J., Duff, G.C., 1999. Interaction of cattle health/immunity and nutrition. *Journal of Animal Science* 77, 1120.
- Garcia, M.D., Thallman, R.M., Wheeler, T.L., Shackelford, S.D., Casas, E., 2010. Effect of bovine respiratory disease and overall pathogenic disease incidence on carcass traits 1,2. *Journal of Animal Science* 88, 491–496.
<https://doi.org/10.2527/1999.7751120x>.

- Gardner, B.A., Dolezal, H.G., Bryant, L.K., Owens, F.N., Smith, R.A., 1999. Health of finishing steers: effects on performance, carcass traits, and meat tenderness. *Journal of Animal Science* 77, 3168. <https://doi.org/10.2527/1999.77123168x>.
- Genther, O.N., Hansen, S.L., 2014. Effect of dietary TM supplementation and a multi-element TM injection on shipping response and growth performance of beef cattle. *J Anim Sci* 92, 2522–2530. <https://doi.org/10.2527/jas.2013-7426>.
- Georgievskii, V.I., Annenkov, B.N., Samokhin, V.T., 1982. Mineral nutrition of animals, Studies in the agricultural and food sciences. Butterworths, London; Boston.
- Gerloff, B.J., 1992. Effect of selenium supplementation on dairy cattle. *J Anim Sci* 70, 3934–3940. <https://doi.org/10.2527/1992.70123934x>.
- Gibbons, R.A., Dixon, S.N., Hallis, K., Russell, A.M., Sansom, B.F., Symonds, H.W., 1976. Manganese metabolism in cows and goats. *Biochim. Biophys. Acta* 444, 1–10. [https://doi.org/10.1016/0304-4165\(76\)90218-X](https://doi.org/10.1016/0304-4165(76)90218-X).
- Gifford, C.A., Holland, B.P., Mills, R.L., Maxwell, C.L., Farney, J.K., Terrill, S.J., Step, D.L., Richards, C.J., Burciaga Robles, L.O., Krehbiel, C.R., 2012. Growth and Development Symposium: Impacts of inflammation on cattle growth and carcass merit. *J Anim Sci* 90, 1438–1451. <https://doi.org/10.2527/jas.2011-4846>.
- Goswami, T. K., R. Bhar, S. E. Jadhav, S. N. Joardr and G. C. Ram. 2005. Role of dietary zinc as a nutritional modulator. *Asian-Aust. J. Anim. Sci.* 18:439-452. <https://doi.org/10.5713/ajas.2005.439>.

- Greene, L.W., Lunt, D.K., Byers, F.M., Chirase, N.K., Richmond, C.E., Knutson, R.E., Schelling, G.T., 1988. Performance and carcass quality of steers supplemented with zinc oxide or zinc methionine. *J. Anim. Sci.* 66, 1818–1823.
<https://doi.org/10.2527/jas1988.6671818x>.
- Griffin, D. 2016a. “Learn now which products require a VFD.” *Progressive Cattleman* 6(4): 9–11.
- Griffin, D. 2016b. “Starting points to help apply new VFD rules.” *Progressive Cattleman* 6(1): 41–43.
- Gulliksen, S.M., Lie, K.I., Løken, T., Østerås, O., 2009. Calf mortality in Norwegian dairy herds. *Journal of Dairy Science* 92, 2782–2795.
<https://doi.org/10.3168/jds.2008-1807>.
- Harison, J. H. and H. R. Conrad. 1984. Effect of selenium intake on selenium utilization by the nonlactating dairy cow. *J. Dairy Sci.* 67:219-223.
[https://doi.org/10.3168/jds.s0022-0302\(84\)81288-6](https://doi.org/10.3168/jds.s0022-0302(84)81288-6).
- Healy, J., Tipton, K., 2007. Ceruloplasmin and what it might do. *J Neural Transm* 114, 777–781. Hoehn, K.B., Fowler, A., Lunter, G., Pybus, O.G., 2016. The Diversity and Molecular Evolution of B-Cell Receptors during Infection. *Mol Biol Evol* 33, 1147–1157. <https://doi.org/10.1007/s00702-007-0687-7>.
- Hidiroglou, M., 1979. Manganese in Ruminant Nutrition. *Can. J. Anim. Sci.* 59, 217–236. <https://doi.org/10.4141/cjas79-028>.
- Hidiroglou, M., 1980. Effect of Dietary Manganese on Growth and Manganese Metabolism in Sheep. *Journal of Dairy Science* 63, 385–390.
[https://doi.org/10.3168/jds.s0022-0302\(80\)82944-4](https://doi.org/10.3168/jds.s0022-0302(80)82944-4).

Hoekstra, W.G., 1975. Biochemical function of selenium and its relation to vitamin E.

Fed. Proc. 34, 2083–2089.

Holland, B.P., Burciaga-Robles, L.O., VanOverbeke, D.L., Shook, J.N., Step, D.L.,

Richards, C.J., Krehbiel, C.R., 2010. Effect of bovine respiratory disease during preconditioning on subsequent feedlot performance, carcass characteristics, and beef attributes. *J. Anim.Sci.* 88, 2486–2499. <https://doi.org/10.2527/jas.2009-2428>.

Howes, A.D., Dyer, I.A., 1971. Diet and supplemental mineral effects on manganese

metabolism in newborn calves. *J. Anim. Sci.* 32, 141–145.

<https://doi.org/10.2527/jas1971.321141x>.

Hughes, H.D., Carroll, J.A., Sanchez, N.C.B., Richeson, J.T., 2014. Natural variations in

the stress and acute phase responses of cattle. *Innate Immun* 20, 888–896.

<https://doi.org/10.1177%2F1753425913508993>.

Hurley, L.S., 1981. Teratogenic aspects of manganese, zinc, and copper nutrition.

Physiological Reviews 61, 249–295.

<https://doi.org/10.1152/physrev.1981.61.2.249>.

Hurley, L.S., and C.L. Keen. 1987. Manganese. Pages 185-223 in *Trace Elements in*

Human and Animal Nutrition (Vol. 1). W. Mertz, ed. Academic Press Inc., New York, NY.

Hutcheson, D.P., Cole, N.A., 1986. Management of Transit-Stress Syndrome in Cattle:

Nutritional and Environmental Effects. *J Anim Sci* 62, 555-560.

<https://doi.org/10.2527/jas1986.622555x>.

- Jackson, K.J.L., Kidd, M.J., Wang, Y., Collins, A.M., 2013. The shape of the lymphocyte receptor repertoire: lessons from the B cell receptor. *Front Immunol* 4, 263.
- Johnson, A. B., M. T. Socha, T. M. Fakler and C. K. Swenson. 1998. Role of trace minerals in immune function. ASAS/ADSA Nutrition – Immunity Interaction Symposium, July.
- Johnson W, Norman B, Dunbar J. Selenium improves weight gain of beef calves. *Calif Agr.* 1979. 33(3):14-16.
- Kelley KW: Immunological consequences of changing environmental stimuli. In Moberg, GP (ed): *Animal Stress*. Bethesda, American Physiological Society, 1985, p 193 55.
- Kelley KW: Stress and immune function: A bibliographic review. *Ann Rech Vet* 11:445, 1980 56.
- Kincaid, R. L. 1999. Assessment of trace mineral status of ruminants: A review. Available: <http://www.asas.org/symposia/proceedings/0930.pdf>. Accessed May 14, 2014.
- Kurz, M. W. 2004. Nutritional modulation of immunity and physio-logical responses in beef calves. M.S. thesis. Texas A&M Univ., College Station.
- Lawler, T.L., Taylor, J.B., Finley, J.W., Caton, J.S., 2004. Effect of supranutritional and organically bound selenium on performance, carcass characteristics, and selenium distribution in finishing beef steers. *J. Anim. Sci.* 82, 1488–1493. <https://doi.org/10.2527/2004.8251488x>.

- Lay, D.C., Randel, R.D., Friend, T.H., Jenkins, O.C., Neuendorff, D.A., Bushong, D.M., Lanier, E.K., Bjorge, M.K., 1997. Effects of prenatal stress on suckling calves. *J. Anim. Sci.* 75, 3143–3151. <https://doi.org/10.2527/1997.75123143x>.
- Leach, R. M., Jr., and E. D. Harris. 1997. Manganese. Pages 335– 356 in *Handbook of Nutritionally Essential Mineral Elements*. B. L. O'Dell and R. A. Sunde, ed. Marcel Dekker Inc., New York, NY.
- Legleiter, L.R., Spears, J.W., Lloyd, K.E., 2005. Influence of dietary manganese on performance, lipid metabolism, and carcass composition of growing and finishing steers. *J. Anim. Sci.* 83, 2434–2439. <https://doi.org/10.2527/2005.83102434x>.
- Linder, M.C., Hazegh-Azam, M., 1996. Copper biochemistry and molecular biology. *Am. J. Clin. Nutr.* 63, 797S-811S. <https://doi.org/10.1093/ajcn/63.5.797>.
- Loneragan, G.H., Dargatz, D.A., Morley, P.S., Smith, M.A., 2001. Trends in mortality ratios among cattle in US feedlots. *J. Am. Vet. Med. Assoc.* 219, 1122–1127. <https://doi.org/10.2460/javma.2001.219.1122>.
- Maas, J., Peuroi, J.R., Weber, D.W., Adams, F.W., 1994. Safety, efficacy, and effects on copper metabolism of intrarectally placed selenium boluses in beef heifer calves. *Am. J. Vet. Res.* 55, 247–250.
- Mackenzie, A.M., Drennan, M., Rowan, T.G., Dixon, J.B., Carter, S.D., 1997. Effect of transportation and weaning on humoral immune responses of calves. *Res. Vet. Sci.* 63, 227–230. [https://doi.org/10.1016/s0034-5288\(97\)90025-4](https://doi.org/10.1016/s0034-5288(97)90025-4).
- Mader, T.L., Davis, M.S., Brown-Brandl, T., 2006. Environmental factors influencing heat stress in feedlot cattle. *J. Anim. Sci.* 84, 712–719. <https://doi.org/10.2527/2006.843712x>.

