EFFECTS OF FEEDING RACTOPAMINE HYDROCHLORIDE IN COMBINATION WITH ZINC OR CHROMIUM ON GROWTH PERFORMANCE, CARCASS CHARACTERISTICS, AND MEAT QUALITY OF FINISHING STEERS

BY

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THESIS

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ABSTRACT

Cattle growth rate, feed efficiency, and carcass quality directly influence profitability of beef cattle producers throughout the United States. Volatile feed costs have made it economical for livestock producers to use technologies to improve efficiency and profitability. Beta adrenergic agonists are one such technology and are a class of feed additives that have been widely fed by the feedlot industry in the last decade. One product available on the market is ractopamine hydrochloride (**RAC**), a beta-agonist that acts as a nutrient repartitioning agent to shift nutrient deposition from fat to lean tissue. There are other additives which may work in combination with RAC to enhance lean tissue deposition and improve meat quality. Of particular interest in this thesis are trace minerals, specifically zinc (**Zn**) and chromium (**Cr**) fed in combination with RAC. Zinc is not only involved in protein synthesis through its integral role in all 3 RNA polymerases (Cousins, 1998), but Harris et al. (2012) reported Zn in combination with RAC reduced β-receptor desensitization in bovine muscle satellite cells. This suggests that Zn supplementation may prolong the response of skeletal muscle to RAC. In addition to Zn, Cr is integral to glucose metabolism and has been shown to increase glucose clearance rates following glucose infusion (Sumner et al., 2007). We hypothesized that feeding RAC in combination with Zn would increase efficiency of gain and muscle growth over feeding RAC alone, that RAC in combination with Cr would increase intramuscular fat deposition over RAC alone, and RAC in combination with Zn and Cr would enhance both lean tissue accretion and marbling. Therefore, objectives were to determine the effects of feeding RAC with Zn and Cr on feedlot growth performance, carcass characteristics, and meat quality. Crossbred steers (N = 179; average initial BW = 533 ± 94 kg) were blocked by BW and allotted to 30 pens, 10 pens per block, for a 63 d trial. Pens were randomly assigned to 1 of 5 treatments: (1) control (**CONT**), (2) RAC only

(**RO**), (3) RAC + Zn (**RZ**), (4) RAC + Cr (**RC**), or (5) RAC + Zn + Cr (**RZC**). Steers were fed the same basal diet containing 60% dry rolled corn, 20% corn silage, 10% DDGS, and 10% supplement. Trace minerals were fed from d 0 to 63 and to target 1 g of Zn/steer·d^{-1} (KemTRACE Zn; Kemin Industries, Inc., Des Moines, IA) and 3 mg Cr/steer·d^{-1} (KemTRACE Cr; Kemin Industries, Inc.) for Zn and Cr treatments, respectively. Dry rolled corn, 0.605 kg/steer, was removed from the ration and 400 mg RAC, per 0.605 kg of ground corn carrier, was top dressed per steer immediately following feed delivery to pens fed RAC. Data were analyzed using the MIXED procedure in SAS. There were no effects ($P \geq 0.45$) of trace mineral supplementation during the first 35 d of the trial, prior to RAC supplementation, on DMI, ADG, or G:F. There were also no effects ($P \geq 0.46$) of treatment for the entire 63 d of the trial on DMI, ADG, or G:F. Despite the lack of differences in live performance, steers fed RO and RC averaged 0.10 kg/d greater ($P = 0.10$) carcass ADG than steers fed RZC and CONT, while steers fed RZ were intermediate and not different. Steers fed RO had 13% greater ($P = 0.09$) carcass G:F than steers fed CONT. Steers fed RO and RC averaged 5.5 kg heavier ($P = 0.09$) HCW than steers fed RZC and CONT, while steers fed RZ were intermediate and not different. There were no treatment effects ($P \geq 0.32$) on LM area, 12^{th} rib fat, marbling score, KPH, carcass yield, or USDA yield grade and distribution. However, carcasses from steers fed RC had the greatest ($P = 0.10$) percentage grading USDA Select. There were no treatment effects ($P \geq 0.20$) on shear force, intramuscular fat, pH, a*, and b*. Steaks from steers fed RO and RC had 11.4% greater ($P = 0.08$) cook loss than steaks from steers fed CONT and RZC, whereas steaks from steers fed RZ were intermediate and not different. Also, steaks from steers fed RC had 2.11 units greater ($P = 0.03$) L* values than steaks from steers fed RZ, steaks from steers fed CONT, RO, and RZC were intermediate. In feedlot steers, addition of either Cr and Zn, alone or in combination did not

improve growth performance or meat quality when fed in combination with 28 d of RAC supplementation; however, RAC, fed alone or in combination with Cr, did increase HCW.

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CHAPTER 1: LITERATURE REVIEW

INTRODUCTION

Growth rate, feed efficiency, and carcass quality directly influence the profitability of beef cattle producers throughout the United States. As feed costs have continued to be volatile, livestock producers have utilized technologies to improve efficiency and profitability. One such technology used has been beta agonists. Beta adrenergic agonists are a class of feed additives that have been used by the feedlot industry in the last decade. Beta-agonists act as nutrient repartitioning agents to increase protein synthesis and decrease protein degradation as beta-agonists redirect nutrients away from adipose tissue and toward muscle (Mersmann, 1998). There are 2 Food and Drug Administration (FDA) approved β-agonists for feedlot cattle. Zilpaterol hydrochloride is a category two β-agonist that has been reported to increase ADG, improve gain to feed ratio, increase HCW, increase longissimus muscle area, and decrease carcass adiposity when compared to cattle not supplemented with zilpaterol (Vasconcelos et al., 2008). However, there were anecdotal reports from a slaughterhouse of lameness in cattle that had been supplemented with zilpaterol (Feedstuffs, 2013); thus, zilpaterol was removed from the market in August 2013. Therefore, this review will focus on ractopamine hydrochloride.

Ractopamine hydrochloride is a category one β-agonist that has been shown to increase ADG, improve gain to feed, increase HCW, and increase longissimus muscle area when compared to cattle not supplemented with ractopamine hydrochloride (Gruber et al., 2007; Pyatt, 2013). The mechanisms by which ractopamine hydrochloride affects gene expression and fiber type distribution within skeletal muscle are not fully elucidated. Recently, there has been increased interest in the use of trace minerals, specifically zinc and chromium, in combination with ractopamine hydrochloride.

Zinc (**Zn**) is an essential trace mineral that has been linked to at least 90 enzymes and the hormone insulin (McDowell, 2003). Zinc functions within enzyme systems involved in nucleic acid metabolism, protein synthesis, and carbohydrate metabolism (McDowell, 2003). The NRC requirement for Zn is 30 mg Zn/kg DM (NRC, 1996). Research results of Zn supplementation for cattle have been variable. Greene et al. (1988) reported that finishing steers fed 360 mg Zn/d had increased USDA quality grade, marbling score, external fat, and KPH compared with steers fed a basal diet containing 81 mg Zn/kg DM; while Spears (1989) also reported that Zn supplementation improved ADG and gain to feed for the first 56 d of a heifer finishing trial. However, Malcolm-Callis et al. (2000) reported that Zn supplementation had no effect on ADG, gain to feed, HCW, dressing percentage, and longissimus muscle area. Caldera et al. (2012) also reported no effect of Zn supplementation on various performance measures. The inconsistent data involving the role of Zn on cattle growth performance and carcass characteristics needs to be further evaluated.

Chromium (**Cr**) is an important trace mineral in animal diets because of its role in glucose metabolism (Mertz, 1993). Chromium enhances the ability of insulin to bind to insulin receptor sites and enhances glucose uptake into insulin sensitive tissues (Anderson, 1998a; Anderson, 2003). The increase in glucose uptake by both muscle and adipose tissue may have an effect on body composition (Anderson, 1998b; Anderson, 2003). However, the effects of supplemental Cr on body composition and meat quality of finishing steers are not known. There are conflicting data as Moonsie-Shageer and Mowat (1993), Chang and Mowat (1992), and Chang et al. (1992) reported that Cr supplementation increased ADG, DMI, and gain to feed. But, Mathison and Engstrom (1995) and Bunting et al. (2000) reported that Cr supplementation did not have an effect on any performance variables measured. While it has been determined that

Cr activity is parallel to insulin function (Pechova and Pavlata, 2007), the role of Cr and insulin function on adipogenesis in cattle has not been fully elucidated.

There are very little data on Zn and Cr supplementation in concert with beta-agonist supplementation. At a cellular level, Harris et al. (2012) found that when Zn was supplemented in addition to a beta-agonist, Zn prevented beta receptor desensitization and prolonged the biological response of skeletal muscle cells to beta-agonist. Bohrer et al. (2014) reported that steers supplemented with 300 mg ractopamine hydrochloride per day had improved ADG, feed efficiency, and HCW; but, supplementation of Cr and Zn, combined and fed in addition, did not further improve the response ractopamine hydrochloride supplementation. Individual effects of Zn and Cr and their interactions with ractopamine on cattle growth, meat quality, and carcass characteristics have not been reported in vivo.

RACTOPAMINE HYDROCHLORIDE

Beta-adrenergic receptors

Extracellular signals target receptors on specific tissues to produce physiological responses. Beta-adrenergic receptors are one such group of receptors and are members of the larger superfamily of G-protein coupled receptors (Mersmann, 1998; Johnson et al., 2014). These G-protein coupled receptors consist of 7 transmembrane domains, with a ligand binding site in the middle of the domains (Mersmann, 1998). Beta-adrenergic receptors can be further subdivided into β_1-, β_2-, and β_3-adrenergic receptors (Mersmann, 1998). The predominant receptor type in cattle is the β_2-adrenergic receptor on both skeletal muscle and adipocytes, whereas the β_1-adrenergic receptor is considered to be secondary in total concentration in cattle (Mersmann, 1998).

Beta-agonist mode of action

Beta-agonists are similar in nature to catecholamines, dopamine, norepinephrine, and epinephrine (NRC, 1994; Bell et al., 1998), and are nutrient repartitioning agents that act mainly by increasing protein synthesis and decreasing protein degradation (Mersmann, 1998). Increased protein synthesis with decreased degradation ultimately leads to an increase in lean tissue deposition in animal fed beta-agonists (Mersmann, 1998). Beta-adrenergic agonists bind to beta-adrenergic receptors, to activate the G_s protein which in turn activates andenylyl cyclase, the enzyme that produces cyclic AMP (**cAMP**). A major intracellular signaling molecules, cAMP binds to protein kinase A causing the release of the catalytic subunit, which then phosphorylates a number of intracellular proteins (Mersmann, 1998). Protein kinase A phosphorylates key enzymes, including several metabolic hormones, leading to their activation or inactivation (Mersmann, 1998). For example, phosphorylation of hormone sensitive lipase by protein kinase A activates this enzyme, which results in the partial hydrolysis of triacylglycerol (**TAG**). The partial hydrolysis of TAG leads to the depression of TAG accumulation in adipose tissue (Johnson et al., 2014), i.e. decreased fat deposition. Therefore, nutrient repartitioning, due to beta agonist supplementation, leads to increased lean tissue accretion and, in some cases, a corresponding decrease in fat deposition (Ricks et al., 1984; Anderson et al., 1988).

Commercially available beta-agonists

There are 2 FDA approved beta-agonists for finishing beef cattle, zilpaterol hydrochloride and ractopamine hydrochloride. Zilpaterol hydrochloride which is manufactured under the trade name Zilmax® by Merck Animal Health, Summit, NJ. Feedlot cattle exhibited greater responses to zilpaterol hydrochloride supplementation in comparison to cattle fed RAC, because it targets the β_2 receptors on the cell surface that are more prevalent in beef cattle

(Mersmann, 1998). Vasconcelos et al. (2008) reported that steers supplemented with 8.33 mg zilpaterol/kg of dietary dry matter had an increase of 0.06 kg in ADG, a 4.6% improvement in gain to feed ration, a 17.2 kg increase in HCW, and an increase of 9.6 cm^2 in longissimus muscle area over cattle not supplemented with zilpaterol. Carcass adiposity is also affected by zilpaterol supplementation as steers supplemented with 8.33 mg zilpaterol/kg of dietary dry matter had a decrease in marbling score of 44 units, a decrease in 12^{th} rib back fat of 0.21 cm, and a decrease in kidney, heart, and pelvic fat of 4.7% over cattle not supplemented with zilpaterol hydrochloride (Vasconcelos et al., (2008). However, zilpaterol hydrochloride was taken off the market in August 2013 due to animal welfare concerns reported by commercial abattoirs.

