APPLICATION OF FEED ADDITIVE TECHNOLOGIES TO ENHANCE HEALTH AND PERFORMANCE OF RANGE BEEF COWS

By

SARA KOREN LINNEEN

Bachelor of Science in Animal Science
University of Arizona
Tucson, Arizona
2005

Master of Science in Animal Science
Kansas State University
Manhattan, Kansas
2007

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APPLICATION OF FEED ADDITIVE

TECHNOLOGIES TO ENHANCE HEALTH AND

PERFORMANCE OF FORAGE-FED CATTLE

Dissertation Approved:

Dr. David Lalman

Dr. Gerald Horn

Dr. Scott Carter

Dr. Jerry Ritchey

Dr. Carla Goad

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“Always do the things you fear the most. Courage is an acquired taste, like caviar.” – Erica Jong

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Abstract: A total of 260 cows and 7 cannulated steers were used in four studies to the effects of Bio-Mos, monensin, and slow release urea on cow and calf performance, passive immunity, and in situ digestibility. All experiments were conducted at the Oklahoma State University North Range unit using Angus and Angus X Hereford cows and crossbred steers. All experimental diets were fed as a protein supplement containing at least 38% crude protein and: 10 g/head/d of Bio-Mos, 200 mg/head/d of monensin (cows), 250 mg/head/d monensin (steers), and 526 g of slow release urea. Feeding cows Bio-Mos in late gestation did not improve the transfer of passive immunity to the calf ($P > 0.19$), or calf growth performance ($P > 0.43$), but it improved cow BCS change for the trial duration ($P = 0.05$). Cows consuming monensin during late gestation and lactation did not have improved performance ($P > 0.19$) or milk yield ($P > 0.41$); however, calves born to cows consuming monensin had greater ADG from birth to the end of the feeding period ($P = 0.04$). Monensin also did not improve blood glucose of the cow two hours after eating ($P > 0.16$). When fed to steers, monensin tended to reduce DMI ($P = 0.07$), and increased digestibility of NDF, ADF, and DM ($P < 0.01$). It also significantly increased ($P = 0.01$) propionate at the expense of acetate and propionate indicating that steers had improved energetic efficiency from consuming monensin. Combining monensin and slow-release urea appeared to lessen the BW loss among cows consuming only slow release urea. Replacing a portion of the cottonseed meal with slow release urea did not reduce animal performance, or digestibility, making it a valid and less expensive replacement for true protein in the winter cow supplementation program. Calf growth performance is improved by feeding cows monensin, making it an effective dietary option for improving preweaning efficiency.

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CHAPTER I

INTRODUCTION

Recently cow-calf producers in the Southern Great Plains have faced extreme drought conditions that have made traditional nutritional programs challenging. Using feeding technologies to improve feed efficiency will result in less grass consumption per cow, allowing producers to maintain their herd during time of drought, or increase stocking density in time of high growth. For example, potentially improving health of the calf from birth to weaning using Bio-Mos (Alltech, Inc., Nicholasville, KY) may result in healthier calves at weaning. Producers are currently using monensin (Rumensin 90®; Elanco Animal Health; Greenfield, IN) in protein or mineral supplements to decrease forage intake while maintaining or improving cow condition. A more comprehensive understanding of monensin and its impact on cow calf production is needed to help producers understand the effects of incorporating it into their nutritional programs. Additionally, slow release urea (Optimase®, Alltech, Inc., Nicholasville, KY) can be used as a less expensive source of nitrogen to replace a portion of cottonseed meal or soybean meal in cow supplements. Combining monensin and slow release urea may provide producers yet another nutritional tool to improve cow efficiency and forage utilization while using a lower cost source of crude protein. This research is aimed at

providing information about commonly used nutrition products in the Southern Great Plains that may help producers make sound decisions to improve overall efficiency and decrease cost of production.

CHAPTER II

LITERATURE REVIEW

Passive Immunity

Passive immunity is defined as temporary immunity for the calf after consuming the dams immunoglobulin (Ig) rich milk known as colostrum. Colostrum provides a complete diet at birth and is the sole source of nutrition during this time. Colostrum is particularly important to the health of the calf because the bovine placenta does not allow large protein molecules such as Ig to pass from dam to calf in utero, making the calf relatively defenseless against infectious disease challenges at birth (Waldner and Rosengren, 2009). Calf efficiency of absorption of Ig is incredibly important in ensuring that the calf absorbs the Ig after the colostrum is consumed. Efficiency of absorption is defined as serum Ig divided by Ig intake and is influenced by multiple factors such as age at first feeding and stress level of the calf (Quigley and Drewry, 1998). Previous research has verified the importance of Ig intake for calf health and survival directly after birth. Consequently, current research has focused on analyzing strategies to improve quality and level of Ig in the colostrum along with the efficiency of absorption of passive immunity.

Colostrum contains many constituents including growth factors, cytokines, and

vitamins, to name a few. Hammon et al. (2013) reported that colostrum not only provides immune protection, but it also serves to stimulate protein synthesis, cell growth, and digestive functions. These authors also reported that colostrum impacts both fetal and calf energy supply by increasing glucose absorption. The antibodies included in colostrum include IgG (1 and 2 subclasses), IgA, and IgM, and they are identified based on the structure of the constant region of their heavy chain. Immunoglobulin G functions to bind, neutralize, and promote the removal of antigens (Frandson, 2003) and is concentrated in colostrum (Stelwagen et al., 2009). It has the ability to move from blood to tissues to facilitate destruction of pathogens (Goodier and Williams, 2012). Immunoglobulin G_1 makes up approximately 90% of colostral and 50% of serum IgG, so this response is often used as the primary measurement of the transfer of passive immunity from dam to calf (Franklin et al., 2005; Hurley and Theil, 2011). Immunoglobulin A in colostrum is responsible for protecting mucosal surfaces, whereas IgM is responsible for forming natural antibodies to promote early immune responses (Frandson, 2003). Early immune risks may include cold stress, diarrhea, pneumonia, septicemia, and bacterial challenges (Garry, 1995; Selk, 1998).

The Ig enters the mammary gland through transport mediated by the neonatal constant fragment (Fc) receptor (Stelwagen et al., 2009; Hurley and Theil, 2011). The Ig is transported to the apical end of the mammary secretory cell, released into the alveolar lumen, and then incorporated into the colostrum through the neonatal Fc receptor (Hurley and Theil, 2011). At this time the cow will be immunocompromised because she is transporting all Ig into colostrum (Hurley and Theil, 2011). At birth, the calf consumes colostrum if it is able to stand promptly and nurse. Colostrum is subsequently absorbed

by the intestine without digestion because most Ig are resistant to intestinal enzymes and protected by a trypsin inhibitor. At approximately 24 to 36 hours postpartum, there is a decline in this protection causing reduced absorption capability of the intestinal cells (Hurley and Theil, 2011). This closure process inhibits the calf from absorbing whole Ig and receiving immune protection. Feeding colostrum beyond this point is important because Ig will line the digestive tract to prevent bacterial infection. It has also been suggested that Ig provides immune protection for the mammary gland itself. The mammary gland is appealing to pathogens due to a favorable body temperature and teat opening that exposes the gland to the external environment (Stelwagen et al., 2009).