- Malcolm-Callis, K.J., Duff, G.C., Gunter, S.A., Kegley, E.B., Vermeire, D.A., 2000. Effects of supplemental zinc concentration and source on performance, carcass characteristics, and serum values in finishing beef steers. *J. Anim. Sci.* 78, 2801–2808. <https://doi.org/10.2527/2000.78112801x>.
- Maret, W. (2002). In "Trace Elements in Man and Animals (TEMA-II)" (J. R. Prohaska and J. C. King, eds.), p. 30. Univ. California, Berkeley, CA.
- Martin, J.J., Strasia, C.A., Gill, D.R., Hicks, R.B., Ridenour, K., Dolezal, G., Owens, F.N., 1987. Effect of zinc methionine on live performance and carcass merit of feedlot steers. Miscellaneous publication - Agricultural Experiment Station, Oklahoma State University (USA).
- Marshall, J.S., Warrington, R., Watson, W., Kim, H.L., 2018. An introduction to immunology and immunopathology. *Allergy Asthma Clin Immunol* 14.
- Masters, D.G., Paynter, D.I., Briegel, J., Baker, S.K., Purser, D.B., 1988. Influence of manganese intake on body, wool and testicular growth of young rams and on the concentration of manganese and the activity of manganese enzymes in tissues. *Aust. J. Agric. Res.* 39, 517-524. <https://doi.org/10.1071/AR9880517>.
- Maxwell, C., Hagenmaier, J., Betts, N., Rutten-Ramos, S., 2018. 499 The effects of disease on growth efficiency and carcass composition of beef cattle. *J Anim Sci* 96, 276–276. <https://doi.org/10.1093/jas/sky404.606>.
- McDonald, D.R., Levy, O., 2013. 3 - Innate immunity, in: Rich, R.R., Fleisher, T.A., Shearer, W.T., Schroeder, H.W., Frew, A.J., Weyand, C.M. (Eds.), *Clinical Immunology (Fourth Edition)*, London, pp. 35–46.

- McDowell, L. R. 2003. *Minerals in Animal and Human Nutrition*. 2nd ed. Elsevier Science B.V., Amsterdam, The Netherlands.
- McDowell, L. R. 1996. Feeding minerals to cattle on pasture. *Anim. Feed. Sci. Tech.* 60: 247- 271.
- McDowell, L.R. 1992. *Minerals in Animal and Human Nutrition*. Academic Press Inc., New York. NY.
- McDowell, L. R. 1985. *Nutrition of grazing ruminants in warm climates*. Academic Press, New York.
- McGee, M., Ramirez, J.A., Carstens, G.E., Price, W.J., Hall, J.B., Hill, R.A., 2014. Relationships of feeding behaviors with efficiency in RFI-divergent Japanese Black cattle. *J. Anim. Sci.* 92, 3580–3590. <https://doi.org/10.2527/jas.2013-7360>.
- McNeill, J.W., J.C. Paschal, M.S. McNeill, and W.W. Morgan. 1996. Effect of morbidity on performance and profitability of feedlot steers. *J. Anim. Sci.* 74(Suppl. 1):135.
- Meylan, E., J. Tschopp, and M. Karin. 2006. Intracellular pattern recognition receptors in the host response. *Nature* 442:39-44. <https://doi.org/10.1038/nature04946>.
- Moberg G. & Mench A., 2000, *The biology of animal stress: Basic principles and implications for animal welfare*, CABI, Wallingford, United Kingdom.
- Mogensen, T.H., 2009. Pathogen Recognition and Inflammatory Signaling in Innate Immune Defenses. *Clin Microbiol Rev* 22, 240–273.

- Mormed, P., Soissons, J., Bluthe, R.-M., Raoult, J., Legarff, G., LevieuxE, D., Dantzer, R., Chaillou, J.F., Geffard, M.-C., Armoux, D., Bouyer, J., 1982. Effect of transportation on blood serum composition, disease incidence and production traits in young calves. Influence of the journey duration. *Annales de Recherches Vétérinaires* 13, 369–384.
- Mylrea, P.J., Byrne, D.T., 1974. An Outbreak of Acute Copper Poisoning in Calves. *Australian Veterinary Journal* 50, 169–172. <https://doi.org/10.1111/j.1751-0813.1974.tb06885.x>.
- National Academies of Sciences, E., 2016. *Nutrient Requirements of Beef Cattle: Eighth Revised Edition*.
- NAHMS, 2000a. *Feedlot '99 Part II: Baseline Reference of Feedlot Health and Health Management*. USDA, APHIS, National Animal Health Monitoring Systems. May 15, 2019.
- NRC, 1996. *Nutrient Requirements of Beef Cattle*. 7th rev. ed. Natl. Acad. Press, Washington, D.C
- Niedermayer, E.K., Genter-Schroeder, O.N., Loy, D.D., Hansen, S.L., 2017. The effects of injectable TMs on growth performance and mineral status of Angus beef steers raised in a natural feedlot program. *The Professional Animal Scientist* 33, 186–193. <https://doi.org/10.15232/pas.2016-01570>.
- Nunnery, G. A., G. E. Carstens, and L. W. Greene. 1996. Feedlot performance and carcass characteristics in steers fed different sources and levels of supplemental zinc. *J. Anim. Sci.* 74 (Suppl.1):294.

- Nunnery, G. A. 1998. Effects of source and level of zinc on feedlot performance and carcass characteristics in steers. M.S. Thesis, Texas A&M University College Station.
- Orr, C.L., Hutcheson, D.P., Grainger, R.B., Cummins, J.M., Mock, R.E., 1990. Serum copper, zinc, calcium and phosphorus concentrations of calves stressed by bovine respiratory disease and infectious bovine rhinotracheitis. *J. Anim. Sci.* 68, 2893–2900. <https://doi.org/10.2527/1990.6892893x>.
- Ott, E. A., W. H. Smitii, R. B. Harrington, H. E. Parker, and W. M. Beeson. 1966. Zinc toxicity in ruminants. IV. Physiological changes in tissues of beef cattle. *J. Anim. Sci.* 25:432-438. <https://doi.org/10.2527/jas1966.252432x>.
- Ott, T.L., 2019. Symposium review: Immunological detection of the bovine conceptus during early pregnancy. *Journal of Dairy Science* 102, 3766–3777. <https://doi.org/10.3168/jds.2018-15668>.
- Patterson, J. A. and T. E. Engle. 2005. Trace mineral nutrition in beef cattle. Presented at the 2005 Nutrition Conference. University of Tennessee.
- Petrie, H.T., Klassen, L.W., Klassen, P.S., O'Dell, J.R., Kay, H.D., 1989. Selenium and the immune response: 2. Enhancement of murine cytotoxic T-lymphocyte and natural killer cell cytotoxicity in vivo. *J. Leukoc. Biol.* 45, 215–220. <https://doi.org/10.1002/jlb.45.3.215>.
- Pogge, D.J., Richter, E.L., Drewnoski, M.E., Hansen, S.L., 2012. Mineral concentrations of plasma and liver after injection with a TM complex differ among Angus and Simmental cattle. *J. Anim. Sci.* 90, 2692–2698. <https://doi.org/10.2527/jas.2012-4482>.

- Pringle, W.L., Dawley, W.K., Miltimore, J.E., 1973. Sufficiency of cu and zn in barley, forage, and corn silage rations as measured by response to supplements by beef cattle. *Can. J. Anim. Sci.* 53, 497–502. <https://doi.org/10.4141/cjas73-076>.
- Reffett, J.K., Spears, J.W., Brown, T.T., 1988. Effect of Dietary Selenium on the Primary and Secondary Immune Response in Calves Challenged with Infectious Bovine Rhinotracheitis Virus. *The Journal of Nutrition* 118, 229–235. <https://doi.org/10.1093/jn/118.2.229>.
- Reid, R. L. and D. J. Horvath. 1980. Soil Chemistry and mineral problems in farm livestock. A Review. *Anim. Feed. Sci. Technol.* 5: 95-167. [https://doi.org/10.1016/0377-8401\(80\)90002-4](https://doi.org/10.1016/0377-8401(80)90002-4).
- Rhoads, A. R., T. L. Stanton, T. E. Engle, and C. V. Kimberling. 2003. Effects of concentration and source of TMs on performance, immunity, mineral and lipid metabolism, and carcass characteristics of beef steers. *Prof. Anim. Sci.* 19:150.
- Richards, C.J., Blalock, H.M., Jacques, K.A., Loveday, H.D., 2011. Efficacy of feeding selenium-enriched yeast to finishing beef cattle. *The Professional Animal Scientist* 27, 1–8. [https://doi.org/10.15232/S1080-7446\(15\)30437-X](https://doi.org/10.15232/S1080-7446(15)30437-X).
- Richeson, J.T., Hughes, H.D., Broadway, P.R., Carroll, J.A., 2019. Vaccination Management of Beef Cattle: Delayed Vaccination and Endotoxin Stacking. *Veterinary Clinics: Food Animal Practice* 35, 575–592. <https://doi.org/10.1016/j.cvfa.2019.07.003>.
- Romagnani, S., 1999. Th1/Th2 cells. *Inflamm. Bowel Dis.* 5, 285–294.