Optaflexx® (Elanco Animal Health, Greenfield, IN) and Actogain® (Zoetis, Kalamazoo, MI) are 2 commercial products available with the same active ingredient, ractopamine hydrochloride (**RAC**). Ractopamine hydrochloride is a category 1 beta-agonist that was approved by the FDA for use in cattle in 2003 and became commercially available in 2004 (Pyatt, et al., 2013). While there are multiple reviews on beta-agonists (Pyatt et al., 2013; Johnson et al., 2014) and comparisons of zilpaterol and ractopamine (Avendaño-Reyes et al., 2006; Scramlin et al., 2010) available, the review below will focus only on the effects of ractopamine hydrochloride on growth performance, carcass characteristics, and meat quality in cattle.

Effects of supplemental RAC on performance

The effects of RAC on measures of growth performance vary greatly in published literature due to a number of factors including: dose of RAC supplementation, duration of RAC supplementation, and sex of the animals, to name a few. Ractopamine hydrochloride is approved for feeding in confinement at doses ranging from 90 to 430 mg/animal·d^{-1} for the last 28 to 42 d

on feed (Pyatt et al., 2013). Studies discussed below vary in both dose and duration of RAC supplementation, and the differences reported in the studies cited may be due to this variation in dose and/or duration of RAC supplementation.

The effect of RAC on weight gain and final BW has been studied more than the effects of RAC on other measures of growth performance. Avendaño-Reyes et al. (2006) reported an increase of 10.6 kg in final BW of steers supplemented with 300 mg RAC/hd·d^{-1} when compared with steers not supplemented with RAC. Similarly, Winterholler et al. (2007) reported an increase of 11 kg in final BW when steers supplemented with 200 mg RAC/hd·d^{-1} were compared to steers fed no RAC. Boler et al. (2012) and Bohrer et al. (2014) reported increases in final BW of 15 kg and 8 kg, respectively, in steers supplemented with 300 mg RAC/hd·d^{-1} when compared to steers receiving no RAC. Finally, a meta-analysis (Pyatt et al., 2013) of 32 studies evaluating the effects of RAC supplementation in at 100, 200, and 300 mg RAC/hd·d^{-1} reported increases in live weight gain of 3.4 kg, 6.8 kg, and 10.2 kg, respectively, when steers supplemented with RAC were compared to steers not receiving RAC.

The effects of RAC on ADG, not surprisingly, follow a similar pattern to the effects of RAC on final BW and total weight gain. Avendaño-Reyes et al. (2006) and Pyatt et al. (2013) reported increases in ADG of 0.5 kg/d and 0.6 kg/d, respectively, in steers fed 300 mg RAC/hd·d^{-1} when compared to steers not fed RAC. In steers supplemented with only 200 mg RAC/hd·d^{-1}, reported ADG were 0.07 kg/d, 0.23 kg/d, 0.13 kg/d, and 0.23 kg/d greater than steers not fed RAC (Gruber et al., 2007; Winterholler et al., 2007; Scramlin et al., 2010; Pyatt et al., 2013 respectively). The meta-analysis by Pyatt et al. (2013) reported that steers supplemented with 100 mg RAC/hd·d^{-1} had a 0.14 kg/d advantage in ADG over controls not

receiving RAC. Thus, the evidence to support a conclusion that RAC has an effect on weight gain in feedlot steers is clear; however, reasons for the variation in reported ADG are not.

Despite the improvements on gain, numerous studies reported no difference in DMI between steers fed RAC and those not fed RAC (Winterholler et al., 2007; Gruber et al., 2007; Scramlin et al., 2010; Boler et al., 2012, Pyatt et al., 2013; Arp et al., 2014; Bohrer et al., 2014). In fact, one study reported that steers supplemented with 300 mg RAC/hd·d^{-1} had a 1.6% decrease in DMI in comparison with steers that did not receive RAC (Avendaño-Reyes et al., 2006). This study appears to be an outlier, however, and even feedlot heifer studies have reported that RAC has no effect on DMI (Walker et al., 2006; Quinn et al., 2008).

As feed efficiency is determined by the ratio of ADG to DMI, or G:F, and supplementing RAC increases ADG without affecting DMI, it should be apparent that feeding RAC improves G:F. Studies evaluating the effects of 200 mg RAC/hd·d^{-1} have reported increases in G:F ranging from 4% to 22% in steers fed RAC in comparison to steers not fed RAC (Winterholler et al., 2007; Gruber et al., 2007; Scramlin et al., 2010; Pyatt et al., 2013); while at a supplementation rate of 300 mg RAC/hd·d^{-1} Avendaño-Reyes et al. (2006), Pyatt et al. (2013) and Bohrer et al. (2014) reported increases in G:F of 34%, 14.5%, and 17%, respectively, in steers fed RAC when compared with steers not fed RAC. Therefore, it is well known that RAC improves G:F by increasing ADG without affecting DMI.

Recently, there has been increased interest in measures such as carcass ADG and carcass G:F. These variables measure the effect of treatment on the carcass weight that is commonly used to determine the price received by producers (Tatum et al., 2012). Boler et al. (2012) reported that RAC increased carcass weight gain. Also, Arp et al. (2014) reported that steers fed 300 and 400 mg RAC/hd·d^{-1} had increased carcass ADG and carcass G:F in comparison to steers

fed no RAC and 200 mg RAC/hd·d^{-1}. However, in the United States, efficiency is only part of the equation. In fact, producers are generally paid on carcass value, i.e. a yield and quality grade grid, and HCW. The effects of beta-agonist on yield and quality grade will be discussed in subsequent sections; however, information regarding the effects of beta-agonists are carcass characteristics are discussed below.

Effects of supplemental RAC on carcass characteristics

As one may expect with live weight gain and ADG increases in the feedlot phase when RAC is fed, multiple studies report greater HCW in response to RAC supplementation. At 200 mg RAC/hd·d^{-1}, studies have reported increased HCW ranging from 5 to 8 kg in steers fed RAC when compared with steers not fed RAC (Winterholler et al., 2007; Gruber et al., 2007; Scramlin et al., 2010). At 300 mg RAC/hd·d^{-1}, HCW increased from 6 to 14 kg in steers fed RAC when compared to those not fed RAC (Avendaño-Reyes et al., 2006; Boler et al., 2012; Bohrer et al., 2014). Finally, Arp et al. (2014) reported a 6 kg increase in HCW for steers fed 400 mg RAC/hd·d^{-1} when compared to those steers fed no RAC. Of particular interest to note from the research above, is the variation in HCW. For example, the 6 kg increase obtained by Arp et al. (2014) at 400 mg versus the 14 kg by Avendaño-Reyes et al. (2006) at 300 mg. This variation in HCW could stem from several potential causes. Ractopamine hydrochloride is a beta-agonist that repartitions nutrients from lipid deposition to protein accretion; therefore, a changes in longissimus muscle (**LM**) area are one factor that can affect HCW.

Similar to HCW, there are varying data on the effect of RAC supplementation on LM area. Several studies have reported increased LM area in response to RAC supplementation at both 200 and 300 mg RAC/hd·d^{-1}. For example, Gruber et al. (2007) reported an increase in LM area of 2.3 cm^2 in steers fed 200 mg RAC/hd·d^{-1}, while Boler et al. (2012) reported an increase

of 4.0 cm^2 when steers were fed 300 mg RAC/hd·d^{-1}, both of these studies compared steers supplemented with RAC to steers not fed RAC. However, multiple studies also report no effect of RAC on LM area (Avendaño-Reyes et al., 2006; Winterholler et al., 2007; Scramlin et al., 2010; Bohrer et al., 2014). Certainly, dose of RAC may play a role in its effect on LM area as Arp et al. (2014) reported that steers supplemented with 400 mg RAC/hd·d^{-1} had the greatest LM area, while the LM area of steers supplemented with 200 mg RAC/hd·d^{-1} were not different than those of steers fed no RAC; LM area of steers fed 300 mg RAC/hd·d^{-1} was intermediate and not different from any other group. Pyatt et al. (2013) reported a linear relationship between increasing dose of RAC supplementation and increasing LM area (1.0, 1.9, and 3.0 cm^2 increase in LM area over controls for 100, 200, and 300 mg/hd·d^{-1}, respectively). The variation in LM area alone does not provide a simple answer to the additional HCW found in steers supplemented with RAC, thus, measures of carcass fat and carcass dressing percent must also be addressed.

One mechanism of beta-agonists that was previously discussed is that of decreasing lipid deposition, however, there is very little support in the published data for RAC having an effect on the common measures of carcass fat: back fat (**BF**), KPH, and marbling score (Mersmann, 1998). A meta-analysis evaluating the effects of RAC on 26,483 feedlot steers reported no difference in 12^{th} rib BF between steers supplemented 100, 200, or 300 mg RAC/hd·d^{-1} and steers fed no RAC (Pyatt et al., 2013). Similar results can be found in several studies (Avendaño-Reyes et al., 2006; Walker et al., 2006; Winterholler et al., 2007; Gruber et al., 2007; Scramlin et al., 2010; Boler et al., 2012; Arp et al., 2014; Bohrer et al., 2014). The meta-analysis conducted by Pyatt et al. (2013) did report an inverse linear relationship with KPH, as dose of RAC increased, KPH decreased (0.01, 0.02, and 0.03% decrease in KPH over controls for 100, 200, and 300 mg/hd·d^{-1}, respectively). However, there is very little other data to support this

conclusion as several other studies find no difference in KPH between carcasses of steers fed RAC and those fed no RAC (Winterholler et al., 2007; Gruber et al., 2007, Scramlin et al., 2010; Bohrer et al., 2014). It is likely that the small difference in KPH in the meta-analysis were magnified due to the vast numbers in the study. The final commonly measured carcass fat characteristic is marbling score, which is vitally important to the price received by producers that market their cattle on the grid system of yield and quality grade. The effect of RAC on marbling score follows the trend established by 12th rib BF and KPH. The meta-analysis conducted by Pyatt et al. (2013) reported a linear relationship between increasing dose of RAC and decreasing marbling score; however, the difference between steers supplemented 300 mg RAC/hd·d^{-1} and controls fed no RAC is 9 units. In a marketing system, the difference of 9 units reported by Pyatt et al. (2013) would not be great enough to affect pricing. Arp et al. (2014) also reported that steers fed 300 mg RAC/hd·d^{-1} had decreased marbling scores in comparison to controls fed no RAC, while steers fed 200 and 400 mg RAC/hd·d^{-1} were intermediate and not different from either the controls or the steers fed 200 mg RAC/hd·d^{-1}. The magnitude of difference between the carcasses of steers fed 300 mg RAC/hd·d^{-1} and the carcasses of steers fed no RAC was 17 units (Arp et al., 2014); again, not great enough to affect pricing in the grid system. Several studies found no difference in the marbling scores of carcasses of steers fed RAC and the carcasses of steers not fed RAC (Winterholler et al., 2007; Gruber et al., 2007; Scramlin et al., 2010; Boler et al., 2012; Bohrer et al., 2014).

Similar to the other carcass characteristics, data vary regarding the effects of RAC on dressing percentage. Both Avendaño-Reyes et al. (2006) and Boler et al. (2012) reported increases, 2.4% and 1.5%, respectively, in dressing percentage when steers were supplemented with 300 mg RAC/hd·d^{-1} were compared with steers not fed RAC. In addition, Pyatt et al. (2013)

reported that dressing percentage increased as RAC inclusion in the diet increased from 0 to 300 mg/hd·d^{-1}; and, Arp et al. (2014) reported dressing percentage increased as RAC inclusion in the diet increased from 0 to 400 mg/hd·d^{-1}. The effects of RAC on dressing percent may be due to the previously discussed increases observed in LM area with increasing supplementation of RAC (Pyatt et al., 2013); however, several studies also report no difference in dressing percentage between steers fed RAC and steers fed no RAC (Walker et al., 2006; Winterholler et al., 2007; Scramlin et al., 2010; Bohrer et al., 2014).

The varying data with regards to the effect of carcass adiposity and lean deposition lends itself to the conclusion that the effects of RAC in steers are multifaceted. Ultimately, however, the majority of carcasses in the United States are still marketed on the grid system, as mentioned above. Therefore, the effects of RAC supplementation on characteristics of the grid, and not individual characteristics, may be more meaningful. Thus, the effects of RAC supplementation on yield and quality grad are discussed in greater detail below. .

Effects of supplemental RAC on quality grade and yield grade

A common carcass based pricing system is that of the quality and yield grade grid. This grid rewards those carcasses with increased cutability and marbling (USDA, 1997). Thus, in order to maximize profits in this system, it is essential to produce heavy muscled cattle, to increase cutability, while, at the same time, not sacrificing carcass quality, as measured by marbling (Smith and Crouse, 1984). The effect of RAC on marbling score is discussed above, and the effect of RAC on USDA YG follows the same trend. The meta-analysis conducted by Pyatt et al. (2013) reported an inverse linear relationship between dose of RAC and YG, as dose of RAC fed to cattle increased from 0 to 100 to 200 to 300 mg/hd·d^{-1}, YG decreased 0.04, 0.08, and 0.12 units, respectively. However, there are also several individual studies that reported no

difference in YG between carcasses from steers fed RAC and the carcasses from steers not fed RAC (Avendaño-Reyes., 2006; Winterholler et al., 2007; Gruber et al., 2007; Scramlin et al., 2010; Boler et al., 2012; Arp et al., 2014; Bohrer et al., 2014).