There are many factors that affect the Ig concentration in colostrum and the calf's ability to efficiently absorb an adequate amount of Ig. The concentration of Ig in colostrum is heavily influenced by the previous health status of the dam (Quigley, 2007). Cattle produce Ig in response to health challenges such as mastitis, immunization, or any specific diseases (Hurley and Theil, 2011). Animals exposed to various health challenges over their lifetime, often due to unsanitary conditions, will have increased immune components in colostrum (Stelwagen et al., 2009). Age and breed of the cow can heavily influence the amount of colostrum produced and the concentration of Ig within the colostrum. Dairy breeds produce much more colostrum and milk, but with less concentration of Ig. Alternatively, beef breeds produce less milk, but have a greater concentration of Ig within the colostrum and milk. High parity cows will produce higher quality colostrum than heifers, but often a more ideal udder and teat composition of the heifer makes up for lower colostrum and their poorer mothering skills (Selk, 1998).

Both Quigley (2007) and Herr et al. (2011) have emphasized great sensitivity in Ig measurements collected around parturition and the importance of maximizing the number of experimental animals. There are great differences in published research in the time of collection of colostrum samples relative to birth, especially in an extensively managed system such as beef cow-calf production systems. This results in variability among published Ig colostrum values simply due to experimental design and management.

Even with an adequate concentration of Ig in the colostrum, the calf may not always be able to consume colostrum or efficiently absorb Ig. This may be due to the age at first feeding, health status of the calf, mothering ability of the cow, and stress (Quigley and Drewry, 1998; Herr et al., 2011). The age at first feeding of the calf is important because the amount of time for Ig to be absorbed by the calf is restricted. This is especially important in the dairy industry, where calves are removed from their dam at birth and fed stored colostrum or milk replacer. The health status of the calf affects Ig absorption because they need to be healthy enough to nurse to receive colostrum. Furthermore, calf vigor at birth often determines how quickly they stand to nurse. Selk (1998) suggested that if the cow does not lick the calf to establish a bond, the cow may not let the calf nurse at all. Older cows often have a pendulous udder that is difficult for the calf to find and suckle. Extreme weather is another stressor that has been shown to influence absorption of Ig if the calf cannot quickly nurse (Quigley, 2007) or if heat stress decreases efficiency of absorption (Selk, 1998).

Fetal program, characterized by nutritional influences of the cow in gestation on the health and productivity of her offspring, is currently a popular area of research.

Thoroughly discussing this topic is beyond the scope of this review; however, Table 1 summarizes the effects of beef cattle prepartum nutrition on dam and calf passive immunity. Research in this area is limited because it is difficult to collect responses indicative of passive immunity transfer in unconfined animals. The high variation in reported results from these studies makes it very difficult to draw any definitive conclusions. One interesting characteristic of the summary is that calf serum IgG does not always follow the pattern of change of IgG level in the dam's colostrum (Shell et al., 1995; Hough et al., 1990; Blecha et al., 1981), suggesting that the calf does not absorb all Ig present in the colostrum. This may simply be due to experimental design and time of sample collection relative to parturition. Sasaki et al. (1976) reported a steady increase in IgG_1 production up to time of parturition that may explain why the quantity of IgG_1 in the colostrum, or calf serum, does not match that of the cow serum.

Two studies reported in Table 1 used Holstein dairy cows as their subjects, rather than a beef breed (Hook et al., 1989; Burton et al., 1984). Dairy calf management requires that the calf be removed from the dam and most often they are fed a blend of colostrum and milk replacer. Calves are left on their dam much longer in beef production systems and this ensures that they typically receive the necessary colostrum from their birth dam, to establish immunity that the calf needs to be healthy and productive. These two dairy studies were designed similarly to the beef cattle studies and also reported inconclusive results for the impact of nutritional restriction during gestation on IgG level of calf colostrum.

Mannan Oligosaccharide

Calf health and nutrition immediately following birth is extremely important, regardless of whether the calf will be consuming their dams milk, a mixed colostrum source, or milk replacer. There has been an abundance of overuse of antibiotics in milk replacer for dairy calves and this has lead to investigation into alternative sources to antibiotics (Terré, et al., 2006), such as prebiotics. As summarized by Zhao et al. (2012), Gibson and Roberfroid (1995) defined prebiotics as any indigestible food ingredient that increases bacteria growth in the digestive system. Prebiotics are recognized to improve gastrointestinal tract (GIT) health resulting in healthier and more productive animals. Mannan oligosaccharide is a prebiotic comprised of mannose sugars from a yeast cell wall that blocks colonization of pathogens in the digestive tract (Che et al., 2011; Franklin et al., 2005).

There are multiple yeast products currently marketed to the livestock industry. One mannan oligosaccharide, called Bio-Mos® (Alltech, Inc., Nicholasville, KY), is a product derived from the cell wall of *Saccharmyces cerevisiae*. Bio-Mos acts in the GIT by blocking the pathogen colonization of the intestinal mucosa or directly binding to the pathogen, carrying them out of the gut because mannan oligosaccharide is indigestible. It has been postulated that Bio-Mos improves gut health, allowing the dam to use nutrients and energy for immunoglobulin production (Alltech, Inc., Nicholasville, KY, personal communication) rather than an immune response. Other authors have proposed that Bio-Mos directly affects immunity by suppressing prolonged inflammation in pigs infected with Porcine Reproductive and Respiratory Syndrome (Che et al., 2011). Multiple studies have established that Bio-Mos does improve performance and immune function when consumed by monogastric animals especially. Bio-Mos fed to sows in late

gestation (12 to 14 d prefarrowing) has been shown to increase piglet weaning weight (Newman et al., 2001; O'Quinn et al., 2001) compared to sows fed a control diet without Bio-Mos. It has also been shown to either numerically (Newman et al., 2001) or significantly (Quinn et al., 2001) increase the level of IgG in the colostrum of sows fed a gestation diet containing Bio-Mos compared to those fed without Bio-Mos.

Bio-Mos has been studied in a variety of species including poultry, swine, and cattle. The most recent studies on feeding Bio-Mos to cattle have focused on the dairy industry and used either Holstein or Jersey calves. In one study, Bio-Mos was included in Holstein calf milk replacer (4 g/d) and compared alongside milk replacer containing no additives or antibiotics. The study reported a significant increase in grain feed intake at week six of age, a decline in the probability of calf scours, and that Bio-Mos provided similar results to that of an antibiotic addition to milk replacer, qualifying it as a good replacement for antibiotics (Heinrichs et al., 2003). Similar to the study by Heinrichs et al. (2003), Bio-Mos was included in the milk replacer of Holstein calves at a higher rate of 10 g/d in a study by Morrison et al. (2010). The author compared Bio-Mos inclusion with a control (no additive), probiotic, and probiotic plus Bio-Mos. The authors found that Bio-Mos did in fact increase feed intake of calves at four weeks of life; however, this did not translate into an increase in live weight or earlier wean age, nor did they see a decline in the number of scour episodes due to the addition of Bio-Mos compared to the other treatments (Morrison et al., 2010). These results were supported in a study by Terré et al. (2006) when they compared Bio-Mos in the milk replacer at 4 g/d to a control, no additive milk replacer and found an improvement in feed efficiency, an increase in starter

intake, and no significant effects on reducing fecal bacterial counts as compared to calves fed a control milk replacer diet.