- Roth, J.A., 1985. Cortisol as Mediator of Stress-Associated Immunosuppression in Cattle, in: Moberg, G.P. (Ed.), *Animal Stress*. Springer, New York, NY, pp. 225–243.
https://www.researchgate.net/deref/http%3A%2F%2Fdx.doi.org%2F10.1007%2F978-1-4614-7544-6_13.
- Roth, J.A., Perino, L.J., 1998. Immunology and Prevention of Infection in Feedlot Cattle. *Veterinary Clinics of North America: Food Animal Practice* 14, 233–256.
[https://doi.org/10.1016/s0749-0720\(15\)30252-8](https://doi.org/10.1016/s0749-0720(15)30252-8).
- Rust, S. R., and M. L. Schlegel. 1993. Effect of dietary zinc level and source on steer intake, growth and carcass characteristics. *J. Anim. Sci.* 71(Suppl. 1):249.
- Samuelson, K.L., Hubbert, M.E., Galyean, M.L., Löest, C.A., 2016. Nutritional recommendations of feedlot consulting nutritionists: The 2015 New Mexico State and Texas Tech University survey. *J Anim Sci* 94, 2648–2663.
<https://doi.org/10.2527/jas.2016-0282>
- Sarmah, A.K., Meyer, M.T., Boxall, A.B.A., 2006. A global perspective on the use, sales, exposure pathways, occurrence, fate and effects of veterinary antibiotics (VAs) in the environment. *Chemosphere* 65, 725-759.
<https://doi.org/10.1016/j.chemosphere.2006.03.026>.
- Schneider, M.J., Tait, R.G., Busby, W.D., Reecy, J.M., 2009. An evaluation of bovine respiratory disease complex in feedlot cattle: Impact on performance and carcass traits using treatment records and lung lesion scores,. *J Anim Sci* 87, 1821–1827.
<https://doi.org/10.2527/jas.2008-1283>.

- Serra, A.B., Nakamura, K., Matsui, T., Harumoto, T., Fujihara, T., 1994. Inorganic selenium for sheep. Selenium balance and selenium levels in the different ruminal fluid fractions. *Asian-Australasian Journal of Animal Sciences* 7, 83–89. <https://doi.org/10.5713/ajas.1994.83>.
- Shand, A., Lewis, G., 1957. Chronic copper poisoning in young calves. *Veterinary Record* 69, 618–620.
- Smith, D.L., Harris, A.D., Johnson, J.A., Silbergeld, E.K., Morris, J.G., 2002. Animal antibiotic use has an early but important impact on the emergence of antibiotic resistance in human commensal bacteria. *Proc Natl Acad Sci U S A* 99, 6434–6439. <https://dx.doi.org/10.1073%2Fpnas.082188899>
- Smith, R.A., 1998. Impact of disease on feedlot performance: a review. *J Anim Sci* 76, 272–274. <https://doi.org/10.2527/1998.761272x>.
- Snowder, G.D., 2006. Bovine respiratory disease in feedlot cattle: Environmental, genetic, and economic factors. *Journal of Animal Science* 84, 1999–2008. <https://doi.org/10.2527/jas.2006-046>.
- Snyder, P.W., 2017. Chapter 5 - Diseases of Immunity¹, in: Zachary, J.F. (Ed.), *Pathologic Basis of Veterinary Disease (Sixth Edition)*. Mosby, pp. 242-285.e5.
- Spears, J. W., and L. J. Samsell. 1984. Effect of zinc supplementation on performance and zinc status of growing heifers. *J. Anim. Sci.* 49(Suppl. 1):407 (Abstr.).
- Spears J. W. 1991. Chelated trace minerals in ruminant nutrition. *Proc. 2nd Annual Florida Ruminant Nutrition Sym.* Gainesville, FL. p. 1-13.

- Spears, J. W. 1995. An update on mineral nutrition of growing and finishing beef cattle. In: Proc. Plains Nutrition Council Symposium Update on Mineral Nutrition of Beef Cattle. Publ. No. TAMUS-AREC-95-2, Texas A&M Univ. Res. and Ext. Center, Amarillo. p 17.
- Spears, J.W., Kegley, E.B., 2002. Effect of zinc source (zinc oxide vs zinc proteinate) and level on performance, carcass characteristics, and immune response of growing and finishing steers. *J. Anim. Sci.* 80, 2747–2752.
<https://doi.org/10.2527/2002.80102747x>.
- Stabel, J.R., Spears, J.W., Brown, T.T., 1993. Effect of copper deficiency on tissue, blood characteristics, and immune function of calves challenged with infectious bovine rhinotracheitis virus and *Pasteurella hemolytica*. *Journal of Animal Science* 71, 1247. <https://doi.org/10.2527/1993.7151247x>.
- Suttle, N.F., 2010. Mineral nutrition of livestock, 4. ed. ed. CABI, Wallingford, Oxfordshire.
- Suttle, N. F. and D. G. Jones. 1989. Recent developments in trace mineral element metabolism and function: trace elements disease resistance and immune responsiveness in ruminants. *J. Nutri.* 119: 1055-1061.
<https://doi.org/10.1093/jn/119.7.1055>.
- Taylor, J.D., Fulton, R.W., Lehenbauer, T.W., Step, D.L., Confer, A.W., 2010. The epidemiology of bovine respiratory disease: What is the evidence for preventative measures? *Can Vet J* 51, 1351–1359.
- Tizard, I.R., 2012. *Veterinary Immunology*. Elsevier/Saunders.

- Thompson, K.M., Haibach, H., Sunde, R.A., 1995. Growth and plasma triiodothyronine concentrations are modified by selenium deficiency and repletion in second-generation selenium-deficient rats. *J. Nutr.* 125, 864–873.
<https://doi.org/10.1093/jn/125.4.864>.
- Tomlinson, D., Socha, M., Defrain, J., 2008. Role of trace minerals in the immune system. *Proc. Penn State Dairy Cattle Nutrition Workshop* 39–52.
- Tuncer, P., Yeşilbağ, K., 2015. Serological detection of infection dynamics for respiratory viruses among dairy calves. *Veterinary Microbiology* 180, 180–185.
<https://doi.org/10.1016/j.vetmic.2015.08.025>.
- USDA - NASS, 2020. URL
https://www.nass.usda.gov/Surveys/Guide_to_NASS_Surveys/Cattle_On_Feed/index.php (accessed 4.6.20).
- Underwood, E. J. 1971. *Trace Elements in Human and Animal Nutrition*. 3rd Ed., Academic Press, New York.
- Underwood, E.J., Suttle, N.F., 1999. *The mineral nutrition of livestock*, 3rd ed. ed. CABI Pub, Wallingford, Oxon, UK; New York.
- Underwood, E.J. 1981 L. *The Mineral Nutrition of Livestock*, 2nd edition. Slough, Commonwealth Agricultural Bureaux.
- Valdivia, R., Ammerman, C.B., Wilcox, C.J., Henry, P.R., 1978. Effect of Dietary Aluminum on Animal Performance and Tissue Mineral Levels in Growing Steers. *J Anim Sci* 47, 1351–1356. <https://doi.org/10.2527/jas1978.4761351x>.

- Watson, L.T., Ammerman, C.B., Feaster, J.P., Roessler, C.E., 1973. Influence of manganese intake on metabolism of manganese and other minerals in sheep. *J. Anim. Sci.* 36, 131–136. <https://doi.org/10.2527/jas1973.361131x>.
- Webster Marketon, J.I., Glaser, R., 2008. Stress hormones and immune function. *Cellular Immunology* 252, 16–26. <https://doi.org/10.1016/j.cellimm.2007.09.006>
- Wessely-Szponder, J., 2008. The influence of TNF α and IL-8 on secretory action of neutrophils isolated from heifers in the course of bovine respiratory disease. *Acta Veterinaria Hungarica* 56, 187–196. <https://doi.org/10.1556/avet.56.2008.2.6>.
- Wherry, E.J., Masopust, D., 2016. Chapter 5 - Adaptive Immunity: Neutralizing, Eliminating, and Remembering for the Next Time, in: Katze, M.G., Korth, M.J., Law, G.L., Nathanson, N. (Eds.), *Viral Pathogenesis (Third Edition)*. Academic Press, Boston, pp. 57–69.
- Zust, J., Hrovatin, B., Simundić, B., 1996. Assessment of selenium and vitamin E deficiencies in dairy herds and clinical disease in calves. *Vet. Rec.* 139, 391-394. <https://doi.org/10.1136/vr.139.16.391>.

**Table 1. Composition and chemical analysis
(DM basis) of the finishing ration**

Item		
Ingredient, % DM		SEM ¹
Alfalfa Hay	10.7	
Corn Silage	17.9	
Barley	71.4	
Chemical Analysis		
DM, % as fed	79	0.27
CP, %	13.1	0.09
Fat, %	2.19	0.02
Ash, %	6.59	0.06
Nem, Mcal/lb	0.82	0.003
Neg, Mcal/lb	0.54	0.003
TDN, %	77.22	0.25
Mineral composition		
Calcium, %	0.3	0.01
Phosphorous, %	0.32	0.003
Magnesium, %	0.18	0.004
Potassium, %	0.97	0.02
Sodium, %	0.04	0.002
Iron, ppm	124	28.42
Manganese, ppm	43	2.77
Zinc, ppm	49	1.95
Copper, ppm	12	0.63
Selenium, ppm	0.14	0.01

¹Standard error of the mean (SEM).

Table 2. Composition of mineral mix (DM basis) added to ration¹

Item	
Ingredient, %DM	
Calcium, %	5.81
Chlorine, %	0.11
Magnesium, %	1.47
Sulphur, %	0.11
Cobalt, ppm	178.76
Copper, ppm	7,156.28
Copper, ppm (chelate)	1,788.69
Iodine, ppm	463.70
EDDI, mg/lb	262.36
Manganese, ppm	17,075.81
Manganese, ppm (chelate)	4,268.95
Selenium, ppm	142.30
Selenium, ppm (chelate)	46.90
Zinc, ppm	23,881.04
Zinc, ppm (chelate)	5,970.07
Vit A, KIU/lb	540.32
Vit D, KIU/lb	129.47
Vit D-3, KICU/ lb	129.47
Vit E, IU/lb	5,174.52

¹Mineral was fed at 182.2 g to HM group (n = 10) and 91.1 g to AM group (n = 10).