Several studies also evaluated the effect of RAC on YG and QG frequency. Not surprisingly, YG and QG frequencies follow the same trend as the YG and QG main effects discussed above. The meta-analysis conducted by Pyatt et al. (2013) reported a linear relationship between increasing RAC dose and increasing YG 1 and 2 with corresponding decreases in YG 3, 4, and 5. Also, Pyatt et al. (2013) reported a linear relationship between increasing RAC dose and the increase in select and standard carcasses with corresponding decreases in carcasses grading prime and choice. However, 2 studies reported no differences in YG and QG frequency between carcasses of steers supplemented with RAC and the carcasses of steers not supplemented with RAC (Boler et al., 2012; Bohrer et al., 2014). Winterholler et al. (2007) reported that while there was no difference in QG distribution, there was an increase in YG 2 carcasses from those steers fed RAC in comparison to those steers not fed RAC. Finally, Arp et al. (2014) reported no difference in YG, but did report a decrease in carcasses grading in the upper 2/3 of choice from steers supplemented with 300 mg RAC/hd·d^{-1} in comparison to carcasses from steers not supplemented with RAC, while carcasses from steers supplemented with 200 and 400 mg RAC/hd·d^{-1} were intermediate and not different from either the control carcasses or from the carcasses of steers fed 300 mg RAC/hd·d^{-1}. Again, the variation in the data makes them difficult to interpret.

Effects of supplemental RAC on meat quality

A common concern with any feed additive is its possible effect on meat quality. Ractopamine hydrochloride has had a range of effects on measures of carcass quality, including

Warner-Bratzler shear force (**WBSF**), color, pH, and water holding capacity. The variation in these measures can be linked to the inconsistent effects of RAC on meat quality.

Any attempt to discuss the effect of RAC or any feed additive on WBSF must first be prefaced with a discussion on the threshold for meat to be considered tender. Platter et al. (2003) reported that a WBSF value of 4.5 kg was the consumer threshold to consider a steak tender. Also, the American Society for Testing and Material (ASTM, 2011) set the WBSF threshold for certification of tender steaks at 4.4 kg. Thus, WBSF values should be discussed in comparison to these thresholds as consumer acceptability is an important factor affecting profitability. Regardless of the threshold values, there is variation in the effect of feeding RAC on WBSF in the published literature, and it may be confounded by time of post mortem aging time and RAC dose, making studies difficult to compare. Avendaño-Reyes et al. (2006) reported that steaks from steers supplemented with 300 mg RAC/hd·d^{-1} and aged for 24 hr had increased WBSF values in comparison to steaks from steers not supplemented with RAC, 4.833 kg vs 4.397 kg. However, increased aging time seems to limit the effect of RAC on WBSF. Boler et al. (2012) reported that steaks from steers supplemented 300 mg RAC/hd·d^{-1} had increased WBSF at 21 d post mortem in comparison to steaks from steers not fed RAC, but were not different at 28 d; however, all shear force values were less than 3.62 kg, placing them well below the maximum value to be considered tender. Bohrer et al. (2014) reported an increase of 0.31 kg in WBSF in steaks from steers supplemented with 300 mg RAC/hd·d^{-1} in comparison to controls not fed RAC at 14 d. However, all steaks were again below the accepted tenderness threshold, as steaks from steers supplemented with RAC averaged 3.01 kg, and thus would have been acceptable to consumers. Scramlin et al. (2010) reported that while WBSF values from steaks from steers supplemented 200 mg RAC/hd·d^{-1} were increased at d 7 post mortem in comparison to steaks

from steers not fed RAC, there was no difference in WBSF values by 14 d post mortem. These data, in combination with the data from Boler et al. (2012) suggest that while RAC may increase WBSF values post mortem, post-mortem aging removes differences in WBSF.

Other common measures of meat quality are pH, measures of color including L*, a*, and b*, and water holding capacity. However, in the case of RAC supplementation in beef cattle, there is limited data on these measures. The lack of effect on the pH of steaks from steers supplemented RAC in comparison to steaks from steers not fed RAC has been reported in several studies (Avendaño-Reyes et al., 2006; Scramlin et al., 2010; Bohrer et al., 2014). The measures of color, L*, a*, and b*, evaluate the lightness from white to black, the redness from red to green, and the blueness from yellow to blue of a steak, respectively. Gonzalez et al. (2009) conducted a study evaluating the effect of 200 mg RAC/hd·d^{-1} on the variables of meat quality in carcasses of steers fed RAC in comparison to controls not fed RAC; they reported no difference in L*, a*, and b* values in the adductor, gracilis, longissimus lumborum, rectus fermoris, semimembranosus, and vastus lateralis in steaks from steers supplemented RAC in comparison to steaks from steers not supplemented RAC. Other studies are in agreement that RAC does not have an effect on color (Avendaño-Reyes et al., 2006; Bohrer et al., 2014). Avendaño-Reyes et al. (2006) also evaluated the effects of RAC on water holding capacity and reported that steaks from steers supplemented 300 mg RAC/hd·d^{-1} were not different than steaks from steers not fed RAC. Furthermore, Boler et al. (2012) reported no differences in cook loss between steaks from steers supplemented with RAC and steaks from steers not supplemented with RAC.

There is limited literature available on the effects of RAC on pH, measures of color, and water holding capacity. What is available suggests that there are no negative effects of feeding RAC. Meanwhile, data on the effects RAC supplementation on WBSF of steaks suggest that

there may be potential negatives there, i.e. WBSF increased in some instances in steaks from steers supplemented with RAC compared to those steaks from steers not supplemented with RAC. However, these reports on WBSF further suggested that issues were overcome with increased time of post-mortem aging.

Effects of supplemental RAC on muscle biology

Studies evaluating the effect of RAC on various factors of muscle biology in beef cattle have become increasingly prevalent in the past decade. The majority of these studies evaluated the effects of RAC on muscle fiber type and fiber type distribution. The mechanism by which RAC increases muscle cell size, and consequently increases characteristics like HCW and LM area, is connected with its ability to alter the metabolic profile of individual fibers (Gonzalez et al., 2009). Type I slow-twitch muscle fibers are more adapted to mitochondrial oxidative phosphorylation for the generation of ATP, thus these cells are predisposed to elevated concentrations of NADH; type II fast-twitch fibers contain fewer mitochondria and less NADH (Howlett and Willis, 1998) and are more adapted to generate ATP from glycolysis. This is important because NADH serves as an electron donor for the process of altering muscle fiber type, suggesting RAC may have an effect on shifting muscle fiber type from type I to type II (Gonzalez et al., 2009).

Gonzalez et al. (2008) reported supplementing cull cows with 200 mg RAC/hd·d^{-1} increased of type IIA fibers in the longissimus muscle, semimembranosus, and vastus lateralis. There was also a distribution shift towards increased type I fibers in the infraspinatus of cull cows supplemented with either 100 mg RAC/hd·d^{-1} or 300 mg RAC/hd·d^{-1}. In a later study, Gonzalez et al. (2009) reported that supplementation of 200 mg RAC/hd·d^{-1} changed the fiber type isoform distribution from type I to type II in the longissimus lumborum, adductor, gracilis,

and vastus lateralis. There was also a shift in fiber type isoform from type II to type I in the rectus femoris. The muscle-specific fiber type shifts noted above suggest that RAC supplementation does not have the same effect on all muscles in young beef cattle. However, there was no change in the cross-section area of type I or II fibers in any of the 6 muscles that were analyzed, thus, muscle fiber size did not differ. Gonzalez et al. (2007) reported no change in the cross sectional area or the diameters of type II fibers or in the percentage of type I and type II fibers in cull cows supplemented with 15 mg RAC/kg diet DM·d^{-1}. The shifts in fiber type isoform distribution may be the influence behind the increase in LM area. As type I fibers are oxidative fibers that are smaller in diameter than the more glycolytic type II fibers, the shift in muscle fiber type isoforms may be linked to the changes in IGF expression and the expression of the various myosin heavy chain isoforms.

Walker et al. (2010) reported that steers supplemented with RAC had decreased expression of IGFBP-5 and β_2-receptor mRNA in the longissimus muscle when compared to steers not supplemented with RAC. In contrast, the expression of myosin heavy chain IIA was not affected by RAC supplementation (Walker et al., 2010). Winterholler et al. (2007) reported that the abundance of β_1-agonist receptor mRNA or β_3-agonist receptor mRNA did not change in cattle fed 200 mg RAC/hd·d^{-1}, but RAC tended to increase the abundance of β_2-agonist receptor mRNA in cattle fed 200 mg RAC/hd·$d^{-1}$when compared to cattle not fed RAC. Winterholler et al. (2008) reported that circulating IGF-I concentrations were not affected by RAC supplementation, but, in contrast to an earlier study, the abundance of β_1-agonist receptor mRNA tended to increase, in comparison to cattle not fed RAC. Furthermore, RAC supplementation did not affect β_2 - agonist receptor, β_3-agonist receptor, or IGF-I mRNA.

Weber et al. (2012) reported that although few significant changes in carcass characteristics were observed between cull cows supplemented with RAC versus those fed a RAC free diet, there were differences observed in cellular expression, including myosin heavy chain IIA. The loss of myosin heavy chain IIA function may be a precursor to the desensitization of the beta-adrenergic receptors, which will decrease the effect of RAC on the individual muscle cells. There is little data on how high dose inclusion rates of RAC in feedlot steers may influence beta adrenergic receptor concentration and muscle fiber type. It is hypothesized that beta-adrenergic receptors may become overwhelmed during a long term, high inclusion rate beta-agonist feeding period. Receptor desensitization would cause a decrease in the effectiveness of RAC, thus it would be beneficial to evaluate potential feed additives that could prevent receptor desensitization, which could potentially improve the efficacy of RAC. One solution to the problem of receptor desensitization may be zinc, as at a cellular level, Harris et al. (2012) reported that zinc when fed in combination with a beta-agonist may help to prevent desensitization of the receptors, which may prolong the biological response in skeletal muscle to beta-adrenergic receptors.

ZINC

Properties and metabolism

Zinc is a divalent cation that is relatively evenly distributed throughout tissues in the body (Hambidge et al., 1986; McDowell, 2003). However, the greatest concentrations can be found in the epidermal tissues, such as skin, hair, and feathers (Hambidge et al., 1986). Zinc is involved in diverse cellular process including catalysis, gene expression, and protein synthesis (Hambidge et al., 1986; NRC, 2000; McDowell, 2003). Zinc also functions as the central metal ion for several enzymes, such as DNA and RNA polymerases, and peptidases (Miller, 1970;

Kimura, 1993). Another role of Zn is as a component of Zn-fingers on binding proteins required to enhance transcription for specific genes (McDowell, 2003).

Zinc concentration in the diet is the greatest factor affecting zinc absorption. Zinc deficient animals absorb more dietary Zn than animals consuming adequate zinc diets (McDowell, 2003; Stake et al., 1975; Miller, 1970). In many species, Zn is absorbed in the small intestine, specifically in the duodenum (Brody, 1999; Cousins, 1998); however, Arora et al. (1969) reported greater Zn absorption in the rumen of sheep than in the duodenum. The process of Zn metabolism once it has been absorbed by the intestinal mucosal cells, and possibly rumen papillae, was broken down into 4 phases by Cousins et al. (1998). The first phase is the absorption of Zn across the epithelial layer of intestinal mucosal cells by a carrier mediated process which is regulated by a variety of binding ligands (McDowell, 2003). One binding ligands, metallothionein, can be influenced by dietary Zn and plasma Zn concentrations and functions as a regulator for the absorption of Zn within the intestinal cells when dietary Zn concentrations are high (McDowell, 2003; Cousins, 1998; Underwood and Suttle, 1999). Once Zn is absorbed into the enterocyte the absorption of Zn into the blood stream is dependent on Zn concentrations in the circulatory system, in addition to the regulation of metallothionein; when Zn concentrations are low in the blood, Zn transport into the blood stream will increase (Hambidge et al., 1986). The bidirectional process of Zn passage between the intestinal lumen and the blood stream is still not clearly elucidated (Hambidge et al., 1998). In ruminants, Zn that is absorbed through the rumen wall can be reabsorbed into the lumen of the small intestine; within the lumen, Zn binds to metallothionein unless the concentrations of Zn are lower in the blood (McDowell, 2003). When Zn concentrations in the blood are low, Zn bind to the cysteine rich binding protein (**CRIP**). The binding protein CRIP moves Zn across the enterocyte to the

basolateral side, and then Zn is attached to a carrier molecule like albumin; albumin is then able to transport Zn throughout the body (Hempe and Cousins, 1991).