A study completed by Franklin et al. (2005) included a control diet compared with Bio-Mos at 10 g/d in a total mixed ration for the cows the last three weeks of gestation, rather than including it in the milk replacer like previous studies. The experiment used a combination of Holstein and Jersey cow-calf pairs (at least 19 cows/treatment) with data from a total of 39 cows and 41 calves used for analysis. There were no treatment effects on cow body weight, white blood cell count, the level of total Ig in the blood, or the amount of colostrum produced. There was also no treatment affect for calf birth weight, level of IgG_1 (which makes up 90% of Ig) in the calf serum, or packed cell volume. There was, however, a decrease in the level of IgA found in the serum of calves born to cows on the Bio-Mos feeding treatment. The authors indicated that they vaccinated the cows twice for rotavirus and found that the neutralization titers were greater for cows fed Bio-Mos. The calves belonging to these cows had a numerically higher serum protein concentration from birth to 24 hours old, which the authors suggested may imply a better transfer of passive immunity from dam to offspring for cows fed the Bio-Mos dietary treatment. There was a limited amount of evidence that Bio-Mos, when fed in late gestation, may improve passive immunity in the dairy calf.

Improvement of calf passive immunity by dairy cows and heifers through the addition of Bio-Mos in either the milk replacer or the cow gestation diet is limited. There is currently no published research on feeding Bio-Mos to beef cows. Perhaps in extensively managed herds like cow-calf production, the responses necessary to improve immune status, health, or animal performance are difficult to measure. Moreover,

nutritional strategies for beef cows often intentionally place them in an energy or protein deficient status. Bohnert et al. (2013) found that cows unsupplemented during winter dormancy gave birth to calves that had lighter BW, and subsequently lighter wean weight, than calves born to protein supplemented cows. Blecha et al. (1981) also reported a linear reduction in calf serum IgG_1 as cow protein intake declined, indicating that transfer of passive immunity is influenced by protein intake. Collectively these results indicate that beef cows may have nutritional challenges that are not present in studies with dairy cattle consuming Bio-Mos. This may determine whether Bio-Mos would have a similar mode of action in beef cows as it has been demonstrated in monogastrics and dairy cows.

Monensin

Monensin is a growth promoting ionophore derived from *Streptomyces cinnamonensis* (Clanton et al., 1981) that is widely used in diets for feedlot cattle under the trade name Rumensin® (Elanco Animal Health, Greenfield, IN). Approved by the Federal Drug Administration for use in feedlot cattle in 1976, by 1978 over 80% of feedlot cattle were receiving monensin (Owens, 1980). Monensin has been well established to increase propionate at the expense of acetate and butyrate, resulting in improved energetic efficiency by acting on the system according to three main mechanisms. Monensin disrupts ion channels in gram positive bacteria, due to lack of lipopolysaccharide outer membrane (Russell and Strobel, 1989), although some gram positive bacteria are resistant. These would include small and large *Entodinia* (van der Merwe, 2001) and *Streptococcus bovis* (Dawson and Boling, 1987). Bacteria use energy during this disruption to remove hydrogen from the cell, while pumping sodium and

potassium back into the cell (Wallace et al., 1980). Hydrogen cannot be removed from the cell fast enough resulting in a decreased pH causing lysing of gram positive bacteria. Without gram positive bacteria proliferating, gram negative bacteria proliferate causing an increase in succinate production. Increased production of succinate is beneficial because it leads to increased propionate levels and ultimately more glucose, as propionate is a precursor to glucose (Weimer, 1998). Propionate is the most efficient precursor of glucose among all three VFAs, which results in improved energetic efficiency (Schelling, 1984).

Many gram positive microorganisms use amino acids and peptides as a source of energy; therefore, monensin spares protein by decreasing the number of gram positive bacteria. Turner et al. (1988) reported that cows maintain more BW through calving and early lactation when they consume 90% of the protein requirement plus monensin compared to cows fed 100% of the protein requirement with no monensin, suggesting improved protein and forage utilization by beef cows. Similarly, Muntifering et al. (1980) reported that feeding monensin to steers improved crude protein digestibility potentially through the increase in propionate in the rumen, sparing amino acids that may otherwise be deaminated for gluconeogenesis. Finally, monensin reduces metabolic disorders, such as acidosis, and acts as a coccidiostat by decreasing coccidia prior to reaching the small intestine (Goodrich, 1984).

Monensin consistently reduces ruminal ammonia concentration (Lemenager et al., 1978b; Tolbet at al., 1977; Dinius et al., 1976) when fed to cattle. Yang and Russell (1993) suggested that microorganisms sensitive to monensin produce ammonia more than those not sensitive to monensin. Deceased rumen ammonia concentration may be

advantageous for maintaining optimum ruminal ammonia level at 5-8 mg·100 ml^{-1} rumen fluid for maximum rate of microbial growth (Satter and Slyter, 1974; Owens and Zinn, 1993). This is especially important in times when rumen ammonia levels may be excessively high, such as when cattle are fed urea (Lemenager et al., 1978b).

Research indicates that monensin reduces the protozoa population in the rumen (Ankrah et al., 1990; van der Merwe et al., 2001), resulting in an increase in propionate, bacterial protein, and bypass protein, as well as a decrease in pH (Lana and Russell, 1998). Since protozoa are beneficial for degrading fibrous ingredients when cattle consume high fiber rations, defauntinization is both favorable for sparing protein and unfavorable by reducing forage digestion. This may partially explain the more variable monensin response in grazing animals compared to feedlot animals (Sprott et al., 1988; Beauchemin et al., 2004).

In a monensin meta-analysis on feedlot animals, ADG increased linearly with increasing monensin in the diet while DMI linearly decreased (Duffield et al., 2012). It has been postulated that the response to monensin in feedlot animals is more consistently a reduction in DMI, rather than an improvement in ADG (Owens, 1980), but generally recognized that monensin serves to improve growth efficiency. Monensin as a coccidiostat is especially important in the feedlot segment. In feedlot animals where lactate production is higher because of cereal grain consumption, monensin increases pH (Vagnoni et al., 1995) by decreasing lactate production (Russell and Strobel, 1989). Monensin has also been shown to reduce daily pH variation in feedlot animals, lending itself to decreased propensity for digestive disorders (Cooper et al., 1997).

The effects of feeding monensin to grazing animals are highly variable, especially for beef cows. Feeding monensin to cows during gestation decreased forage intake, accompanied by either an improvement (Barnett et al., 1982; Sexten et al., 2011) or no change in cow gain (Walker et al., 1980; Moseley et al., 1977). Although Lemenager et al. (1978a) reported reductions in cow DMI when cows consumed monensin during lactation, other studies did not measure intake (Hixon et al., 1982; Bailey et al., 2008). Turner et al. (1980) allotted cows consuming monensin to consume either 92 or 89% of the hay that Control cows (100% hay and no monensin) consumed. They reported no reductions in cow gain or subsequent calf performance among all of the cows suggesting that less hay can be fed and body condition maintained with monensin.

Increased milk production efficiency from reduced forage DMI and no change in milk yield has been identified as a common result of feeding monensin in the dairy industry (Duffield et al., 2008b). Duffield et al. (2008a) determined that precalving blood glucose concentrations were lower when cows consumed monensin, and this may be indicative of the fetus receiving the glucose generated from monensin. Increases in milk yield of 2.3% (Duffield et al., 2008b) and 5% (McGuffey et al., 2011) have been reported when monensin is fed to dairy cows. Therefore, it is reasonable to speculate that the increased energy from monensin is being partitioned for lactation. In beef cows, Lemenager et al. (1978a) reported a decrease in forage DMI accompanied by no change in milk yield causing improved milk production efficiency. Others also reported no improvement in milk yield when feeding monensin to beef cows (Randel and Rouquette, 1976; Hixon et al., 1982; Grings and Males, 1988). Sprott et al. (1988) acknowledged the great variation in breed differences and time of milk collection relative to parturition

among studies measuring milk parameters in beef cows. Very few beef cow studies have reported milk yield, so the inconsistent results in this area may be a function of too little data.

