EFFECTS OF CHROMIUM SUPPLEMENTATION ON DAM PERFORMANCE AND PROGENY GROWTH AND DEVELOPMENT

BY

SAMANTHA G KNEESKERN

THESIS

Submitted in partial fulfillment of the requirements
for the degree of Master of Science in Animal Sciences
in the Graduate College of the
University of Illinois at Urbana-Champaign, 2015

Urbana, Illinois

Master's Committee:

Assistant Professor Tara L. Felix, Chair
Assistant Professor Daniel W. Shike
Assistant Professor Anna C. Dilger
Professor Steven C. Loerch

ABSTRACT

Chromium has been shown to have several positive effects on animal growth and insulin function. Currently, there is only 1 FDA approved source of Cr to be fed to cattle, chromium-propionate (Cr-prop). We hypothesized that feeding Cr-prop to cows during gestation would increase dam insulin sensitivity and glucose metabolism in gestation and increase subsequent milk production, thereby increasing weaning weights of calves from dams supplemented with Cr-prop compared to those not supplemented with Cr-prop. Furthermore, we hypothesized that feeding Cr-prop to steers in the feedlot would increase growth performance, increase insulin sensitivity, and improve marbling in the carcasses of steers fed Cr-prop when compared to steers not fed Cr-prop. To test these hypotheses, 2 experiments were conducted.

In experiment 1, the objectives were to determine the effects of supplementing Cr-prop through mid- and late gestation on beef cow BW and BCS, milk production, and progeny development, pre-weaning. Spring-calving, Angus-cross cows (n = 66) were fed 1 of 2 supplements through mid- and late-gestation: (1) 1.81 kg corn/hd·d^{-1} as fed (**Control**), or (2) 1.81 kg corn fortified with 3 mg Cr/hd·d^{-1} as fed (**CrP**). Chromium supplementation did not affect ($P \geq 0.24$) pre- or postpartum cow BW, BCS, or BW and BCS change. There was an interaction ($P < 0.01$) of d in gestation and CrP supplementation for plasma glucose concentrations only. During mid-gestation, plasma glucose concentrations in cows fed CrP decreased 0.282 mmol/L compared to cows fed Control; however, by late-gestation, glucose concentrations in cows fed CrP increased 0.321 mmol/L compared to cows fed Control. Supplementation with CrP did not affect ($P \geq 0.58$) mean insulin or insulin:glucose; however, insulin and glucose concentrations were reduced ($P <0.01$) as d in gestation increased, regardless of CrP supplementation. There was no effect of dam treatment on calf BW at birth ($P = 0.40$) or

weaning ($P = 0.56$). Chromium supplementation did not affect ($P \geq 0.20$) 24-h milk weight in mid- or late lactation. Milk composition was not affected ($P \geq 0.25$) by CrP at mid-lactation. In late lactation, cows fed CrP had greater ($P = 0.01$) milk urea nitrogen compared to cows fed Control, with no other differences observed ($P \geq 0.23$). In this experiment, supplemental CrP did not affect cow BW, BCS, milk production, or calf BW at birth or weaning. Furthermore, cow insulin sensitivity did not change; however, concentrations of both glucose and insulin decreased as days in gestation increased, regardless of CrP supplementation. The steer progeny from these supplemented dams were used in a second experiment to determine the effects of CrP during the finishing period.

In experiment 2, objectives were to test the effects of feeding Cr-prop to finishing steers in the feedlot on growth performance, insulin sensitivity, and carcass characteristics. Angus-cross steers (n = 34) from the dams fed in experiment 1 were stratified by BW and dam treatment and assigned to 1 of 2 treatments: (1) no supplemental Cr (**Cont**), or (2) 3 mg supplemental Cr/hd·d^{-1} (**CrP**). Both supplements, Cont and CrP, were delivered via 0.454 kg ground corn top dressed on the basal diet. There was no effect ($P \geq 0.45$) of CrP on ADG, DMI, G:F, or final BW. However, steers fed CrP had greater ($P = 0.10$) days on feed (**DOF**) than steers fed Cont. There were no effects ($P \geq 0.41$) of CrP on HCW, back fat, or KPH. Steers fed CrP had increased ($P = 0.01$) dressing percentage (**DP**) and tended to have increased LM area ($P = 0.15$), decreased marbling scores ($P = 0.11$), and intramuscular fat ($P = 0.11$) compared to steers fed Cont. There were no differences ($P \geq 0.25$) in Quality or Yield Grade distributions. A glucose tolerance test (**GTT**) was conducted early in the finishing phase (21 DOF) and later in the finishing phase (98 DOF). There was a Feedlot Treatment **(FT**) × Time × DOF interaction ($P = 0.08$) for glucose concentrations. There were no other interactions ($P \geq 0.21$) for glucose or

insulin concentrations over time. There was no FT × DOF ($P \geq 0.21$) for insulin area under the curve (**iAUC**), insulin:glucose, insulin or glucose baseline, or peak insulin or glucose concentrations. A FT × DOF interaction was observed for glucose area under the curve (**gAUC**; $P = 0.01$), glucose clearance rate (***k***; $P = 0.02$), and glucose half-life (**T½**; $P = 0.07$); however, DOF seemed to have the greatest effect on GTT. At 98 DOF, all steers had increased ($P < 0.01$) peak glucose and insulin, *k*, iAUC, insulin:glucose, and baseline insulin, when compared to 21 DOF, regardless of treatment, but, gAUC and $T_{1/2}$ decreased ($P < 0.01$). While steers fed CrP tended ($P = 0.11$) to have increased baseline glucose concentrations compared to steers fed Cont, CrP supplementation did not affect ($P \geq 0.17$) other measures of glucose or insulin. Results of this experiment indicate that, contrary to our hypothesis, CrP increased LM area and DP, but decreased marbling scores and intramuscular fat, with no effect on growth performance. With increased DOF, all steers became more insulin resistant, indicated by the use of more insulin to clear less glucose; and these effects were not mitigated by CrP supplementation.

Although the effectiveness of Cr supplementation has been proven in nonruminant animals and diabetic humans, the efficacy of Cr supplementation to gestating cows and feedlot finishing cattle remains to be determined. Factors limiting research in this area are many; however, the largest challenges may be determining Cr levels in the basal diet and the FDA limit on Cr inclusions in the diet. Future research with increasing Cr concentrations and greater animal numbers may be warranted based off of these preliminary findings.

ACKNOWLEDGEMENT

There are many people I would like to acknowledge for their guidance and assistance throughout my research at the University of Illinois. Most importantly, I would like to thank my husband, Brett Kneeskern, for his love, friendship, and his selfless, continuous support. I would also like to thank my family and in-laws for their support throughout this journey. I would like to thank my fellow graduate students for primarily their friendship, but also their assistance throughout my project: Chris Cassady, Bailey Edenburn, Blake Lehman, Chance Meteer, Lindsay Shoup, and Bain Wilson. Furthermore, I would like to thank Gary Lowe, Doug Clevenger, Wayne Shriver, and all of the other employees at EARS and the Beef Research Station at OARDC for their innumerable hours of feeding and assistance when I could and couldn't make the trip back to Ohio for my research. Lastly, I would like to thank my committee for their advice, knowledge, and dedication in making me an animal scientist.

TABLE OF CONTENTS

Page

LIST OF TABLES……...viii

LIST OF FIGURES……ix

CHAPTER 1: LITERATURE REVIEW……1

INTRODUCTION……...1

CHROMIUM……...1

CHROMIUM UPTAKE AND MECHANISM OF ACTION...……..3

FETAL PROGRAMMING……..7

CHROMIUM AND STRESSED/TRANSPORTED CATTLE……...9

BEEF QUALITY AND YIELD……12

INSULIN RESISTANCE……..14

SUMMARY……...17

LITERATURE CITED……..18

CHAPTER 2: EFFECTS OF CHROMIUM SUPPLEMENTATION TO BEEF COWS DURING GESTATION ON BEEF COW PERFORMANCE AND PROGENY DEVELOPMENT PRE-WEANING……25

ABSTRACT……...25

INTRODUCTION ……26

MATERIALS AND METHODS……...27

RESULTS AND DISCUSSION……31

IMPLICATIONS……...35

LITERATURE CITED……..37

TABLES AND FIGURES…….……41

CHAPTER 3: EFFECTS OF CHROMIUM SUPPLEMENTATION TO FEEDLOT STEERS ON GROWTH PERFORMANCE, INSULIN SENSITIVITY, AND CARCASS CHARACTERISTICS……...47

ABSTRACT……...47

INTRODUCTION ……48

MATERIALS AND METHODS……...49

RESULTS……..56

DISCUSSION……58

LITERATURE CITED……..64

TABLES AND FIGURES…….68

CHAPTER 4: CONCLUSIONS……..……..74

LIST OF TABLES

Page

Table 2.1. Composition of supplementation to cows……41

Table 2.2. Effects of CrP supplementation on cow performance…..……42

Table 2.3. Effect of CrP supplementation on milk production and milk composition as determined via weigh-suckle-weigh…….……...43

Table 2.4. Effects of CrP supplementation on prepartum cow glucose and insulin concentrations…….....44

Table 3.1. Composition of diet fed to steers fed 0 mg Cr/d (Cont) or 3 mg Cr/d (CrP)…..…….68

Table 3.2. Effects of Cr supplementation on growth performance of steers fed 0 mg Cr/d (Cont) or 3 mg Cr/d (CrP)……...69

Table 3.3. Effects of chromium supplementation on carcass characteristics and USDA Yield and Quality Grade distributions of steers fed 0 mg Cr/d (Cont) or 3 mg Cr/d (CrP)…..……...……70

Table 3.4. Effects of chromium supplementation on glucose and insulin kinetics following and intravenous glucose tolerance test in steers fed 0 mg Cr/d (Cont) or 3 mg Cr/d (CrP)…….71

LIST OF FIGURES

Page

Figure 2.1. Timeline of cow CrP supplementation……..…….45

Figure 3.1. Effects of CrP supplementation on glucose change over time following an intravenous glucose tolerance test (GTT).....….....72

Figure 3.2. Effects of CrP supplementation on insulin change over time following an intravenous glucose tolerance test (GTT).....…..73

CHAPTER 1: LITERATURE REVIEW

Introduction

Chromium (**Cr**) is an important trace mineral in human and animal nutrition because of its role in carbohydrate and lipid metabolism (Vincent, 1999). Supplementation of Cr to livestock may improve health and increase immune response (Spears, 2000), amplify growth performance in stressed feeder calves (Kegley et al., 1997), increase milk production in dairy cattle (McNamara and Valdez, 2005), and improve carcass traits in swine (Jackson et al., 2009). These positive effects on animal performance may be related to Cr effects on insulin. Chromium acts on insulin to enhance, or improve, glucose use (Spears and Weiss, 2014). However, the exact mode of action of Cr has not been well elucidated. Some researchers suggest Cr enhances the response to insulin receptors by increased sensitivity (Mertz et al., 1974), while others propose Cr increases the number of insulin receptors (Anderson, 2003). In growing beef heifers, Spears et al. (2012) found Cr supplementation increased glucose clearance rates, decreased glucose concentrations, and decreased insulin:glucose, all signs of improved insulin function. In cattle, increased days on feed results in insulin resistance and gestational energy source plays a role in the development of insulin resistance (Radunz et al., 2012). There is a lack of information investigating the effects of Cr supplementation during gestation on pre- and post-natal development of beef cattle progeny.

Chromium

Chromium ore mainly consists of chromite, a mineral composed of Fe and Cr^{3+} with minute amounts of Al and Mg (NRC, 2005). Chromium has not been mined in the United States

since 1961; therefore, Cr is imported from South America, Turkey, and Russia (NRC, 2005). Hexavalent Cr, a strong oxidizing agent, is used in the production of metals and stainless steel (NRC, 2005). Trivalent Cr is more stable, less toxic, and is supplemented to humans and livestock because of its role in metabolic activity (Anderson, 2003). In fact, trivalent Cr is found naturally in many common food items, including egg yolks, coffee, nuts, whole-grain products, broccoli, and meat (Cefalu and Hu, 2004). Chromium is also in tissue, blood, river, and soil samples at low concentrations, ≤100 ng/g, ≤ 0.3 μg/L, 10 μg /L, and 37 mg/kg, respectively (NRC, 2005).

The role of Cr in human nutrition has been studied for almost 4 decades. In 1977, Cr was added to a patient's total parenteral nutrition (**TPN**) and determined to be essential when exogenous insulin failed to relieve diabetic-like symptoms (Jeejeebhoy et al., 1977). Upon receiving Cr, the patient no longer needed exogenous insulin, and her diabetic-symptoms were alleviated (Jeejeebhoy et al., 1977). Chromium is now regularly added to TPN solutions (Anderson, 1998).

Currently, no Cr requirement or maximum tolerable level has been established for cattle diets (NRC, 2000). However, the only FDA approved source of Cr in cattle diets is chromium-propionate (**Cr-Prop**). Chromium can only be legally added to the diet at levels up to 0.5 mg Cr/kg DM, or to provide 3 mg/hd per d. Chromium supplementation greater than 3 mg/d has elicited the same response as supplementation at 3 mg/d in growing cattle (Spears et al., 2012), and Cr has been supplemented up to 15 mg/hd per d in cattle and no linear increases in performance were found (Sumner et al., 2007). Furthermore, the findings on immune function and health have been too inconsistent to estimate a requirement (Chang and Mowat, 1992; Lindell et al., 1994; Mathison and Engstrom, 1995). Growth performance, supplementation

levels, and source of Cr supplementation has also varied greatly between studies (Moonsie-Shageer and Mowat, 1993; Spears, 2000; Pollard et al., 2002; Bernhard et al., 2012), complicating the process to make a recommendation; these variations will be discussed in detail later. Therefore, determining the optimal Cr supplementation level for cattle has been challenging.

Both Cr-Prop and Chromium-Picolinate (**Cr-Pic**) are approved for swine diets at an inclusion up to 0.2 mg Cr/kg DM (NRC, 2012). Other than swine and cattle, Cr supplementation is not permitted in any other species' diet (Spears, 2000). When Cr is fed (not injected), there have been no reports of toxic effects in humans or animals (Anderson, 2003). Organic forms of Cr (Cr-Prop and Cr-Pic) are more bioavailable when compared to inorganic forms (Cr-Chloride or Cr nicotinic acid) of Cr (Kegley and Spears, 1995).

Chromium deficiency in cattle, even though it is hard to achieve, can cause symptoms similar to diabetes in humans (Kawachi, 2006). Diabetic humans have increased absorption rates of Cr, but cannot use Cr. Thus, diabetics have increased Cr losses. Similar to cattle, humans who have Cr deficiencies and/or problems with insulin functionality will most efficiently use Cr (Anderson, 1997). The only way to quantify Cr action in beef cattle is through supplementation and alterations in insulin and glucose as measured via glucose tolerance and insulin sensitivity tests (Anderson, 1997). Quantifying Cr action via these tests is discussed below with an emphasis on Cr efficacy in cattle feeding.

Chromium Uptake and Mechanism of Action

Mode of Cr action is still not fully known, but we do know that Cr improves insulin function. There has been much speculation and each proposed mode of action may be associated

with the other. For example, studies have suggested Cr may increase insulin receptor number and the activity of insulin binding to its receptor (Anderson, 2003), enhance insulin receptor sensitivity (Mertz et al., 1974), increase the receptor tyrosine kinase activity after insulin binding (Vincent, 1999), or enhance membrane fluidity to allow for effective trafficking of GLUT4 (Chen et al., 2006). Chromium has also been shown to increase pancreatic beta-cell sensitivity, thus, enhancing insulin secretion (Potter et al., 1985; Anderson, 1998). Our knowledge of Cr functionality and how Cr affects insulin has evolved over the years; however, the basics of Cr action remains fairly constant.

In 1974, Cr was thought to be a part of a Glucose Tolerance Factor (**GTF**; Mertz et al., 1974) that potentiated the action of insulin. The GTF compound was later renamed and is now known as chromodulin (coined by Davis and Vincent, 1997), a low-molecular-weight Cr-containing oligopeptide, made up of 4 types of amino acid residues: glycine, cysteine, glutamate, and aspartate, some of the main compounds that composed GTF (Yamamoto et al., 1989). Once Cr is absorbed, it moves from the blood into insulin-dependent cells at the beta receptor in muscle and adipose tissues (Morris et al., 1992). Transferrin binds and assists in Cr movement. Transferrin receptors are sensitive to insulin; as insulin increases, transferrin moves towards the membrane (Anderson, 2003).

Insulin receptors are transmembrane receptors that have 2 extracellular alpha subunits and 2 intermembrane beta subunits. Anderson (2003) showed that once insulin binds to the alpha subunit of an insulin receptor, phosphorylation of the transmembrane beta subunit occurs automatically and simultaneously. The enzyme that is regularly responsible for the intermolecular phosphorylation reactions is tyrosine kinase (Anderson, 1998). Tyrosine kinase is activated by chromodulin, and Anderson (2003) reported 4 mol of Cr/mol of chromodulin were

needed to activate tyrosine kinase receptor. Once chromodulin binds 4 mol Cr, chromodulin is fully activated and can activate tyrosine kinase. In fact, in rat adipocytes, chromodulin increased tyrosine kinase activity, in the presence of 100 n*M* insulin by 8 fold compared to 0 n*M* of insulin (Davis and Vincent, 1997). Therefore, with an increase in phosphorylation of the insulin receptor by tyrosine kinase, insulin sensitivity is increased. Chromium also increases insulin sensitivity by inhibiting phosphotyrosine phosphatase (**PTP-1**; Davis et al., 1996; Anderson, 1997). This protein removes a phosphate group from the beta subunit of the insulin receptor, inactivating the receptor (Anderson, 1998). The Cr-chromodulin complex inhibits PTP-1, allowing for increased activation of the insulin receptor, i.e. phosphorylation is allowed to occur at the beta subunit, and is associated with increased insulin sensitivity. Thus, insulin sensitivity is enhanced when the Cr-chromodulin complex 1) increases insulin receptor kinase activity, and 2) inhibits PTP-1 from inactivating the insulin receptor.

The transporter GLUT4 is 1 of 4 glucose transporters that is highly expressed in muscle and adipose tissue. This plasma membrane protein, in its basal state, is cycled between the plasma membrane and vesicular compartments within the cell interior (Fernyhough et al., 2007). In the presence of insulin, GLUT4 is translocated to the plasma membrane and stimulates glucose uptake via facultative diffusion (Chen et al., 2006). Once insulin binds to an insulin receptor, GLUT4 protein levels increase. Furthermore, glucose transport and GLUT4 trafficking within the cell may be increased as membrane fluidity is increased (Pilch et al., 1980). Chen et al. (2006) observed that Cr mobilized GLUT4 to the plasma membrane in adipocytes and accumulated adjacent to the inner cell surface membrane. In the presence of insulin, GLUT4 was incorporated into the membrane. The authors believe this may be due to the aforementioned increase in receptor kinase activity by Cr, thus, enhancing GLUT4 movement with the addition

of improved membrane fluidity. Obese rats fed Cr-Pic had significantly reduced glucose and insulin AUC and had an enhancement of membrane-associated GLUT4 following a GTT compared to control rats (Cefalu et al., 2002). Although the increase in tyrosine kinase activity leading to increased GLUT4 movement has been reported, the mechanisms behind the movements is still unknown.