The bioavailability of Zn must also be taken into consideration when evaluating the ability of the body to effectively utilize dietary Zn. Evaluating the bioavailability of any trace mineral is a difficult endeavor, and comparing studies that evaluate the bioavailability is also difficult due to differing methods and the potential presence of dietary inhibitors of Zn absorption. A compilation of the data from 2 studies (Wedekind et al., 1994; Cao et al., 2000) generates a list of Zn complexes from most available to least available as follows: Zn-proteinate, Zn-amino acid chelate, Zn-methionine, Zn-sulfate, Zn-Oxide, and Zn-Lysine. The organic sources of Zn, those sources bound to amino acid or protein complexes appear to be more digestible than the inorganic sources of Zn, those sources bound to sulfates, oxides, and chlorides. Thus, the evaluation of the effect of a dietary treatment including Zn must be done with care as a different source of Zn with a different bioavailability may have a different effect.

Animals do not have capacity to store Zn long-term. The major source of readily mobilized Zn storage is metallothionein in the liver (Richard and Cousins, 1976); however, when dietary concentrations of Zn are increased, Zn storage increases in the hair, bone, and pancreas, but these stores are not readily mobilized (Miller, 1970).

Lack Zn of storage could exacerbate periods of zinc deficiency (McDowell, 2003). In cattle, Zn deficiency is characterized by reduced growth, feed intake, feed efficiency, swollen feed with open lesions, listlessness, and excessive salivation (McDowell, 2003). However, the most obvious clinical sign of severe Zn deficiency is parakeratosis (Miller, 1979). Diagnosing a severe Zn deficiency can be accomplished by measuring plasma concentrations of Zn, however this method is of little use in detecting marginal deficiencies. Zinc toxicity is not of great concern

in beef cattle, due to the fact that the amount of Zn necessary to cause toxicity is much greater than the NRC requirements. The NRC (2000) sets the maximum tolerable level of Zn concentration at 500 mg Zn/kg diet, whereas the requirement for Zn in beef cattle is 30 mg Zn/kg diet. Ott et al. (1966) evaluated the effects of over feeding Zn, which resulted in reduced gain, feed consumption, and feed efficiency. Engle et al. (1997) reported the results of 2 studies which investigated the effects of zinc deficiency and repletion in feedlot steers. The results of these studies indicate that steers fed diets sufficient in zinc for the entire trial had a greater rate of protein turnover than steers fed diets deficient in zinc initially and then fed adequate Zn; however, there was no difference in overall muscle mass between the treatment groups.

Zn requirement

As reported earlier, the Zn requirement for beef cattle is 30 mg Zn/kg diet DM (NRC, 2000). However, this requirement is not well defined as Pond and Oldjen (1988) reported no performance benefits in steers, i.e. no additional weight gain or increased efficiency, that were supplemented with Zn in addition to basal diets that contained 20 to 28 mg/kg Zn, indicating that the Zn requirement for growing steers may be lower than anticipated. Perry et al. (1968) added differing doses of Zn to basal feedlot diets, from 75 to 346 mg Zn/kg ration, in a series of 4 experiments. They reported that in 2 of the 4 experiments there were increases in daily gain; however, when steers consumed 132 mg Zn/kg diet, they had decreased rate of gain and feed intake. This pattern was not seen in at either greater or lesser supplementation levels. Beeson et al. (1977) reported the results of 7 experiments in which Zn was supplemented at levels from 0 to 620 mg Zn/kg of the ration to basal diets containing approximately 20 mg Zn/kg ration. At only 1 level, 75 mg additional Zn/kg diet in addition to the 20 mg Zn/kg in the basal diet, was there a significant increase in gain; there was also a decrease in growth at 620 mg additional Zn/kg

ration. The 30 mg Zn/ kg diet DM, while appropriate in most cases, may not be the point at which returns can be maximized.

Effect of Zn on growth performance

Several studies have reported Zn supplementation having an effect on immune response (Chirase et al., 1991; Salyer et al., 2004) which may, subsequently affect performance of immunocompromised animals; however, Zn actions on immune responses will not be reviewed in this discussion. Like RAC, however, the effects of Zn on various measures of growth performance have been inconsistent. Several studies report no differences in final BW in cattle supplemented with varying levels of Zn in comparison to cattle not supplemented with Zn (Spears, 1989; Malcolm-Callis et al., 2000; Spears and Kegley, 2002); however, Nunnery et al., (2007) reported that receiving heifers supplemented with 75 mg Zn/kg DM had 8 kg lighter final BW than heifers not receiving supplemental Zn. The effects of Zn on ADG are also inconsistent. Again, several studies report no differences in ADG between cattle being supplemented varying levels of Zn and cattle supplemented with no Zn (Malcolm-Callis et al., 2000; Spears and Kegley, 2002; Nunnery et al., 2007); however, Spears (1989) reported that cattle supplemented with 25 mg Zn/kg DM had a 15.6% increase in ADG over the first 56 d of the finishing phase. Likewise, there is also variation in the effect of Zn supplementation on DMI. Multiple studies report no differences in DMI between cattle fed varying levels of Zn and cattle not fed Zn (Greene et al., 1988; Spears, 1989; Spears and Kegley, 2002; Nunnery et al., 2007). Malcolm-Callis et al. (2000) reported, however, a linear decrease in DMI with increasing Zn inclusion rate from 20 to 100 to 200 mg Zn/kg DM. However, despite the variations found in ADG and DMI, the magnitude of variation is not enough to effect feed efficiency as multiple studies report no differences between cattle supplemented varying levels of Zn and cattle supplemented no Zn

(Spears, 1989; Malcolm-Callis et al., 2000; Spears and Kegley, 2002). The variation in response of growth performance characteristics to Zn supplementation lends credence to the fact that trace mineral supplementation is a complex undertaking that can be effected by multiple factors.

Effect on carcass characteristics

The effect of Zn on carcass characteristics is similar to the effect of Zn on growth performance, in as much as there is significant variation in certain measures. Three characteristics that do not exhibit that variation are HCW, LM area, and dressing percentage. Previous reports are unanimous in the conclusion that varying levels of Zn supplementation have no effect on HCW, LM area, and dressing percentage (Greene et al., 1988; Malcolm-Callis et al., 2000; Nunnery et al., 2007; Spears and Kegley, 2002). Variation, however, can be found in the measures of carcass adiposity, QG, and YG. Nunnery et al. (2007) reported no differences in BF, KPH, or marbling, and no resulting differences in QG and YG. In contrast, Malcolm-Callis et al. (2000) reported no differences in KPH and marbling, but did report a quadratic response in BF and YG as cattle supplemented with 100 mg Zn/kg DM had increased BF and resulting YG scores in comparison to cattle supplemented 20 and 200 mg Zn/kg DM. Also, Spears and Kegley (2002) reported no differences in KPH, but did report an 11% increase in marbling and subsequent 4.0% increase in QG scores in cattle supplemented with 25 mg Zn/kg DM in comparison to controls not supplemented with Zn. As with the growth performance results, there is variation in some carcass characteristics that may be explained by the effect of Zn on meat quality or muscle biology.

Effect on meat quality and muscle biology

There is no data regarding the effect of Zn supplementation on measures of meat quality in cattle. In swine, however, Zn supplementation has been shown to have no effect on WBSF

(Paulk et al., 2014); however, that same study reported that pork chops from pigs fed 10 mg/kg RAC in combination with 75, 150, or 225 mg/kg of supplemental Zn as either ZnO or Availa-Zn for 35 d had a linear response of increasing cook loss as Zn concentration in the diet increased and that pork chops from pigs fed supplemental Zn at varying levels for 35 d had decreased L* values in comparison to pork chops from pigs fed a control diet. There is also little to no data on the effect of Zn on muscle biology in cattle. In swine, once source evaluated the effects of a Zn metallothionein injection prior to harvest and reported that injection of Zn metallothionein improved tissue anti-oxidative ability (Li et al., 2007). In addition, Oh and Choi (2004) investigated the effect of Zn supplementation on lipogenesis in an *in vitro* experiment using bovine intramuscular adipocytes. Zinc supplementation in levels from 5 to 100μM increased the number and size of lipid droplets in the cytosol and stimulated the effects on triglyceride synthesis. It was hypothesized that Zn role in stimulating lipogenesis could be due to reduced nitrogen oxide production, which was also a result of Zn supplementation in this study. However, PPARγ2 expression was also enhanced by Zn supplementation levels of 50 and 100 μM. The increase in PPARγ2 expression could signal that Zn is enhancing the expression of the transcription factors that are responsible for the synthesis of triglycerides; however, this mode of action has not been documented. Data on the effects of Zn on lipogenesis and PPAR expression in vivo are lacking.

Trace mineral studies are inherently difficult due to small concentrations and the number of factors that can influence changes throughout supplement manufacturing and laboratory analysis. The conflicting results of experiments above supplementing Zn, and the lack of knowledge on how Zn may effect lipogenesis and protein accretion, are relatively commonplace

in the field of trace mineral nutrition. Studies of another trace mineral, chromium, have had similar variable results in beef cattle trials.

CHROMIUM

Properties and Metabolism

Chromium (**Cr**) is an important trace mineral in both human and animal nutrition because of its role in glucose metabolism (Mertz, 1993). Schwarz and Mertz (1959) were among the first to demonstrate the Cr is a required mineral. Rats fed Torula yeast were unable to efficiently remove glucose from their bloodstream; however, when greater Cr concentrations were fed, the rats could effectively remove glucose. Whether the inability to remove glucose from the bloodstream was due to low levels of insulin or excess circulating glucose was not determined. Roginski and Mertz (1969) reported that Cr supplementation in rats increased amino acid uptake into heart proteins and other tissues; however, there have been no studies in ruminants that have explored the relationship of Cr and protein metabolism. One explanation of the early finding by Schwarz and Mertz (1959) is Cr interactions with insulin. Chromium acts as a cofactor for insulin, as Cr activity in the organism is parallel to insulin function (Pechova and Pavlata, 2007). It was first believed that Cr potentiated insulin's actions through the glucose tolerance factor (Toepfer et al., 1977); currently, it is accepted that chromodulin is the active form of Cr. Chromodulin is made up of glycine, cysteine, aspartate, and glutamate and is found in the cytosol and nucleus of insulin sensitive cells (Yamamoto et al., 1987; Vincent, 2000; Vincent, 2004). It is hypothesized that as blood concentrations of insulin increase due to increases in blood sugar, there is an influx of Cr from the blood into insulin sensitive cells. The Cr entering the cells binds to the inactive form of chromodulin, apochromodulin. When Cr binds to apochromodulin, it changes conformation to holochromodulin, which then binds to an insulin-stimulated insulin

receptor. This binding aids the receptor in maintaining its active conformation and the chromodulin acts as an amplifier of insulin signaling (Vincent, 2000). The activation of insulin receptor kinase activity and the resulting inhibition of insulin receptor tyrosine phosphatase would invariably lead to increased phosphorylation of the insulin receptor. This increase in phosphorylation is associated with increased insulin sensitivity, which would lead to an increase in glucose uptake by insulin sensitive cells (Anderson, 1998a; Anderson, 2003). Kegley et al. (2000) reported that steers fed Cr as Cr-L-methionine had increased glucose clearance rates after an insulin infusion and increased insulin response to a glucose challenge in the form of a glucose tolerance test. Kegley et al. (2000) also reported a linear effect of increasing the dose of Cr supplementation on serum insulin concentrations following a glucose tolerance test. Radunz et al. (2012) reported a relationship between insulin resistance and adipogenesis late in the finishing phase of feedlot cattle; however, this relationship needs to be further elucidated as there is little published work that supports this hypothesis In humans and pigs, an increase of circulating insulin increases weight gain and obesity through insulin resistance and hyperinsulinemia without increased energy intake (Anderson, 1998b; NRC, 1997). Because Cr increases insulin sensitivity and, thus, leads to a decrease in circulating insulin, Cr may have an effect on body composition (Anderson, 1998b; Anderson, 2003). In humans, insulin resistance and hyperinsulinemia can cause increased weight gain and obesity (Barnard and Wen, 1994). In cattle, Cr propionate supplementation has been shown to increase glucose clearance rates following glucose infusion (Sumner et al., 2007). Cefalu et al. (2002) reported obese rats fed Cr had significantly lower fasting insulin levels and improved glucose disappearance in comparison to obese rats not given supplemental Cr; furthermore, they hypothesized that these results signaled a change in insulin sensitivity which could have an effect on glucose uptake by insulin

sensitive tissues. While the role of Cr in regards to insulin function is known, there is a lack of data regarding how changes in insulin sensitivity and uptake may effect adipogenesis in cattle.