Very few studies have fed beef cows monensin during lactation and reported subsequent calf growth performance. Clanton et al. (1981) reported a significant increase in calf birth weight among calves born to cows consuming monensin and Lemenager et al. (1978a) reported an increased ADG of calves among calves from cows consuming monensin, although the authors acknowledged that the calves had access to the supplement containing monensin. Conversely, Burrell, (1980), Turner et al. (1980), and Walker et al. (1980) found no improvement in calf gain when cows consumed monensin, even when forage was fed at 90% of the control allotment in these studies.

The literature on feeding monensin to beef cows is rather inconclusive on what benefits it offers in measurable cow performance, milk yield, or calf growth performance. Many of the studies on this topic are dated, used very few experimental units, did not measure forage intake, and had substantial differences in forage quality. All of these factors could have contributed to the inconsistent responses among the limited number of studies. Regardless, monensin appears to improve energetic efficiency of the cow, which is translated to improvements in cow performance in some studies, or calf performance in others.

Urea

Nonprotein nitrogen (NPN) compounds contain nitrogen (N) that is not associated with protein (Akay et al., 2004). These compounds include amines, amino acids, nucleic acids, nitrates, and urea, to name a few (Huntington and Archibeque, 2000). Urea

contains 287% CP (45.6% N) and is the most commonly fed NPN because it is often less expensive per unit of crude protein ($0.06/unit protein for urea) compared to an oilseed protein such as soybean meal ($0.46/unit protein) or cottonseed meal ($0.39/unit protein) using April 2014 prices. When ruminants consume urea, urease converts it to ammonia in the rumen which is subsequently used to meet the N requirement of the microbes. Excessive ammonia will be excreted through the urine. Recycling of urea often masks a deficiency in degradable intake protein (DIP) if the metabolizable protein requirement is being met (NRC, 1996). If diets are formulated to contain a surplus of DIP, urea will not be beneficial (Russell et al., 1992). Urea has traditionally been used to replace a portion of plant protein to reduce dietary costs, while maintaining or slightly reducing animal performance.

Multiple experiments testing the effectiveness of NPN in a protein supplement included a negative control treatment for comparison. Rush et al. (1976) reported that cows lost less BW when they received a 30% CP supplement with either urea or biuret (slow release urea) compared to a negative control treatment containing 15% CP. Similarly, Currier et al. (2004) reported that unsupplemented cows lost more BW and condition than cows provided at least 28% CP from a supplement containing either urea or biuret. These authors also reported no differences in forage intake among cows. Forero et al. (1980) also used a negative control supplement containing 15% CP to compare performance of animals consuming a supplement with 40% CP from soybean meal, slow release urea, or urea. They reported an increase in forage intake and less BW and BCS loss among cows receiving urea or slow-release urea in comparison to cows being fed a diet deficient in protein.

Among studies comparing NPN inclusion to a positive control treatment, Ammerman et al. (1972) also reported no reduction in forage intake among wethers when comparing a treatment containing 37% CP from cottonseed meal to a 37% CP supplement containing biuret. Farmer et al. (2004) reported similar results to that study when they supplied 30% of the supplement DIP from urea compared to a supplement with all DIP supplement from soybean meal. Not only was DMI not affected by including urea in the diet at this level, but cow and calf performance was not different among animals on either treatment. In the second experiment in Farmer et al. (2004), the authors reported more BW and BCS loss when urea supplement 45% of the supplement DIP indicating that inclusion greater than 30% of supplied DIP is not acceptable. In an older study, Forero et al. (1980) also reported negative results when comparing urea, slow release urea, and soybean meal as protein sources in a 40% CP supplement. Cows consuming urea or slow release urea lost significantly more BW and BCS than cows consuming soybean meal, indicating that urea cannot completely replace oilseed in a protein supplement. Similarly, Lemenager et al. (1978b) found more weight and condition loss among cows consuming slow-release urea compared to cows on a positive control. Urea is not as efficiently utilized by the animal because of the rapid release of ammonia in the rumen (Chalupa et al., 1968), and this is reflected in the performance of grazing animals in studies comparing a treatment containing urea to a positive control group of cattle. The cost effectiveness of protein per unit N that urea has compared to an oilseed protein should be considered, as it may compensate for the negative impact on animal performance of feeding urea.

An advantage of including urea in the diets is that bacteria fermenting structural carbohydrates prefer ammonia as a source of N compared to amino acids (Köster et al., 2002). Unfortunately, urea is rapidly hydrolyzed in the rumen causing quick ammonia release and asynchrony between ammonia release and carbohydrate degradation. Rumen microorganisms use ammonia for growth, but growth is dependent on energy availability, and this is often an obstacle in using urea in high forage diets (Oltjen et al., 1968; Johnson, 1976). In theory, synergy is established by matching ammonia release with the rate of digestion of energy yielding dietary components for maximum microbial efficiency (Owens and Zinn, 1988). Many researchers have suggested that a slower ammonia release will allow for more ammonia to be assimilated to microbial protein and less lost to escape (Chalupa, 1968; Akay et al., 2004; Taylor-Edwards et al., 2009). This has lead to the idea of slow-release urea in order to match carbohydrate digestion with ammonia release.

Slow-release urea products that have been studied include biuret, Starea®, linseed oil-coated urea, isobutylidine monourea, formaldehyde-treated urea, and others. Biuret has been researched extensively in comparison to urea, and is less water soluble as well as more slowly released to ammonia than urea (Currier et al., 2004). Oltjen et al. (1968) demonstrated the rumen ammonia release rate of urea versus biuret, eliciting further research in this area. Since that time, many of the slow-release products have been discredited because the slow-release rumen ammonia response did not translate to any improvement in performance (Males et al., 1979; Martin et al., 1976; Forero et al., 1980). The idea of synchrony has also been questioned and Krehbiel et al. (2007) suggested that synchrony in the rumen is not possible because of urea recycling. Without improvements

in animal performance over a natural protein, it appears that synchrony between carbohydrate digestion and ammonia release is either not achieved using slow-release products, or it is not possible.

Optimase® (Alltech, Nicholasville, KY) is a feed additive product containing 256% CP for cattle that combines a urea coated in a biodegradable polymer and fibrolytic enzyme technology. There is no published research evaluating the efficacy of Optimase® as a feed additive for beef cows; however experiments have studied Optigen® (Alltech, Inc., Nicholasville, KY), which is the trade name for the slow-release NPN product in Optimase®. Optigen® is urea coated in a biodegradable polymer, which causes controlled release of the urea (Akay et al., 2004; García-González et al., 2007). While having a similar N content as urea, Optigen® has been shown to effectively partially replace soybean meal in supplements when heifers consumed low quality forage diets (Kononoff et al., 2006). In an in-situ digestibility study, Akay et al. (2004) suggested that Optigen® and soybean meal have similar rate of N release. The authors found that nitrogen disappearance of Optigen® over 30 h more closely matched disappearance of soybean meal than urea. Consequently, the suggested use of Optigen® is as a partail replacement for soybean meal, or an oilseed equivalent.