Table 3. Comparison of trace mineral content between treatments and NRC requirements

	NRC req.	MM ¹	AM ¹	HM ¹
Cobalt, mg/kg	0.15	-	96.48%	180.81%
Copper, mg/kg	10	150%	73.66%	138.03%
Iodine, mg/kg	0.5	-	119.59%	224%
Manganese, ppm	20	50%	87.89%	164.68%
Selenium, mg/kg	0.1	5000%	155.81%	291.94%
Zinc, mg/kg	30	200%	81.94%	153.54%

¹These are the amount of each mineral found in the supplement relative to NRC requirements.

Table 4. Average cortisol level of steers after travel

	Treatment ¹				SEM ³	P-value ²
	AM	CON	HM	MM		
Cortisol, ng/mL	34.385	35.591	35.465	35.277	3.430	0.99

¹Treatments include: a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; n = 10), oral supplementation of minerals provided at levels above NRC requirements (HM; n = 10), a MultiMin® injection at labeled dose (MM; n = 10) or no mineral supplementation (CON; n = 10) for the first 40 d of the trial.

²P-values indicate effect of treatment.

³Standard error of the mean (SEM).

Table 5. Average daily gain of steers during the feedlot phase

	Treatment ^{1,2}				SEM ⁴	P-value ³
	AM	CON	HM	MM		
Average daily gain, kg						
Days 0-13	2.5	2.53	2.81	2.97	0.27	0.55
Days 14-27	1.84	2.22	1.8	1.56	0.26	0.37
Days 28-40	1.8	2.03	2.19	0.71	0.46	0.12
Days 41-55	1.98	1.017	1.12	2.32	0.47	0.16
Days 56 - 69	0.82 ^{ab}	1.72 ^{ab}	3.05 ^b	0.34 ^a	0.46	0.05
Days 70 - 83	2.13	1.29	1.435	2.53	0.44	0.17
Days 84-109	1.29	1.45	2.06	1.41	0.26	0.18
Days 0-109	1.7	1.72	1.94	1.65	0.10	0.18

¹Treatments include: a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; n = 10), oral supplementation of minerals provided at levels above NRC requirements (HM; n = 10), a MultiMin® injection at labeled dose (MM; n = 10) or no mineral supplementation (CON; n = 10) for the first 40 d of the trial.

²Values with different letter superscripts indicate differences ($P < 0.05$) between treatments within that row.

³P-values indicate effect of treatment.

⁴Standard error of the mean (SEM).

Table 6. Carcass Data

	Treatment ¹				SEM ³	P-value ²
	AM	CON	HM	MM		
HCW, kg	333.4	333.5	338.5	327.1	17.2	0.08
Marbling Score	410.9	424.2	551.2	405.7	24.9	0.75
Ribeye Area, cm ²⁰	73.6	75.4	74.6	73.0	2.1	0.88
Ribeye fat thickness, mm	7.62	7.44	7.65	7.14	0.25	0.50
Yield grade	2.3	2.3	2.3	2.1	0.16	0.76
Marb:BF ratio ⁴	-0.33	0.10	0.10	0.12	0.32	0.72

¹Treatments include: a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; n = 10), oral supplementation of minerals provided at levels above NRC requirements (HM; n = 10), a MultiMin® injection at labeled dose (MM; n = 10) or no mineral supplementation (CON; n = 10). for the first 40 d of the trial.

²P-Values indicate differences between treatments.

³Standard error of the mean (SEM).

⁴Marbling to backfat ratio was determined using the calculations previously described by Mohrhauser et al., 2015a. [(observation marbling score - marbling score \bar{x})/marbling SD]- [(observation backfat - backfat \bar{x})/backfat SD]

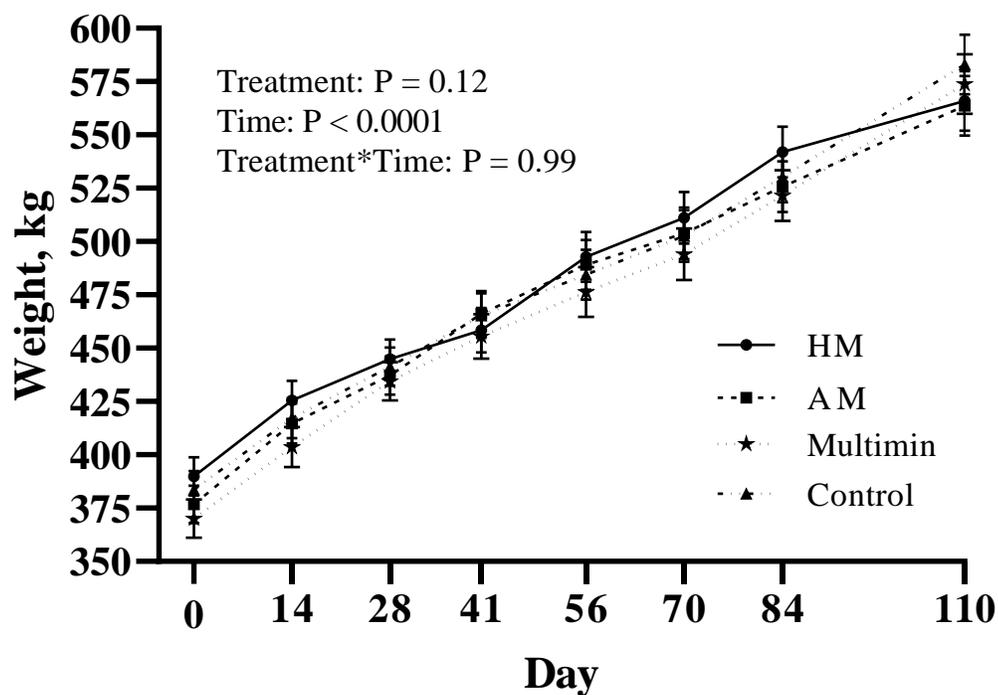


Figure 1. Average weight gain over 110 d finishing period in steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; $n = 10$), oral supplementation of minerals provided at levels above NRC requirements (HM; $n = 10$), a MultiMin® injection at labeled dose (MM; $n = 10$) or no mineral supplementation (CON; $n = 10$) for the first 40 d of the trial. Values are represented as the least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the top left corner of the graph.

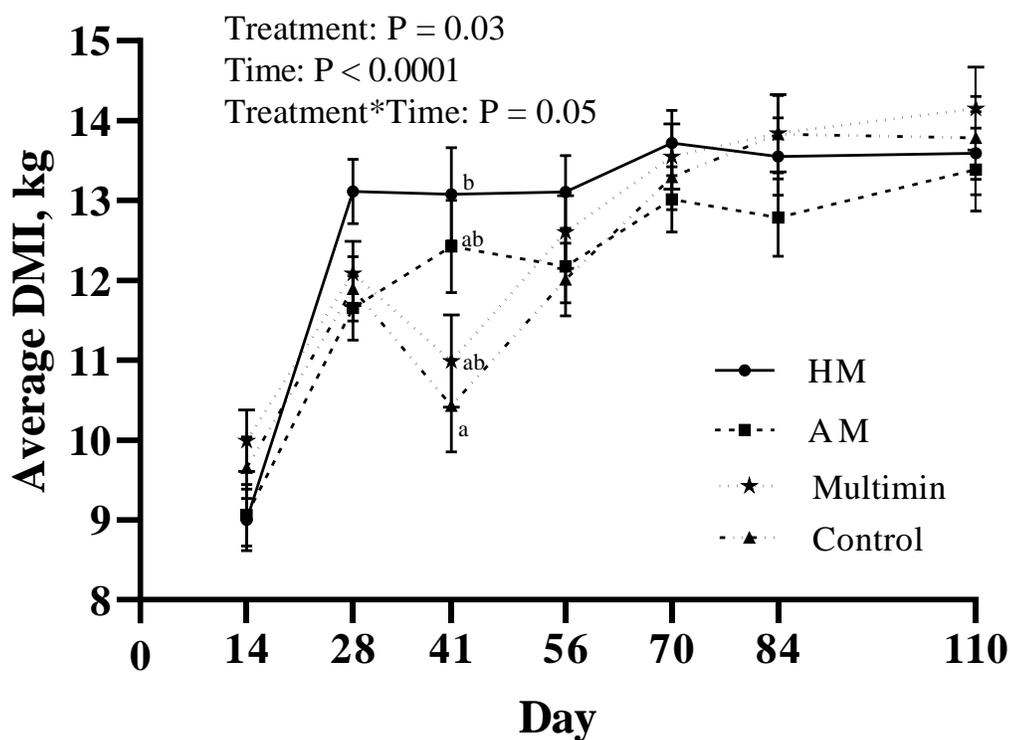


Figure 2. Average dry matter intake (DMI) over 110 d finishing period in steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; $n = 10$), oral supplementation of minerals provided at levels above NRC requirements (HM; $n = 10$), a MultiMin® injection at labeled dose (MM; $n = 10$) or no mineral supplementation (CON; $n = 10$) for the first 40 d of the trial. Values are represented as the least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the top left corner of the graph. Time points with different letters indicate a difference ($P < 0.05$) between the different treatments at that specific time point.