Although these mechanisms are well understood in human and mouse models, the effects of Cr on mode of action of insulin and insulin responses in cattle has not been as well studied. Spears et al. (2012) determined that Cr-Prop enhances insulin sensitivity, as determined via glucose tolerance test. Angus-cross heifers were supplemented with Cr-Prop at levels of either 0, 3, 6, or 9 mg Cr/d. The authors made comparisons of control vs Cr treatments (3, 6, and 9 mg Cr/d), 3 versus 6 mg Cr/d, and 6 versus 9 mg Cr/d. Heifers that were not supplemented with Cr had an insulin:glucose ratio of 68.6 pmol/L:mmol/L and heifers that were fed 3, 6, or 9 had an insulin:glucose of $\leq$ 57.7 pmol/L:mmol/L. From 15 to 45 min post-infusion, heifers fed Cr, regardless of level, had a faster glucose clearance rate compared to heifers not fed Cr. From 0 to 45 min post-infusion, Cr supplementation decreased insulin and glucose area under the curve by $\geq$ 1,316 μIU/mL and 158 mg/dL respectively, compared to heifers not supplemented with Cr. In a similar experiment, Holstein heifers were supplemented with 0, 5, 10, or 15 mg Cr/d supplied as Cr-Prop (Sumner et al., 2007). Glucose clearance was increased by Cr supplementation, as characterized by a decreased glucose half-life, time to nadir, and AUC, regardless of whether Holsteins were fed 5, 10 or 15 mg Cr/d. Basal glucose was increased in Holsteins fed 10 and 15 mg Cr /d when compared to Holsteins fed 0 or 5 mg Cr/d, but supplemental Cr did not affect any insulin variables. Similar insulin values in conjunction with a greater glucose clearance rate would indicate an improved sensitivity to insulin in tissues of those Holsteins fed 10 or 15 mg

Cr/d. In newly received beef calves, chromium-L-methionine at either 0.4 or 0.8 mg/kg of diet has been shown to linearly increase glucose clearance rates from 5 to 10 min (Kegley et al., 2000). However, steers fed Cr had a greater insulin AUC from 0 to 60 min and had a linear increase in insulin:glucose, and therefore, steers used more insulin to remove glucose and were less sensitive to insulin even though they had an increase in glucose clearance rates after infusion. To date, the only Cr supplementation work to look at insulin sensitivity has been done in growing, newly received calves (above) or lactating cows (described below). There is lack of information on the effects of Cr supplementation to beef cows during gestation on the growth and development of the progeny before entering the feedlot or the effects of Cr supplementation during the feedlot finishing phase on growth and insulin sensitivity.

Fetal Programming

"Fetal Programming" was first coined by Godfrey and Barker in 2001 when they found that changes occur in the fetus due to nutrient supply in utero, and that these modifications affected postnatal growth and development. Godfrey and Barker's work was with human nutrient deficiency and evidence showed glucose tolerance and insulin sensitivity were reduced at birth, and increased blood pressure and cardiovascular disease were observed later in life in the progeny from mothers that suffered nutrient restriction during gestation.

Critical to animal nutrition, the number of muscle fibers formed during pre-natal development is crucial for increasing muscle growth postnatally and overall muscle development (Rehfeldt et al., 1999). In rats, offspring from mothers fed Cr-restricted diets had decreased percentages of lean body mass and fat-free mass (Padmavathi et al., 2010) compared to offspring from mothers fed a control diet that included Cr during pregnancy. Authors suggested the

decrease in muscle was due to decreased expression of the myogenic genes MyoD, Myf5, and MyoG. No differences were observed in offspring insulin sensitivity with varying maternal Cr nutrition (Padmavathi et al., 2010). However, in beef cattle, the effects of Cr supplementation to increase glucose supply to the fetus on increasing lean tissue and growth, has not been previously explored.

Some Cr research has been conducted in mature cows. Nearing parturition, cows naturally become more insulin resistant in order to partition nutrients to the growing fetus and for milk production, and therefore, may be the best time to supplement with Cr. Chromium supplementation increased tissue sensitivity to insulin in gestating beef cows (Stahlhut et al., 2006a) and dairy cows (Hayirli et al., 2001) and increased subsequent pregnancy rates (Stahlhut et al., 2006b). Furthermore, Cr supplementation during gestation reduced cow BW after parturition (Stahlhut et al., 2006b). Authors concluded this could have an impact on lifetime reproduction effectiveness and milk production of those cattle supplemented with Cr. In dairy cattle, Cr supplementation during both gestation and lactation has been shown to increase milk production in numerous studies (Besong et al., 1996; Hayirli et al., 2001; McNamara and Valdez, 2005; Smith et al., 2005). Zebu cows in Brazil supplemented with Cr-yeast had increased percent estrus detected and a tendency for a higher pregnancy rate, when compared to cows receiving no supplementation (Aragon et al., 2000). The period of anestrus was also decreased for those Zebu cows supplemented with Cr. In nonlactating dairy cows, excessive energy intake is common during the dry period, with insulin resistance and impaired glucose tolerance soon to follow. A recent study in Brazil aimed at mitigating or alleviating insulin resistance for cows exceeding their ME requirement (Leiva et al., 2014). Gir × Holstein cows (n = 13) were fed 1 of 3 dietary treatments: to meet ME requirement without Cr, to exceed ME requirement (177%) with 10 mg

Cr as Cr-Prop/hd·d^{-1}, or to exceed ME requirement (177%) without Cr-Prop. Following a GTT, cows fed Cr-Prop had lower mean insulin and insulin:glucose values compared to cows not fed Cr-Prop. Insulin AUC was greatest for cows fed diets exceeding their energy requirements and not receiving Cr; cows fed diets exceeding their energy requirements and fed Cr were not different from cows meeting ME requirements. These data suggest that cows receiving excessive energy may have reduced insulin sensitivity, but supplementation with Cr-Prop prevented the decrease in insulin sensitivity following a GTT. However, results from this study could be discounted do to the small number of cows per treatment and high level of Cr supplementation. Overall, the role of Cr supplementation in beef cows on BW and BCS, milk production, and subsequent calf performance is not well elucidated.

Chromium and Stressed/Transported Cattle

Cattle transportation at any distance can cause stress, but particularly in young, growing calves. The majority of research on Cr supplementation to cattle has studied Cr effects on stress as a means of promoting the immune system. In general, animal requirements are increased when in a state of stress, rather caused from shipping, immunization, weaning, etc.; however, Cr may counteract the effects of stress (Chang and Mowat, 1992). For example, Kegley et al. (1997) conducted a study on transported versus non-shipped feeder calves. Steers that were transported had a 6.5% BW shrink compared to calves that were not shipped that had a 0.6% shrink. Shipping also decreased DMI after arrival for 23 days and serum cortisol levels were also increased at arrival. In order to try and alleviate some of the stressors from shipping, Kegley et al. (1997) fed Cr as Cr nicotinic acid complex to supply 0.4 mg Cr/kg of DM. Calves supplemented with Cr, shipped or not, had an increased ADG in the first 80 days in the feedlot.

In stressed cattle, Cr supplementation has reduced morbidity by as much as 38.1% (Moonsie-Shageer and Mowat, 1993; Mowat et al., 1993; Lindell et al., 1994). However, Cr supplementation also resulted in no changes in morbidity in other studies (Chang and Mowat, 1992; Chang et al., 1995; Mathison and Engstrom, 1995). After inoculation with IBRV and Pasteurella haemolytica, calves supplemented with 0.4 mg Cr/kg DM (either $CrCl_3$ or Cr-nicotinic acid complex) tended to have lower body temperatures (Kegley et al., 1996). However, at the same levels of Cr in another study, no effects of Cr on body temperature or intake were observed (Kegley et al., 1997). Arthington et al. (1997) supplemented high-Cr yeast to calves inoculated with bovine herpes virus-1 with no effects on rectal temperatures after inoculation. Therefore, effects of Cr supplementation on subsequent calf morbidity in "stressed" calves are variable.

Cortisol is a hormone used to attempt to quantify "stress" (Chang and Mowat, 1992), i.e. cortisol concentrations are greater in stressed animals than in those that are not stressed. Chromium supplementation to ameliorate serum cortisol levels in cattle has been variable. In several studies, Cr decreased serum cortisol levels in cattle (Chang and Mowat, 1992; Moonsie-Shageer and Mowat, 1993; Kegley et al., 1996); however, in others, Cr supplementation had no effect on serum cortisol levels (Lindell et al., 1994; Kegley and Spears, 1995; Arthington et al., 1997; Kegley et al., 1997). In addition to decreased cortisol, calves supplemented with Cr had improved growth performance. Chang and Mowat (1992) transported Charolais growing feeder calves just over 3,000 km, before beginning an initial 28 day Cr-yeast and corn silage diet. Supplementation of 0.4 mg Cr/kg of DM increased ADG by 30% and G:F by 27%. Chromium increased calf growth, but did not affect morbidity during this high-stress, 28 day feeding period. The calves were then switched to a 70 day growing period with 0.2 mg of Cr per kg DM fed.

There was no effect of Cr on ADG or G:F during the second period. However, Cr decreased serum cortisol (75.0 vs 55.6 nmol/L) and improved immune response, as shown by an increase in immunoglobulin M during the last 70 days. Calves supplemented with Cr also tended to have lower serum glucose concentrations, without changes in insulin concentrations, indicating a possible increase in insulin sensitivity. The authors cited a lack of stress and lack of Cr deficiency as the reason for the lack of Cr response during the 70 day growing period.

Similar results were shown by Moonsie-Shageer and Mowat (1993) with calves transported over 44 hours. Calves supplemented with Cr-yeast to supply 0.2 and 1.0 mg Cr/kg of diet DM had an ADG increase of 27% over the 30 day trial compared to those calves that received no Cr supplementation. Chromium-yeast supplemented to supply 0.2 and 0.5 mg Cr/kg of diet DM also increased DMI. The authors alluded to low dietary CP (11%) as a source of additional stress, on top of transportation. The 236 kg steers in the current study would have a CP requirement of 700 g/d (NRC, 2000); however, in the first 10 days, on average, the steers were only consuming 260 g CP/d.

Because of research findings of improved growth performance in stressed cattle, other scientists pursued research in hopes of having similar results. Again, however, variable results have been shown with Cr supplementation to non-stressed calves. Kegley and Spears (1995) investigated efficacy of Cr to increase growth performance in crossbred Angus steer calves. They supplemented three types of Cr at 0.4 mg Cr/kg of DM: chromium choloride hexahydrate, Cr-yeast, and Cr nicotinic acid complex. However, performance was not affected by any source of Cr. Chromium nicotinic acid, the only Cr source that had an effect, increased IgG by 251 mg/dL compared to calves fed Cr-yeast or no Cr. However, no morbidity was observed throughout the trial, so quantifying changes in immune response was not possible. Chang et al. (1992) and

Bunting et al. (1994) also reported no changes in ADG, DM, and G:F in non-stressed, growing calves. Because calves were not stressed nor did they have a Cr deficiency, Cr supplementation did not have an effect. Overall, there are inconsistent results for Cr effect on health status, immunity, and growth performance.

Beef Quality and Yield

Carcass weight, USDA Yield Grade, and USDA Quality Grade are used to determine the value of beef carcasses. Quality Grade is determined by the amount of intramuscular (**IM**) fat in the beef loin (Tatum et al., 2007). More IM fat will result in carcasses grading Prime and Choice and quality premiums for the producer, whereas less IM fat will result in carcasses grading Standard and Select with discounts for the producer. Yield Grades are determined by the amount of boneless, closely trimmed retail cuts of meat and producers receive discounts for carcasses with more trim, or subcutaneous (**SC**) fat (Tatum et al., 2007). Researchers have also found that consumers prefer less SC fat on their retail beef product (Savell et al., 1989; Jeremiah, 1996).

Varying the nutrition and supplementation during late gestation may divert energy to either the IM or SC fat depots in subsequent progeny (Larson et al., 2009). However, there is a gap in knowledge regarding the mechanistic factors that control and regulate the site of fat deposition. It is known that concentrations of insulin receptors vary among different types of cells; with adipocytes and hepatocytes having the most with at least 200,000 receptors and almost all cells having at least one insulin receptor (Anderson, 1997). Glucose is the major substrate for adipogenesis in the IM fat depot and glucose uptake plays a critical role in increasing USDA Quality Grade (Schoonmaker et al., 2004), ultimately controlled by insulin sensitivity and uptake. Thus, Cr effects on insulin may indirectly have effects on adipogenesis.

Radunz et al. (2012) reported that insulin resistant cattle may have enhanced marbling. But, one study in beef cattle on the effects of Cr supplementation on carcass characteristics and growth performance showed negative effects on growth and carcass characteristics of steers supplemented with 0.4 mg Cr/kg of DM (Pollard et al., 2002). Chromium-yeast was supplemented to provide 0.0, 0.2, and 0.4 mg Cr/kg of diet DM. Calves receiving 0.4 mg/kg of DM had the lowest ADG, G:F, final live weight, dressing percentage, marbling scores, YG, and HCW compared to the other two treatments. There were no differences in ADG, G:F, marbling score, dressing percentage, and YG in calves receiving 0.0 and 0.2 mg Cr/kg. Calves receiving 0.2 mg/kg had greater HCW compared to calves fed 0.0 and 0.4 mg/kg. Chromium-yeast at any level tended to increase longissimus muscle area. Thus, Pollard et al. (2002) suggested feeding Cr at 0.2 mg/kg of DM because of the improvements of HCW and LM area, without the reductions noted in performance at the 0.4 mg/kg inclusion. However, once again, effects of Cr supplementation have varied. Only 3 other studies in beef cattle have aimed at feeding Cr to feedlot steers to improve carcass traits and all 3 reported no effects of Cr on carcass characteristics (Chang et al., 1992; Mathison and Engstrom, 2005; Bohrer et al., 2014). Similar to Cr supplementation to cattle, swine studies have been variable; however, the swine industry has conducted more research of Cr supplementation on carcass characteristics than the beef industry.

Swine studies have shown that Cr supplementation increases longissimus muscle area (Page et al., 1993; Kornegay et al., 1997) and percentage of muscle in the carcass (Boleman et al., 1995; Jackson et al., 2009). And, in fact, percentages of fat tissue has been decreased by at least 1.5% (Mooney and Cromwell, 1997) and SC fat depth at the 10th rib has also been decreased by 0.38 cm, 0.50 cm, and 0.52 cm (Page et al., 1993; Boleman et al., 1995; Jackson et

al., 2009, respectively) in swine supplemented with Cr when compared to those not supplemented. However, results are conflicting as Matthews et al. (2003) supplemented 0.2 mg Cr/kg of DM as Cr-Prop to growing-finishing pigs; swine supplemented with Cr had an increase in marbling. Another study by Matthews et al. (2001) fed either no Cr, 0.2 mg Cr/kg DM as Cr-Pic, or 0.2 mg Cr/kg DM as Cr-Prop and observed a tendency for Cr-Pic to increase carcass length. However, there were no differences in backfat, LM area, dressing percentage, percent muscle, lean:fat, leaf fat weight (internal fat), or percent lean. For those growing barrows, both Cr-Pic and Cr-Prop decreased ADG and average daily feed intake by 0.12 and 0.10 kg, respectively. During an insulin challenge test, Cr-Prop supplementation increased glucose clearance by 0.87%/min. Growth performance also varied between studies, with some presenting increased G:F (Lindemann et al., 1995; Jackson et al., 2009), others seeing no differences (Mooney and Cromwell, 1997) or decreased ADG and DMI (Page et al., 1993) in swine fed Cr compared to those that were not. Variability between studies has created the need for more research to be conducted on the effects of Cr on muscle growth, carcass characteristics, and adipogenesis. The mechanisms of Cr function in cattle are not known; whether Cr is enhancing adipogenesis, myogenesis, or both, is unclear.

Insulin resistance

Insulin resistance, as defined by Anderson in 1998, is when there is little control, utilization, and storage of glucose. Anderson posed that a human becomes insulin resistant because of glucose intolerance and/or insulin levels becoming elevated. If insulin levels are elevated, or there are excess levels of insulin circulating in the blood, also called hyperinsulinaemia, then there is a possibility of increased adipogenesis and decreased lean tissue

(Anderson, 1998). In cattle, increased subcutaneous fat and decreased muscle area would negatively affect carcass values. Insulin resistance can also impair body weight gain (Saltiel, 2012) and mitigating insulin resistance during the growing phase may improve muscle growth and marbling before slaughter.

The cause of insulin resistance or decreased insulin sensitivity can be multifaceted. For example, both excessive and deficient energy intake can lead to insulin resistance (Leiva et al., 2014). Decreased insulin sensitivity can also be caused by mobilization of lipids and increased lipogenesis (Prior and Scott, 1980; Vernon, 1980) because lipids interfere with insulin and insulin receptor binding (Ferezou and Bach, 1999; Lewis et al., 2002). Non-esterified fatty acids can interfere with insulin binding to its receptor and the downstream signaling pathway, causing adipocytes to become more insulin resistant (Lewis et al., 2002; Pires et al., 2007). Insulin resistance could be due to a defective chromodulin not binding to 4 chromic ions and therefore, receptor kinase would not be fully activated (Davis and Vincent, 1997). Shulman (2000) hypothesized that insulin may not be delivered to the tissue causing faulty downstream signaling and defective muscle glycogen synthesis. Glucose transporter type 4 may be inefficiently stimulated by insulin because of decreased glucose-6-phosphate and free fatty acids (Shulman, 2000). Normally, with any substrate and its receptor, problems arise by a downstream signaling defect. Le Roith and Zick (2001) believe a reduction in receptor kinase activity could begin the signaling process problematically.

In cattle, Radunz et al. (2012) studied the effect of days on feed (**DOF**) on glucose and insulin response. Their data suggest that increasing DOF increased cattle needs for insulin to metabolize glucose, i.e. cattle became more insulin resistant the longer they were fed. In this same report, the authors also suggested that improving insulin sensitivity may increase lean

muscle growth and improve feed efficiency. In agreement with Radunz et al. (2012), an earlier report showed that insulin sensitivity decreased as BW and fat content in hindquarters of beef steers increased (Eisemann et al., 1997). The combination of these results suggests that tissue insulin resistance increases with BW, age, and body fat content in beef steers.

Furthermore, studies from Radunz suggest that progeny from cows that were limit-fed corn had reduced insulin sensitivity, increased birth and weaning weights, and increased IM fat percentage compared to progeny from cows fed hay-based diets (Radunz et al., 2010; Radunz et al., 2012). Certainly, postpartum growth and glucose metabolism is affected by the source of energy available to the dam; however, the mechanisms by which maternal nutrition affects progeny insulin response and IM fat deposition are unknown.

Bernhard et al. (2012) hypothesized increased insulin function would increase glucose uptake. However, following a glucose tolerance test, steers supplemented with 0.2 mg Cr/kg DM as Cr-Prop had an increased insulin:glucose ratio and their insulin concentration tended to be greater than calves not fed Cr. This contradictory finding suggests these calves actually became more insulin resistant or had reduced insulin sensitivity. An insulin sensitivity test (**IST**) revealed that glucose concentrations were greater in calves supplemented with Cr at 15 min, aligning with decreased insulin sensitivity and a "saturation effect of glucose at sensitive tissues." Other results from the IST complicated the matter. Calves receiving Cr had an increased glucose clearance rate at 30 to 45 min and glucose levels returned to baseline faster. The authors imply that these calves might have had glucosuria because of their light weight; therefore, Cr was excreted in the urine at these time points and prevented the calves from having higher glucose levels. There is a lack of information regarding the supplementation of Cr-prop in feedlot cattle and the subsequent effects on insulin sensitivity.