Inclusion of Optigen® in cow supplements often results in no reduction in cow performance, blood metabolites, or milk yield, indicating that it successfully replaces other sources of protein in the diet. Wahrmund et al. (2007) fed beef cows no supplement, urea, or Optigen® while having ad libitum access to bahiagrass and reported no differences in cow BW, BCS, blood glucose or blood urea nitrogen. The authors reported an improvement in DMI the last 4 weeks of the 8 week study for cows

consuming NPN compared to those consuming no supplemental protein. Kononoff et al. (2006) fed high forage diets containing Optigen® and also reported no change in performance, plasma urea nitrogen, or DMI in dairy heifers compared to heifers consuming a supplement containing soybean meal. Inclusion of Optigen® in isonitrogenous total mixed rations (TMR) also has no impact on milk yield (Galo et al., 2003; dos Santos et al., 2008). The limited body of work on Optigen® in cow diets indicates that Optigen® is a suitable partial replacement of an oilseed meal for forage fed cattle.

A novel characteristic of Optimase® is the unique combination of the coated urea with a fibrolytic feed enzyme (FFE) in the form of xylanase (minimum 40 Xu $XU \cdot g^{-1}$). The addition of fibrolytic feed enzymes in the diet improves fiber digestion, often resulting in increased passage rate (Murillo et al., 2000; Beauchemin et al., 2004). Other FFE currently available for livestock feeding include cellulase, amylase, ferulic acid esterase, and any combination of those (Adesogan et al., 2014). Although FFE have the ability to improve digestion, the rumen environment and physiological status of the animal must be ideal for this response to be significant (Eun et al., 2009). In a recent review on the use of FFE in livestock diets,Adesogan et al. (2014) highlighted causes of the variable responses among studies testing FFE. The correct enzyme must be used that has specificity for the substrate, with special consideration of the enzyme potency and the rumen pH as influenced by diet (White et al., 1993; Adesogan et al., 2014). Adesogan et al (2014) reported that xylanase activity is most optimal at a pH of 5, which would suggest that the rumen pH of cattle grazing forage may not be conducive to using a FFE containing xylanase.

Perhaps the ideal rumen environment for the FFE being fed was not achieved in the multiple studies that reported no impact of FFE on digestibility (Pinos-Rodríguez et al., 2002; Ware et al., 2005; Avellaneda et al., 2007). Furthermore, FFE has been shown to have no impact on rumen ammonia concentration (Avellaneda et al., 2008; Giraldo et al., 2008; Hristov et al., 2008). In contrast, Rode et al. (1999) and Hristov et al. (2007) found an improvement in fiber digestion when xylanase was fed. Giraldo et al. (2008), using a xylanase and endoglucanase enzyme, and Phakachoed et al. (2012) using xylanase alone reported improvements in NDF digestion. Feeding a FFE in combination with urea would in theory improve forage digestibility by providing a steady supply of fiber degrading enzymes and rumen ammonia necessary to synchronize ammonia release with carbohydrate degradation (Alltech, Inc., Nicholasville, KY, personal communication).

Davis and Erhart (1976), Lemenager et al. (1978b), Poos et al. (1979), and Vagnoni et al. (1995) conducted studies that combined monensin and urea in a high fiber diets. Both Davis and Erhart (1976) and Poos et al. (1979) used high concentrate diets fed to steers and lambs, respectively. When urea was combined with monensin, Davis and Erhart (1976) reported that steers were more efficient than when monensin was fed with cottonseed meal. Conversely, Lemenager et al. (1978b) demonstrated that cows consuming Starea® lost more weight, regardless of whether they were also consuming monensin, than cows being fed an oilseed protein. Lemenager et al. (1978b), Poos et al. (1979), and Vagnoni et al. (1995) found that combining monensin and urea reduced rumen ammonia concentration compared to a treatment only containing urea. This may

be advantageous for less ammonia to be wasted and presumably more microbial protein production.

Historically, urea has been included in the ruminant diet because it is less expensive source of crude protein (per unit N) than other protein sources, such as soybean meal. It is recognized that urea may reduce animal performance, especially with the quick release of rumen ammonia from urea, and this has elicited interest in slow-release urea products. Even in instances when ammonia released is slowed, slow release urea products generally have not translated to an improvement in animal performance suggesting that synchronizing ammonia release with carbohydrate fermentation may not be possible. Finally, the combination of urea and monensin does reduce rumen ammonia concentration, potentially leading to increased microbial protein production.

Summary

In summary, cow-calf producers are facing drought conditions in the Southern Great Plains that are making traditional nutrition programs for cows challenging. Using feeding technologies to improve feed efficiency will result in less grass consumption per cow, allow producers to maintain their herd during a drought, increase stocking density in a time of growth, and have healthier livestock. Using Bio-Mos in the cow supplement may improve gut health by blocking the colonization of pathogens to the gut cell wall or directly binding to the pathogen and washing it out of the gut. Although the research on this product in beef cattle is nonexistent, feeding it to dairy cows suggests that it will improve the transfer of passive immunity from dam to offspring. Monensin is another feeding technology option for producers to improve forage efficiency by increasing energetic efficiency of the animal. Feeding monensin to grazing animals is sometime

shown to increase cow gain, increase calf gain when nursing from a cow fed monensin, or it may only increase feed efficiency. Monensin improves energetic efficiency of the cow, but the mechanism of this improvement is currently unclear. Finally, replacing a portion of oilseed protein with NPN in the form of urea will reduce the cost of supplementation because urea is less expensive per unit of N than both cottonseed meal and soybean meal. Optimase® is also a form of NPN that has been shown to maintain cow performance compared to feeding cows soybean meal in the supplement. All of these feeding technology options are intended to help producers in the Southern Great Plains make sound decision to improve cow efficiency and health to promote overall animal performance at a lower cost.

Table 1. Summary of research studying the effects of energy and protein supplementation on passive immunity in late gestation cattle diets.

Authors	Breed	N (per trt)	Treatments	Treatment description	Colostrum IgG	Calf serum IgG
Shell et al. (1995) Beef	Bos taurus	4-5 cows, 4 to 11 years old	1. Shade/Low feed intake 2. Shade/High feed intake 3. No shade/Low feed intake 4. No shade/Low feed intake	Final 190 d in gestation: Shade = 5.5 m x 6.9 m High feed = 8.6 kg of Sudangrass/d Low feed = 5.5 kg of Sudangrass/d.	Numeric reduction for cows fed restricted diet	No differences
Hough et al.(1990) Beef	Angus	13 cows, 4 to 8 years old	1. Dam fed control/calf control[1] 2. Dam fed control/calf restricted[2] 3. Dam fed restricted/calf control 4. Dam fed restricted/calf restricted	Final190 d in gestation: Control = ~1.7% of BW DM Intake (100% of protein and energy req'r of the NRC), Restricted = ~0.78% Of BW DM Intake (57% of the protein and energy req'r of the NRC	Significant increase in IgG for cows fed restricted diet	No differences
Blecha et al. (1981) Beef	Hereford or Hereford x Angus bred to Charolais x Red Angus	10-11 heifers	1. 0.52 kg CP DM Basis daily 2. 0.61 kg CP DM Basis daily 3. 0.71 kg CP DM Basis daily 4. 0.80 kg CP DM Basis daily 5. 0.89 kg CP DM Basis daily 6. 0.98 kg CP DM Basis daily	Final 100 d of gestation	No differences	Linear reduction in IgG_1 as CP consumption per d declined
Olson et al. (1981) Beef	Arberdeen Angus	15 cows, 2 years old	1. Control 2. Protein restricted 3. ME energy restricted 4. Protein and ME energy restricted	Final 156 d of gestation: Control = 50.88 MJ ME, 0.96 kg CP Protein restr = 50.88 MJ ME, 0.32 kg CP ME restr = 36.4 MJ ME, 0.96 kg CP Protein and ME restriction = 36.4 MJ ME, 0.32 kg CP	No differences	No differences
Fishwick[3] et al. (1975) Beef	?	14-16 heifers	1. 2.7 kg/d molasses sugar-beet pulp 2. #1 with 30 g urea/kg	Final 114 d of gestation: Oat straw ad-lib from d 14-11 gestation, then hay for the final gestation.	No differences	No differences
Hook[3] et al. (1989) Dairy	Holstein	22 heifers/trt	1. High protein, 2. Low protein	Final 20 d of gestation: High protein = 13%, Low = 9.9%	No differences	No differences
Burton et al. (1984) Dairy	Holstein	13 heifers/trt	1. Protein deficient, 2. Positive control	Final 102 d of gestation: Deficient = 918 g/d, adequate = 1598 g/d	No differences	Dec IgG_1 level in serum of calf w/ protein deficient dam