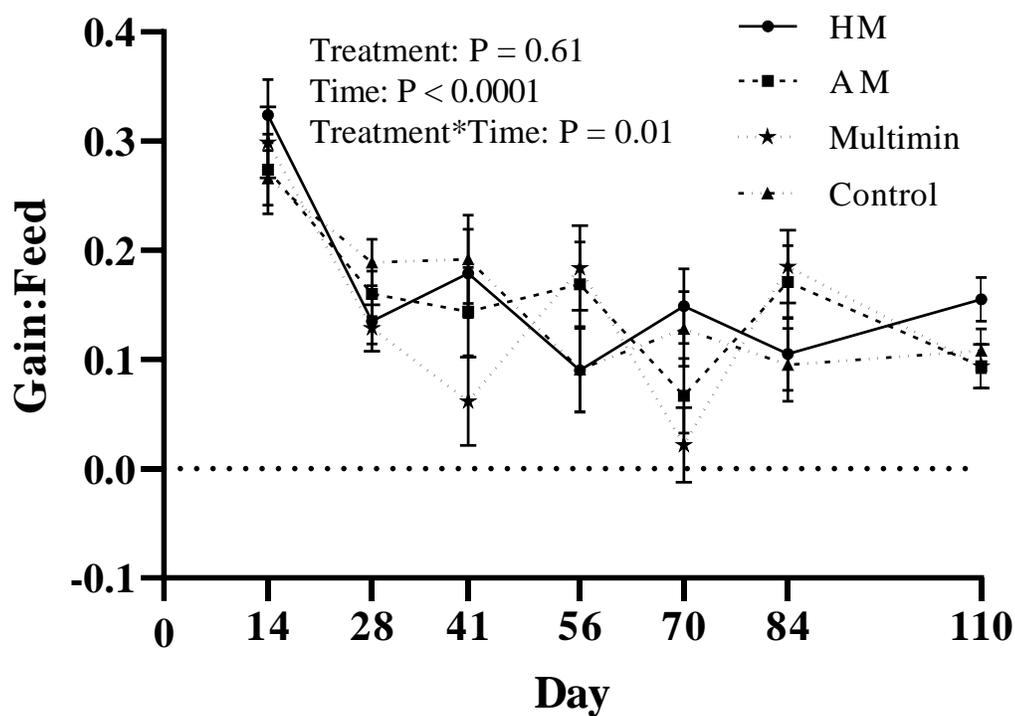


Figure 3. Average gain:feed ratio over 110 d finishing period in steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; n = 10), oral supplementation of minerals provided at levels above NRC requirements (HM; n = 10), a MultiMin® injection at labeled dose (MM; n = 10) or no mineral supplementation (CON; n = 10) for the first 40 d of the trial. Values are represented as the least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the top left corner of the graph.

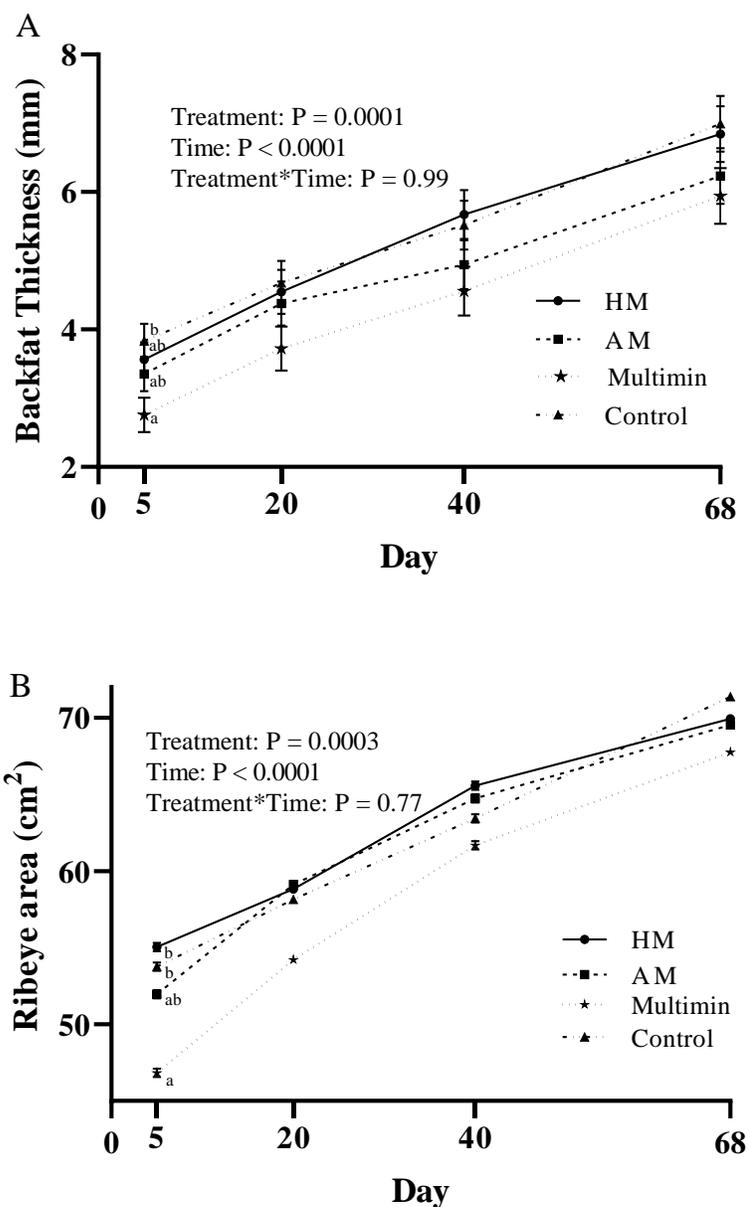


Figure 4. Mean backfat thickness (A) and ribeye area (B) of finishing steers as measured by ultrasound over the 110 d finishing period in steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; $n = 10$), oral supplementation of minerals provided at levels above NRC requirements (HM; $n = 10$), a MultiMin® injection at labeled dose (MM; $n = 10$) or no mineral supplementation (CON; $n = 10$) for the first 40 d of the trial. Values are represented as the least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the top left corner of each graph. Time points with different letters indicate a difference ($P < 0.05$) between the different treatments at that specific time point.

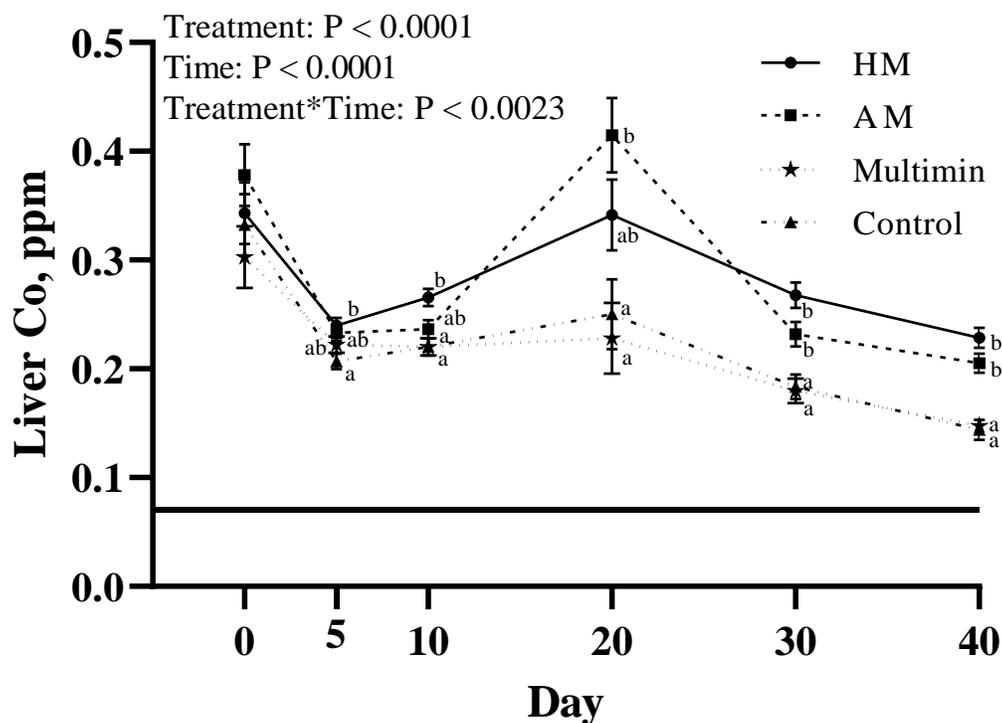


Figure 5. Mean hepatic cobalt (Co) concentrations of finishing steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; $n = 10$), oral supplementation of minerals provided at levels above NRC requirements (HM; $n = 10$), a MultiMin® injection at labeled dose (MM; $n = 10$) or no mineral supplementation (CON; $n = 10$) for the first 40 d of the trial. Any value below the bold black line is indicative of a clinical deficiency. Values are represented as least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the top left corner of the graph. Time points with different letters indicate a difference ($P < 0.05$) between the different treatments at that specific time point.