Summary

Chromium is an important trace mineral that beef producers could utilize as a supplement that may be beneficial to certain types of production systems. It has been most explored as an agent to improve health in stressed animals. However, there has been a lot of variability in Cr effects not only on stressed cattle, immunity, and antibody response, but also on growth performance, milk production, and reproduction. The variability may be due to severity of deficiencies, Cr in the basal diet, Cr supplementation inclusion levels, degree of stress, and initial Cr status (Spears, 2000). Regardless, there is mounting evidence that Cr may improve insulin function either through the enhanced response of insulin receptors or by increasing insulin receptor number in certain species. Insulin sends signals to organs, tissues, and muscles to absorb glucose from the blood. A relationship exists between insulin resistance and adipogenesis late in the finishing phase of growth (Radunz et al., 2012). Evidence suggests Cr supplementation may be effective during the feedlot finishing phase, because of enhanced glucose uptake by the tissues, to increase IM fat deposition and muscle hypertrophy. In addition to the effects of postnatal nutrition, prenatal energy and nutrition can affect progeny growth and development. However, there is a lack of information regarding the effects of Cr supplementation during pre- and post-natal development on dam insulin sensitivity and performance or Cr effects on growth performance, insulin sensitivity, and carcass characteristics in beef feedlot steers.

LITERATURE CITED

Anderson, R. A. 1997. Nutritional factors influencing the Glucose/Insulin system: Chromium. J of the American College of Nutrition. 16:5, 404-410.

Anderson, R. A. 1998. Chromium, Glucose Intolerance and Diabetes. J of the American College of Nutrition. 17:6, 548-555. doi:10.1080/07315724.1998.10718802.

Anderson, R. A. 2003. Chromium and Insulin resistance. Nutrition Research Reviews. 16: 267-275. doi:10.1079/NRR200366.

Aragon, V. E. F., D. S. Graca, A. L. North, G. S. Santiago, and O. J. Paula. 2000. Chromium supplementation and reproductive performance of primiparous Zebu cows kept on pasture. Arq. Bras. Vet Med Zootec. 2001:5. doi:10.1590/S0102-09352001000500019.

Arthington, J. D., L. R. Corah, J. E. Minton, T. H. Elsasser, and F. Blecha. 1997. Supplemental dietary chromium does not influence ACTH, cortisol, or immune responses in young calves inoculated with bovine herpesvirus-1. J. Anim. Sci. 75: 217-223.

Bernhard, B. C., N. C. Burdick, R. J. Rathmann, J. A. Carroll, D. N. Finck, M. A. Jennings, T. R. Young, and B. J. Johnson. 2012. Chromium supplementation alters both glucose and lipid metabolism in feedlot cattle during the receiving period. J. Anim. Sci. 90:4857-4865. doi:10.2527/jas.2011-4982.

Besong, S., J. Jackson, S. Trammell, and D. Amaral-Phillips. 1996. Effect of supplemental chromium picolinate on liver triglycerides, blood metabolites, milk yield and milk composition in early lactation cows. J. Dairy Sci. 79 (Suppl. 1):196.

Bohrer, B. M., B. M. Edenburn, D. D. Boler, A. C. Dilger, and T. L. Felix. 2014. Effect of feeding ractopamine hydrochloride (Optaflexx) with or without supplemental zinc and chromium propionate on growth performance, carcass characteristics, and meat quality of finishing steers. J. Anim. Sci. 92: 3988-3996.

Boleman, S. L., S. J. Boleman, T. D. Bidner, L. L. Southern, T. L. Ward, J. E. Pontif, and M. M. Pike. 1995. Effect of chromium picolinate on growth, body composition, and tissue accretion in pigs. J. Anim. Sci. 73:2033-2042. doi:/1995.7372033x.

Bunting, L. D., J. M. Fernandez, D. L. Thompson, Jr., and L. L. Southern. 1994. Influence of chromium picolinate on glucose usage and metabolic criteria in growing Holstein calves. J. Anim. Sci. 72:1591-1599. doi:/1994.7261591x.

Cefalu, W. T., Z. Q. Wang, X. H. Zhang, L. C. Baldor, and J. C. Russell. 2002. Oral chromium picolinate improves carbohydrate and lipid metabolism and enhances skeletal muscle Glut-4 translocation in obese, hyperinsulinemic (JCR-LA Corpulent) rats. Journal of Nutrition. 132:1107-1114.

Cefalu, W. T., and F. B. Hu. 2004. Role of Chromium in Human Health and in Diabetes. Diabetes Care. 11:2741-2751.

Chang, X., and D. N. Mowat. 1992. Supplemental chromium for stressed and growing feeder calves. J. Anim. Sci. 70:559-565. doi:/1992.702559x.

Chang, X., D. N. Mowat, and G. A. Spiers. 1992. Carcass characteristics and tissue-mineral contents of steers fed supplemental Chromium. Can. J. Anim. Sci. 72:663-669. doi:/1992.702559x.

Chang, X., D. N. Mowat, and Mallard B. A. 1995. Supplemental chromium and niacin for stressed feeder calves. Can. J of Anim. Sci. 75:351-358.

Chen, G., P. Liu, G. R. Pattar, L. Tackett, P. Bhonagiri, A. B. Strawbridge, and J. S. Elmendorf. 2006. Chromium activates glucose transporter 4 trafficking and enhances insulin-stimulated glucose transport in 3T3-L1 adipocytes via a cholesterol-dependent mechanism. Molecular Endocrinology. 20:857-870.

Davis, C. M., K. H. Sumrall, and J. B. Vincent. 1996. A biologically active form of chromium may activate a membrane Phosphotyrosine Phosphatase (PTP). Biochemistry. 35:12963-12969.

Davis, C. M. and J. B. Vincent. 1997. Chromium oligopeptide activates insulin receptor tyrosine kinase activity. Biochemistry. 36:4382-4385.

Eisemann, J. H., G. B. Huntington, and D. R. Catherman. 1997. Insulin sensitivity and responsiveness of portal-drained viscera, liver, hindquarters, and whole body of beef steers weighing 275 or 490 kilograms. J. Anim. Sci. 75:2084-2091.

Ferezou, J., and A. C. Bach. 1999. Structure and metabolic fate of triacylglycerol- and phospholipid-rich particles of commercial parenteral fat emulsions. Nutrition 15:44-50.

Fernyhough, M. E., E. Okine, G. Hausman, J. L. Vierck, and M. V. Dodson. 2007. PPARy and GLUT-4 expression as developmental regulators/markers for preadipocyte differentiation into an adipocyte. Dom. Animal Edocrinology 33:367-378.

Godfrey, K. M., and D. J. P. Barker. 2001. Fetal programming and adult health. Public Health Nutrition. 4:611-624.

Hayirli, A., D. R. Bremmer, S. J. Bertics, M. T. Socha, and R. R. Grummer. 2001. Effect of Chromium supplementation on production and metabolic parameters in periparturient dairy cows. J Dairy Sci. 84:1218-1230. doi:10.3168/jds.S0022-0302(01)74583-3.

Jackson, A. R., S. Powell, S. L. Johnston, J. O. Matthews, T. D. Bidner, F. R. Valdez, and L. J. Southern. 2009. The effect of chromium as chromium propionate on growth performance, carcass traits, meat quality, and the fatty acid profile of fat from pigs fed no

supplemented dietary fat, choice white grease, or tallow. J. Anim. Sci. 87:4032-4041. doi:10.2527/jas.2009-2168.

Jeejeebhoy, K. N., R. C. Chu, E. B. Marliss, G. R. Greenburg, and A. Bruce-Robertson. 1977. Chromium deficiency, glucose intolerance, and neuropathy reversed by chromium supplementation in a patient receiving long-term total parenteral nutrition. Am J Clin Nutr. 30:531-538.

Jeremiah, L. E. 1996. The influence of subcutaneous fat thickness and marbling on beef: Palatability and consumer acceptability. Food Research International. 29:513-520.

Kawachi, H. 2006. Micronutrients affecting adipogenesis in beef cattle. Animal Science Journal 77:463-471.

Kegley, E. B. and J. W. Spears. 1995. Immune response, glucose metabolism, and performance of stressed feeder calves fed inorganic or organic chromium. J. Anim. Sci. 73: 2721-2726. doi:/1995.7392721x.

Kegley, E. B., J. W. Spears, and T. T. Brown, Jr. 1996. Immune response and disease resistance of calves fed chromium nicotinic acid complex or chromium chloride. J. Dairy Sci. 79:1278-1283.

Kegley, E. B., J. W. Spears, and T.T. Brown, Jr. 1997. Effect of shipping and Chromium supplementation on performance, immune response, and disease resistance of steers. J. Anim. Sci. 75:1956-1964. doi:/1997.7571956x.

Kegley, E. B., D. L. Galloway, and T. M. Fakler. 2000. Effect of dietary chromium-L-methionine on glucose metabolism in beef steers. J. Anim. Sci. 78:3177-3183. doi: /2000.78123177x.

Kornegay, E. T., Z. Wang, C. M. Wood, and M. D. Lindemann. 1997. Supplemental chromium picolinate influences nitrogen balance, dry matter digestibility, and carcass traits in growing-finishing pigs. J. Anim. Sci. 75:1319-1323. doi:/1997.7551319x.

Larson, D. M., J. L. Martin, D. C. Adams, and R. N. Funston. 2009. Winter grazing system and supplementation during late gestation influence performance of beef cows and steer progeny. J. Anim. Sci. 87:1147-1155.

Le Roith, D., and Y. Zick. 2001. Recent advances in our understanding of insulin action and insulin resistance. Diabetes Care. 24: 588-597.

Leiva, T., R. F. Cooke, A. C. Aboin, F. L. Drago, R. Gennari, and J. L. M. Vasconcelos. 2014. Effects of excessive energy intake and supplementation with chromium propionate on insulin resistance parameters in nonlactating dairy cows. J. Anim. Sci. 92:775-782. doi:10.2527/jas.2013-6852.

Lewis, G. F., A. Carpentier, K. Adehi, and A. Giacca. 2002. Disordered fat storage and mobilization in the pathogenesis of insulin resistance and type 2 diabetes. Endocr. Rev. 23: 201-229.

Lindell, S. A., R. T. Brandt Jr., J. E. Milton, F. Blecha, G. L. Stokka, and C. T. Milton. 1994. Supplemental Cr and revaccination effects on performance and health of newly weaned calves. J. Anim. Sci. 72, Suppl. 1, 133.

Lindemann, M. D., C. M. Wood, A. F. Harper, E. T. Kornegay, and R. A. Anderson. 1995. Dietary chromium picolinate additions improve gain:feed and carcass characteristics in growing-finishing pigs and increase litter size in reproducing sows. J. Anim. Sci. 73:457-465.

Mathison, G. M. and D. F. Engstrom. 1995. Chromium and protein supplements for growing-finishing beef steers fed barley-based diets. Can. J. of Anim. Sci. 75:549–558. doi:10.4141/cjas95-083.

Matthews, J. O., L. L. Southern, J. M. Fernandez, J. E. Pontif, T. D. Bidner, and R. L. Odgaard. 2001. Effect of chromium picolinate and chromium propionate on glucose and insulin kinetics of growing barrows and on growth and carcass traits of growing-finishing barrows. J. Anim. Sci. 79:2172-2178.

Matthews, J. O., A. D. Higbie, L. L. Southern, D. F. Coombs, T. D. Bidner, and R. L. Odgaard. 2003. Effect of chromium propionate and metabolizable energy on growth, carcass traits, and pork quality of growing-finishing pigs. J. Anim. Sci. 81:191-196. doi:/2003.811191x.

McNamara, J. P. and F. Valdez. 2005. Adipose tissue metabolism and production responses to calcium propionate and chromium propionate. J. Dairy Sci. 88:2498-2507. doi:10.3168/jds.S0022-0302(05)72927-1.

Mertz, W., E. W. Toepfer, E. E. Roginski, and M. M. Polansky. 1974. Present knowledge of the role of Cr. Fed. Proc. 33:2275.

Mooney, K. W. and G. L. Cromwell. 1997. Efficacy of chromium picolinate and chromium chloride as potential carcass modifiers in swine. J. Anim. Sci. 75:2661-2671.

Moonsie-Shageer, S. and D. N. Mowat. 1993. Effect of level of supplemental chromium on performance, serum constituents, and immune status of stressed feeder calves. J. Anim. Sci. 71:232-238. doi:/1993.711232x.

Morris, B. W., A. Blumsohn, S. MacNeil, and T. A. Gray. 1992. The trace element chromium – a role in glucose homeostasis. Am. J. of Clinical Nutrition. 55:989–991.

Mowat, D. N., X. Chang, and W. Z. Yang. 1993. Chelate chromium for stressed feeder calves. Can. J. Anim Sci. 73:49-55. doi:10.4141/cjas93-004.

NRC. 2000. Nutrient Requirements of Beef Cattle. 7th Rev. Ed., 1996. Natl. Acad. Press, Washington, DC.

NRC. 2005. Mineral Tolerance of Animals. 2nd rev. ed. Natl. Acad. Press, Washington, DC.

NRC. 2012. Nutrient Requirements of Swine. 11th Rev. Ed., 2012. Natl. Acad. Press, Washington, DC.

Padmavathi, J. N. I., K. R. Rao, L. Venu, A. Ismail, and M. Raghunath. 2010. Maternal dietary chromium restriction programs muscle development and function in the rat offspring. Exp. Biology and Medicine. 235:349-355. doi:10.1258/ebm.2009.009199.

Page, T. G., L. L. Southern, T. L. Ward, and D. L. Thompson, Jr. 1993. Effect of chromium picolinate on growth and serum and carcass traits of growing-finishing pigs. J. Anim. Sci. 71:656-662. doi:/1993.713656x.

Pilch, P. F., P. A. Thompson, and M. P. Czech. 1980. Coordinate modulation of D-glucose transport activity and bilayer fluidity in plasma membranes derived from control and insulin-treated adipocytes. Proc Natl Acad Sci USA. 77: 915-918.

Pires, J. A. A., A. H. Souza, and R. R. Grummer. 2007. Induction of hyperlipidemia and intravenous infusion of tallow emulsion causes insulin resistance in Holstein cows. J. Dairy Sci. 90:2735-2744.

Pollard, G. V., C. R. Richardson, and T. P. Karnezos. 2002. Effects of supplemental organic chromium on growth, feed efficiency and carcass characteristics of feedlot steers. Animal Feed Science and Technology. 98:121-128. doi:10.1016/S0377-8401(02)00010-X.

Potter, J. F., P. Levin, R. A. Anderson, J. M. Freiberg, R. Andres, and D. Elahi. 1985. Glucose metabolism in glucose-intolerant older people during chromium supplementation. Metabolism 34:199-204.

Prior, R. L., and R. A. Scott. 1980. Effects of intravenous infusion of glucose, lactate, propionate or acetate on the induction of lipogenesis in bovine adipose tissue. J. Nutr. 110:2011-2019.

Radunz, A. E., F. L. Fluharty, M. L. Day, H. N. Zerby, and S. C. Loerch. 2010. Prepartum dietary energy source fed to beef cows: I. Effects on pre-and postpartum cow performance. J. Anim. Sci. 88:2717-2728. doi:10.2527/jas.2009-2744.

Radunz, A. E., F. L. Fluharty, A. E. Relling, T. L. Felix, L. M. Shoup, H. N. Zerby, and S. C. Loerch. 2012. Prepartum dietary energy source fed to beef cows: II. Effects on progeny postnatal growth, glucose tolerance, and carcass composition. J. Anim. Sci. 90:4962-4974. doi: 10.2527/jas.2012-5098.

Rehfeldt, C., N. C. Strickland, I. Fiedler, and J. Wegner. 1999. Environmental and genetic factors as sources of variation in skeletal muscle fibre number. Basic Appl. Myol. 9:235-253.

Saltiel, A. R. 2012. Insulin resistance in the defense against obesity. Cell Metab. 15:798-804.

Savell, J. W., H. R. Cross, J. J. Francis, J. W. Wise, D. S. Hale, D. L. Wilkes, and G. C. Smith. 1989. National Consumer Retail Beef Study: interaction of trim level, price and grade on consumer acceptance of beef steaks and roasts. Journal of Food Quality. 12:251-274.

Schoonmaker, J. P., M. J. Cecava, F. L. Fluharty, H. N. Zerby, and S. C. Loerch. 2004. Effect of source and amount of energy and rate of growth in the growing phase on performance and carcass characteristics of early- and normal-weaned steers. J. Anim. Sci. 82:273-282.

Shulman, G. I. 2000. Cellular mechanisms of insulin resistance. J. Clinical Invest. 106:171-176.

Smith, K. L., M. R. Waldron, J. K. Drackley, M. T. Socha, and T. R. Overton. 2005. Performance of dairy cows as affected by prepartum dietary carbohydrate source and supplementation of chromium throughout the transition period. J Dairy Sci. 88:255-263. doi:10.3168/jds.S0022-0302(05)72683-7.

Spears, J. W. 2000. Micronutrients and immune function in cattle. Proceedings of the Nutrition Society. 59:587-594.

Spears, J. W., C. S. Whisnant, G. B. Huntington, K. E. Lloyd, R. S. Fry, K. Krafka, A. Lamptey, and J. Hyda. 2012. Chromium Propionate enhances insulin sensitivity in growing cattle. J. Dairy Sci. 95:2037-2045. doi:10.3168/jds.2011-4845.

Spears, J. W. and W. P. Weiss. 2014. Invited Review: Mineral and vitamin nutrition in ruminants. Prof. Anim. Sci. 30:180-191.

Stahlhut, H. S., C. S. Whisnant, K. E. Lloyd, E. J. Baird, L. R. Legleiter, S. L. Hansen, and J. W. Spears. 2006a. Effect of chromium supplementation and copper status on glucose and lipid metabolism in Angus and Simmental beef cows. Animal Feed Science and Tech. 128:253-265. doi:10.1016/j.anifeedsci.2005.11.002.

Stahlhut, H. S., C. S. Whisnant, and J. W. Spears. 2006b. Effects of chromium supplementation and copper status on performance and reproduction of beef cows. Animal Feed Science and Tech. 128:266-275. doi:10.1016/j.anifeedsci.2005.11.003.

Sumner, J. M., F. Valdez and J. P. McNamara. 2007. Effects of chromium propionate on response to an intravenous glucose tolerance test in growing Holstein heifers. J. Dairy Sci. 90:3467-3474. doi:10.3168/jds.2006-623.

Tatum, J. D., K. E. Belk, T. G. Field, J. A. Scanga, and G. C. Smith. 2007. Relative Importance of Weight, Quality Grade, and Yield Grade as Drivers of Beef Carcass Value in Two Grid Pricing Systems. The Professional Animal Scientist. 22:41-47.

Vernon, R. G. 1980. Lipid metabolism in the adipose tissue of ruminant animals. Prog. Lipid Res. 19:23-106.

Vincent, J. B. 1999. Mechanisms of Chromium Action: Low-Molecular-Weight Chromium-Binding Substance. J. of the Am. College of Nutrition. 18:1, 6-12. doi:10.1080/07315724.1999.10718821.

Yamamoto, A., O. Wada, and S. Manabe. 1989. Evidence that chromium is an essential factor for biological activity of low-molecular-weight, chromium-binding substance. Biochemical and Biophysical Research Communications. 163:1, 189-193.