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CHAPTER III

EFFECTS OF MANNAN OLIGOSACCHARIDE ON BEEF COW PERFORMANCE AND PASSIVE IMMUNITY TRANSFER TO CALVES

S.K. Linneen,* G.L. Mourer,* J.D. Sparks,* J.S. Jennings,† C.L. Goad‡, and D.L. Lalman,*

*Department of Animal Science, Oklahoma State University, Stillwater 74078; †Alltech, Inc., Nicholasville 40356; and ‡Department of Statistics, Oklahoma State University, Stillwater 74078

ABSTRACT

This experiment investigated the effects of feeding mannan oligosaccharide to beef cows during late gestation through 30 d of lactation on cow and calf performance and calf passive immunity. Angus and Angus x Hereford cows (n = 69; BW = 569 ± 68 kg; Age = 5.3 ± 7 yr) were allotted by BW and age in a completely randomized designed. Cows were assigned to 1 of 2 treatments including 1) 1.36 kg/d during gestation of a cottonseed meal-based 30% CP supplement and 1.81 kg/d during lactation of a cottonseed meal-based 38% CP supplement (**Control**); or 2) Control plus 10 g/d Bio-Mos® (**Bio-Mos**; Alltech, Inc., Nicholasville, KY). Experimental

supplementation began on February 14, 2012 and was terminated after cows consumed the lactation diet for at least 30 d. Cow and calf blood and colostrum were collected within 12 h of parturition. Cows fed Bio-Mos tended to maintain more BW from parturition through the end of the feeding period ($P = 0.10$). Similarly, cows consuming Bio-Mos were better able to maintain BCS from initiation of the experiment through weaning ($P = 0.05$). At parturition, no differences for IgG_1 concentrations in colostrum ($P = 0.28$), cow serum ($P = 0.19$) or calf serum ($P = 0.70$) were detected. Similarly, parturition calf serum IgG_2, IgA, or IgM concentrations were not different ($P > 0.14$). Adding Bio-Mos to winter supplement may limit BCS loss following parturition in spring calving beef cows, however there was no impact on passive immunity characteristics.

Key words: calves, cows, immunity, mannan oligosaccharide

INTRODUCTION

Colostrum is particularly important to the health of the calf because the bovine placenta does not allow immunoglobulins (Ig) to pass from dam to calf in-utero, making the calf relatively defenseless against infectious disease challenges at birth (Waldner and Rosengren, 2009). Colostrum provides a complete diet after birth, as well as providing the antibodies necessary for calf survival. Improving the quality and quantity of Ig may alleviate morbidity and mortality among calves in the first weeks of life. Nutritional modifications to milk replacers in the dairy industry to enhance Ig in colostrum have included using antibiotic alternatives such as direct-fed microbials or mannan oligosaccharide (Bio-Mos). Mannan oligosaccharide, in the form of Bio-Mos® (Alltech, Nicholasville, KY), comes from the cell wall of *Saccharmyces cerevisiae* yeast and is known to block colonization of pathogens in the digestive tract while improving immune function (Che et al., 2011; Franklin et al., 2005). An affordable non-antibiotic that may increase health and growth performance such as Bio-Mos, may be valuable to the livestock industry (Franklin et al., 2005). Feeding Bio-Mos to sows in late gestation (12 to 14 d prefarrowing) has been shown to increase piglet weaning BW compared to piglets from sows fed a control diet without Bio-Mos (Newman and Newman 2001; O'Quinn et al., 2001). Including Bio-Mos in milk replacers for dairy calves has provided mixed results, in either improving intake (Morrison et al., 2010; Terré et al., 2006) or improving gain (Heinrichs et al., 2003) by comparison to antibiotics, probiotics or no additions to the milk in the 3 experiments, respectively. Franklin et al. (2005) found no improvements in dairy cow BW or Ig concentration of cow serum, calf serum, or colostrum when they fed Bio-Mos to cows 30 d before parturition. Research indicating

an improvement in the transfer of high quality passive immunity, as measured by Ig concentration, from dairy cows to offspring due to the addition of Bio-Mos is limited. Currently there is no published research evaluating the impact of Bio-Mos on passive immunity characteristics from beef cows to their offspring. Therefore, the objective of this experiment was to investigate the effects of feeding Bio-Mos to beef cows during late gestation through 30 d lactation on cow and calf growth performance and passive immunity transfer to the calf.

MATERIALS AND METHODS

All animal procedures were conducted in accordance with the approved Oklahoma State University Animal Care and Use Protocol. This experiment was conducted at the Range Cow Research Center, North Range Unit, located approximately 16 km West of Stillwater, Oklahoma. Spring calving Angus and Angus X Hereford cows (n = 69; 569 kg initial BW; SE = 8.14 kg; 5.5 initial BCS; SE = 0.07; 5.3 yr initial age; SE = 0.38 yr) were assigned to 1 of 2 dietary supplements in a completely randomized design. Cows were ranked by BW and age and randomly allocated so that BW and age were similar across treatments. Treatment supplements (DM basis) included 1) 1.36 kg/d during gestation of a cottonseed meal-based 30% CP supplement and 1.81 kg/d during lactation of a cottonseed meal-based 38% CP supplement (**Control**); or 2) Control plus 10 g/d (**Bio-Mos**). Supplements were fed as 0.97-cm diam. Pellets, balanced for Ca, P, and Vitamin A, and formulated to meet or exceed NRC (1996) protein requirement of the cow. The gestation control supplement was formulated to provide 27% NDF, 14% ADF, 2.4% fat, 1.11% P, and 0.21% Ca. All cows had ad libitum access to prairie hay (5% CP, 74% NDF, DM basis) for the duration of the experiment.

Individual animal supplementation began on February 14, 2012 and was terminated after cows consumed the lactation diet for at least 30 d. Cows that had not calved by April 10, 2012 were removed from the study resulting in 38 and 31 cows on the Control and Bio-Mos treatments, respectively. All supplementation ended on May 1, 2012 resulting in an experimental treatment period ranging from 52 to 80 d.