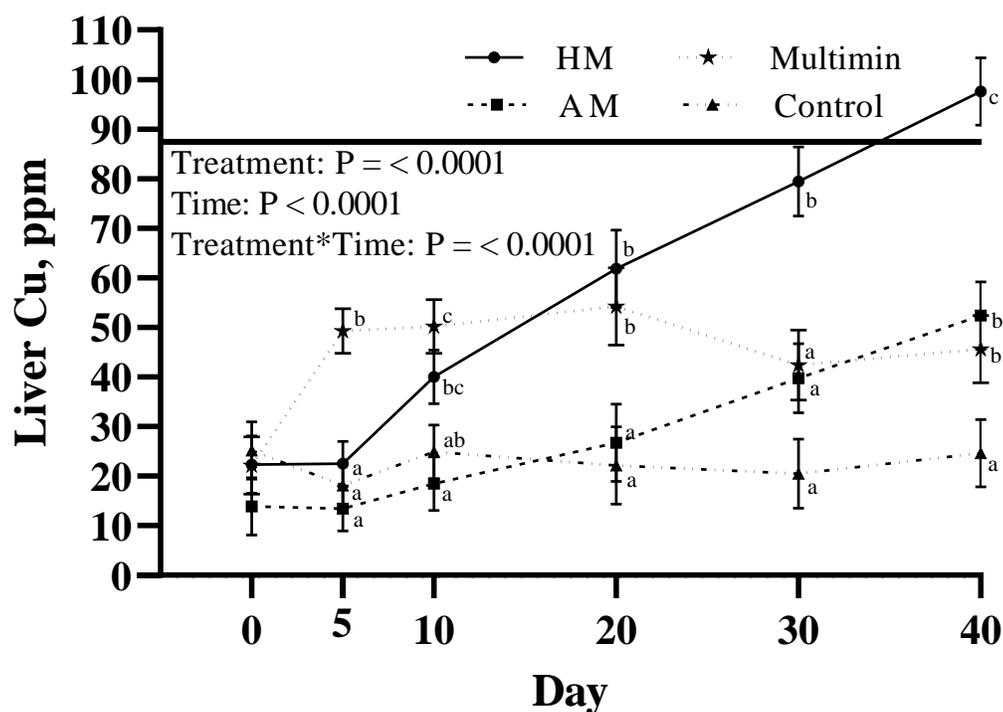


Figure 6. Mean hepatic copper (Cu) concentrations of finishing steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; $n = 10$), oral supplementation of minerals provided at levels above NRC requirements (HM; $n = 10$), a MultiMin® injection at labeled dose (MM; $n = 10$) or no mineral supplementation (CON; $n = 10$) for the first 40 d of the trial. Any value below the bold black line is indicative of a clinical deficiency. Values are represented as least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the top left corner of the graph. Time points with different letters indicate a difference ($P < 0.05$) between the different treatments at that specific time point.

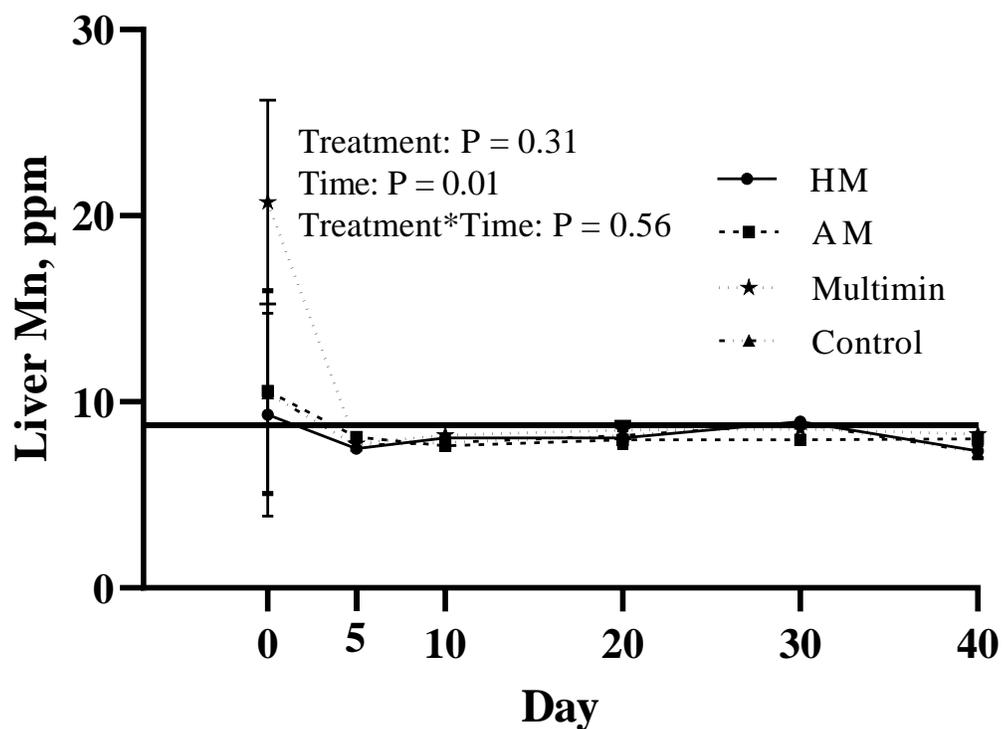


Figure 7. Mean hepatic manganese (Mn) concentrations of finishing steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; n = 10), oral supplementation of minerals provided at levels above NRC requirements (HM; n = 10), a MultiMin® injection at labeled dose (MM; n = 10) or no mineral supplementation (CON; n = 10) for the first 40 d of the trial. Any value below the bold black line is indicative of a clinical deficiency. Values are represented as least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the top left corner of the graph.

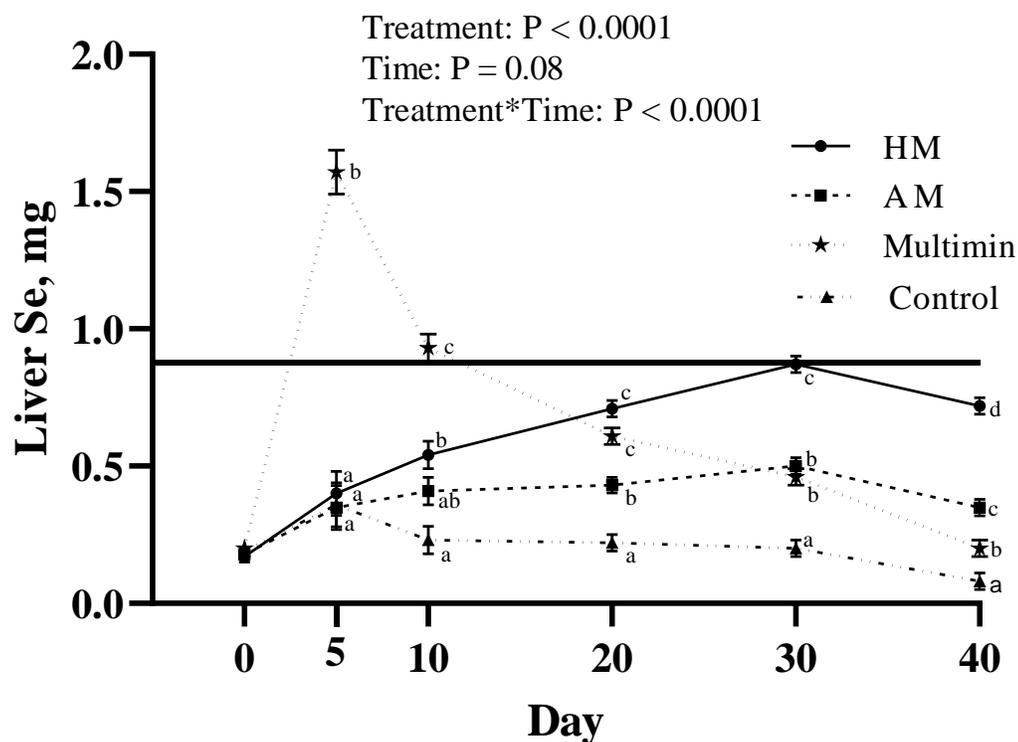


Figure 8. Mean hepatic selenium (Se) concentrations of finishing steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; $n = 10$), oral supplementation of minerals provided at levels above NRC requirements (HM; $n = 10$), a MultiMin® injection at labeled dose (MM; $n = 10$) or no mineral supplementation (CON; $n = 10$) for the first 40 d of the trial. Any value below the bold black line is indicative of a clinical deficiency. Values are represented as least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the top left corner of the graph. Time points with different letters indicate a difference ($P < 0.05$) between the different treatments at that specific time point.

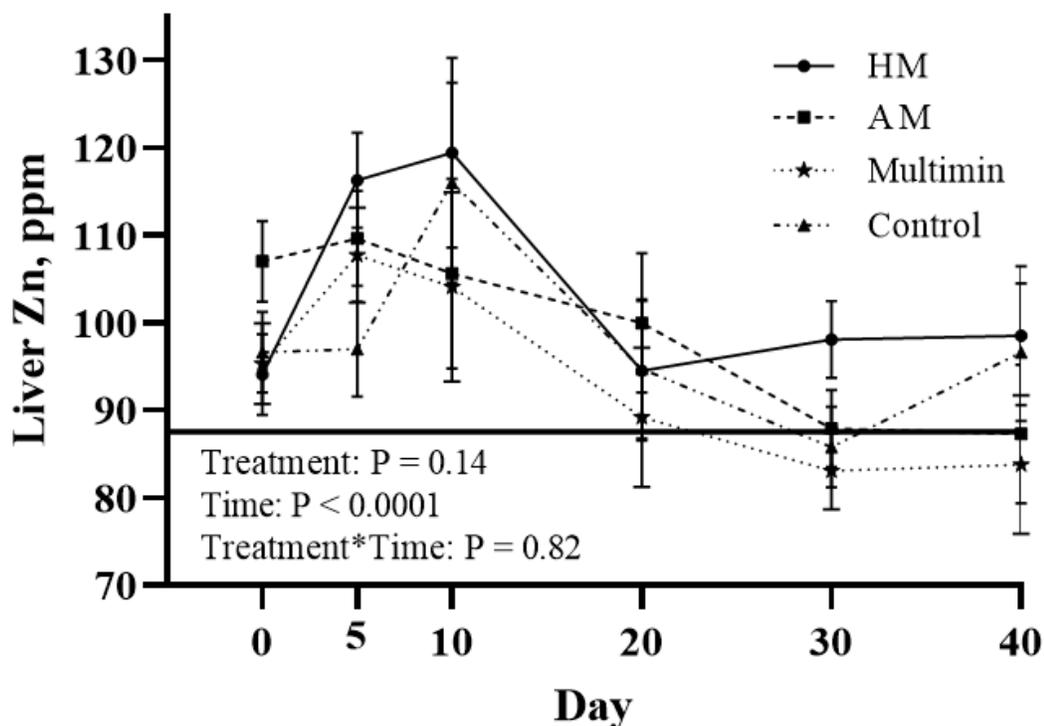


Figure 9. Mean hepatic zinc (Zn) concentrations of finishing steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; $n = 10$), oral supplementation of minerals provided at levels above NRC requirements (HM; $n = 10$), a MultiMin® injection at labeled dose (MM; $n = 10$) or no mineral supplementation (CON; $n = 10$) for the first 40 d of the trial. Any value below the bold black line is indicative of a clinical deficiency. Values are represented as least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the bottom left corner of the graph.