CHAPTER 2: EFFECTS OF CHROMIUM SUPPLEMENTATION TO BEEF COWS DURING GESTATION ON BEEF COW PERFORMANCE AND PROGENY DEVELOPMENT PRE-WEANING

ABSTRACT

Angus-cross cows (n = 66) were fed 1 of 2 supplements through mid- and late-gestation: (1) 1.81 kg corn/hd·d^{-1} as fed (**Control**), or (2) 1.81 kg corn fortified with 3 mg Cr/hd·d^{-1} as fed (**CrP**). Chromium-propionate supplementation did not affect ($P \geq 0.24$) pre- or postpartum cow BW, BCS, or BW and BCS change. There was an interaction ($P < 0.01$) of d in gestation and CrP supplementation for glucose concentrations only. During mid-gestation, plasma glucose concentrations in cows fed CrP decreased 0.282 mmol/L compared to cows fed Control; however, by late-gestation, glucose concentrations in cows fed CrP increased 0.321 mmol/L compared to cows fed Control. Supplementation with CrP did not affect ($P \geq 0.58$) mean insulin or insulin:glucose; however, insulin and glucose concentrations were reduced ($P <0.01$) as d in gestation increased, regardless of CrP supplementation. There was no effect of dam treatment on calf BW at birth ($P = 0.40$) or weaning ($P = 0.56$). Supplementation of CrP did not affect ($P \geq 0.20$) 24-h milk weight in mid- or late lactation. Milk composition was not affected ($P \geq 0.25$) by CrP at mid-lactation. In late lactation, cows fed CrP had greater ($P = 0.01$) milk urea nitrogen compared to cows fed Control, with no other differences observed ($P \geq 0.23$). Supplemental CrP did not affect cow BW, BCS, milk production, or calf birth and weaning BW. Regardless of treatment, as cows neared parturition, plasma glucose and insulin concentrations of cows decreased.

Key words: beef, chromium, cows, fetal programming

INTRODUCTION

Adequate dam nutrition is critical for proper fetal development and has lasting effects on progeny development later in life (Barker, 1997). During gestation, nutrient deficiencies in dams are common, particularly in minerals, because of nutrient allocation to fetal development (Anderson, 1998). Chromium is an important trace mineral in animal nutrition because of its role in carbohydrate and lipid metabolism (Vincent, 1999). Chromium supplementation has been shown to improve immune response, decrease morbidity, and improve ADG and DMI in transported, newly received calves (Mowat et al., 1993; Moonsie-Shageer and Mowat, 1993; Kegley and Spears, 1995; Kegley et al., 1996). In addition, Cr supplementation increased insulin function and glucose metabolism in newly received steer calves (Kegley et al., 2000), growing heifers (Spears et al., 2012), and mature dairy cows (Leiva et al., 2014), and increased ADG and DMI in young calves (Moonsie-Shageer and Mowat, 1993; Kegley et al., 1997). Furthermore, feeding Cr during gestation reduced postpartum BW loss in 2 and 3 yr old cows (Stahlhut et al., 2006b) and increased postpartum BW gain (Aragon et al., 2001). In dairy cows, Cr increased milk production (McNamara and Valdez, 2005; Smith et al., 2005). However, limited research has been conducted on the effects of prepartum Cr supplementation on beef cow BW, BCS, or milk production. We hypothesized that Cr would improve insulin and glucose metabolism during mid- and late-gestation and, therefore, improve cow BW and BCS and increase milk production. In turn, this improvement in glucose and insulin sensitivity would increase calf BW at weaning. Therefore, objectives of this experiment were to determine the effects of gestational Cr supplementation to beef cows on glucose and insulin concentrations, cow BW and BCS, milk production, and the effects on resulting progeny birth and weaning BW.

MATERIALS AND METHODS

All animal procedures in this experiment followed guidelines recommended in the Guide for the Care and Use of Agricultural Animals in Agricultural Research and Teaching (FASS, 2010).

Animal and Diet Management

Angus-cross cows (n = 66; primiparous, n = 18; multiparous, n = 48; age = 2 to 13 yr) were used in an experiment at the Eastern Agricultural Research Station (EARS) in Belle Valley, OH, beginning on October 31, 2012 (164 ± 13 d prepartum). Cows were bred to 1 of 5 Angus bulls. Cows were stratified by age and randomly assigned to 1 of 2 treatments: (1) 1.81 kg pelleted corn/hd·d^{-1} as fed (**Control**), or (2) 1.81 kg pelleted corn fortified with 3 mg Cr/hd·d^{-1} as fed (**CrP**, supplied as Cr propionate; KemTRACE Cr, Kemin Industries Inc., Des Moines, IA). Treatments were fed for 133 d, from 164 ± 13 d to 32 ± 13 d prepartum (until March 12, 2013). Concentrations of Cr in the corn ranged from 0.5 mg/kg to 1.6 mg/kg.

Cows were managed as 2 separate groups throughout the trial and were grazed on mixed grass-legume pasture until fall forage was depleted in late November. Cows in each treatment group were rotated among pastures 3 times per wk to eliminate any potential confounding effects of location and treatment. When forage in the pasture began to be depleted and was not in sufficient quantities to meet animal's maintenance need, round baled, mixed grass hay was offered. Hay intake was regulated by limiting access and limit-feeding when necessary to maintain acceptable BW gain and BCS throughout gestation, for approximately 4 mo. Limiting access was accomplished by allowing 6 h access to round baled hay racks Monday through Friday. On weekends, cows were allowed access for 24 h to reduce labor requirements. Cows

were offered free choice mineral supplement throughout the trial (Table 2.1). Initial cow BW were taken at 0800 (before daily supplementation) at the start of the trial (163 ± 13 d prepartum) and then approximately every 4 wk until the termination of CrP supplementation (32 ± 13 d prepartum; Figure 2.1). Final cow BW were taken 39 ± 13 d prepartum (1 wk before the end of Cr supplementation). Cow BCS was also recorded approximately every 4 wk. The same, trained Ohio State University personnel measured cow BCS throughout the trial using a whole numbered scale of 1 to 9 with 1 = all ribs visible, visible spine, no brisket fat, no tail head fat, and muscle loss; 5 = 1 to 2 visible ribs, spine not visible, no brisket fat, no tail head fat, no muscle loss; and 9 = no visible ribs, no visible spine, abundant brisket fat, abundant tail head fat, and no muscle loss. The first calf was born on March 24, 2013 and the last calves were born on May 15, for a 51 d calving season, and an average calving date of April 13. Cows were removed (n = 17) from the experiment for reasons unrelated to Cr supplementation: open or bred too late for a timely arrival (n = 7), calving difficulties/birth defects (n = 4), and not calving within the 51 d season (n = 6), leaving 66 cows (from an original 83) to be used in the experiment.

Corn and CrP supplementation ended 32 ± 13 d prepartum and all 66 cows were grouped together. Feed and hay core samples were collected monthly and composited for nutrient analysis. Composite samples were analyzed and results were used to record nutrient composition of the diets (Table 2.1). All samples were analyzed for DM (24 h at 105˚C), ADF and NDF (using Ankom Technology method 5 and 6, respectively; Ankom^{200} Fiber Analyzer, Ankom Technology, Macedon, NY), CP (method 930.15; AOAC, 1996), and fat (using Ankom Technology method 2; Ankom Technology).

To determine progeny differences, calves were weighed at birth and at weaning, approximately 6 mo of age (184 ± 13 d postpartum). Steer calves were castrated and vaccinated

with Alpha 7-MB-1® (Boehringer Ingelheim, Germany) and Leptoferm-5® (Zoetis, Flroham Park, NJ) on May 23, 2013. On July 22, 2013, 12 wk before weaning, calves received Bovi-Shield® Gold FP 5L5 (Zoetis, Florham Park, NJ) and Ultrabac 7/Somubac® (Pfizer Animal Health, New York City, NY). On August 22, approximately 7 wk before weaning, calves received Bovi-Shield® Gold FP 5L5 booster, Ultrabac 7®/Somubac® booster, and One Shot Ultra® (Zoetis, Florham Park, NJ).

Sampling and Analysis

Cows were bled from the jugular vein (K_2 EDTA Vacutainer tubes, REF 366643, BD Vacutainer ®, Franklin Lakes, NJ) for plasma insulin and glucose determination 4 h after supplementation on 3 separate d: 163 ± 13, 95 ± 13, and 39 ± 13 d prepartum. Blood samples were placed on wet ice for no longer than 30 min and then centrifuged at 3,000 × g for 20 min at 4°C. Plasma was aliquoted into 2-mL tubes in duplicate and placed in a -80°C freezer until analysis. Plasma glucose concentrations were determined via colorimetric assay (Glucose Liquicolor; Procedure No. 1070, Stanbio Laboratory, Boerne, TX) following the kit instructions. Plasma concentrations of insulin were determined by ELISA (Mercodia Bovine Insulin ELISA; Mercodia, Sweden) following the instructions provided and using a pooled-sample control. Glucose and insulin absorbance was measured at 500 nm and 450 nm, respectively using a plate reader (Syndergy HT, BioTek, Winooski, VT). Results for glucose and insulin analysis were deemed acceptable at a coefficient of variation (**CV**) of ≤ 5% and ≤ 10%, respectively, within duplicates.

Glucose and insulin values were converted from traditional units to the International System of Units (**SI**). Glucose was converted using the formula mg/dL = 0.05551 mmol/L

(Young, 1987). Insulin was converted using the molecular weight of bovine insulin, 5,734 g/mol (Darby et al., 2001), and subsequently, a final conversion rate of 1 ng/mL = 174.398 pmol/L was used.

Cow milk production was measured by performing a weigh-suckle-weigh procedure (**WSW**; Radunz et al., 2010) in mid-lactation (93 ± 13 d postpartum) and late lactation (170 ± 13 d postpartum). During the WSW, calves were separated from their dams for 3 h. During that time, a milk sample (approximately 50 mL) was collected by hand from each cow, strained through cheese cloth to remove any debris and added to a DHIA tube treated with bronopol and natamycin as preservatives. Tubes were stored on wet ice until sent to a local commercial laboratory (DHI Cooperative, Inc., Columbus, OH) to be analyzed for composition of fat, crude protein, lactose, and milk urea nitrogen as described by Beckman and Weiss (2005). After the 3 h separation, calves were allowed to nurse their mothers for 15 to 20 min, or until calves lost interest, and were separated again for 12 h. The next mornings, on d 94 and 171, empty calf BW was collected. After weighing, calves were allowed to nurse their mothers to completion and then calves were reweighed. Milk production was calculated by taking the difference between the full calf BW and empty calf BW, and multiplying by 2 to represent 24-h milk weight.

Statistical Analysis

This experiment was a randomized complete block design with 2 treatments. Individual animal was the experimental unit. The milk composition and yield, BW, and BCS data were analyzed using the MIXED procedures of SAS (SAS Institute, Cary, NC). Treatment was included as a fixed effect. The covariance matrix of the fixed-effect estimates and denominator degrees of freedom for the *t* and *F* tests were determined using the Kenward and Roger

adjustment. Glucose and insulin data were analyzed using the MIXED procedures of SAS with repeated measures where fixed effects were Treatment, Day, and Treatment × Day, the covariance structure was compound symmetry, as determined by lowest Bayesian information criterion, and cow was the subject. Because insulin and glucose concentrations were significant ($P < 0.05$) at d 163 ± 13 prepartum, glucose and insulin at d 163 ± 13 prepartum were covariates (fixed effects) in the model statements. Significance was declared at $P \leq 0.05$ and trends are discussed at $0.05 < P \leq 0.10$.

RESULTS AND DISCUSSION

Supplementation of CrP did not affect ($P \geq 0.24$) initial, final, or change in cow BW or BCS during gestation (Table 2.2). There was also no carry-over effect ($P = 0.71$) of CrP supplementation on postpartum cow BW recorded at time of weaning. We hypothesized that CrP supplementation would improve cow BW and BCS at the end of gestation. At this time, cows are diverting their energy and resources to the developing fetus, a demanding situation that can lead to Cr deficiency (Anderson, 2003). Limited research has been conducted on the effects of Cr supplementation on prepartum beef cow BW and BCS. However, contrary to the current study, Stahlhut et al. (2006a,b) supplemented Cr as Cr-Picolinate to Angus and Simmental cows starting 75 d prepartum. They observed that young (2 to 3 yr old) cows supplemented with Cr lost less BW throughout the trial when compared to cows not fed Cr. However, postpartum BW loss tended to be greater for 4 and 5 yr old cows supplemented with Cr when compared to the controls in the same age block. In addition, Stahlhut et al. (2006a) supplemented Cr to cows in gestation for a shorter period of time when compared to cows on the current study. In dairy cattle, Cr supplementation before and after calving has been shown to increase postpartum BW

gain (Smith et al., 2005), increase BW loss (Yang et al., 1996), or have no effect on BW change (Hayirli et al., 2001). Thus, there is a wide variation of cow responses to Cr supplementation in the limited literature. The lack of difference shown in the current study may have resulted from all cows, regardless of whether or not they received CrP, being fed 1.81 kg of corn each day as a glucogenic precursor. Furthermore, as grazing resources were depleted, cows were supplemented with hay. Feeding this additional energy to the cows in the current experiment may have masked any potential pressure of limited energy during mid- and late-gestation, as all cows had adequate BW and BCS throughout gestation. Therefore, CrP may not have been able to elicit a response, as prior research has demonstrated that responses to Cr are more often observed in dairy cows suffering from negative energy balance (Hayirli et al., 2001; Spears and Weiss, 2008).

There was no difference in calf BW at birth ($P = 0.24$) or weaning ($P \geq 0.56$) between treatments (Table 2.2). We hypothesized that CrP supplementation would increase calf BW at weaning because prepartum Cr supplementation to primiparous dams has increased insulin efficiency and glucose utilization (Subiyatno et al., 1996). These data suggest that increased glucose utilization by the dam would increase glucose supplied to the fetus for growth and development. However, this was not the case. It is possible that potential effects of CrP were masked, as calves were still nursing their dams with ample milk available and had adequate forage throughout development. Furthermore, supplementing Cr to dams during late-gestation has shown no effect on calf BW at weaning (Aragon et al., 2001; Stahlhut et al., 2006a).

Similar calf BW at weaning may also be due to the fact there were no differences ($P \geq 0.20$) in 24-h milk weight estimated at 2 WSW events (Table 2.3). No other research has been conducted on Cr effect on milk yield or milk production in beef cows. We had hypothesized that fluid milk yield would increase because of Cr role in glucose metabolism and, in dairy cows, Cr

supplemented during gestation and early lactation has increased milk production in several studies (Yang et al., 1996; Hayirli et al., 2001; Smith et al., 2005). Chromium supplementation to cattle has been shown to increase insulin function and tissue sensitivity to insulin (Chang and Mowat, 1992; Sumner et al., 2007; Leiva et al., 2014), thereby increasing glucose metabolism and uptake by cells. Glucose is the main driver of lactose synthesis, and lactose production is linked to fluid milk yield (Linzell and Peaker, 1971; Hanigan et al., 2001).

Similar to milk yield, differences in milk composition were also limited in the present trial. During the first WSW, in mid-lactation, no differences ($P \geq 0.25$) were observed in fat, protein, somatic cell count (SCC), lactose, other solids, or milk urea nitrogen (**MUN**). However, during the second WSW, in late lactation, cows fed CrP had greater ($P = 0.01$) MUN (mg/dL) compared to cows fed Control. No other differences were observed for milk composition during late lactation ($P \geq 0.23$). The increase in MUN by CrP is difficult to explain and has not been previously observed. Protein levels in the diet normally cause differences in MUN; however, protein levels were identical for both supplementation treatments. Furthermore, CrP supplementation was terminated approximately 200 d prior to the observed increase in MUN. Nevertheless, it is important to note that MUN levels were still within the optimal range for cows being fed alfalfa hay and forage diets. In dairy cattle, pre- and postpartum Cr supplementation has failed to affect milk composition, including MUN (Yang et al., 1996; McNamara and Valdez, 2005; Smith et al., 2005).

To investigate the effect of CrP supplementation on glucose utilization, insulin and glucose concentrations were measured at the beginning, middle, and end of the CrP supplementation period. Glucose concentrations at 163 ± 13 d prepartum were used as a covariate because cows fed CrP had decreased ($P = 0.05$) plasma glucose concentration (3.455

mmol/L) compared to cows fed Control (3.705 mmol/L). In the present study, there was a Treatment × Day interaction for glucose concentrations ($P < 0.01$; Table 2.4). On d 95 ± 13 prepartum (mid-gestation), cows fed CrP had 7% lower glucose concentrations compared to cows fed Control; by 39 ± 13 d prepartum (late gestation), cows fed CrP had 11% greater glucose concentrations when compared to cows fed Control. However, the average prepartum cow blood glucose levels in the current study are within a normal range of 2.5 to 4.2 mmol/L (Kaneko, 2008), and the effect of CrP on glucose concentrations was not likely biologically relevant. Perhaps more importantly, as number of d in gestation and d on corn supplementation increased, glucose concentrations decreased ($P < 0.01$), regardless of treatment.

At 163 ± 13 d prepartum, cows fed CrP and Control had a plasma insulin concentrations of 45.5 and 49.5 pmol/L, respectively, and d 163 ± 13 used as covariate for analysis of d 95 ± 13 and 39 ± 13 prepartum. There was no interaction of Treatment × Day ($P = 0.70$), nor main effect of Treatment ($P = 0.58$), on insulin concentrations. However, similar to glucose responses, there was a main effect of Day on insulin concentrations. From d 95 ± 13 to d 39 ± 13 prepartum, insulin concentrations decreased ($P < 0.01$) in all cows, regardless of treatment. Despite the Treatment × Day interaction for glucose concentration, there was no interaction of Treatment × Day ($P = 0.62$), nor main effects Treatment ($P = 0.60$) or Day ($P = 0.14$) on insulin:glucose ratio. Vincent (1999) and Anderson (2003) defined increasing insulin sensitivity as the same amount of insulin being used to clear more glucose. In the current study, both plasma insulin and glucose concentrations decreased as d in gestation increased, and the ratio of insulin:glucose was unaffected, suggesting no changes in insulin sensitivity. Previous research is variable comparing insulin and glucose concentrations before and after parturition. Decreased insulin and glucose concentrations (Regnault et al., 2004; Stahlhut et al., 2006a) and decreased insulin sensitivity

(Debrass et al., 1989; Faulkner and Pollock, 1990; Sano et al., 1991; Hayirli et al., 2001) have been observed in ruminants when plasma samples from late gestation are compared to postpartum samples.

Furthermore, insulin concentrations in bovine can vary between 90 to 300 pmol/L (Trenkle, 1972). In pregnant beef cows, non-fasted insulin concentration have been shown to vary between 60 to 200 pmol/L and can be considered normal (Sano et al., 1991; Lents et al., 2005; Stahlhut et al., 2006a). The pregnant beef cows in the current study had lower than normal non-fasted insulin concentrations compared to previous research.

In conclusion, we hypothesized that Cr-prop supplementation would enhance insulin function and, thus, improve cow BW and BCS during gestation and improve calf BW at birth. We also hypothesized that Cr-prop would increase milk production and thereby, increase calf BW at weaning. In this study, while CrP increased glucose concentrations in mid-gestation, cows fed CrP had decreased glucose concentrations by late-gestation. Supplementation with CrP did not affect insulin or insulin:glucose. Over time, regardless of whether cows were fed CrP or not, all cows had decreased insulin and glucose concentrations a month before the average parturition date compared to earlier in gestation. Pre- and postpartum cow BW and BCS was not affected by CrP. There was no effect of CrP on cow milk yield and no effect of CrP on calf BW at birth or weaning.