Each morning at approximately 0800 cows were fed individually in a barn containing 31 individual feeding stalls to ensure that each cow received the assigned amount of feed. Each d the cows were gathered from a pasture adjacent to the feeding barn and placed into a feeding stall, restrained, and allowed 20 min to consume their dietary supplement. Each cow thoroughly consumed the dietary supplement for the duration of the experiment. Cows were fed the gestation supplement until parturition, when they were switched to the lactation supplement for the duration of the experiment. Cow-calf pairs were separated while the dam consumed the supplement and then rejoined each d.

Cows were managed as a contemporary group during both gestation and lactation. During gestation, cows remained in a single pasture (6 ha) with free access to tall-grass prairie hay (5% CP, 74% NDF, DM basis). At parturition, pairs were moved to a nearby pasture (6 ha) where they had access to tall-grass prairie hay matching the nutrient composition as described above.

Individual cow BW, BCS (scale 1 through 9; Wagner et al., 1988), blood, and fecal samples were collected at initiation of the experiment on February 10, 2012. Blood was collected via coccygeal venipuncture into vacuum tubes (BD Vacutainer) to establish

immune system parameters of the dam. Blood samples were analyzed for serum Ig concentrations and serum protein concentrations (SPC). Approximately 40 g of feces was collected by rectal grab and analyzed for presence of *Salmonella* and coccidiosis. All cows calved without assistance. Within 3 to 12 h from parturition, an individual BW was recorded and a blood sample was collected from each cow (coccygeal venipunture) and calf (jugular venipuncture). A colostrum sample was also collected from the cow at this time. Cows received 1.0 mL injection of oxytocin (20 USP units/mL, intramuscularly; Phoenix Pharmaceutical, Inc., St. Joseph, MO) to facilitate milk letdown. Two hundred and fifty mL of colostrum were collected uniformly from all quarters from each cow. Colostrum was immediately analyzed for colostrum quality using a Colostrometer (BIOGENICS, Mapleton, Oregon) and colostrum samples were frozen at -20° C for later analysis of Ig concentration. Rectal grab samples of feces were collected from the cow approximately 14 d after parturition and at the end of the experiment. After approximately 30 d of consuming the lactation supplement, cows were removed from dietary treatments. At this time, individual BW and BCS were recorded and a fecal sample was collected from each cow. Individual BW, fecal sample, and jugular venipuncture sample were collected from each calf also at this time, which concluded the feeding portion of the experiment. At weaning on September 11, 2012, cow BW and BCS, along with calf BW was also recorded. Calf BW at weaning was adjusted to a 205 d BW with a dam age adjustment factor according to the Beef Improvement Federation and Guidelines (2002).

On February 10, 2012 all cows received an injection of Endovac-Bovi (IMMVAC, Inc., Columbia, MO) for protection against E-Coli mastitis. On May 9, 2012

the cows underwent a pre-breeding vaccination program that included Safe Guard dewormer (Merck Animal Health, Summitt, NJ), Express FP-10 vaccine (Boehringer Ingelheim Vetmedica, St. Joseph, MO), and MultiMin 90 injectable trace mineral (MultiMin USA, Inc., Ft. Collins, CO). The calves were vaccinated with Caliber 7, Presponse SQ, and Pyramid 5 (Boehringer Ingelheim Vetmedica, St. Joseph, MO).

All morbidity, mortality, and medical treatments were recorded throughout the experiment. Cow and calf health was determined by visual appraisal by trained herdsmen once daily. One cow-calf pair from each treatment was removed from the data due to cow mortality (mastitis and unknown cause). Three cows were removed from the Control treatment due to morbidity (1 from mastitis and 2 from *Salmonella* infection) as determined by a veterinarian and 2 cows were removed from the Bio-Mos treatment due to failure to collect data. Three calves from the Control treatment died (2 from *Salmonella* infection, 1 from unknown causes) and 3 from the Bio-Mos treatment died (unknown causes, bone infection, and accidental drowning).

Sample Analysis

Serum was harvested from 10 mL vacuum tubes for immediate determination of SPC using a refractometer (Reichert VET 360, New York). Samples of the serum and colostrum were frozen at -20°C until being shipped on ice to University of California-Davis School of Veterinary Medicine Immunology and Virology Laboratory (Davis, CA) for analysis of IgG_1, IgG_2, IgA, and IgM using ELISA test kits from Bethyl Laboratory, Inc. (Montgomery, TX).

A portion of the cow and calf fecal samples were shipped on ice immediately after collection to Circle H Laboratory (Dalhart, TX) for analysis of *Salmonella* according to the procedure outlined by Kunze et al. (2008). All samples were stored temporarily at 0-4°C until analysis. For the *Salmonella* analysis, approximately 1 g of feces was placed into 9 ± 0.1 mL of tetrathionate broth (TET) and approximately 1 g of feces was placed into 9 ± 0.1 mL buffered peptone water. The peptone water was vortexed and 1 mL of the peptone mixture was placed into 9 ± 0.1 mL of rappaport-vassiliadis (RV) broth. The TET and RV solutions were incubated at 42°C for 24 ± 2 h. Each broth was then streaked with sterile swabs on half xylose-lysine-tergitol 4 (XLT-4) agar plates for isolation. Plates were incubated at 37°C for 24 ± 2 h. Samples containing presumptively positive colonies were identified as having yellow or red with centers on the growth on the plate. Presumptive positive samples were subjected to the most-probably-number (MPN) technique in a 3 dilution scheme. Using 9 ± 0.1 mL of RV broth, serial dilutions of 1/100 to 1/10,000 were made and incubated at 37°C for 24 ± 2 h. The plates were then examined for positive growth and the data recorded. An MPN calculator was used to calculate the MPN of organisms per unit of substrate (1 g of feces; www.i2workout.com/mcuriale/mpn/index.html).

The other portion of fecal sample was submitted immediately after collection to the Oklahoma Animal Disease Diagnostic Laboratory (Stillwater, OK) for analysis of coccidiosis. Samples were analyzed with a modified Wisconsin Egg-Counting technique using sheather's sugar solution as the float. Samples with abnormally high egg counts were analyzed using modified McMaster Egg-Counting test (Zajac and Conboy, 2006).

Statistical Analysis

A preliminary power analysis was completed to estimate the number of experimental units to determine differences between treatments in the current study. The Cow IgG_1 responses from Franklin et al. (2005) using the DATA rep procedure in SAS (SAS Inst., Inc., Cary, NC) were used for the power analysis. For the experiment data, animal was considered the experimental unit for all analysis because the treatment supplement was fed individually to each cow. Data were analyzed using the MIXED procedure. The model included treatment as a fixed effect and cow age, parturition d, initial BCS, d consuming dietary treatment, or initial cow Ig serum concentration score as potential covariates. Type 1 tests of fixed effects were interpreted for significance ($P < 0.05$) to determine if the covariate could be used in the model.

Fecal sample data are reported as the proportion of animals showing presence of the respective microorganism using the GLIMMIX procedure of SAS. Animal remained the experimental unit and the model included treatment. For all analyses, when the *P*-Value for the F-Statistic was ≤ 0.05, least square means were separated and reported. Tendencies were reported at $0.05 < P\text{-value} \leq 0.10$.

RESULTS AND DISCUSSION

Adding Bio-Mos to winter supplement tended ($P = 0.10$) to improve BW gain from parturition to 30 d of lactation (Table 1). Body condition score was not recorded at the time of calving, therefore, BCS change data during this same time period is not available. There was no difference ($P > 0.36$) in BCS change from the initiation of the treatment period to treatment termination. However, Bio-Mos supplemented cows were better able to maintain BCS ($P = 0.05$) when evaluated from treatment initiation through

weaning (Table 1). No other cow growth performance measurements were affected ($P > 0.14$), nor was calf growth performance affected by dam treatment ($P > 0.29$; Table 5). Neither BW change nor BCS are regularly reported for dairy cows, so the improvement in BCS in this experiment among beef cows consuming supplements containing Bio-Mos from initiation to weaning is not comparable to other literature.