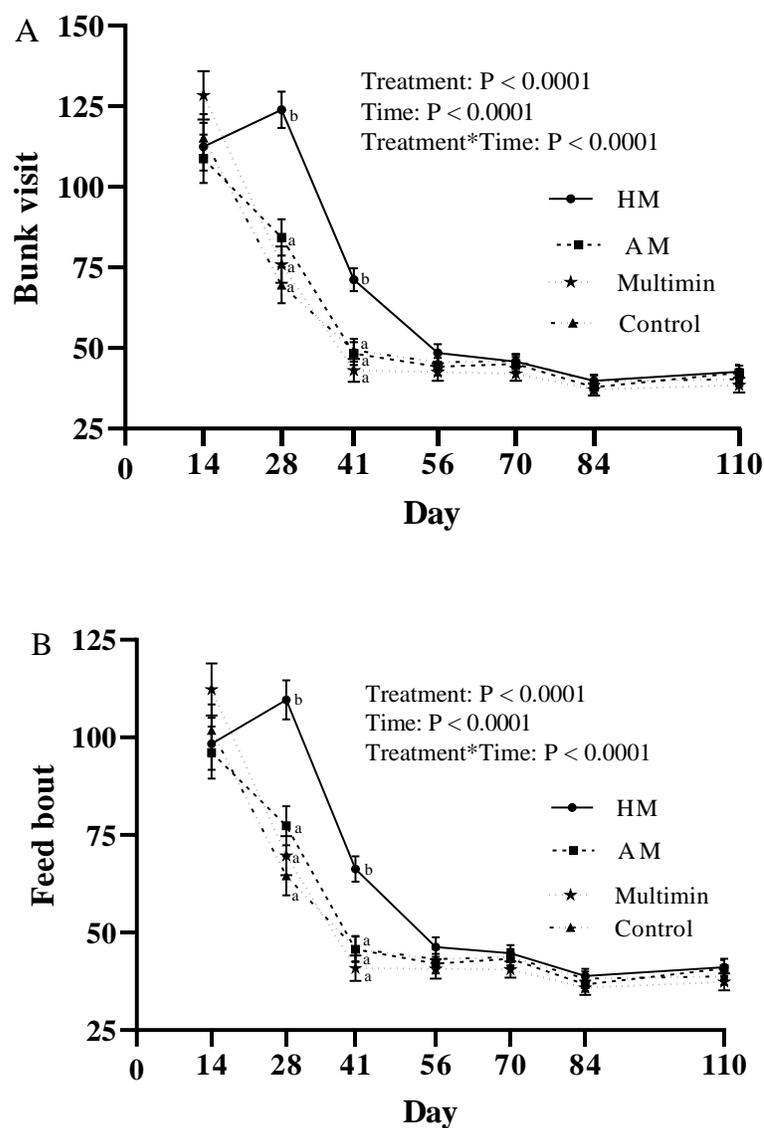


Figure 10. Average time each animal went up to the bunk defined as mean bunk visit (reading of a single animal eid tag when entering at a bunk whether it consumed feed or not, A) and feed bout (reading of a single animal eid tag when entering a bunk and consumed at least 10 g of feed, B) over 110 d finishing period in steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; $n = 10$), oral supplementation of minerals provided at levels above NRC requirements (HM; $n = 10$), a MultiMin® injection at labeled dose (MM; $n = 10$) or no mineral supplementation (CON; $n = 10$) for the first 40 d of the trial. Values are represented as least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the top right corner of the graph. Time points with different letters indicate a difference ($P < 0.05$) between the different treatments at that specific time point.

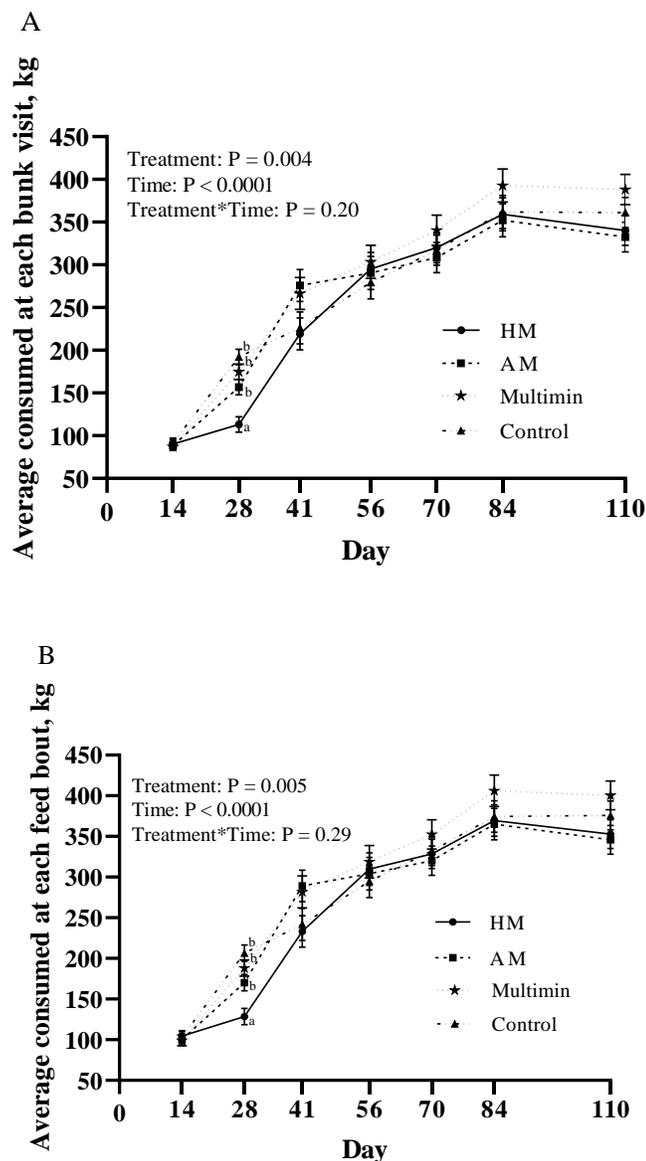


Figure 11. Average amount consumed each time the animal goes to the bunk measured as mean consumption per bunk visit (each time an animal went up to the bunk, A) and consumption per feed bout (when an animal entered a bunk and consumed at least 10 g of feed, B) over 110 d finishing period in steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; n = 10), oral supplementation of minerals provided at levels above NRC requirements (HM; n = 10), a MultiMin® injection at labeled dose (MM; n = 10) or no mineral supplementation (CON; n = 10) for the first 40 d of the trial. Values are represented as least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the top left corner of the graph. Time points with different letters indicate a difference ($P < 0.05$) between the different treatments at that specific time point.

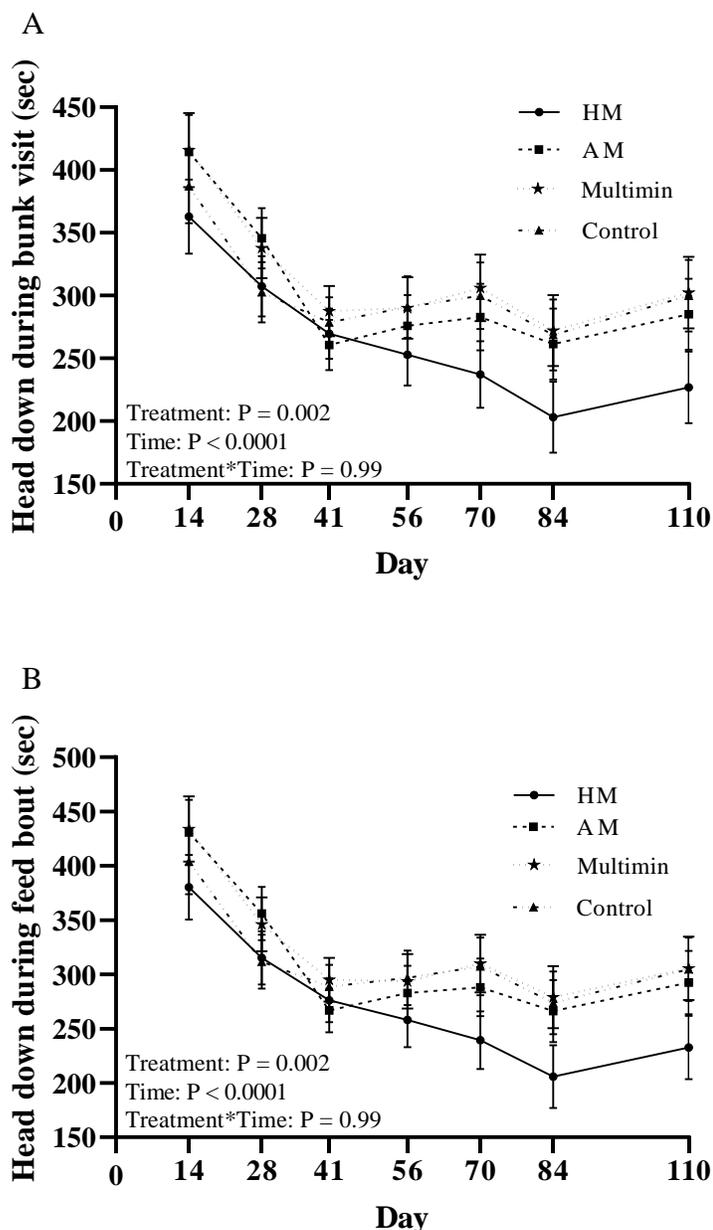


Figure 12. Mean time spent with head down per bunk visit (each time an animal went up to the bunk, A) and time spent with head down per feed bout (when an animal entered a bunk and consumed at least 10 g of feed, B) over 110 d finishing period in steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; n = 10), oral supplementation of minerals provided at levels above NRC requirements (HM; n = 10), a MultiMin® injection at labeled dose (MM; n = 10) or no mineral supplementation (CON; n = 10) for the first 40 d of the trial. Values are represented as least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the bottom left of the graph.