IMPLICATIONS

In the present trial, supplementing cows in mid- and late-gestation with CrP did not enhance insulin sensitivity during gestation, and therefore, no positive effects of CrP supplementation on cow BW, BCS, or progeny BW at weaning were observed. The present

research would suggest no benefit of supplementing 3 mg Cr/hd·d^{-1} to spring-calving, Angus-cross cows during the last 6 mo of gestation. More research in beef cows may be necessary to quantify the effects of Cr on cow BW, milk production, insulin sensitivity, and calf growth.

LITERATURE CITED

AOAC. 1996. Official Methods of Analysis. 16th ed. Assoc. Off. Anal. Chem., Gaithersburg, MD.

Anderson, R. A. 1998. Chromium, Glucose Intolerance and Diabetes. J Am Coll Nutr. 17:548-555. doi:10.1080/07315724.1998.10718802.

Anderson, R. A. 2003. Chromium and Insulin resistance. Nutr Res Rev. 16:267-275. doi:10.1079/NRR200366.

Aragon, V. E. F., D. S. Graca, A. L. North, G. S. Santiago, and O. J. Paula. 2001. Chromium supplementation and reproductive performance of primiparous Zebu cows kept on pasture. Arq. Bras. Vet Med Zootec. 2001:5. doi:10.1590/S0102-09352001000500019.

Barker, D. J. P. 1997. Maternal nutrition, fetal nutrition, and disease later in life. J. Nutr. 13:807-813. doi:10.1016/S0899-9007(97)00193-7.

Beckman, J. L., and W. P. Weiss. 2005. Nutrient digestibility of diets with different fiber to starch ratios when fed to lactating dairy cows. J. Dairy Sci. 88:1015-1023. doi:10.3168/jds.S0022-0302(05)72769-7.

Chang, X., and D. N. Mowat. 1992. Supplemental chromium for stressed and growing feeder calves. J. Anim. Sci. 70:559-565. doi:/1992.702559x.

Darby, S. M., M. L. Miller, R. O. Allen, and M. LeBeau. 2001. A mass spectrometric method for quantification of intact insulin in blood samples. J. of Analyt. Toxico. 25:8-14. doi:10.1093/jat/25.1.8.

Debras, E., J. Grizard, E. Aina, S. Tesseraud, C. Champredon, and M. Arnal. 1989. Insulin sensitivity and responsiveness during lactation and dry period in goats. Am. J. Physiol. 256:295-302. doi:/0193-1849/89.

FASS, Federation of Animal Science Societies. 2010. Guide for the care and use of agricultural animals in research and teaching. 3rd ed.

Faulkner, A., and H. T. Pollock. 1990. Metabolic responses to euglycaemic hyperinsulinaemia in lactating and non-lactating sheep *in vivo*. J. of Endocrinol. 124:59-66. doi:/0022-0795/90/0124-0059.

Hanigan, M. D., L. A. Crompton, J. A. Metcalf, and J. France. 2001. Modelling mammary metabolism in the dairy cow to predict milk constituent yield, with emphasis on amino acid metabolism and milk protein production: Model construction. J. Theor. Biol. 213:223-239. doi:10.1006/jtbi.2001.2417.

Hayirli, A., D. R. Bremmer, S. J. Bertics, M. T. Socha, and R. R. Grummer. 2001. Effect of Chromium supplementation on production and metabolic parameters in periparturient dairy cows. J. Dairy Sci. 84:1218-1230. doi:10.3168/jds.S0022-0302(01)74583-3.

Kaneko, J. J. 2008. Carbohydrate Metabolism and Its Diseases. In: J. J. Kaneko, J. W. Harvey, and M. L. Bruss, editors, Clinical Biochemistry of Domestic Animals. 6th ed. Elsevier, Inc. p. 45-81.

Kegley, E. B. and J. W. Spears. 1995. Immune response, glucose metabolism, and performance of stressed feeder calves fed inorganic or organic chromium. J. Anim. Sci. 73:2721-2726. doi:/1995.7392721x.

Kegley, E. B., J. W. Spears, and T. T. Brown, Jr. 1996. Immune response and disease resistance of calves fed chromium nicotinic acid complex or chromium chloride. J. Dairy Sci. 79:1278-1283.

Kegley, E. B., J. W. Spears, and T.T. Brown, Jr. 1997. Effect of shipping and Chromium supplementation on performance, immune response, and disease resistance of steers. J. Anim. Sci. 75:1956-1964. doi:/1997.7571956x.

Kegley, E. B., D. L. Galloway, and T. M. Fakler. 2000. Effect of dietary chromium-L-methionine on glucose metabolism in beef steers. J. Anim. Sci. 78:3177-3183. doi: /2000.78123177x.

Leiva, T., R. F. Cooke, A. C. Aboin, F. L. Drago, R. Gennari, and J. L. M. Vasconcelos. 2014. Effects of excessive energy intake and supplementation with chromium propionate on insulin resistance parameters in nonlactating dairy cows. J. Anim. Sci. 92:775-782. doi:10.2527/jas.2013-6852.

Lents, C. A., R. P. Wettemann, F. J. White, I. Rubio, N. H. Ciccioli, L. J. Spicer, D. H. Keisler, and M. E. Payton. 2005. Influence of nutrient intake and body fat on concentrations of insulin-like growth factor-I, insulin, thyroxine, and leptin in plasma of gestating beef cows. J. Anim. Sci. 83:586-596. doi:/2005.833586x.

Linzell, J. L., and M. Peaker. 1971. Mechanisms of milk secretion. Physiol. Rev. 51:564-597.

McNamara, J. P. and F. Valdez. 2005. Adipose tissue metabolism and production responses to calcium propionate and chromium propionate. J. Dairy Sci. 88:2498-2507. doi:10.3168/jds.S0022-0302(05)72927-1.

Moonsie-Shageer, S. and D. N. Mowat. 1993. Effect of level of supplemental chromium on performance, serum constituents, and immune status of stressed feeder calves. J. Anim. Sci. 71:232-238. doi:/1993.711232x.

Mowat, D. N., X. Chang, and W. Z. Yang. 1993. Chelate chromium for stressed feeder calves. Can. J. Anim Sci. 73:49-55. doi:10.4141/cjas93-004.

Radunz, A. E., F. L. Fluharty, M. L. Day, H. N. Zerby, and S. C. Loerch. 2010. Prepartum dietary energy source fed to beef cows: I. Effects on pre-and postpartum cow performance. J. Anim. Sci. 88:2717-2728. doi:10.2527/jas.2009-2744.

Regnault, T. R. H., H. V. Oddy, C. Nancarrow, N. Sriskandarajah, and R. J. Scaramuzzi. 2004. Glucose-stimulated insulin response in pregnant sheep following acute suppression of plasma non-esterified fatty acid concentrations. Reprod. Biol. Endocrinol. 2:64-74. doi:10.1186/1477-7827-2-64

Sano, H., M. Nakai, T. Kondo, and Y. Terashima. 1991. Insulin responsiveness to glucose and tissue responsiveness to insulin in lactating, pregnant, and nonpregnant, nonlactating, beef cows. J. Anim. Sci. 69:1122-1127. doi:/1991.6931122x.

Smith, K. L., M. R. Waldron, J. K. Drackley, M. T. Socha, and T. R. Overton. 2005. Performance of dairy cows as affected by prepartum dietary carbohydrate source and supplementation of chromium throughout the transition period. J Dairy Sci. 88:255-263. doi:10.3168/jds.S0022-0302(05)72683-7.

Spears, J. W., and W. P. Weiss. 2008. Role of antioxidants and trace elements in health and immunity of transition dairy cows. Vet. J. 176:70-76.

Spears, J. W., C. S. Whisnant, G. B. Huntington, K. E. Lloyd, R. S. Fry, K. Krafka, A. Lamptey, and J. Hyda. 2012. Chromium Propionate enhances insulin sensitivity in growing cattle. J Dairy Sci. 95:2037-2045. doi:10.3168/jds.2011-4845.

Stahlhut, H. S., C. S. Whisnant, K. E. Lloyd, E. J. Baird, L. R. Legleiter, S. L. Hansen, and J. W. Spears. 2006a. Effect of chromium supplementation and copper status on glucose and lipid metabolism in Angus and Simmental beef cows. Anim. Feed Sci. Technol. 128:253-265. doi:10.1016/j.anifeedsci.2005.11.002.

Stahlhut, H. S., C. S. Whisnant, and J. W. Spears. 2006b. Effects of chromium supplementation and copper status on performance and reproduction of beef cows. Anim. Feed Sci. Technol. 128:266-275. doi:10.1016/j.anifeedsci.2005.11.003.

Subiyatno, A., D. N. Mowat, and W. Z. Yang. 1996. Metabolite and hormonal responses to glucose or propionate infusions in periparturient dairy cows supplemented with chromium. J. Dairy Sci. 79:1436-1445. doi:10.3168/jds.S0022-0302(96)76502-5.

Sumner, J. M., F. Valdez and J. P. McNamara. 2007. Effects of chromium propionate on response to an intravenous glucose tolerance test in growing Holstein heifers. J. Dairy Sci. 90:3467-3474. doi:10.3168/jds.2006-623.

Trenkle, A. 1972. Radioimmunoassay of plasma hormones: Review of plasma insulin in ruminants. J. Dairy Sci. 55:1200-1211. doi:10.3168/jds.S0022-0302(72)85648-0.

Vincent, J. B. 1999. Mechanisms of Chromium Action: Low-Molecular-Weight Chromium-Binding Substance. J. Am. Coll. Nutr. 18:1, 6-12. doi:10.1080/07315724.1999.10718821.

Yang, W. Z., D. N. Mowat, A. Subiyatno, and R. M. Liptrap. 1996. Effects of chromium supplementation on early lactation performance of Holstein cows. Can. J. Anim. Sci. 76:221-230. doi:10.4141/cjas96-034.

Young, D. S. 1987. Implementation of SI units for clinical laboratory analysis. Annals of Int. Med. 106:114-129.

TABLES AND FIGURES

Table 2.1. Composition of supplementation to cows

Item	Supplement[1]		Hay[3]
	Control	CrP[2]	
Corn	100	99.96	-
Cr-Prop	-	0.04	-
Mixed grass hay	-	-	100
Analyzed nutrient content, % DM			
ADF	7.26	6.36	42.82
NDF	15.02	16.07	69.21
CP	10.26	9.30	7.38
Ether extract	5.01	5.50	0.94

[1]All cows had access to free choice mineral containing 29.5% white salt, 25% Dicalcium Phosphate, 25% Magnesium Oxide, 10% limestone, 10% ground corn, and 3.5% trace mineral package (included: 98% NaCl; 0.35% Zn, as ZnO; 0.28% Mn, as MnO_2; 0.175% Fe, as $FeCO_3$; 0.035% Cu, as Cu_2O; 0.007% I, as $Ca_5(IO_6)_2$; and 0.007% Co, as $CoCO_3$).

[2]Cows supplemented with Chromium (CrP) received 1.81 kg corn/hd·d^{-1} to provide 3 mg Cr as Cr-propionate (KemTRACE Cr; Kemin Industries Inc., Des Moines, IA); supplementation began 10/31/12 and ended 3/12/13. Control cows were fed 1.81 kg corn/hd·d^{-1} without additional Cr.

[3]All cows were supplemented with hay on an as-needed basis during the winter months; core samples were taken at the start of the trial (10/31/12) to determine nutrient content.

Table 2.2. Effects of CrP supplementation on cow performance

Item	Control	CrP[1]	SEM	*P*-value
Animals, n	30	36	--	--
BW				
Initial, kg[2]	511	506	19	0.71
Final, kg[3]	612	601	21	0.42
Change, kg	101	95	9	0.24
BCS[4]				
Initial[2]	5.1	5.1	0.1	0.76
Final[3]	5.6	5.5	0.1	0.31
Change	0.5	0.4	0.1	0.67
Calf Birth BW, kg	36	35	2	0.40
BW at Weaning[5]				
Cow, kg	533	529	19	0.71
Calf, kg	202	199	10	0.56

[1]Cows supplemented with Chromium (CrP) received 1.81 kg corn/hd·d^{-1} to provide 3 mg Cr as Cr-propionate (KemTRACE Cr; Kemin Industries Inc., Des Moines, IA); supplementation began 10/31/12 and ended 3/12/13. Control cows were fed 1.81 kg corn/hd·d^{-1} without additional Cr.

[2]Taken 163 ± 13 d prepartum (11/1/12)

[3]Taken 39 ± 13 d prepartum (3/5/13)

[4]On 1 to 9 scale

[5]10/14/13; calf age = 184 ± 13 d

Table 2.3. Carry over effects of CrP supplementation on milk production and milk composition as determined via weigh-suckle-weigh

Item	Control	CrP[1]	SEM	*P*-value
Animals, n	30	36	--	--
93 to 94 ± 13 d postpartum[2]				
24-h milk weight, kg	9.0	10.2	0.7	0.20
Fat, %	4.33	4.11	0.33	0.62
Protein, %	2.70	2.76	0.34	0.25
SCC, cells/mL[3]	313	294	143	0.92
Lactose, %	4.92	4.94	0.04	0.65
Other Solids, %	5.85	5.88	0.05	0.68
MUN, mg/dL[4]	16.29	16.23	0.44	0.92
170 to 171 ± 13 d postpartum[5]				
24-h milk weight, kg	6.1	6.0	0.5	0.89
Fat, %	5.40	4.83	0.35	0.23
Protein,%	3.21	3.32	0.08	0.32
SCC, cells/mL	301	351	152	0.81
Lactose,%	4.71	4.69	0.04	0.58
Other Solids,%	5.62	5.61	0.04	0.80
MUN, mg/dL	14.59	16.06	0.41	0.01

[1]Cows supplemented with CrP during gestation received 1.81 kg corn/hd·d^{-1} to provide 3 mg Cr as Cr-propionate (KemTRACE Cr; Kemin Industries Inc., Des Moines, IA); supplementation began 10/31/12 and ended 3/12/13. Control cows were fed 1.81 kg corn/hd·d^{-1} without additional Cr during gestation.

[2]Weigh-Suckle-Weigh procedure conducted on July 15 to 16, 2013

[3]SCC = Somatic Cell Count

[4]MUN = Milk Urea Nitrogen

[5] Weigh-Suckle-Weigh procedure conducted on Sept 30 to Oct 1, 2013

Table 2.4. Effects of CrP supplementation on prepartum cow glucose and insulin concentrations

				P-value[2]		
Item	Control	CrP[1]	SEM	T	D	T × D
Animals, n	30	36	--	--	--	--
Glucose, mmol/L[3]			0.074	0.78	<0.01	<0.01
95 ± 13 d prepartum	3.792	3.510		<0.01		
39 ± 13 d prepartum	3.006	3.327		<0.01		
Insulin, pmol/L			4.32	0.58	<0.01	0.70
95 ± 13 d prepartum	47.52	48.23				
39 ± 13 d prepartum	33.67	37.46				
Insulin:Glucose			1.26	0.60	0.14	0.62
95 ± 13 d prepartum	12.44	13.65				
39 ± 13 d prepartum	11.29	11.32				

[1]Cows supplemented with CrP received 1.81 kg corn/hd·d^{-1} to provide 3 mg Cr as Cr-propionate (KemTRACE Cr; Kemin Industries Inc., Des Moines, IA); supplementation began 10/31/12 and ended 3/12/13. Control cows were fed 1.81 kg corn/hd·d^{-1} without additional Cr.

[2]Where T = the main effect of treatment, D = the main effect of day, and T × D = the interaction of treatment × day. When an interaction occurred ($P \leq 0.05$), the SLICE option (SAS Inst. Inc., Cary, NC) was used to compare treatments at each day. SEM shown is associated with T × D interaction.

[3]Blood samples were taken prepartum at d 163 ± 13 (11/1/12), d 95 ± 13 (1/8/13), and d 39 ± 13 (3/5/13) to be analyzed for glucose and insulin concentrations. Glucose concentrations differed ($P = 0.05$) at d 163 ± 13 prepartum; thus, this initial time point was used as a covariate.

Figure 2.1. Timeline of cow CrP supplementation

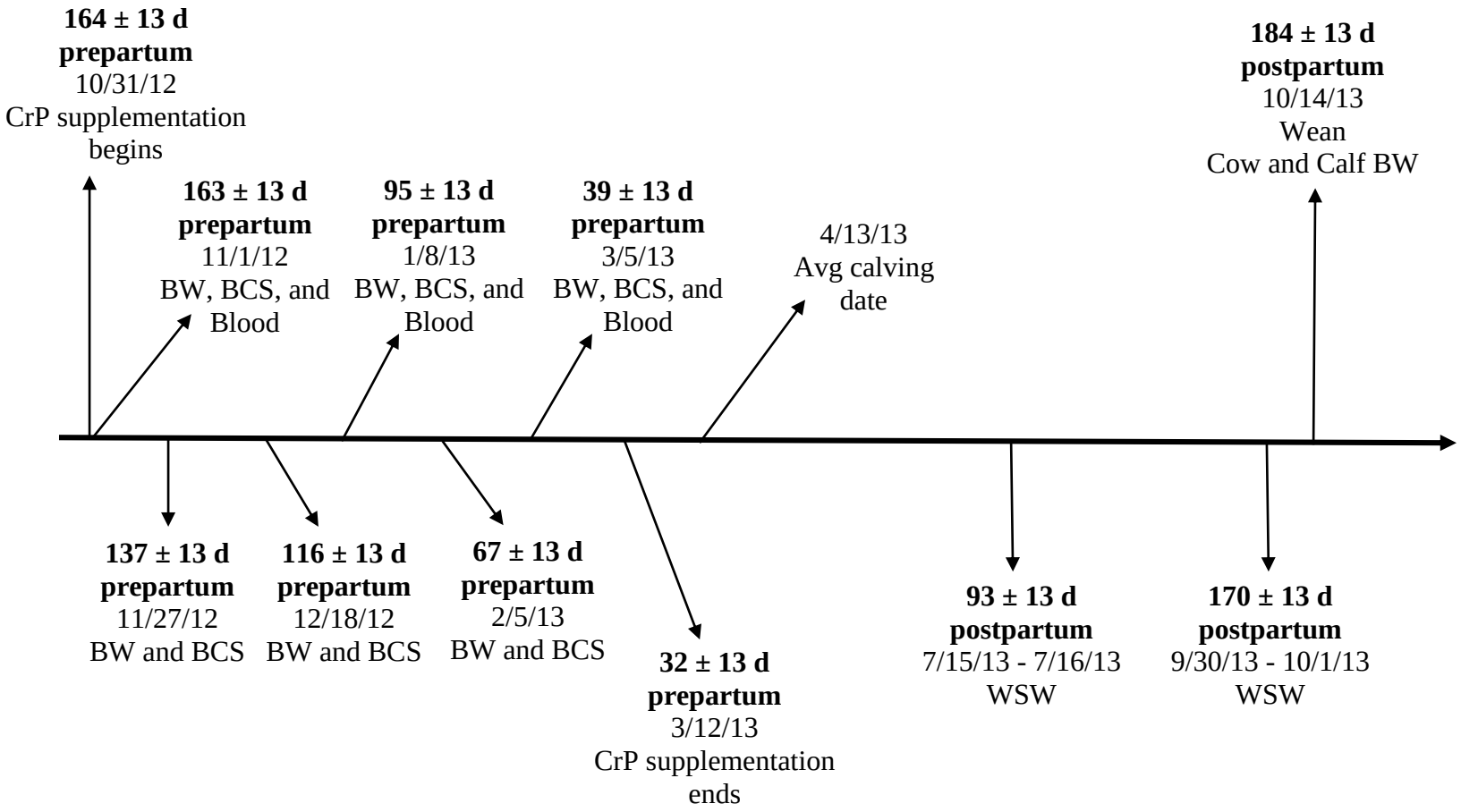

Figure 2.1. (Cont.) Timeline of cow CrP supplementation. Chromium-propionate supplementation to cows began 164 ± 13 d prepartum. Cow BW and BCS was taken approximately every 4 wk until CrP supplementation ended 32 ± 13 d prepartum. Blood samples were collected every 2 mo during mid- and late gestation for plasma insulin and glucose determination. The first calf was born on March 24, 2013 and the last calves were born on May 15, for a 51 d calving season, and an average calving date of April 13. Two weigh-suckle-weighs (WSW) were conducted in order to estimate 24-h milk weight. The first WSW was performed 93 ± 13 d postpartum during peak lactation and the second WSW was conducted 170 ± 13 d postpartum during late lactation. Calves were weaned at approximately 6 mo of age.