Supplementation and sources

Currently, the only approved source of Cr supplementation in beef cattle is Cr propionate, which can be added at levels up to 0.5 mg of Cr per kg of dry matter (Spears et al., 2012). The source of Cr in a diet is important as it may affect its ability to increase insulin binding. Kegley and Spears (1995) reported that inorganic Cr sources, such as CrO, are essentially unavailable, while organic complexes, such as Cr fortified yeast and Cr propionate are highly available.

Effect on growth performance

As with RAC and Zn, the growth performance results with regards to Cr are highly variable and greatly influenced by time of supplementation within the animals life and amount of Cr supplemented. When a high Cr yeast product was fed to finishing cattle at a rate of 0.2 mg/kg Cr, average daily gain, dry matter intake, and carcass characteristics were improved (Chang et al., 1992). Also, Valdés-García et al. (2011) reported a linear increase in final BW, ADG, DMI and G:F as the inclusion rate of a Cr yeast increased from 10 to 30 g/hd·d^{-1}. Moonsie-Shageer and Mowat (1993) reported that Cr supplementation at levels of 0.2 mg/kg DM and 1 mg/kg DM increased ADG by 27% and Cr supplementation at levels of 0.2 mg/kg DM and 0.5 mg/kg DM increased DMI in stressed feeder calves by 0.5 kg over the course of a 30 d trial over steers fed just a basal diet that consisted of 0.16 mg Cr/kg DM. Chang and Mowat (1992) reported results of a 2 part study that investigated the effect of Cr supplementation on stressed and growing feeder calves. Calves that received 4 mg Cr/hd·d^{-1} for the first 28 d in the feedlot had increased ADG by 30%, and G:F by 27%. Calves that then received 0.2 mg Cr/kg DM·d^{-1} during a 70 d growing period did not have an increase in ADG, DMI, or feed efficiency. Furthermore,

Mathison and Engstrom (1995) found that Cr supplementation had no influence on rate of gain, efficiency of gain, or on morbidity during the initial 28 days in the feedlot. Bunting et al. (2000) reported that Cr propionate supplementation at 0.5 mg/kg ration in Holstein calves did not affect body weight gain; however, glucose disappearance rates were greater for those calves supplemented with Cr. Finally, Swanson et al. (2000) reported no differences in ADG and G:F between growing steers supplemented varying levels of Cr and controls supplemented with no Cr.

Effect on carcass characteristics

Along with the variability found in the growth performance results, there is also variability in the effects of Cr supplementation on carcass characteristics. Mathison and Engstrom (1995) reported no differences in HCW, dressing percentage, BF, LM area, lean yield, and marbling between feedlot steers supplemented with 0.5 mg Cr/kg DM and controls that were not supplemented with Cr. Valdés-García et al. (2011) also reported no differences in dressing percentage, LM area, and marbling score in heifers supplemented with varying levels of Cr and controls not supplemented with Cr; however, as Cr inclusion increased from 10 to 20 to 30 g Cr/hd·d^{-1}, HCW also increased linearly and fat thickness and KPH decreased linearly. Change et al. (1992) reported no differences in dressing percentage, LM area, BF thickness, marbling score, and kidney fat between steers receiving 0.2 mg Cr/kg DM and controls not receiving Cr.

Effect on meat quality and muscle biology

There are no data regarding the effect of Cr supplementation on meat quality and muscle biology in finishing cattle. However, in sheep, Arvizu et al. (2011) reported no differences in WBSF, moisture content, and protein content between steaks from lambs supplemented with 0.25 mg Cr/kg DM and steaks from lambs not supplemented with Cr; however, Cr

supplementation did reduce fat content of the steaks by 15.3%. In swine, a meta-analysis conducted by Sales and Jančík (2011) reported no differences in L*, a*, b*, drip loss, cook loss, or shear force in steaks from pigs supplemented Cr and steaks from pigs not supplemented Cr. However, the mechanism of action of Cr on muscle biology is not known.

ZINC AND CHROMIUM IN COMBINATION WITH RACTOPAMINE

Recently, interest has risen in evaluating the effect of trace mineral supplementation in combination with RAC. In vitro, Harris et al. (2012) reported that bovine satellite cells treated with 1 µM of Zn in combination with 10 µM RAC had increased concentration of cAMP in comparison to bovine satellite cells treated with only 10 µM RAC and satellite cells treated with no RAC or Zn. The authors suggested that the change in cAMP response may prevent receptor desensitization and prolong the response of skeletal muscle cells to the beta-agonist. However, this study was an in vitro trial with purified cultures and these results may not be biologically relevant in a large scale feeding trial.

Bohrer et al. (2014) evaluated the effects of supplementing 1 g Zn/hd·d^{-1} as Zn-propionate and 3 mg Cr/hd·d^{-1} as Cr-propionate in combination with 300 mg RAC/hd·d^{-1}. There were no differences in final BW, total weight gain, ADG, DMI, or G:F between steers supplemented with both Zn and Cr in addition to RAC and those steers supplemented only RAC. There were also no differences reported in HCW, dressing percentage, BF, KPH, LM area, YG, marbling score, or quality and yield grade distribution between steers supplemented with both Zn and Cr in addition to RAC and steers just supplemented with RAC. Bohrer et al. (2014) also reported no differences in loin pH, L*, a*, b*, and WBSF between steaks from steers supplemented with both Zn and Cr in addition to RAC and steaks from steers just supplemented with RAC. There are no other published reports evaluating the effects of either Zn and/or Cr

supplementation in combination with RAC supplementation, thus, it is prudent to further explore any potential individual responses and/or interactions at different doses of RAC.

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CHAPTER 2: EFFECTS OF FEEDING RACTOPAMINE HYDROCHLORIDE IN COMBINATION WITH ZINC OR CHROMIUM ON GROWTH PERFORMANCE, CARCASS CHARACTERISTICS, AND MEAT QUALITY OF FINISHING STEERS

ABSTRACT

Objectives were to determine effects of feeding ractopamine hydrochloride (**RAC**) with zinc (**Zn**) and chromium (**Cr**) on feedlot growth performance, carcass characteristics, and meat quality. Steers (N = 179; initial BW = 533 ± 94 kg) were blocked by BW, allotted to 30 pens, and pens were randomly assigned 1 of 5 treatments: (1) control (**CONT**), (2) RAC only (**RO**), (3) RAC + Zn (**RZ**), (4) RAC + Cr (**RC**), or (5) RAC + Zn + Cr (**RZC**). Trace minerals were fed from d 0 to 63 and to target 1 g of Zn/steer·d^{-1} (KemTRACE Zn; Kemin Industries, Inc., Des Moines, IA) and 3 mg Cr/steer·d^{-1} (KemTRACE Cr; Kemin Industries, Inc.) for Zn and Cr treatments, respectively. Dry rolled corn, 0.605 kg/steer, was removed from the diet and 400 mg RAC, per 0.605 kg of ground corn carrier, was top dressed per steer immediately following feed delivery to pens fed RAC. There were no effects ($P \geq 0.45$) of trace mineral supplementation on DMI, ADG, or G:F prior to RAC feeding. There were also no treatment effects ($P \geq 0.46$) over all 63 d of the trial on DMI, ADG, or G:F. Despite the lack of differences in live performance, steers fed RO and RC averaged 0.10 kg/d greater ($P = 0.10$) carcass ADG than steers fed RZC and CONT, while steers fed RZ were intermediate and not different. Steers fed RO had the greatest ($P = 0.09$) carcass G:F while steers fed CONT had the least carcass G:F, 0.0875 and 0.0774, respectively. Steers fed RO and RC averaged 5.5 kg heavier ($P = 0.09$) HCW than steers fed RZC and CONT, while steers fed RZ were intermediate and not different. There were no treatment effects ($P \geq 0.32$) on LM area, 12th rib fat, marbling score, KPH, carcass yield, or USDA yield grade and distribution. However, carcasses from steers fed RC had the greatest ($P = 0.10$) percentage grading USDA Select. There were no treatment effects ($P \geq 0.20$) on shear force, intramuscular fat, pH, a*, and b*. Steaks from steers fed RO and RC had 11.4% greater (*P*

= 0.08) cook loss than steaks from steers fed CONT and RZC, whereas steaks from steers fed RZ were intermediate and not different. Also, steaks from steers fed RC had 2.11 units greater (*P* = 0.03) L* values than steaks from steers fed RZ, steaks from steers fed CONT, RO, and RZC were intermediate. In feedlot steers, addition of both Cr and Zn supplementation did not improve growth performance or meat quality when fed in combination with 28 d of RAC supplementation; however, RAC, fed alone or in combination with Cr, did increase HCW.

Key words: beta-agonist, cattle, chromium, Optaflexx, zinc

INTRODUCTION

Ractopamine hydrochloride (**RAC**) is a β_1-agonist approved for use at doses ranging from 90 to 430 mg/animal·d^{-1} for the last 28 to 42 d of the finishing phase in feedlot cattle (Pyatt et al., 2013). A meta-analysis including 26,483 steers evaluated doses of 100, 200, or 300 mg RAC/hd·d^{-1} and reported that supplementing 300 mg RAC/steer·d^{-1} increased ADG by 20.5%, improved feed conversion by 16.4%, increased HCW by 9.2 kg, and increased LM area 3.0 cm^2 compared with controls not fed RAC (Pyatt et al., 2013); however, the meta-analysis did not evaluate cattle fed 400 mg RAC/steer·d^{-1}. Recently, there has been increased interest in supplementing trace minerals in combination with RAC to enhance growth and intramuscular fat (Harris et al., 2012; Bohrer et al., 2014).

Both zinc (**Zn**) and chromium (**Cr**) are of particular interest due to their relationships with protein synthesis and glucose metabolism, respectively. Zinc is integral to all 3 of the RNA polymerases (Cousins, 1998) that affect protein synthesis, and Zn inhibited muscle protein degradation (Engle et al., 1997). Furthermore, Harris et al. (2012) reported Zn supplied in combination with RAC to bovine satellite cells prevented β-receptor desensitization, and suggested Zn may prolong the response of skeletal muscle to RAC. Chromium may increase insulin action by binding to, and increasing the kinase activity of, the insulin receptor and, thus, support glucose metabolism (Mertz, 1993; Vincent, 2004). In cattle, Cr-propionate increased glucose clearance rates following glucose infusion (Sumner et al., 2007). We hypothesized that feeding RAC in combination with Zn would increase efficiency of gain and muscle growth over feeding RAC alone, that RAC in combination with Cr would increase intramuscular fat deposition over RAC alone, and RAC in combination with Zn and Cr would enhance both lean

tissue accretion and marbling. Objectives were to determine effects of feeding RAC with Zn and Cr on feedlot growth performance, carcass characteristics, and meat quality.

MATERIALS AND METHODS

All animal procedures were approved by the University of Illinois Institutional Animal Care and Use Committee (IACUC protocol #12009) and followed guidelines recommended in the Guide for the Care and Use of Agricultural Animals in Agricultural Research and Teaching (FASS, 2010).

Animals and diets

There were 210 crossbred steers from the Dixon Spring Agricultural Center in Simpson, IL housed in confinement barns on concrete slatted floors covered in 1.91 cm thick rubber matting at University of Illinois Beef Cattle and Sheep Field Laboratory in Urbana, IL. Pen dimensions were 4.88 m × 4.88 m constructed of 5.08 cm galvanized steel tubing. Steers were fed a corn-based diet for 188 d prior to the initiation of this study and were implanted with Component TE-IS (80 mg trenbolone acetate, 16 mg estradiol; Elanco Animal Health, Greenfield, IN) 104 d prior to the initiation of the study. Steers were individually weighed on 2 consecutive d. A subset of 179 steers were selected from the original 210 on d -1 and stratified by BW into 3 blocks: heavy (avg initial BW = 552 ± 59 kg), medium (avg initial BW = 508 ± 35 kg), and light (avg initial BW = 459 ± 60 kg). Each BW block was comprised of 10 pens and there were 5 to 6 steers/pen, such that there were 60 steers in the light and heavy blocks and 59 steers in the medium block. Pens within block were randomly assigned to 1 of 5 treatments on d 0: (1) control (**CONT**) (2) RAC only (Optaflexx 45 to provide 400 mg RAC/hd·d^{-1}, Elanco Animal Health, Greenfield, IN; **RO**), (3) RAC (Optaflexx 45 to provide 400 mg RAC/hd·d^{-1}, Elanco Animal Health) + Zn (KemTRACE Zn; Kemin Industries Inc., Des Moines, IA; to

provide approximately 1.0 g Zn/hd·d^{-1}; **RZ**), (4) RAC (Optaflexx 45 to provide 400 mg RAC/hd·d^{-1}, Elanco Animal Health) + Cr (KemTRACE Cr; Kemin Industries Inc., Des Moines, IA; to provide approximately 3 mg Cr/hd·d^{-1}; **RC**), and (5) RAC (Optaflexx 45 to provide 400 mg RAC/hd·d^{-1}, Elanco Animal Health) + Zn (KemTRACE Zn; Kemin Industries Inc., Des Moines, IA; to provide approximately 1.0 g Zn/hd·d^{-1}) + Cr (KemTRACE Cr; Kemin Industries Inc., Des Moines, IA to provide approximately 3 mg Cr/hd·d^{-1}; **RZC**).