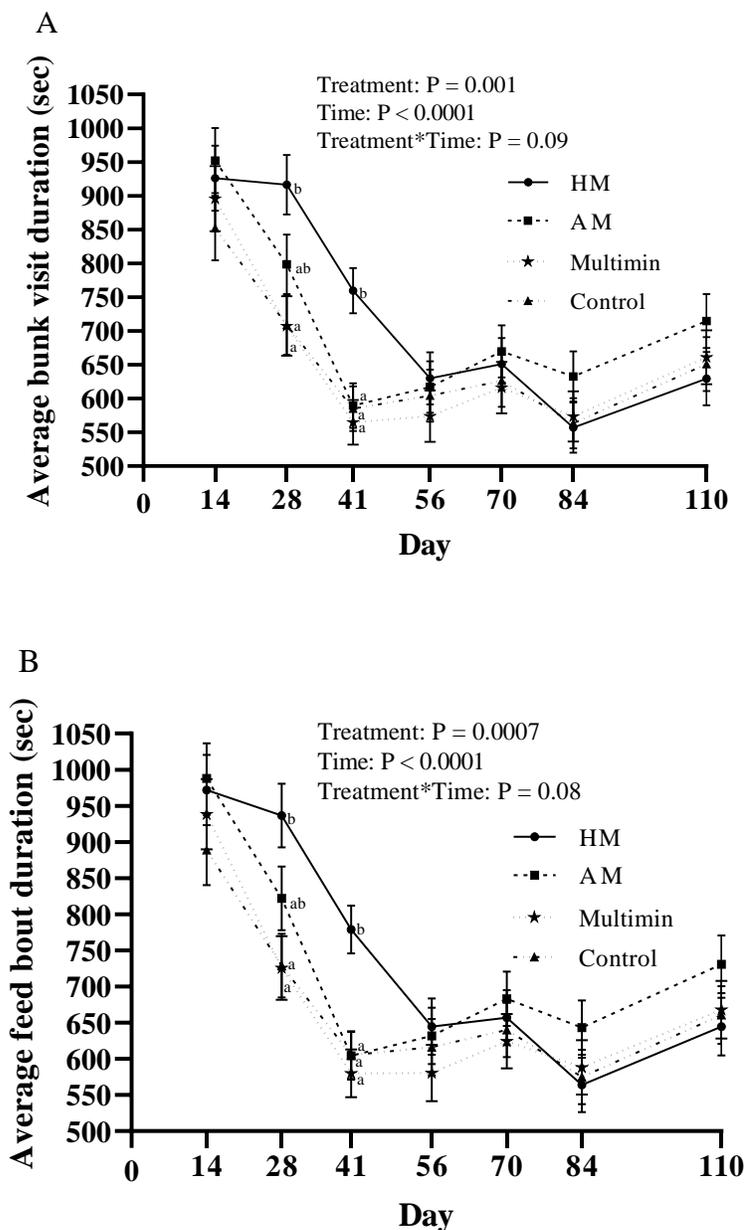


Figure 13. Mean bunk visit duration (each time an animal went up to the bunk, A) (when an animal entered a bunk and consumed at least 10 g of feed, B) over 110 d finishing period in steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; n = 10), oral supplementation of minerals provided at levels above NRC requirements (HM; n = 10), a MultiMin® injection at labeled dose (MM; n = 10) or no mineral supplementation (CON; n = 10) for the first 40 d of the trial. Values are represented as least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the top middle of the graph. Time points with different letters indicate a difference.

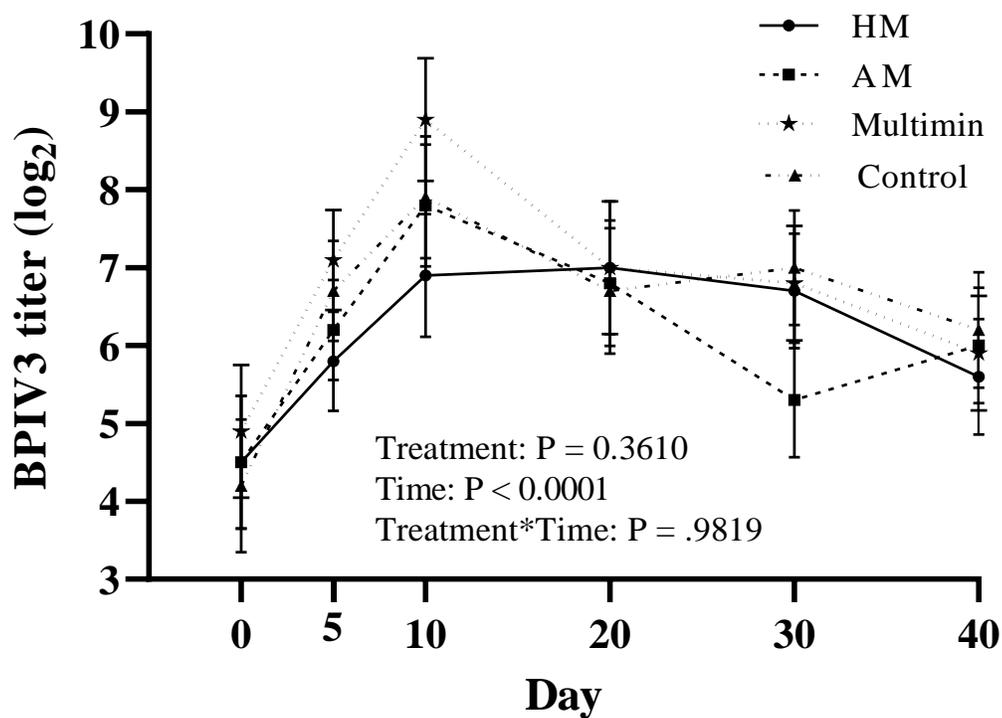


Figure 14. Average antibody titers for BPIV3 of finishing steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; n = 10), oral supplementation of minerals provided at levels above NRC requirements (HM; n = 10), a MultiMin® injection at labeled dose (MM; n = 10) or no mineral supplementation (CON; n = 10) for the first 40 d of the trial. Values are represented as least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the top left corner of the graph.

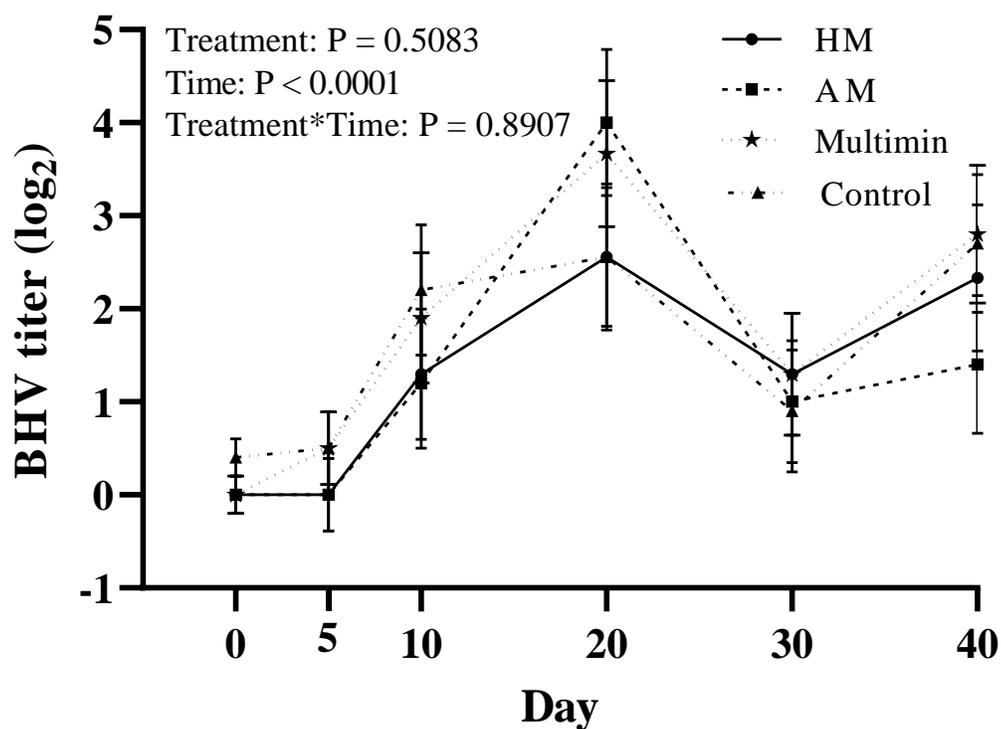


Figure 15. Average antibody titers for BHV of finishing steers receiving a diet containing oral supplementation of minerals provided at levels similar to NRC requirements (AM; n = 10), oral supplementation of minerals provided at levels above NRC requirements (HM; n = 10), a MultiMin® injection at labeled dose (MM; n = 10) or no mineral supplementation (CON; n = 10) for the first 40 d of the trial. Values are represented as least squares mean \pm SEM. P-values of repeated measure analysis for the fixed effects of treatment, time and their interaction are indicated in the top left corner of the graph.