CHAPTER 3: EFFECTS OF CHROMIUM SUPPLEMENTATION TO FEEDLOT STEERS ON GROWTH PERFORMANCE, INSULIN SENSITIVITY, AND CARCASS CHARACTERISTICS

ABSTRACT

Objectives were to determine the effects of Chromium-propionate supplementation on growth performance, insulin and glucose metabolism, and carcass characteristics of beef cattle. Angus-cross steers (n = 34) were stratified by BW and dam treatment and assigned to 1 of 2 treatments: (1) no supplemental Cr (**Cont**), or (2) 3 mg supplemental Cr/hd·d^{-1} (**CrP**). Both supplements, Cont and CrP, were delivered via 0.454 kg ground corn top dressed on the basal diet. There was no effect ($P \geq 0.45$) of CrP on ADG, DMI, G:F, or final BW. However, steers fed CrP had greater ($P = 0.10$) days on feed (**DOF**) to achieve the same carcass back fat as steers fed Cont. There were no effects ($P \geq 0.41$) of CrP on HCW, back fat, or KPH. Steers fed CrP had increased ($P = 0.01$) dressing percentage (**DP**) and tended to have a 4.21 cm^2 greater LM area ($P = 0.15$), decreased marbling scores ($P = 0.11$), and decreased intramuscular fat by 0.71% ($P = 0.11$) compared to steers fed Cont. There were no differences ($P \geq 0.25$) in Quality or Yield Grade distributions. A glucose tolerance test (**GTT**) was conducted early (21 DOF) and late (98 DOF) in the finishing phase. There was a Feedlot Treatment **(FT**) × Time × DOF interaction ($P = 0.08$) for glucose concentrations. There were no other interactions ($P \geq 0.21$) for glucose or insulin concentrations. There was no FT × DOF ($P \geq 0.21$) for insulin area under the curve (**iAUC**), insulin:glucose, insulin or glucose baseline, or peak insulin or glucose concentrations. At 21 DOF, steers fed CrP had decreased glucose area under the curve (**gAUC**; $P = 0.01$), decreased glucose clearance rate (***k***; $P = 0.02$), and increased glucose half-life (**T½**; $P = 0.07$) compared to steers fed Cont; however, by 98 DOF, no differences were observed between

treatments. At 98 DOF, all steers, regardless of treatment, had increased ($P < 0.01$) peak glucose and insulin, *k*, iAUC, insulin:glucose, and baseline insulin, when compared to 21 DOF, but, gAUC and $T_{1/2}$ decreased ($P < 0.01$). While steers fed CrP tended ($P = 0.11$) to have increased baseline glucose concentrations compared to steers fed Cont, CrP supplementation did not affect ($P \geq 0.17$) other measures of glucose or insulin. Results of this study indicate that CrP increased DP and tended to increase LM area and, but tended to decrease intramuscular fat, with no effect on growth performance. With increased DOF, all steers became more insulin resistant, using more insulin to clear less glucose; and these effects were not mitigated by CrP supplementation.
Key words: beef, carcass characteristics, chromium, glucose, insulin

INTRODUCTION

Chromium is an essential trace mineral recognized for increasing insulin function and insulin sensitivity, not only in humans and laboratory rodents (Vincent, 2000; Anderson, 2003; Padmavathi et al., 2010), but also in livestock (Kegley et al., 1997b; Sumner et al., 2007; Spears et al., 2012). Supplementing Cr increased insulin sensitivity in ≤ 12 mo old Holstein and beef heifers (Sumner et al., 2007; Spears et al., 2012, respectively) but decreased insulin sensitivity in beef feeder calves during the receiving period (Kegley et al., 2000; Bernhard et al., 2012, respectively). Sumner et al. (2007) and Spears et al. (2012) both observed that heifers fed Cr produced less insulin and cleared glucose at a faster rate than those not supplemented with Cr. Conversely, Kegley et al. (2000) and Bernhard et al. (2012) reported that feeder steers fed Cr during the receiving period had increased insulin and insulin:glucose, with no differences in glucose, when compared to steers fed no Cr. However, there are no data on the effects of feeding Cr to feedlot beef steers during the finishing period on insulin and glucose metabolism.

Supplementing Cr improved growth performance in newly received, stressed calves (Chang and Mowat, 1992; Moonsie-Shageer and Mowat, 1993; Kegley et al., 1997a), and increased LM area (Page et al., 1993; Kornegay et al., 1997) and marbling in swine (Matthews et al., 2003). However, data regarding the effects of Cr supplementation to feedlot steers during the finishing period is limited and variable (Chang et al., 1992; Mathison and Engstrom, 1995; Pollard et al., 2002). We hypothesized that feeding Cr to feedlot steers would increase insulin sensitivity, and thereby, improve ADG, DMI, G:F, and BW, and increase intramuscular fat deposition when compared to steers that were fed no Cr. Therefore, our objectives were to determine the effects of feeding 3 mg Cr/hd·d^{-1} to beef steers throughout the finishing phase on growth performance, carcass characteristics, and glucose and insulin metabolism.

MATERIALS AND METHODS

All procedures in this experiment followed guidelines recommended in the Guide for the Care and Use of Agricultural Animals in Agricultural Research and Teaching (FASS, 2010).

Animal Diet and Management

Angus-cross steers (n = 34; BW = 454 ± 130 kg) were weaned at approximately 6 mo of age, at the Eastern Agricultural Research Station (**EARS**) in Belle Valley, OH, preconditioned for 28 d, and then transported (142 km) to the Beef Research Station at the Ohio Agricultural Research and Development Center (**OARDC**) in Wooster, OH on Nov. 18, 2013. After approximately 24 h rest at the feedlot, steers received Inforce® 3 (Zoetis, Florham Park, NJ) and were dewormed with Ivomec® pour-on (Merial, Duluth, GA). Steers were housed in 34 individual pens. Pens are made of metal gates on slatted concrete floors. Pens are 2.6 × 1.5 m and

have individual, self-watering cups. A common receiving diet was fed for 29 d (45% corn silage, 20% soybean hulls, 10% dry-rolled corn, 25% protein supplement, on DM basis). During that time, steers were transitioned to a corn-based finishing diet in 2 step-up rations.

After transition, steers were stratified by BW (initial average BW = 246 ± 59 kg) and dam treatment and assigned to 1 of 2 treatments: (1) no supplemental Cr (**Cont**), or (2) 3 mg supplemental Cr/hd·d^{-1} (**CrP**, KemTRACE Cr, 0.4% Cr as Cr-propionate; Kemin Industries Inc., Des Moines, IA; Table 3.1). Both supplements, Cont and CrP, were delivered via 0.454 kg ground corn top dressed on the basal diet. The finishing diet started on Dec. 17, 2013 and contained 55% corn, 20% corn silage, 15% dried distillers grains, and 10% trace mineral and vitamin supplement (Table 3.1). Steers were weighed every 28 d during the finishing phase (Powder River Chutes, Provo, UT; SR2000 Tru-Test Scale Head). Steers were implanted with Compudose (Elanco Animal Health; Greenfield, IN) at the beginning of the finishing phase (Dec. 17, 2013) and implant retention was checked 29 d later.

Feed samples were collected every 14 d and individual ingredient samples were dried at 55˚C and composited over the course of the trial. Composites of individual samples were analyzed for nutrient composition and results were used to calculate nutrient composition of the diets. All samples were analyzed for DM (24 h at 103˚C), ADF and NDF (using Ankom Technology method 5 and 6, respectively; Ankom^{200} Fiber Analyzer, Ankom Technology, Macedon, NY), CP (Leco TruMac, LECO Corporation, St. Joseph, MI), fat (using Ankom Technology method 2; Ankom Technology), and total ash (600˚C for 2 h, Thermolyne muffle oven model: F30420C, Thermo Scientific, Waltham, MA). For complete analysis of Cr content, top-dress samples for the CrP and Cont treatments were subjected to atomic absorption spectroscopy (**AA**) as described by Williams et al. (1962).

Glucose Tolerance Test and Blood Analysis

A Glucose Tolerance Test (**GTT**) was performed from January 7 to 9, 2014 (21 to 23 DOF). A second GTT was conducted from March 25 to 27, 2014 (98 to 100 DOF). Steers (n = 12) from each feedlot treatment closest to the mean treatment BW were selected for the first GTT. Due to the intensive sample collection, 4 steers within each treatment were catheterized on January 6 to be sampled January 7 in the morning. Then, on the afternoon of January 7, 4 different steers from each treatment were catheterized for sampling on January 8. On January 8, 4 additional steers from each treatment were catheterized for blood sampling on January 9. The same 24 steers were then used in the subsequent GTT, in March. The same schedule previously described was used in March, and the first blood sample was taken on March 25. Feed was withheld from the steers for 18 h, prior to taking the first blood sample. Steers were restrained in a chute and an aseptic procedure was used to catheterize the jugular vein the day before the GTT was performed (approximately 18 h). A 60 cm indwelling, venous catheter was inserted through a 3.81 cm long, 12 gauge needle. Half of the catheter (Tygon Tubing, AAD02127-CP, Saint-Gobain, Valley Forge, PA) was inserted into the steer, leaving the other half outside of the calf to be used for blood collection. The catheter was fitted with a hub to allow for blood collection via syringe. To prevent overnight clotting, catheters were flushed with 2 mL of heparin stock solution (1,000 µg/mL). Catheters were secured to the neck with a velcro patch, covered with vet wrap, and steers were returned to their home pens. The next morning, prior to infusion and sampling, calf empty BW was recorded to determine dose of sterile 50% dextrose solution (AgriLaboratories, Ltd., St. Joseph, MO) required to deliver 0.25g dextrose/kg of BW. Blood samples were taken at -5 and -2 min (prior to infusion) via the catheter to determine baseline

glucose and insulin concentrations. Steers were infused with dextrose in the chute and the time at the end of infusion was recorded as the reference point for initiating each blood collection. Following infusion, 5 ml of sterile saline and 1 ml of flush solution (sterile heparinized saline; 9 g/L of NaCl) was flushed through the catheter to ensure dextrose was cleared from the catheter. Steers were then returned to their pens. Subsequent blood samples were drawn at 5, 10, 15, 20, 30, 60, 90 and 120 min post-infusion. Prior to each blood sample, 4 mL of blood was drawn into a syringe and discarded, to clear the catheter. In a new syringe, 10 mL of blood was drawn for the sample and placed in Vacutainer tubes (K_2 EDTA, REF 366643, BD Vacuatainer ®, Franklin Lakes, NJ). The catheters were subsequently flushed with 1 mL of the flush solution to prevent clotting. Blood samples were placed on ice for no longer than 30 min until centrifuged at 3,000 × g for 20 min at 4°C. Plasma was aliquoted into 2-mL tubes in duplicate and placed in a -20°C freezer for the remainder of the day. Plasma samples were stored at -80°C until analyzed for glucose (Glucose Liquicolor; Procedure No. 1070; Stanbio Laboratory, Boerne, TX) and insulin (Mercodia Bovine Insulin ELISA; Mercodia, Sweden). Glucose and insulin were analyzed according to the manufacturer's instructions. A pooled-sample of all steers, from all treatments, and all time points was used as a control. Most plasma insulin samples were diluted using the provided 0 standard (0.0 µg/L) prior to measuring insulin in order to be in the range of the standard curve, 0 to 3.0 ng/mL. Glucose and insulin absorbance was measured at 500 nm and 450 nm, respectively, with a 96-well plate reader (Syndergy HT, BioTek, Winooski, VT). A coefficient of variation (**CV**) of ≤ 10% within duplicates for glucose and insulin was deemed acceptable.

Glucose and insulin values were converted from traditional units (ng/mL and mg/dL, respectively) to the International System of Units (SI; mmol/L and pmol/ L, respectively), the

global standard. Glucose was converted using the formula mg/dL = 0.05551 mmol/L (Young, 1987). Insulin was converted using the molecular bovine insulin weight, 5734 g/mol (Darby et al., 2001), and subsequently, a conversion rate of 1 ng/mL = 174.398 pmol/L was used for insulin conversions.

Glucose clearance rates (*k*) were calculated as described by Bernhard et al. (2012). Values were determined using incremental serum glucose concentrations between 5 (t_1) and 20 (t_2) min post-infusion during GTT. This time increment best describes the sharpest decrease in glucose concentrations following the peak at 5 min. Clearance rate (***k***) of glucose is calculated from the natural log (ln) of circulating glucose concentrations (**Glu**) between 2 time points (t_2 and t_1), as shown in the equation below.

$$k = \frac{\ln[\text{Glu1}] - \ln[\text{Glu2}]}{t_2 - t_1} \times 100 = \frac{\%}{\text{min}}$$

Glucose half-life ($\mathbf{T_{½}}$) was calculated during the same time points and used the calculated *k* from the equation above.

$$T_{½} = \frac{0.693}{k} \times 100 = \text{min}$$

Glucose and insulin AUC were calculated as described by Cardoso et al. (2011) using positive incremental AUC. Baseline values were determined to be the mean of -5 and -2 min relative to infusion. Insulin:Glucose was calculated using the AUC values.

Carcass Data Collection

Steers were slaughtered when individual animals reached 1.27 cm of back fat (**BF**) as determined by ultrasound (Aloka SSD-500V, Probe: Aloka UST-5049-3.5 MHz). Eight steers

reached the targeted 1.27 cm on 4/30/14 (134 days on finishing diet; **DOF**), 12 steers on 5/19/14 (153 DOF), 7 steers on 6/2/14 (167 DOF), and the last 7 steers on 6/9/14 (174 DOF), where DOF represents just those days the steers were fed their respective Cont or CrP supplements, not the 29 d of transition, prior to supplementation, discussed previously. Steers were transported approximately 25 km and slaughtered at a commercial abattoir under USDA inspection. Hot carcass weights were recorded at the end of the slaughter line before the carcass entered the cooler. Carcasses were chilled for 24 h at -4°C. After chill, USDA Yield Grade (**YG**) and Quality Grade (**QG**) were collected by a trained technician.

A 2.54 cm slice of the LM was taken posterior to the 12th rib. Muscle samples were transported back to the University of Illinois Meat Science Lab on wet ice to be processed. Half of the 2.54 cm LM slice from each calf was trimmed of intermuscular and subcutaneous fat and individually frozen and stored at -20°C until further analysis. These samples of LM were ground and intramuscular fat (**IMF**) concentrations were determined by ether extract (Method 2, Ankom XT10 Fat Extractor; Ankom Technologies, Macedon, NY).

The other half of 2.54 cm LM slice was used for histological analysis. Samples were taken from the center, cut into a 1 cm cube, oriented perpendicular to the muscle fibers, and placed in liquid nitrogen-cooled isopentane. Once the tissue was frozen, it was stored in a foil packet in -80°C until further analysis. These frozen samples were later cut 5µm thick using a cryostat and sections were fixed to charged glass slides at the University of Illinois Veterinary Lab by a trained technician. The glass slides were then stained using a standard hematoxylin and eosin protocol for frozen slides. Once slides were stained and dried for at least 24 h, multiple images were captured of the stained slides using a digital inverted microscope (20× objective; EVOS XL; Life Technologies, Carlsbad, CA). Three representative images per calf were

selected and analyzed in Photoshop (Adobe Photoshop CC 2014). Prior to fiber area measurement of each image, trained personnel and Photoshop were calibrated to be within 5% of a known area of a circle. The fibers in each image were labeled and counted. Fiber area of each cell was measured and analyzed with the pixel length of 514 by tracing the perimeter of each cell with the lasso tool function. For each steer, the average area of all muscle fibers present in 3 images per steer were reported.

Statistical Analysis

This experiment was a randomized complete block design with 2 dietary treatments. Individual steer served as the experimental unit. Average daily gain, DMI, G:F, BW, DOF, HCW, dressing percentage (**DP**), BF, KPH, marbling score, muscle fiber area, and IMF were analyzed using the MIXED procedures of SAS (SAS 9.4; SAS Inst. Inc., Cary, NC). Feedlot Treatment (**FT**) was included as a fixed effect. Dams of steers were on a trial during steer fetal development; therefore, steers were stratified to their feedlot treatments in an attempt to account for carry over effects of Dam Treatment and Dam Treatment was included as a fixed effect in the model. The covariance matrix of the fixed-effect estimates and denominator degrees of freedom for the *t* and *F* tests were determined according to the Kenward and Roger adjustment.

Glucose and insulin concentrations were analyzed using the MIXED procedure (SAS 9.4; SAS Inst. Inc., Cary, NC) with repeated measures, where time of sampling post-infusion was repeated within calf (Time). Thus, Feedlot Treatment, Dam Treatment, DOF, Baseline, and Time × DOF × FT, Time × FT, and DOF × FT were included as fixed effects. Glucose Baseline concentrations and insulin Baseline concentrations were used as covariates (fixed effects) when analyzing their respective concentrations over time. The covariance structure was compound

symmetry, as determined by lowest Bayesian information criterion, and calf was the subject. The SLICE statement was used to separate the effects of the interactions of Time, DOF, and FT.

Insulin:glucose, glucose *k*, glucose $T_{½}$ and peak, baseline, and AUC for glucose and insulin, were analyzed using the MIXED procedure (SAS 9.4; SAS Inst. Inc., Cary, NC) specific for repeated measures (DOF). Dam Treatment, FT, DOF, and DOF × FT were included as fixed effects, the covariance structure was compound symmetry, as determined by lowest Bayesian information criterion, and calf was the subject. The SLICE statement was used to separate the effects of the interactions of DOF and FT.

The GLIMMIX procedure (SAS 9.4; SAS Inst. Inc., Cary, NC) was used to analyze USDA YG and QG distributions. Dam Treatment and FT were fixed effects. The distribution was binomial and denominator degrees of freedom was calculated by the Satterthwaite method.

For all analyses, individual steer served as the experimental unit. Significance was declared at $P \leq 0.10$. Trends were discussed at $0.10 < P \leq 0.15$.

RESULTS

Because steers were stratified by BW at trial allotment, there were no differences (P = 1.00) in initial BW (Table 3.2). There were no differences ($P \geq 0.45$) in ADG, DMI, G:F, or final BW between steers fed CrP and steers fed Cont. Steers fed CrP were on feed 8 d longer (P = 0.10) than steers fed Cont to reach the same targeted BF.