Steers were fed in 3 m concrete bunks. Trace mineral supplementation began 35 d prior to RAC supplementation. To target a similar final BW across blocks, treatment initiation was staggered across BW blocks. The heavy weight block started first, the middle weight block started 7 d later, and the light weight block started 7 d after the middle weight block. Steers were fed the same basal diet of 60% dry rolled corn, 20% corn silage (approximately 50:50 grain:forage), 10% dry distillers grains, and 10% supplement on a DM basis; each diet was formulated to meet or exceed NRC guidelines (NRC, 2000). Steers were fed once daily at 0800 and were managed for slick bunks. Bunks were visually evaluated at 0630 and were considered slick if less than 0.2 kg of feed was remaining. Ractopamine was delivered with a ground corn carrier at a rate of 0.605 kg/steer·d^{-1}, which was formulated to provide 400 mg RAC/steer·d^{-1}. The RAC dose was top dressed at the bunk immediately following the delivery of the total mixed ration by the feed mixing truck. Steers were weighed every 14 d throughout the duration of the study. A 2 d consecutive final BW was taken prior to slaughter to allow calculation of live ADG.

Feed sampling and analysis

Individual feed ingredients were collected every 2 wk, dried at 55° C for 3 d, and composited at the end of the trial. Ingredients were individually analyzed for nutrient composition and then used to formulate diet nutrient composition (Table 2.1). Diet ingredients

were ground using a Wiley mill (1-mm screen, Arthur H. Thomas, Philadelphia, PA). All ingredients were analyzed for DM (100°C for 24 h), CP (Leco TruMac, LECO Corporation, St. Joseph, MI), ADF and NDF (Ankom Technology method 5 and 6, respectively; Ankom Technology), fat (method 2; Ankom Technology), and total ash (600° C for 2 h, Thermolyne muffle oven model: F30420C, Thermo Scientific, Waltham, MA). Diet ingredients were also analyzed for mineral composition by a commercial lab using inductively coupled plasma atomic emission spectroscopy analysis after perchloric acid/nitric acid digestion (Method 975.03; AOAC, 1988; OARDC Star Lab, The Ohio State University, Wooster, OH). Ractopamine hydrochloride concentrations were verified by Covance Laboratories (Greenfield, IN) from individual samples of both the control supplement and the RAC top dress that were collected at 2 time points during the trial, the first at the initiation of RAC supplementation on 10/16/2013 and the second on 11/6/2013; both analyses were within the acceptable margin of error, within 80% to 110% of claim, to supply the 400 mg RAC/hd·d^{-1}. Chromium concentrations were verified prior to the start of the trial using atomic absorption spectroscopy (Williams et al., 1962). Zinc concentrations within the supplements were verified prior to the beginning of the study using atomic absorption spectroscopy (Method 968.08; AOAC, 1988). The background concentrations of trace minerals in the basal diet were 32.59 mg Zn/kg DM and 0.78 mg Cr/kg DM. The concentration of Zn in RZ and RZC was 96.98 mg Zn/kg DM and 100.98 mg Zn/kg DM. The concentration of Cr in RC and RZC was 1.13 mg Cr/kg DM and 1.15 mg Cr/kg DM.

Harvest and carcass data collection

Cattle were transported approximately 430 km to a U.S.D.A. Food Safety and Inspection Service facility. Cattle were fasted for approximately 16 h, but provided water until slaughter. Each BW block was slaughtered at the end of the 28 d RAC supplementation, such that the

heavy block was slaughtered first, the medium block was slaughtered 7 d later, and the light block was slaughtered 7 d after the medium block. Hot carcass weights were recorded after passing federal inspection and just prior to the carcasses entering the cooler. Equations from Tatum et al. (2012) including HCW recorded at the slaughter facility were utilized to evaluate carcass ADG and carcass G:F. Carcasses were cut between the 12th and 13th rib by facility personnel. Back fat thickness (**BF**) was measured at the 12th rib, and KPH was recorded. Loin muscle area was determined using 0.005 matte Dura-Lar film (Grafix Arts; Maple Heights, OH) for tracing. Then, tracings were measured in duplicate with a digitizer tablet (Wacom Co., Ltd; Vancouver, WA), and the average of two measurements were reported. Marbling and maturity scores were determined and recorded by trained university personnel. These data were used to calculate USDA yield grade (**YG**) with the yield grade equation from the USDA beef grading standards (USDA, 1997). The USDA Quality Grade (**QG**) was determined by trained university personnel for each steer. Additionally, a 10 cm long sample of longissimus muscle was collected from each steer and transported to the University of Illinois Meat Science Laboratory to determine intramuscular fat content (**IMF**), ultimate pH, and color. Ultimate pH was measured using a hand held pH star probe fitted with a glass electrode (SFK Technologies Inc., Cedar Rapids, IA; 2 point calibration – pH 4 and 7). Objective CIE L*, a*, and b* [CIE (Commission international de l'eclairage), 1978] values were collected with a Minolta CR-400 (Minolta Camera Company, Osaka, Japan) utilizing a D65 light source and a 0° observer and an aperture size of 8 mm. To determine IMF content, LM samples were trimmed of subcutaneous fat, homogenized in a food processor (Cuisinart model CUI DFP-7BC, Cuisinart; East Windsor, NJ) and duplicate 10 g samples of tissue were oven dried at 100° C for at least 24 h to determine

moisture content. Dried samples were washed multiple times in a mixture of warm chloroform: methanol as described by Novakofski et al. (1989) to determine total extractable lipid.

Additionally, strip loins from the 3 steers closest to the pen mean of BW (90 total steers, 18 steers per treatment) were used to determine Warner-Bratzler shear force (**WBSF**). One steak, was collected, vacuum-packaged, and aged at 4°C for 14 d postmortem and then frozen after aging and held at 20°C for shear force evaluation. Prior to analysis, steaks were removed from the freezer and placed in a cooler at 4° C to thaw for 24 h. Steaks were trimmed of excess fat and cooked on a Farberware Open Hearth grill (Model 455N, Walter Kidde, Bronx, NY) to a final internal temperature of 70 °C. Internal temperature was monitored using copper-constantan thermocouples (Type T, Omega Engineering, Stanford, CT) connected to a digital scanning thermometer (Model 92000-00 Barnant Co., Barington, IL). Cook loss was determined by weighing steaks used for shear force immediately before and after cooking. Steaks were allowed to cool to approximately 25° C, then, 6 cores, 1.25-cm in diameter, were removed parallel to the orientation of the muscle fibers. Cores were sheared using a Texture Analyzer TA.HD Plus (Texture Technologies Corp., Scarsdale, NY/Stable Microsystems, Godalming, UK) fitted with a Warner–Bratzler shear head and a blade speed of 3.3 mm/s and a load cell capacity of 100 kg. Results from the 6 cores were averaged and results were reported as kg of force.

Statistical Analysis

Data were analyzed using the MIXED procedure of SAS (SAS Inst. Inc. v. 9.4 Cary, NC) as a randomized complete block design with the fixed effects of dietary treatment and block. Pen was the experimental unit. Treatment means were separated using the Least Squared Means option in SAS. Quality and yield grade distributions were analyzed by the GLIMMIX procedure

of SAS using a binomial distribution and a Satterthwaite adjustment. Statistical differences were considered significant at $P \leq 0.10$.

RESULTS AND DISCUSSION

A wealth of research regarding the effects of Zn and Cr response in growing cattle has been conducted (Chang and Mowet, 1992; Kegley and Spears, 1995; Salyer et al., 2004, Bernhard, 2012). However, these results have been variable and dependent on immunological status of the calves (Chang and Mowet, 1992; Moonsie-Shageer and Mowet, 1993; Kegley and Spears, 1995; Salyer et al., 2004). However, data are more limited regarding the effects of Zn and Cr on finishing cattle. Therefore, for the purposes of this discussion, highlights of only those trials feeding Zn and Cr throughout the finishing phase or at the end of life will be discussed.

In the current study, trace minerals were supplemented for 35 d prior to the initiation of RAC. During this period of trace mineral supplementation, there were no differences ($P \geq 0.45$, Table 2.2) in DMI, ADG, G:F, or 2 d BW averaged at the end of the 35 d supplementation. Differences were not expected during this initial period of only trace mineral supplementation; rather, the initial period served to maximize tissue deposition of Zn and Cr prior to RAC feeding. Several studies have reported that level and source of Zn did not affect cattle ADG (Malcolm-Callis et al., 2000; Spears and Kegley, 2002; Nunnery et al., 2007), DMI (Spears, 1988; Pollard et al., 2002; Spears and Kegley, 2002; Nunnery et al., 2007), or G:F (Spears, 1988; Malcolm-Callis et al., 2000; Spears and Kegley, 2002). However, Malcolm-Callis et al. (2000) reported a linear decrease in DMI from 20 to 200 mg Zn/kg DM as $ZnSO_4$ during a 112 d finishing phase. Also, Malcolm-Callis et al. (2000) noted decreased DMI in steers receiving 1,910 mg Zn/d, 569 mg more Zn than the amount consumed by steers during the first 35 d in the current study. Sheep

fed elevated dietary Zn often exhibit appetite depression as a result (NRC, 2005). However, the maximum tolerable level of Zn for cattle is 500 mg/kg (NRC, 2005), above what the cattle in Malcom-Callis et al. (2000) or the present study were consuming, and may be the reason why no changes in DMI were observed in the present trial.

Data are limited regarding the effects of Cr-propionate on growth performance in finishing cattle and comparisons to differing sources of supplemental chromium are imperfect. Both Chang et al. (1992) and Pollard et al. (2002) reported that cattle supplemented 0.2 mg Cr/kg DM, as a high Cr-yeast products, had similar ADG, DMI, and G:F compared with cattle fed no supplemental Cr; however, in the same study Pollard et al. (2002) noted that cattle supplemented 0.4 mg Cr/kg DM, as a high Cr-yeast product, had reduced ADG and, consequently, reduced G:F compared with cattle supplemented with 0 or 0.2 mg Cr/kg DM. The cattle that responded to 0.4 mg Cr/kg as Cr yeast consumed 13.5 mg of Cr/d, whereas non-supplemented cattle consumed 10.9 mg Cr/d (Pollard et al., 2002). The steers in the present trial consumed 15.9 mg Cr/d and those fed no additional Cr consumed 11.2 mg Cr/d during the first 35 d. The reason for the lack of response in the current study in steers fed 15.9 mg Cr/d, while Pollard et al. (2002) reported effects of Cr at 13.5 mg Cr/d is not obvious. The relatively short duration, 35 d, of trace mineral supplementation may have played a role in the lack of response to treatment in the current study as most trace mineral studies have fed for longer than 50 d.

After the initial 35 d mineral feeding, cattle fed the RO, RZ, RC and RZC treatments remained on their mineral treatments (no minerals, 1g Zn/hd·d^{-1}, 3 mg Cr/hd·d^{-1}, and 1 g Zn/hd·d^{-1} with 3 mg of Cr/hd·d^{-1}, respectively), and were also fed 400 mg of RAC/hd·d^{-1} in a top dress for an additional 28 d. To date, there has only been 1 published feeding trial in cattle investigating the effects of Zn and Cr fed in combination with RAC (Bohrer et al., 2014); however, Bohrer et

al. (2014) supplemented a combination of Zn and Cr with 300 mg RAC/hd·d^{-1} for only the last 35 d of the finishing phase with no preliminary trace mineral feeding period; in contrast, the present study included a 35 d pre-RAC phase during which either Zn or Cr was fed in order to maximize Zn and Cr deposition in the tissue and evaluated the effects of 400 mg RAC/hd·d^{-1}.

Over the entire 63 d of the study, there were no differences between treatments ($P \geq 0.46$) in DMI, ADG, and G:F. However, there was a numeric increase of 0.10 kg/d in ADG in steers supplemented with RO compared with CONT. Bohrer et al. (2014) reported that steers fed 300 mg RAC/hd·d^{-1} for 35 d had increased ADG and G:F, without altering DMI, when compared with steers fed no RAC. However, there were no additive benefits of supplementing steers fed RAC with additional Zn in combination with Cr. There are limited data evaluating the effects of 400 mg RAC/hd·d^{-1} in feedlot steers, and no data evaluating the effects of feeding Zn and Cr in combination with 400 mg RAC/hd·d^{-1}. Thus, the comparison to other studies is limited.