Just as final BW did not differ, HCW also did not differ (P = 0.41; Table 3.3). Steers fed CrP had HCW of 315 kg and steers fed Cont had a HCW of 306 kg. Because steers were slaughtered as they reached 1.27 cm of BF, there were no differences (P = 0.81) in BF between treatments. There were also no differences (P = 0.76) in KPH fat. However, steers fed CrP had a

greater (P = 0.01) DP than steers fed Cont (62.0 vs 60.7%, respectively). The increase in DP may be influenced by a 4.21 cm^2 larger (trend; P = 0.15) LM area in carcasses from steers fed CrP compared to carcasses from steers fed Cont.

Despite the fact that steers fed CrP had increased DP and tended to have greater LM area, there were no differences ($P \geq 0.25$) in USDA YG distributions (Table 3.3). The majority of carcasses graded YG 3, regardless of treatment. There were also no effects ($P \geq 0.25$) of feeding Cr on QG distributions. Carcasses from steers fed CrP and Cont graded 28.4% and 46.9% Average Choice or greater, respectively. Even though QG did not differ, carcasses from steers supplemented with CrP tended (P = 0.11) to have lower marbling scores when compared to carcasses from steers fed Cont. This tendency for carcasses from steers supplemented with CrP to have 54 units less marbling corresponded to a tendency (P = 0.11) for those same carcasses to have 18% less IMF compared to carcasses from steers fed Cont.

There was a FT × Time × DOF interaction (P = 0.08) for change in glucose concentrations following glucose infusion (Figure 3.1). However, there were no effects of FT × Time × DOF (P = 1.00) on insulin concentrations following a glucose infusion (Figure 3.2). Furthermore, no other interactions ($P \geq 0.50$) were observed for glucose or insulin concentrations following the GTT.

As glucose is tightly regulated, measures other than concentration can be better indicators of glucose status. A FT × DOF interaction ($P \leq 0.07$) was observed for glucose area under the curve (**gAUC**), k, and $T_{½}$ (Table 3.4). At 21 DOF, steers fed CrP had lower (P = 0.01) gAUC, slower (P = 0.02) k, and longer (P = 0.07) $T_{½}$ compared to steers fed Cont. However, at 98 DOF, there were no differences between treatments. Furthermore, a main effect of DOF was observed for gAUC, k, and $T_{½}$. All steers, regardless of treatment, had decreased gAUC, faster k, and

shorter $T_{½}$ ($P < 0.01$) at the GTT conducted at 98 DOF compared to 21 DOF. Steers fed CrP in the present trial tended ($P = 0.11$) to have lower baseline glucose concentrations compared to steers fed Cont, regardless of DOF. Peak glucose concentrations (5 min post infusion) only differed ($P < 0.01$) by DOF; there was no effect ($P = 0.17$) of CrP, or the interaction with DOF observed ($P = 0.27$).

There were no FT × DOF interactions, nor effects of FT ($P \geq 0.21$), on insulin area under the curve (**iAUC**), peak or baseline insulin concentrations, or insulin:glucose ratio; however, there was a main effect of DOF (Table 3.4). Regardless of whether or not steers were fed CrP, at 98 DOF, steers had increased iAUC ($P < 0.01$), increased peak and baseline insulin concentrations ($P < 0.01$), and increased insulin:glucose ($P < 0.01$) compared to 21 DOF.

DISCUSSION

Previous research involving beef feedlot steers and Cr effects on performance and carcass characteristics has been variable and limited (Chang et al., 1992; Mathison and Engstrom, 1995; Pollard et al., 2002). Type of Cr, level and duration of Cr supplementation, and even differences in amount of glucose and infusion rates during GTT (Kegley et al., 2000; Sumner et al., 2007; Bernhard et al., 2012), make discussion and comparison of papers on insulin sensitivity difficult. Furthermore, basal diet Cr concentrations vary widely within the literature and are not always reported, most likely due to the difficulty of analysis. Normally, Cr concentrations in the diet are approximately 1 to 3 mg Cr/kg DM (Vincent, 2000). However, the basal diet in the current study contained 5.61 mg Cr/kg DM. However, the bioavailability of Cr in the basal dietary ingredients is less than the bioavailability of Cr in the supplemented form, Cr-Prop (Vincent, 2000). We had attempted to supplement an additional 3 mg of Cr per d to steers fed CrP to represent the

maximum supplementation allowed. However, according to the analysis, the supplement provided 4.5 mg Cr per d to each steer, via the 0.454 kg top-dress.

Chromium supplementation has been shown to increase DMI, ADG, and G:F in stressed, receiving feedlot calves (Chang and Mowat, 1992; Moonsie-Shageer and Mowat, 1993; Kegley et al., 1997a). Therefore, we hypothesized that steers fed CrP would have increased ADG and final BW, and have a more efficient G:F; however, this was not the case. There were no differences in growth or final BW nor differences in HCW in the present trial. Little work has been done to investigate the effects of feeding Cr to steers on feedlot finishing diets. Some of the work that has been done has also shown no differences in ADG, DMI, G:F, final BW, or HCW of steers fed either 0.0 or 0.2 mg Cr/kg DM as Cr-yeast (Chang et al., 1992), nor in ADG, DMI, G:F, or final BW of steers fed either 0 or 3 mg Cr/kg DM as a Cr-amino acid chelate (Mathison and Engstrom, 1995). In addition, steers fed Cr-yeast at either 0.0, 0.2, or 0.4 mg Cr/kg DM during the feedlot finishing period had similar in ADG, G:F, DMI, and final BW; but, steers fed 0.4 mg Cr/kg DM had decreased ADG, G:F, DMI, and final BW and tended to decrease HCW when compared to those fed 0.0 and 0.2 mg Cr/kg DM (Pollard et al., 2012). However, Pollard et al. (2002) also reported that feeding 0.2 mg Cr/kg DM as Cr-yeast increased HCW by 13 kg, whereas feeding 3 mg Cr/kg DM as a Cr-amino acid chelate (Mathison and Engstrom, 1995) decreased HCW when compared to feed 0 mg Cr. Our data more closely agree with Chang et al. (1992) as there were no differences in growth performance or HCW with Cr supplementation to steers in the present trial.

We had hypothesized that CrP supplementation would enhance marbling because of an increased availability of glucose for intramuscular fat deposition. Therefore, all steers were slaughtered at a targeted 1.27 cm back fat (**BF**), across a 40 d range, to determine the effects of

CrP on intramuscular fat deposition at a common BF end point. Steers fed CrP had greater DOF. This has not been shown in previous research, as calves in previous trials have either been fed Cr during the receiving period and not during finishing (Moonsie-Shageer and Mowat, 1993), or were slaughtered as a group at a targeted final BW (Chang et al., 1992; Pollard et al., 2002).

One of the reasons we had hypothesized increased glucose availability for IMF is because Cr potentiates the action of insulin (Anderson, 2003). In fact, Matthews et al. (2003) observed that growing-finishing pigs supplemented with 0.2 mg Cr/kg of DM as Cr-propionate had a 0.41 unit increase in marbling scores. Furthermore, increased conversion of propionate to glucose has been reported in rams fed concentrate-based diets and supplemented with Cr when compared to those not supplemented (Sano et al., 1997). Increased propionate conversion to glucose may lead to increased marbling (Smith and Crouse, 1984). However, previous research has shown no effect of Cr on marbling scores in beef cattle (Chang et al., 1992; Mathison and Engstrom, 1995; Pollard et al., 2002) and, in fact, in the current study we observed a tendency for decreased marbling and IMF.

This may be due to the fact that the IMF depot is the last to develop and the primary user of glucose in development is muscle. In the present trial, steers fed CrP had an increased DP compared to steers fed Cont. This increased DP by Cr has not been reported previously in beef feedlot steers. Chang et al. (1992) and Mathison and Engstrom (1995) noted no effect of Cr-yeast and a Cr-amino acid chelate on DP. And, in fact, Pollard et al. (2002) reported a decrease in DP by 1.2% with feeding Cr-yeast to supply 0.4 mg Cr/kg DM. But, in the current study, the increase in DP is driven by the tendency for carcasses from steers fed CrP to have a larger LM area compared to carcasses of steers fed Cont. This increase in LM area has also not been previously observed in feedlot steers fed Cr; however, an increase in LM area (Page et al., 1993;

Kornegay et al., 1997) and total percentage of muscle in the carcass (Boleman et al., 1995; Jackson et al., 2009) has been reported in swine fed Cr.

Because Cr effects glucose and insulin metabolism, as discussed previously, 2 GTT were conducted in the present trial to examine the effects of CrP supplementation on glucose concentrations and insulin function in steers. There has been no previous research comparing the effects of feeding Cr to beef steers throughout the finishing phase on glucose and insulin metabolism. Previous Cr research on glucose and insulin metabolism of cattle has been conducted in the receiving phase of the feedlot (Bunting et al., 1994; Kegley and Spears, 1995; Kegley et al., 2000; Sumner et al., 2007; Bernhard et al., 2012). However, these data do not represent an accurate comparison to the current trial because DOF may affect insulin sensitivity and glucose (Radunz et al., 2012). In the most comparable study, Spears et al. (2012) supplemented growing beef heifers, averaging 291 kg at the start of the trial, either 0, 3, 6, or 9 mg Cr per d as Cr-Prop in a ground corn carrier that was top-dressed on a silage-based diet. A GTT was conducted at d 44 on the study, a mid-point between the GTT conducted in the current study. However, treatments were fed for 43 d, less than half of the time that Cr was fed in the current study. In the present trial, the effects of the FT × Time × DOF interaction on glucose may not have as much biological relevance as gAUC, peak and baseline glucose concentrations, *k*, or $T_{½}$.

Similar to the current data, previous research has shown no effect of Cr supplementation on peak glucose concentrations in growing cattle (Spears et al., 2012). In addition, there were also no effects of Cr (at any level) on baseline glucose concentrations (Spears et al., 2012). Whereas, in the current study, steers fed CrP tended to have decreased basal glucose concentrations compared to steers fed Cont, regardless of DOF. Baseline glucose values of

steers in the current study were in the upper range of what is considered normal, 3 to 6 mmol/L for fasted feedlot steers (Kegley et al., 2000; Schoonmaker et al., 2003; Vasconcelos et al., 2009; Radunz et al. 2012).

Increased *k* and decreased T½ are measures that suggest improved insulin sensitivity. However, in the current study, steers fed CrP had decreased gAUC, decreased *k*, and increased T½ when compared to steers fed Cont early in the finishing phase (21 DOF), suggesting a reduction in insulin sensitivity; but, late in the finishing phase (98 DOF), there were no differences between treatments. Regardless of CrP supplementation, all steers in the present study had decreased gAUC, faster *k*, and decreased T½ at 98 DOF compared to 21 DOF. Previous research reported growing heifers had increased *k* from 15 to 45 min after glucose infusion and increased T½ with supplementation of Cr-propionate (Spears et al., 2012), but, again, heifers in the previous experiment were only fed Cr for 43 d and only 1 GTT was conducted. Therefore, comparison of DOF effects with Cr supplementation may require additional research.

While there were no effects of CrP on iAUC, insulin peak or baseline concentrations, or insulin:glucose ratio in the current study, baseline insulin values for steers were considered within the normal range of 100 to 350 pmol/L for beef steers on a finishing diet (Trenkle, 1972; Hersom et al., 2004; Vasconcelos et al., 2009). Contrary to these findings, 12 mo old growing beef heifers fed Cr-propionate had decreased iAUC, insulin:glucose, and peak insulin concentrations with no effect on basal insulin concentrations (Spears et al., 2012).

Overall, Spears et al. (2012) reported that heifers supplemented with Cr for 43 d were more insulin sensitive with Cr-propionate supplementation, due to decreased insulin and glucose concentrations with a faster clearance of glucose and decreased insulin:glucose. In the current study, CrP also decreased gAUC but decreased *k* and increased T½ without affecting

insulin:glucose at 21 DOF; however, by d 98 there were no effects of CrP on any glucose or insulin variables. The differences observed between our study and Spears et al. (2012) may be due to duration of feeding Cr or differences in insulin sensitivity between sex, as growing heifers have been shown to be more insulin sensitive than growing steers (Radunz et al., 2012).

Perhaps one of the most compelling findings from the current study is the powerful effect of DOF on glucose and insulin metabolism. The same amount of insulin being used to clear more glucose suggests increased insulin sensitivity or the tissues becoming more sensitive to the effects of insulin (Reaven, 1988; Wilcox, 2005). The steers in the present trial used *more* insulin to clear *less* glucose at 98 DOF compared to 21 DOF, regardless of Cr supplementation; thus, steers were more insulin resistant, with no mitigation from CrP supplementation. Supporting the current research, Radunz et al. (2012) reported that steers had increased insulin resistance the longer they were on a corn-based finishing diet. No other trials have conducted multiple GTT in feedlot cattle when supplementing Cr.

In conclusion, there were no effects of CrP on any measures of growth performance. While steers fed CrP tended to have increased LM area and increased DP, they also tended to have decreased marbling scores and intramuscular fat. In the present trial, CrP did not improve measures of insulin sensitivity over time. As steers were on feed longer, they became insulin resistant, regardless of whether or not they were supplemented with CPr.

LITERATURE CITED

Anderson, R. A. 2003. Chromium and Insulin resistance. Nutr Res Rev. 16: 267-275. doi:10.1079/NRR200366.

Bernhard, B. C., N. C. Burdick, R. J. Rathmann, J. A. Carroll, D. N. Finck, M. A. Jennings, T. R. Young, and B. J. Johnson. 2012. Chromium supplementation alters both glucose and lipid metabolism in feedlot cattle during the receiving period. J. Anim. Sci. 90:4857-4865. doi:10.2527/jas.2011-4982.

Boleman, S. L., S. J. Boleman, T. D. Bidner, L. L. Southern, T. L. Ward, J. E. Pontif, and M. M. Pike. 1995. Effect of chromium picolinate on growth, body composition, and tissue accretion in pigs. J. Anim. Sci. 73:2033-2042. doi:/1995.7372033x.

Bunting, L. D., J. M. Fernandez, D. L. Thompson, Jr., and L. L. Southern. 1994. Influence of chromium picolinate on glucose usage and metabolic criteria in growing Holstein calves. J. Anim. Sci. 72:1591-1599. doi:/1994.7261591x.

Cardoso, F. C., W. Sears, S. J. LeBlanc, and J. K. Drackley. 2011. *Technical note*: Comparison of 3 methods for analyzing areas under the curve for glucose and nonesterified fatty acids concentrations following epinephrine challenge in dairy cows. J. Dairy Sci. 94:6111-6115. doi:10.3168/jds.2011-4627.

Chang, X., and D. N. Mowat. 1992. Supplemental chromium for stressed and growing feeder calves. J. Anim. Sci. 70:559-565. doi:/1992.702559x.

Chang, X., Mowat D. N., and G. A. Spiers. 1992. Carcass characteristics and tissue-mineral contents of steers fed supplemental Chromium. Can. J. Anim. Sci. 72:663-669. doi:10.4141/cjas92-077.

Darby, S. M., M. L. Miller, R. O. Allen, and M. LeBeau. 2001. A mass spectrometric method for quantification of intact insulin in blood samples. J. of Analyt. Toxico. 25:8-14. doi:10.1093/jat/25.1.8.

FASS, Federation of Animal Science Societies. 2010. Guide for the care and use of agricultural animals in research and teaching. 3rd ed.

Hersom, M. J., R. P. Wettemann, C. R. Krehbiel, G. W. Horn, and D. H. Keisler. 2004. Effect of live weight gain of steers during winter grazing: III. Blood metabolites and hormones during feedlot finishing. J. Anim. Sci. 82:2059-2068. doi:/2004.8272059x.

Jackson, A. R., S. Powell, S. L. Johnston, J. O. Matthews, T. D. Bidner, F. R. Valdez, and L. J. Southern. 2009. The effect of chromium as chromium propionate on growth performance, carcass traits, meat quality, and the fatty acid profile of fat from pigs fed no supplemented dietary fat, choice white grease, or tallow. J. Anim. Sci. 87:4032-4041. doi:10.2527/jas.2009-2168.

Kegley, E. B. and J. W. Spears. 1995. Immune response, glucose metabolism, and performance of stressed feeder calves fed inorganic or organic chromium. J. Anim. Sci. 73: 2721-2726. doi:/1995.7392721x.

Kegley, E. B., J. W. Spears, and T.T. Brown, Jr. 1997a. Effect of shipping and Chromium supplementation on performance, immune response, and disease resistance of steers. J. Anim. Sci. 75:1956-1964. doi:/1997.7571956x.

Kegley, E. B., J. W. Spears, and J. H. Eisemann. 1997b. Performance and glucose metabolism in calves fed Chromium-Nicotinic acid complex or chromium chloride. J. Dairy Sci. 80:1744-1750. doi:10.3168/jds.S0022-0302(97)76107-1.

Kegley, E. B., D. L. Galloway, and T. M. Fakler. 2000. Effect of dietary chromium-L-methionine on glucose metabolism in beef steers. J. Anim. Sci. 78:3177-3183. doi:/2000.78123177x.

Kornegay, E. T., Z. Wang, C. M. Wood, and M. D. Lindemann. 1997. Supplemental chromium picolinate influences nitrogen balance, dry matter digestibility, and carcass traits in growing-finishing pigs. J. Anim. Sci. 75:1319-1323. doi:/1997.7551319x.

Mathison, G. M. and D. F. Engstrom. 1995. Chromium and protein supplements for growing-finishing beef steers fed barley-based diets. Can. J. of Anim. Sci. 75:549-558. doi:10.4141/cjas95-083.

Matthews, J. O., A. D. Higbie, L. L. Southern, D. F. Coombs, T. D. Bidner, and R. L. Odgaard. 2003. Effect of chromium propionate and metabolizable energy on growth, carcass traits, and pork quality of growing-finishing pigs. J. Anim. Sci. 81:191-196. doi:/2003.811191x.

Moonsie-Shageer, S. and D. N. Mowat. 1993. Effect of level of supplemental chromium on performance, serum constituents, and immune status of stressed feeder calves. J. Anim. Sci. 71:232-238. doi:/1993.711232x.

NRC. 2000. Nutrient Requirements of Beef Cattle. 7th Rev. Ed., 1996. Natl. Acad. Press, Washington, DC.

Padmavathi, J. N. I., K. R. Rao, L. Venu, A. Ismail, and M. Raghunath. 2010. Maternal dietary chromium restriction programs muscle development and function in the rat offspring. Exp. Biol. Med. 235:349-355. doi:10.1258/ebm.2009.009199.

Page, T. G., L. L. Southern, T. L. Ward, and D. L. Thompson, Jr. 1993. Effect of chromium picolinate on growth and serum and carcass traits of growing-finishing pigs. J. Anim. Sci. 71:656-662. doi:/1993.713656x.

Pollard, G. V., C. R. Richardson, and T. P. Karnezos. 2002. Effects of supplemental organic chromium on growth, feed efficiency and carcass characteristics of feedlot steers. Anim Feed Sci Technol. 98:121-128. doi:10.1016/S0377-8401(02)00010-X.

Radunz, A. E., F. L. Fluharty, A. E. Relling, T. L. Felix, L. M. Shoup, H. N. Zerby, and S. C. Loerch. 2012. Prepartum dietary energy source fed to beef cows: II. Effects on progeny postnatal growth, glucose tolerance, and carcass composition. J. Anim. Sci. 90:4962-4974. doi: 10.2527/jas.2012-5098.

Reaven, G. M. 1988. Role of insulin resistance in human disease. Diabetes. 37:1595-1607. doi:10.2337/diab.37.12.1595.

Sano, H., D. N. Mowat, R. O. Ball, and D. R. Trout. 1997. Effect of supplemental Chromium on whole-body kinetics of glucose, lactate, and propionate in rams fed a high grain diet. Comp. Biochem. Physiol. 118B:117-121. doi:10.1016/S0305-0491(97)00023-0.

Schoonmaker, J. P., M. J. Cecava, D. B. Faulkner, F. L. Fluharty, H. N. Zerby, and S. C. Loerch. 2003. Effect of source of energy and rate of growth on performance, carcass characteristics, ruminal fermentation, and serum glucose and insulin of early-weaned steers. J. Anim. Sci. 81:843-855. doi:/2003.814843x.

Smith, S. B., and J. D. Crouse. 1984. Relative contributions of acetate, lactate and glucose to lipogenesis in bovine intramuscular and subcutaneous adipose tissue. J. Nutr. 114:792-800.

Spears, J. W., C. S. Whisnant, G. B. Huntington, K. E. Lloyd, R. S. Fry, K. Krafka, A. Lamptey, and J. Hyda. 2012. Chromium Propionate enhances insulin sensitivity in growing cattle. J. Dairy Sci. 95:2037-2045. doi:10.3168/jds.2011-4845.

Sumner, J. M., F. Valdez and J. P. McNamara. 2007. Effects of chromium propionate on response to an intravenous glucose tolerance test in growing Holstein heifers. J. Dairy Sci. 90:3467-3474. doi:10.3168/jds.2006-623.

Trenkle, A. 1972. Radioimmunoassay of plasma hormones: Review of plasma insulin in ruminants. J. Dairy Sci. 55:1200-1211. doi:10.3168/jds.S0022-0302(72)85648-0.

Vasconcelos, J. T., J. E. Sawyer, L. O. Tedeshi, F. T. McCollum, and L. W. Greene. 2009. Effects of different growing diets on performance, carcass characteristics, insulin sensitivity, and accretion of intramuscular and subcutaneous adipose tissue of feedlot cattle. J. Anim. Sci. 87:1540-1547. doi:10.2527/jas.2008-0934.

Vincent, J. B. 2000. The biochemistry of chromium. J. Nutr. 130:715-718. doi:10.1080/07315724.1999.10718821.

Wilcox, G. 2005. Insulin and insulin resistance. Clin Biochem Rev. 26:19-39.

Williams, C. H., D. J. David, and O. Iismaa. 1962. The determination of chromic oxide in faeces samples by atomic absorption spectrophotometry. J. Agric. Sci. 59:381-385.

Young, D. S. 1987. Implementation of SI units for clinical laboratory analysis. Annals of Int. Med. 106:114-129.

TABLES AND FIGURES

Table 3.1. Composition of diet to steers fed 0 mg Cr/d (Cont) or 3 mg Cr/d (CrP)

Item	Cont	CrP[1]
Dietary Inclusion, % DM		
Corn	55	55
Corn Silage	20	20
DDGS	15	15
Supplement	10	10
Ground Corn	67.700	67.700
Urea	5.000	5.000
Limestone	18.000	18.000
Trace Mineral Salt[2]	5.000	5.000
Vitamin A-30	0.074	0.074
Vitamin D-3	0.074	0.074
Vitamin E	0.222	0.222
Selenium	0.380	0.380
Rumensin 90	0.170	0.170
Tylosin	0.380	0.380
Potassium Chloride	3.000	3.000
Analyzed nutrient content, DM		
NDF, %	19.73	19.73
ADF, %	9.54	9.54
CP, %	12.04	12.04
Fat, %	3.36	3.36
Cr, mg/kg	5.61	5.61
Additional Top-Dress[3]		
Corn, %	100%	99.83%
Cr, %	--	0.17%
Analyzed nutrient content, DM		
NDF, %	10.85	10.33
ADF, %	3.38	2.81
CP, %	7.91	7.81
Fat, %	3.82	3.62
Cr, mg/kg	3.63	13.47

[1]Steers supplemented with Chromium (CrP) received 0.454 kg ground corn/hd·d^{-1} fortified with 3 mg Cr (KemTRACE Chromium-propionate 0.4%; Kemin Industries Inc., Des Moines, IA) as a top-dress. Control (Cont) steers were fed 0.454 kg ground corn/hd·d^{-1} with no additional Cr.

[2]Composition: 95% NaCl; 0.35% Zn, as ZnO; 0.28% Mn, as MnO_2; 0.175% Fe, as $FeCO_3$; 0.040% Cu, as Cu_2O; 0.007% I, as $Ca_5(IO_6)_2$; 0.007% Co, as $CoCO_3$.

[3]Supplementation began 12/17/13 and fed at a rate of 0.454 kg/hd^{-1}·d^{-1}

Table 3.2. Effects of chromium supplementation on growth performance of steers fed 0 mg Cr/d (Cont) or 3 mg Cr/d (CrP).

Item	Cont	CrP[1]	SEM	*P*-value
Animals, n	17	17	--	--
Initial BW, kg	246	246	7	1.00
ADG, kg	1.70	1.65	0.05	0.45
DMI, kg	8.6	8.4	0.3	0.57
G:F	0.1977	0.1965	0.0035	0.81
Final BW, kg	504	508	12	0.81
DOF[2]	152	160	3	0.10

[1]Steers supplemented with Chromium (CrP) received 0.454 kg ground corn/hd·d^{-1} fortified with 3 mg Cr (KemTRACE Chromium-propionate 0.4%; Kemin Industries Inc., Des Moines, IA) as a top-dress. Control (Cont) steers were fed 0.454 kg ground corn/hd·d^{-1} with no additional Cr.

[2]DOF = Days on Feed

Table 3.3. Effects of chromium supplementation on carcass characteristics and USDA Yield and Quality Grade distributions of steers fed 0 mg Cr/d (Cont) or 3 mg Cr/d (CrP).

Item	Cont	CrP[1]	SEM	*P*-value
Animals, n	17	17	--	--
HCW, kg	306	315	7.7	0.41
Dressing %	60.7	62.0	0.3	0.01
Back Fat, cm	1.30	1.28	0.03	0.81
KPH, %	3.4	3.5	0.1	0.76
LM area, cm^2	71.92	76.13	2.02	0.15
Muscle Fiber Area, μm^2	2,091	2,052	84	0.75
Yield Grade, %				
2	9.9	15.3	7.4	0.62
3	65.3	83.1	11.8	0.25
4	23.5	0.0	10.3	0.97
Quality Grade, %				
Select	16.9	34.7	11.8	0.25
Low Choice	35.2	35.2	11.6	1.00
≥ Avg Choice[3]	46.9	28.4	12.5	0.29
Marbling Score[2]	600	546	23	0.11
Intramuscular Fat, %	4.55	3.84	0.31	0.11

[1]Steers supplemented with Chromium (Cr) received 0.454 kg ground corn/hd·d^{-1} fortified with 3 mg Cr (KemTRACE Chromium propionate 0.4%; Kemin Industries Inc., Des Moines, IA) as a top-dress. Control (Cont) steers were fed 0.454 kg ground corn/hd·d^{-1} with no additional Cr.

[2]Marbling Scores: 400=slight, 500=small, 600=modest.

[3]Represents all carcasses graded Avg Choice, High Choice, and Prime.

Table 3.4. Effects of chromium supplementation on glucose and insulin kinetics following an intravenous glucose tolerance test in steers fed 0 mg Cr/d (Cont) or 3 mg Cr/d (CrP)

				P-value[2]		
Item	Cont	CrP[1]	SEM	FT	DOF	FT × DOF
Animals, n	17	17	--	--	--	--
Glucose, mmol/L						
AUC[3]			22.5	0.40	<0.01	0.01
21 DOF	354.6	284.2		0.04		
98 DOF	237.4	262.2		0.44		
k[5], %/min			0.229	0.56	<0.01	0.02
21 DOF	1.827	1.235		0.07		
98 DOF	2.121	2.436		0.28		
T½[6], min			6.51	0.48	<0.01	0.07
21 DOF	47.79	63.35		0.09		
98 DOF	36.26	29.44		0.40		
Peak			0.341	0.17	<0.01	0.27
21 DOF	13.143	13.314				
98 DOF	15.148	15.767				
Baseline			0.130	0.11	0.65	0.21
21 DOF	5.907	5.767				
98 DOF	6.075	5.688				
Insulin, pmol/L						
AUC			7,451	0.79	<0.01	0.73
21 DOF	34,489	39,090				
98 DOF	62,539	61,908				
Peak			243	0.71	<0.01	0.87
21 DOF	1,127	1,080				
98 DOF	1,904	1,778				
Baseline			35.2	0.85	<0.01	0.56
21 DOF	96.0	123.4				
98 DOF	218.7	205.0				
Insulin:Glucose[4]			60.0	0.34	<0.01	0.27
21 DOF	117.1	127.2				
98 DOF	378.6	253.6				

[1]Steers supplemented with Chromium (CrP) received 0.454 kg ground corn/hd·d^{-1} fortified with 3 mg Cr (KemTRACE Chromium propionate 0.4%; Kemin Industries Inc., Des Moines, IA) as a top-dress. Control (Cont) steers were fed 0.454 kg ground corn/hd·d^{-1} with no additional Cr.

[2]Where FT = the main effect of feedlot treatment, DOF = the main effect of days on feed, and FT × DOF = the interaction of feedlot treatment × DOF. When an interaction occurred ($P \leq 0.10$), the SLICE option (SAS Inst. Inc., Cary, NC) was used to compare treatments at each time point. SEM shown is associated with T × DOF interaction.

[3]AUC = Area Under the Curve

[4]Insulin:Glucose is the ratio for Insulin and Glucose AUC

[5]*k* = Clearance Rate from 5 to 20 min

[6]T½ = Half-Life from 5 to 20 min; time required for the glucose concentration to fall by one-half

Figure 3.1. Effects of CrP supplementation on glucose change over time following an intravenous glucose tolerance test (GTT**)**

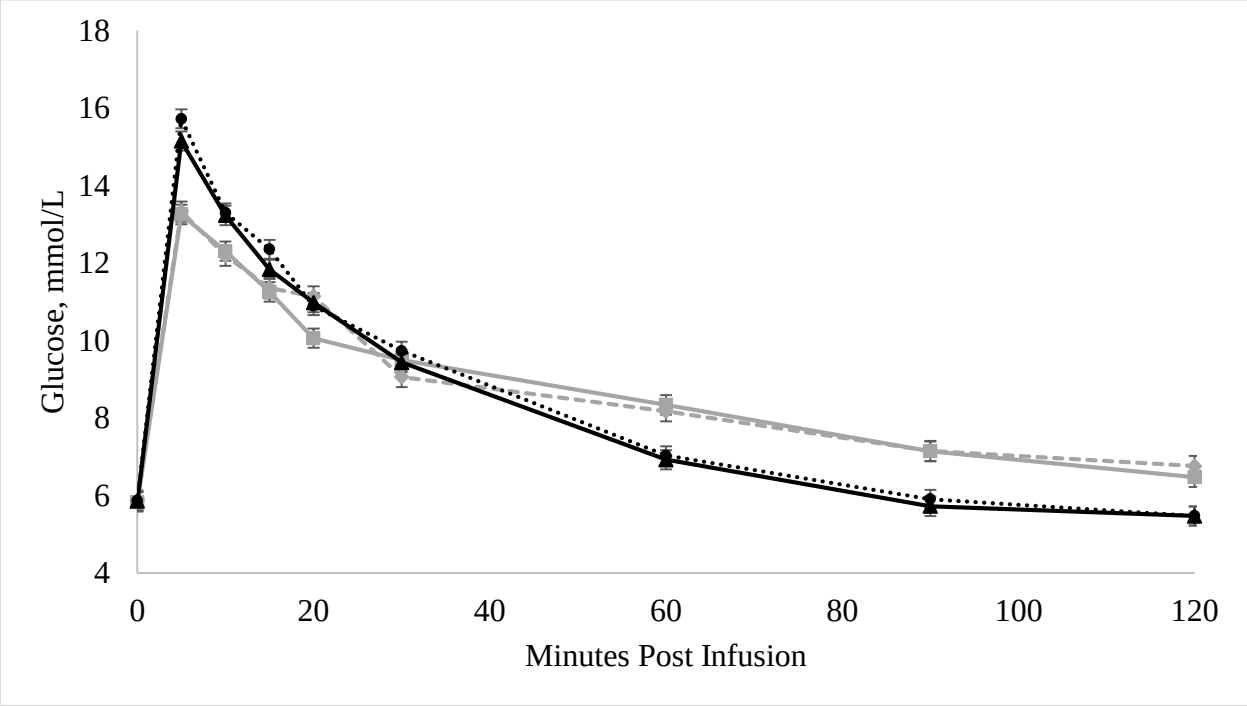

Fig 3.1. Effects of CrP supplementation on glucose change over time following an intravenous glucose tolerance test (GTT). Steers were fed either no Cr-propionate (Cont) or 3 mg supplemental Cr-propionate/d (CrP). Glucose tolerance tests were conducted early in the finishing phase (21 days on feed (**DOF**); grey lines) and later in the finishing period (98 DOF; black lines) (21d CrP, 21d Cont, 98d CrP, 98d Cont). Steers (n = 34) supplemented with CrP received 0.454 kg ground corn/hd·d^{-1} fortified with 3 mg CrP (dashed lines; KemTRACE Chromium Propionate 0.4%; Kemin Industries Inc., Des Moines, IA) as a top-dress; Cont were fed 0.454 kg ground corn/hd·d^{-1} (Cont = solid line). There was a fixed effect FT × Time × DOF (P = 0.08; SEM = 0.251). FT × DOF (P = 0.74) and FT × Time (P = 0.50) did not differ.

Figure 3.2. Effects of CrP supplementation on insulin change over time following an intravenous glucose tolerance test (GTT)

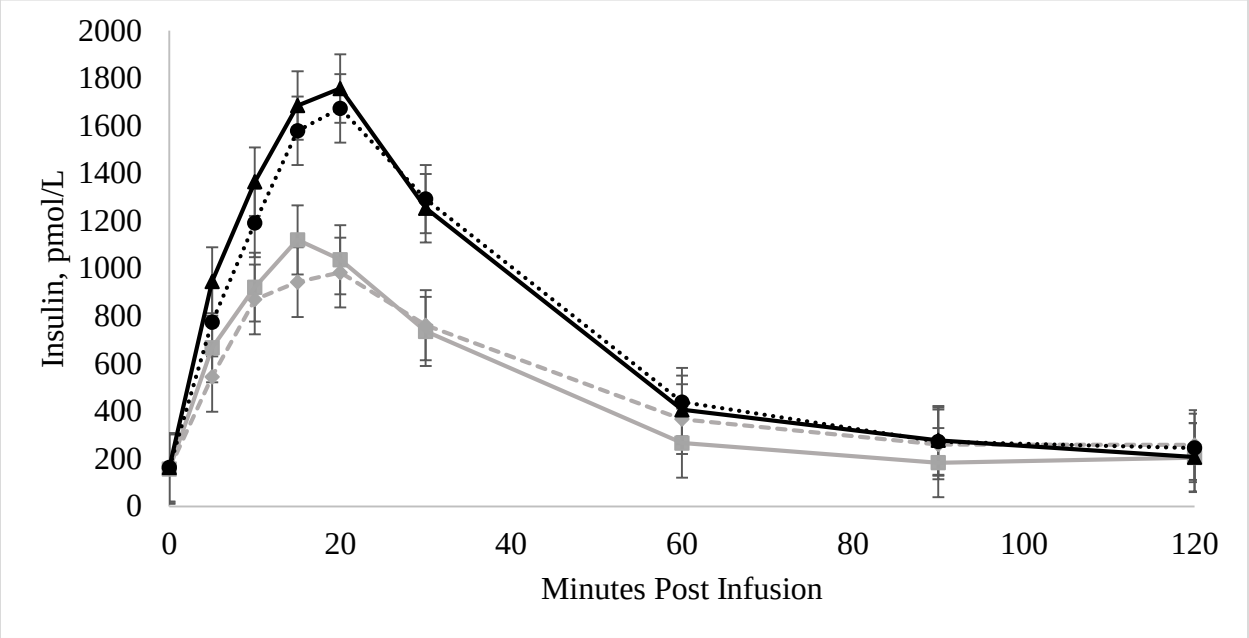

Fig 3.2. Effects of CrP supplementation on insulin change over time following an intravenous glucose tolerance test (GTT). Steers were fed either no Cr-propionate (Cont) or 3 mg supplemental Cr-propionate/d (CrP). Glucose tolerance tests were conducted early in the finishing phase (21 days on feed (**DOF**); grey lines) and later in the finishing period (98 DOF; black lines) (21d Cr, 21d Cont, 98d Cr, 98d Cont). Steers supplemented with CrP received 0.454 kg ground corn/hd·d^{-1} fortified with 3 mg CrP (dashed lines; KemTRACE Chromium Propionate 0.4%; Kemin Industries Inc., Des Moines, IA) as a top-dress; Cont were fed 0.454 kg ground corn/hd·d^{-1} (Cont = solid line). There was no interaction (P = 1.00; SEM = 145.25) of FT × Time × DOF. FT × DOF (P = 0.81) and FT × Time (P = 0.84) did not differ.

CHAPTER 4: CONCLUSIONS

Research with Cr supplementation is limited and Cr is challenging to analyze. There are minute concentrations of Cr in the basal diet and the bioavailability of Cr in basal diet is less than the bioavailability of supplemented Cr-Prop. The amount of supplemental Cr permitted by the FDA is minimal, in most cases, compared to the overall diet. Furthermore, there are no specific cattle requirements established for Cr. In the review of literature preceding the presented research, each study fed a different amount and type of Cr, most fed Cr to younger cattle, and if a GTT was conducted, the glucose infusion rates and doses differed. Only one previous study has examined the effects of Cr during gestation in beef cows, and there is no previous research in beef feedlot steers during the finishing period on the effects of Cr on insulin function. These limitations make study design in Cr supplementation trials challenging. Therefore, 2 experiments were conducted to test the effects of FDA approved feeding levels (target 3 mg Cr/hd·d^{-1}) and source, chromium-propionate, to cows in gestation and feedlot steers throughout the finishing phase.

In the first experiment, we had hypothesized that Cr supplementation would improve cow glucose metabolism and have carry over effects to the fetus, improving growth performance of progeny after parturition. However, we observed no effect of CrP on cow insulin sensitivity and no effect of CrP supplementation on cow or calf performance. In the second experiment, we hypothesized that Cr would increase insulin sensitivity and glucose metabolism in feedlot steers and therefore, improve growth performance and intramuscular fat deposition. However, steers fed CrP took longer to reach an optimal finishing point, tended to have decreased marbling, but had increased dressing percentage and LM area. There were no effects of CrP on insulin

function; however, with increased days on feed, all steers, regardless of treatment, became more insulin resistant, as indicated by steers using more insulin to clear less glucose.

In practical production settings, supplementing Cr to beef cows may not be efficacious, as there were no observed benefits to cows or progeny up to weaning in the present experiment. While contrary to our hypothesis, the observed increase in LM area and dressing percentage suggest feeding Cr-propionate may have advantages in feedlot cattle. However, in the present trial, these changes did not lead to increased HCW, the primary economic driver of finishing cattle.