One reason for the conflicting results between live and carcass adjusted parameters is effects of RAC on carcass characteristics. Although differences in final BW, or lack thereof, are often reflected in HCW, this is not always the case when RAC is fed. In fact, in the present trial there was no effect on final BW ($P = 0.61$), but HCW was increased ($P = 0.09$; Table 2.3) by 7 kg in steers fed RO and RC when compared with steers fed CONT and RZC; steers fed RZ were intermediate and not different. Bohrer et al. (2014) reported that steers fed 300 mg RAC/steer·d^{-1} for 35 d with or without additional Zn-propionate at 1 g Zn/hd·d^{-1} and Cr-propionate at 3 mg Cr/hd·d^{-1} had 12 kg more HCW than steers not fed RAC. The effect of RAC on HCW has been well documented with studies reporting increases in steers fed 400 mg RAC/hd·d^{-1} from 7 to 10 kg when compared with steers not fed RAC (Pyatt et al., 2013; Arp et al., 2014), similar to the findings of the present trial.

Due to these carcass differences, growth performance data from the present 63 d study were analyzed on a carcass-adjusted basis using equations from Tatum et al. (2012). Estimated beginning HCW was similar among treatments ($P = 0.89$; Table 2.4) due to experimental design; but, there was an effect ($P \leq 0.10$) of treatment on both carcass ADG and carcass G:F. Steers fed RO and RC averaged 0.10 kg/d greater ($P = 0.10$) carcass ADG than steers fed RZC and CONT, while steers fed RZ were intermediate and not different. Furthermore, steers fed RO had the greatest ($P = 0.09$) carcass G:F whereas steers fed CONT had the least carcass G:F, 0.0875 and 0.0774, respectively; all other treatments were intermediate and not different from the extremes. Arp et al. (2014) reported that steers fed 300 and 400 mg RAC/hd·d^{-1} for the last 30 d of the finishing phase had increased carcass ADG of 0.31 kg/d and an 18% improvement in G:F in comparison to steers fed no RAC. The magnitude of the effects of RAC noted in the current study are less than those of Arp et al. (2014) with an approximate 9% improvement in carcass ADG and a 13% improvement in carcass G:F

Despite the increase in HCW in steers fed RO or RC compared to HCW in steers fed CONT and RZC, there was no effect ($P = 0.86$) of treatment on LM area. We hypothesized that RAC would increase lean tissue accretion and that supplemental Zn would further increase this accretion by maintaining receptor sensitivity to RAC (Harris et al., 2012). In fact, there was a numerical decrease in LM area in carcasses from steers fed RZ, RC, or RZC when compared to those fed RO, 0.84 cm^2, 1.29 cm^2, and 0.91 cm^2 smaller, respectively. Data reported by Bohrer et al. (2014) are in agreement with the current study: there were no differences in LM area between steers fed 300 mg RAC/hd·d^{-1}, regardless of trace mineral supplementation, and steers fed a control diet with no RAC. We had hypothesized that the additional 35 d of trace mineral

supplementation would maximize tissue deposition of Zn and Cr and allow Zn to maintain receptor sensitivity; however, this was not the case.

There were also no effects ($P \geq 0.32$) of treatment on other carcass characteristics including: 12th rib BF, marbling score, KPH, dressing percentage, and calculated YG or YG distribution (Table 2.5). The lack of improvement in marbling and quality grade in cattle fed Cr was contrary to our hypothesis. In fact, when evaluating the effects of treatment on quality grade distribution, the opposite actually occurred. Carcasses from steers fed RC had a greater (P = 0.10) frequency of carcasses that graded USDA Select than those fed any other treatment (Table 5). However, there were no differences ($P \geq 0.67$) in the frequency of USDA Prime or Choice carcasses. In contrast to the current study, Bohrer et al. (2014) reported no differences in quality grade between carcasses of steers supplemented 300 mg RAC/hd·d^{-1} and carcasses of steers fed 300 mg RAC/hd·d^{-1}in combination with Zn and Cr. Despite the shift in carcasses grading Select, there were no effects (P = 0.89; Table 2.6) of treatment on IMF, and, given the lack of differences in IMF, the shift from 7.9% Select in carcasses from steers fed CONT, RO, and RZC to e 24.3% Select for carcasses from steers fed RC (an additional 6 carcasses) suggests that a small number of carcasses on the border between Select and Low Choice could have greatly influenced the distribution.

There was no effect (P = 0.57) of treatment on Warner-Bratzler shear force (**WBSF**). All treatments in this study produced steaks that were well below the threshold of tenderness of 4.4 kg set by the American Society for Testing and Material (ASTM, 2011) and the 4.5 kg threshold at which a consumer considers a steak tender (Platter et al., 2013). While some have shown an increase in WBSF due to RAC supplementation up to and including the first 14 d post-mortem (Avendaño-Reyes et al., 2006; Scramlin et al., 2010; Bohrer et al., 2014), others are in agreement

with the current study that any potential effects of RAC on WBSF can be mitigated by post-mortem aging (Scramlin et al., 2010; Boler et al., 2012). There is no data regarding the effects of Zn supplementation on measures of meat quality in cattle; however, in swine, Zn supplementation did not affect WBSF (Paulk et al., 2014). Like Zn, there is no data regarding the effects of Cr supplementation on WBSF in cattle; however, Arvizu et al. (2011) and Sales and Jančík (2011) reported that, in sheep and swine, respectively, there was no tenderness differences in WBSF in chops from animals fed Cr and steaks from animals not fed Cr.

There was an effect ($P = 0.08$) of treatment on cook loss as steaks from steers fed RC and RO had 2.7% greatest cook loss compared with steaks from steers fed RZC and CONT; RZ was intermediate and not different. In contrast, Bohrer et al. (2014) reported no differences in cook loss between steaks from steers fed 300 mg RAC/hd·d^{-1} in combination with additional Zn-propionate at 1 g Zn /hd·d^{-1} and Cr-propionate at 3 mg Cr/hd·d^{-1}and steaks from steers fed just 300 mg RAC/hd·d^{-1}. The effect of RAC found in this study varies from the effects reported in previous literature as Avendaño-Reyes et al. (2006) and Boler et al. (2012) reported no differences in water holding capacity and cook loss between steaks from steers fed RAC and steaks from steers not fed RAC. In swine, Paulk et al. (2014) reported that pork chops from pigs fed 10 mg RAC/kg in combination with 75, 150, or 225 mg Zn/kg diet DM, either as ZnO or Availa-Zn for 35 d, had a linear response of increasing cook loss as Zn concentration in the diet increased. However, Sales and Jančík (2011) reported no differences in cook loss in pork chops from pigs supplemented Cr and pork chops from pigs not supplemented Cr. There was also no effect ($P \geq 0.20$) of treatment on the pH, a*, and b*; however, steaks from steers fed RC had the greatest ($P = 0.03$), or lightest, L* values, steaks from steers fed RZ had the least, or darkest, L* values, and all other treatments were intermediate and not different; however, this difference was

only 2.11 L* units, which would not be visible to the average consumer. Several studies have reported that RAC had no effect on L*, a*, or b* value (Avendaño-Reyes et al., 2006; Gonzalez et al., 2009; Bohrer et al., 2014). Therefore, the differences noted in L* value in the current study may be due to trace mineral supplementation. While the effect of Zn supplementation of cattle on beef color is unknown, Paulk et al. (2014) reported decreased L* in pork chops from pigs fed supplemental Zn for 35 d. However, Cr supplementation was reported to not alter color in pork chops (Sales and Jančík, 2011). Therefore, data from this study would suggest that Zn and Cr supplementation in combination with RAC does not have a detrimental impact on measures of meat quality in the longissimus muscle finishing steers.

In this study, RAC supplementation did result in the expected increase in carcass ADG, carcass G:F, and HCW; however, there was no appreciable benefit of adding Zn or Cr, alone or in combination, to the diets. The supplementation of Zn or Cr, alone or in combination, did not alter the carcass characteristics or meat quality of the longissimus muscle of finishing crossbred steers. Therefore, these data suggest that supplementing Zn and Cr into diets, that are already sufficient in Zn and Cr, does not provide any additional benefit in cattle diets containing RAC.

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TABLES

Table 2.1. Composition of diets

Item, % DM Basis	CONT	RO[1]	RZ[1]	RC[1]	RZC[1]
Dry rolled corn	60	60	60	60	60
Dried distillers grains with solubles[2]	10	10	10	10	10
Corn silage	20	20	20	20	20
Basal supplement	10	10	10	10	10
Ground corn	80.020	80.020	79.659	79.941	79.579
Limestone	18.063	18.063	18.072	18.063	18.070
Dairy TM Salt[3]	0.903	0.903	0.904	0.903	0.904
Cr Propionate	0	0	0	0.079	0.079
Zn Propionate	0	0	0.351	0	0.352
Rumensin 90[4]	0.154	0.154	0.154	0.154	0.154
Tylosin 40[5]	0.099	0.099	0.099	0.099	0.099
Grease	0.761	0.761	0.761	0.761	0.761
RAC supplement[6]					
Analyzed Composition, DM basis					
NDF, %	14.95	15.05	15.09	14.97	15.10
ADF, %	7.27	7.29	7.32	7.33	7.32
CP, %	10.51	10.49	10.43	10.42	10.45
EE, %	3.92	3.94	3.91	3.83	3.84
Zn, mg/kg	32.59	36.98	96.98	38.98	100.98
Cr, mg/kg	0.78	0.83	0.84	1.13	1.15

[1]RO: 400 mg ractopamine hydrochloride/hd·d^{-1} (Optaflexx45, 99 g ractopamine hydrochloride/kg DM; Elanco Animal Health, Greenfield, IN) for the final 28 d. RZ: 1.0 g Zn supplied as Zn-propionate (KemTRACE Zn; Kemin Industries Inc., Des Moines, IA)/hd·d^{-1} for the first 35 d + RAC for the final 28 d. RC: 3 mg Cr supplied as Cr-propionate (KemTRACE Cr; Kemin Industries Inc., Des Moines, IA)/hd·d^{-1} for the first 35 d + RAC for the final 28 d. RZC: 1.0 g Zn supplied as Zn-propionate + 3 mg Cr supplied as Cr-propionate/hd·d^{-1} for the first 35 d + RAC for the final 28 d.

[2] Dried distillers grains with solubles: S = 0.43%, ether extract = 9.19%.

[3]Included: 8.5% Ca as $CaCO_3$, 5% Mg as MgO and $MgSO_4$, 7.6% K as KCl_2, 6.7% Cl as KCl_2, 10% S as S_8, prilled, 0.5% Cu as $CuSO_4$, and Availa-4 [Zinpro Performance Minerals; Zinpro Corp., Eden Prairie, MN], 2% Fe as $FeSO_4$, 3% Mn as $MnSO_4$ and Availa-4, 3% Zn as $ZnSO_4$ and Availa-4, 278 mg/kg Co as Availa-4, 350 mg/kg I as $Ca(IO_3)_2$, 150 mg/kg Se as Na_2SeO_3, 2,205 KIU/kg vitamin A as retinyl acetate, 662.5 KIU/kg vitamin D as cholecalciferol, 22,047.5 IU/kg vitamin E as dl-α-tocopheryl acetate, and less than 1% CP, fat, crude fiber, and salt.

[4]Rumensin 90, (198g monensin/kg DM; Elanco Animal Health).

[5]Tylosin 40, (88 g tylan/kg DM; Elanco Animal Health).

[6]RAC supplement contain 99.339% ground corn (DM basis) and 0.661% ractopamine hydrochloride (Optaflexx45, Elanco Animal Health) was top dressed at 0.605 kg/hd·d^{-1} in place of dry rolled corn. Analyzed levels were within acceptable tolerances (80 to 110% of claim) for each diet.

Table 2.2. Effects of feeding ractopamine hydrochloride in combination with zinc or chromium on growth performance of feedlot steers.

	CONT	RO[1]	RZ[1]	RC[1]	RZC[1]	SEM	*P*-value
Pens, n	6	6	6	6	6	-	-
Initial BW, kg[2]	506	506	507	506	506	1.9	0.93
Interim BW, kg[3]	579	584	582	585	581	7.4	0.48
Final BW, kg	628	635	638	636	631	15.8	0.61
Trace Mineral[4]							
DMI, kg/d	13.8	13.8	13.4	14.1	13.7	0.87	0.45
ADG, kg/d[5]	1.45	1.55	1.49	1.56	1.47	0.15	0.52
G:F	0.1051	0.1124	0.1111	0.1106	0.1073	0.006	0.74
RAC[6]							
DMI, kg/d	13.7	13.2	13.6	13.8	13.2	0.97	0.81
ADG, kg/d	1.72	1.81	2.03	1.85	1.81	0.28	0.55
G:F	0.1265	0.1367	0.1507	0.1338	0.1372	0.01	0.44
Total Trial							
DMI, kg/d	13.7	13.5	13.5	13.8	13.2	1.37	0.79
ADG, kg/d[7]	1.55	1.65	1.69	1.66	1.60	0.20	0.61
G:F	0.1131	0.1219	0.1257	0.1188	0.1186	0.006	0.46

[1]RO: ractopamine hydrochloride (Optaflexx, Elanco Animal Health, Greenfield, IN) fed at a rate of 400 mg/steer·d^{-1} for final 28 d. RZ: 1.0 g Zn supplied as Zn-propionate (KemTRACE Zn; Kemin Industries Inc., Des Moines, IA)/hd·d^{-1} for the first 35 d + RAC for the final 28 d. RC: 3 mg Cr supplied as Cr-propionate (KemTRACE Cr; Kemin Industries Inc., Des Moines, IA)/hd·d^{-1} for the first 35 d + RAC for the final 28 d. RZC: 1.0 g Zn supplied as Zn-propionate + 3 mg Cr supplied as Cr-propionate/hd·d^{-1} for the first 35 d + RAC for the final 28 d.

[2]Initial BW = weight at allotment. Cattle were allotted by block such that Initial BW was taken 10 d before start of block 1, 17 d before start of block 2, and 24 d before start of block 3.

[3]Interim BW = 2 d BW prior to the start of RAC feeding, d 34 and 35 of trial for each block

[4]First 35 d, during which CONT and RO were receiving the same diet.

[5]ADG calculated off initial BW and 2 d pre-RAC weight such that block 1 = 44 d, block 2 = 51 d, and block 3 = 58 d.

[6]Last 28 d, during which RO, RZ, RC, and RZC received 400 mg RAC/hd·d^{-1}.

[7]ADG calculated off initial BW and final BW such that block 1 = 73 d, block 2 = 80 d, and block 3 = 87 d.

Table 2.3. Effects of feeding ractopamine hydrochloride in combination with zinc or chromium on carcass characteristics of feedlot steers**.**

	CONT	RO[1]	RZ[1]	RC[1]	RZC[1]	SEM	*P*-value
n, animals (pens)	36 (6)	35 (6)	36 (6)	36 (6)	36 (6)	-	-
HCW, kg	377[b]	384[a]	382[ab]	383[a]	379[b]	6.6	0.09
LM area, cm^2	92.97	95.10	94.26	93.81	94.19	0.30	0.86
12^{th} rib fat, cm	1.30	1.40	1.52	1.30	1.37	0.05	0.32
Marbling score[2]	603	606	605	584	593	25.39	0.90
KPH, %	2.17	2.11	2.08	2.08	2.11	0.11	0.94
Carcass yield %	62.57	63.03	63.29	62.69	62.35	0.39	0.37
Calculated yield grade[3]	2.74	2.80	2.94	2.76	2.77	0.19	0.85

[a-b]Within a row, means without a common superscript letter differ ($P \leq 0.10$).

[1]RO: ractopamine hydrochloride (Optaflexx, Elanco Animal Health, Greenfield, IN) fed at a rate of 400 mg/hd·d^{-1} for final 28 d. RZ: 1.0 g Zn supplied as Zn-propionate (KemTRACE Zn; Kemin Industries Inc., Des Moines, IA)/hd·d^{-1} for the first 35 d + RAC for the final 28 d. RC: 3 mg Cr supplied as Cr-propionate (KemTRACE Cr; Kemin Industries Inc., Des Moines, IA)/hd·d^{-1} for the first 35 d + RAC for the final 28 d. RZC: 1.0 g Zn supplied as Zn-propionate + 3 mg Cr supplied as Cr-propionate/hd·d^{-1} for the first 35 d + RAC for the final 28 d.

[2]Marbling scale: 500 = Small 00, 600 = Modest 00, 700 = Moderate 00.

[3] YG was calculated using the yield grade equation from the USDA beef grading standards (USDA, 1997).

Table 2.4 Effects of feeding ractopamine hydrochloride in combination with zinc or chromium on carcass adjusted measures of efficiency of feedlot steers.

	CONT	RO[1]	RZ[1]	RC[1]	RZC[1]	SEM	*P*-value
n, animals (pens)	36 (6)	35 (6)	36 (6)	36 (6)	36 (6)	-	-
Est. Beginning HCW[2]	310	310	311	310	310	0.59	0.93
Carcass ADG[3]	1.06[b]	1.18[a]	1.13[ab]	1.16[a]	1.07[b]	0.049	0.10
Carcass G:F[4]	0.0774[c]	0.0875[a]	0.0848[ab]	0.0832[abc]	0.0798[bc]	0.004	0.09

[a-c]Within a row, means without a common superscript letter differ ($P \leq 0.10$).
[1]RO: ractopamine hydrochloride (Optaflexx, Elanco Animal Health, Greenfield, IN) fed at a rate of 400 mg/steer·d^{-1} for final 28 d. RZ: 1.0 g Zn supplied as Zn-propionate (KemTRACE Zn; Kemin Industries Inc., Des Moines, IA)/hd·d^{-1} for the first 35 d + RAC for the final 28 d. RC: 3 mg Cr supplied as Cr-propionate (KemTRACE Cr; Kemin Industries Inc., Des Moines, IA)/hd·d^{-1} for the first 35 d + RAC for the final 28 d. RZC: 1.0 g Zn supplied as Zn-propionate + 3 mg Cr supplied as Cr-propionate/hd·d^{-1} for the first 35 d + RAC for the final 28 d.
[2]Est. Beginning HCW = 0.2598(Initial BW$^{1.1378}$) (Tatum et al., 2012).
[3]Carcass ADG = (Average Final HCW – Est. Beginning HCW) / Average Days on Feed (Tatum et al., 2012)
[4]Carcass G:F = Carcass ADG / Average Daily Feed Intake (Tatum et al., 2012).

Table 2.5. Effects of feeding ractopamine hydrochloride in combination with zinc or chromium on quality and yield grade distributions of feedlot steers.

	CONT	RO[1]	RZ[1]	RC[1]	RZC[1]	SEM	*P*-value
USDA Quality Grade, %							
Prime	5.3	< 0.01	5.3	<0.01	<0.01	3.7	1.00
High Choice	4.8	5.1	4.8	7.3	7.3	3.6	0.97
Avg. Choice	36.1	48.6	33.3	33.3	38.9	8.4	0.67
Low Choice	44.4	40.0	44.4	33.3	44.4	8.3	0.78
Select	7.9[a]	5.3[a]	7.9[a]	24.3[b]	7.9[a]	7.2	0.10
Standard	0	0	0	0	0	-	-
$\geq$ Avg. Choice	47.2	54.5	47.2	41.6	47.2	8.4	0.86
Yield Grade[2]							
1	11.1	5.7	8.3	5.5	17.9	6.5	0.44
2	58.5	71.9	44.4	61.3	47.6	8.6	0.15
3	27.5	13.9	38.7	27.5	31.7	8.1	0.25
4	2.3	7.1	7.0	4.6	2.3	4.3	0.74
5	0	0	0	0	0	-	-

[a-b]Within a row, means without a common superscript letter differ ($P \leq 0.10$).

[1]RO: ractopamine hydrochloride (Optaflexx, Elanco Animal Health, Greenfield, IN) fed at a rate of 400 mg/hd·d^{-1} for final 28 d. RZ: 1.0 g Zn supplied as Zn-propionate (KemTRACE Zn; Kemin Industries Inc., Des Moines, IA)/hd·d^{-1} for the first 35 d + RAC for the final 28 d. RC: 3 mg Cr supplied as Cr-propionate (KemTRACE Cr; Kemin Industries Inc., Des Moines, IA)/hd·d^{-1} for the first 35 d + RAC for the final 28 d. RZC: 1.0 g Zn supplied as Zn-propionate + 3 mg Cr supplied as Cr-propionate/hd·d^{-1} for the first 35 d + RAC for the final 28 d.

[2]YG was calculated using the yield grade equation from the USDA beef grading standards (USDA, 1997).

Table 2.6. Effects of feeding ractopamine hydrochloride in combination with zinc or chromium on longissimus muscle quality of feedlot steers.

	CONT	RO[1]	RZ[1]	RC[1]	RZC[1]	SEM	*P*-value
n, animals (pens)	36 (6)	35 (6)	36 (6)	36 (6)	36 (6)	-	-
WBSF, kg	3.17	3.62	3.86	3.61	3.40	0.42	0.57
Cook loss, %	20.8[b]	23.5[a]	22.6[ab]	24.0[a]	21.3[b]	1.21	0.08
IMF, %	5.53	5.20	5.78	5.45	5.40	0.39	0.89
pH	5.60	5.70	5.64	5.64	5.65	0.07	0.75
L*	36.70[ab]	35.52[bc]	35.07[c]	37.18[a]	35.96[ab]	0.64	0.03
a*	19.90	19.08	19.84	19.82	19.19	0.65	0.59
b*	6.71	5.83	6.00	6.89	5.90	0.54	0.20

[a,b,]Within a row, means without a common superscript letter differ (P < 0.10).

[1]RO: ractopamine hydrochloride (Optaflexx, Elanco Animal Health, Greenfield, IN) fed at a rate of 400 mg/hd·d^{-1} for final 28 d. RZ: 1.0 g Zn supplied as Zn-propionate (KemTRACE Zn; Kemin Industries Inc., Des Moines, IA)/hd·d^{-1} for the first 35 d + RAC for the final 28 d. RC: 3 mg Cr supplied as Cr-propionate (KemTRACE Cr; Kemin Industries Inc., Des Moines, IA)/hd·d^{-1} for the first 35 d + RAC for the final 28 d. RZC: 1.0 g Zn supplied as Zn-propionate + 3 mg Cr supplied as Cr-propionate/hd·d^{-1} for the first 35 d + RAC for the final 28 d.

CHAPTER 3: CONCLUSIONS

Technologies, including beta adrenergic agonists like ractopamine hydrochloride (**RAC**), are one way that beef producers can increase profitability of their operations. The potential benefits and pitfalls of RAC have been well documented at doses ranging from 100 to 300 mg RAC/hd·d^{-1} in feedlot steers; however, effects on cattle growth performance and carcass characteristics when feeding RAC at 400 mg RAC/hd·d^{-1} are not as prevalent. In the present thesis, both zinc (**Zn**) and chromium (**Cr**) were supplemented in combination with RAC to examine the interactions between RAC, Zn, and Cr.

Zinc plays an integral role in protein accretion. Previous in vivo research has shown that Zn supplementation in combination with RAC to bovine satellite cells may prevent beta-receptor desensitization (Harris et al., 2012). Chromium plays a vital role in glucose metabolism and insulin action (Vincent, 2004). It has been hypothesized that this metabolic action could influence marbling (Sumner et al., 2007). The only study to date that has evaluated these potential interactions in live feedlot cattle fed both Zn and Cr in combination with 300 mg/hd·d^{-1} (Bohrer et al., 2014), not 400 mg/hd·d^{-1} as was fed in the current study. Therefore, the current study was conducted to evaluate the effects of feeding beta-agonists in combination with zinc or chromium on growth performance, carcass characteristics, and meat quality of finishing steers.

We hypothesized that feeding 400 mg RAC/hd·d^{-1} to steers would increase lean tissue accretion. While there were no differences in live gain or longissimus muscle area in steers fed RAC without additional Zn and Cr, HCW and carcass measures of gain were increased. We also hypothesized that Zn in combination with RAC would increase protein accretion over RAC alone, Cr in combination with Cr would maintain carcass quality over RAC alone, and Zn and Cr in combination with RAC would increase both lean tissue accretion and marbling. However,

these hypothesis were not supported by the data from the current study as neither growth performance, carcass characteristics, or meat quality were improved when Zn and Cr were fed in combination with RAC.

To conclude, Zn and Cr supplementation or the combination of both did not improve growth performance and the addition of Zn and Cr into diets containing 400 mg RAC/hd·d^{-1} did not improve growth performance over the last 28 d of the finishing phase or carcass quality. However, RAC supplementation did increase HCW in comparison to those not fed RAC, but there was no additive benefit of trace mineral supplementation on HCW or any other carcass characteristics. Data from the present trial suggest that supplementing additional Zn and Cr into diets, that are already sufficient in both Zn and Cr, does not improve cattle growth or carcass responses to RAC. However, producers marketing cattle on a HCW basis may profit from the increases in HCW noted in cattle fed RAC compared to cattle not fed RAC.