The positive response in BCS among beef cows consuming a supplement with Bio-Mos and the tendency for increased BW gain during early lactation suggests the need for further research to determine how Bio-Mos may influence nutrient utilization in beef cows. Feeding Bio-Mos to monogastrics improves growth performance by enhancing the host immune system and blocking colonization of pathogenic bacteria (Miguel et al., 2004; Moran, 2004). When Bio-Mos was fed to broiler chickens BW gain and villi height was increased (Iji et al., 2001) while crypt death was reduced (Santin et al., 2001). Perhaps these or similar mechanisms may enhance nutrient absorption and utilization when beef cows are fed Bio-Mos.

Cow serum IgA, IgG_1, and IgG_2 concentrations were not different at experiment initiation ($P > 0.23$); however, cows assigned to the Bio-Mos supplement treatment did have higher ($P = 0.02$) IgM serum concentrations at initiation (Table 2). These measurements were taken on d -1, before any consumption of the dietary treatments. All cows appeared to be healthy at initiation of this experiment. The cause for the significant difference in cow serum IgM at experiment initiation is unclear because no dietary treatments had been applied at this time and the cows were managed as a contemporary group. Consequently, initial Ig serum concentrations were used as covariates where applicable for other responses collected later in the experiment.

At parturition, consumption of Bio-Mos did not improve any Ig concentration measure in cows ($P > 0.19$). Serum protein concentration at parturition was not improved ($P = 0.33$) with the addition of Bio-Mos to the winter supplement. Previous research evaluated the influence of Bio-Mos fed to dairy cows during late gestation (Franklin et al., 2005). These authors reported no improvements in cow parturition serum IgG_1 or colostrum IgG_1 (Franklin et al., 2005). Colostrum concentrations of Ig were not ($P > 0.26$) different among cows consuming either dietary supplement, nor did the colostrometer readings differ ($P = 0.30$; Table 3).

At birth, Ig serum concentrations were not different among calves born to cows consuming a winter supplement with or without Bio-Mos ($P > 0.19$; Table 4). Likewise, Franklin et al. (2005) reported no improvements in calf serum IgG_1. Concentrations of IgA, IgG_1, nor IgG_2 did not differ ($P > 0.63$) at the end of the dietary supplementation period. Supplementing cows with Bio-Mos did not increase calf serum protein concentration at birth ($P = 0.55$) or at the end of the supplementation period ($P = 0.47$). Furthermore, feeding cows a supplement containing Bio-Mos did not improve ($P > 0.29$) progeny growth performance from birth to weaning.

Calves born to cows consuming Bio-Mos had greater ($P = 0.02$) IgM serum concentration at the end of the dietary supplementation period (Table 4). This response was unexpected and is not in agreement with results from Franklin et al. (2005). An increase in IgM is indicative of activation of the complement system in an early immune response (Murphy, 2012), but this experiment was not designed to explain this unforeseen response.

There were no differences ($P > 0.14$) at any time point in the proportions of cows that showed the presence of *Salmonella* or coccidiosis in their feces, nor were there any differences ($P > 0.82$) among the calves for the presence of these two microorganisms (data not shown). Terré et al. (2006) reported a negative result for all fecal samples that were tested for *Salmonella* spp. regardless of whether the calves consumed milk replacer with or without Bio-Mos. Research by Henrichs et al. (2003) did not measure actual counts of microorganisms in the feces, but they reported that dairy calves consuming Bio-Mos in the milk replacer had a greater overall probability of feces with a normal consistency compared to calves consuming replacer without Bio-Mos. In the current experiment, very few animals showed the presence of one or both microorganisms and this did not reflect morbidity.

The Ig concentrations of serum and colostrum are lower than previously reported (Franklin et al., 2005; Norman et al., 1981; Olson et al., 1981), which can be partially attributed to breed differences (Quigley et al., 2007). Dewell et al. (2006) found that 8.6% of beef calves born with an IgG_1 serum concentration ≤ 800 mg/dL died before weaning due to various causes, although this low IgG_1 serum concentration at birth did not impact feedlot morbidity. Likewise, Wittum and Perino (1995) reported a preweaning mortality rate of 8.3% among beef calves with ≤ 800 mg/dL IgG_1 concentration at birth. In the current experiment, 58% of the calves had an IgG_1 serum concentration of ≤ 800 mg/dL at birth and 2.7% among the 58% died before weaning.

Prior to initiation of the experiment, a power analysis was conducted based on the IgG_1 concentration (1490 vs. 1786 mg/dL for control and Bio-Mos treatments respectively) from the calf serum collected at 24 h after birth from Franklin et al. (2005).

The IgG_1 response was chosen because it is the primary indication of passive immunity transfer in cattle. The results indicated being able to detect a difference of 200 mg/dL IgG_1 between treatment levels at power = 0.99% with at least 67 animals per treatment. The lowest power suggested was at 85%, with a maximum difference between treatments being 50 mg/dL and at least 21 animals per treatment. All tests were conducted at the normal $\alpha = 0.05$ level.

The experiment was conducted with 38 and 31 animals per treatment, which is an acceptable number based on the 85% power analysis. Recognizing differences in statistical models, our results indicated a higher degree of variation in IgG_1 measurements than reported by Franklin et al. (2005). This in turn minimized our ability to detect possible significant biological differences because of too few experimental units. Alternatively, there is a greater chance of the presence of type II error (Ott and Longnecker, 2004) in these data due to the unexpected high degree of variation in Ig concentrations. At parturition, cow serum, calf serum, and colostrum IgG_1 response CVs were 39, 93, and 96% respectively. Inherent variation in Ig serum concentrations exist from animal to animal depending on genetics (Detilleux et al., 1995), dam nutrition and disease history, individual calf ability to absorb Ig, and the blood collection time relative to parturition (Quigley, 2007). It has also been proposed that stress can affect Ig concentrations through an interaction between cortisol and Ig (Herr et al., 2011).

Blood and colostrum samples from cows and calves were collected within 12 h of birth ($\mu = 6.4$ h). The collection time was determined with the consideration that efficiency of IgG absorption declines rapidly from birth to 24 h (Quigley, 2007). Furthermore, Quigley (2007) reported that up to 2 h post parturition colostral IgG

concentration declines; therefore, collection of samples within 12 h of birth was most suitable. The time of collection relative to parturition was estimated based on calf mobility, dampness of the calf, and calf hoof development. Variation may be reduced by narrowing the blood collection window, making it more consistent among animals for samples taken at birth. Because of inherent variation in Ig responses, the number of experimental units becomes that much more critical. Herr et al. (2011) reported great sensitivity in Ig measurements collected around parturition and the importance of maximizing the number of experiment animals.

IMPLICATIONS

It was a priority of this experiment to test the impact of feeding Bio-Mos in a beef cattle production scenario that closely matches normal commercial production practices. Adding mannan oligosaccharide in the form of Bio-Mos to winter supplement may limit BCS loss following parturition while improving BW gain during this time in spring calving beef cows. This may have been due to morphological changes in the gut, which have been verified in previous research documenting the improvement in mucosal integrity due to inclusion of mannan oligosaccharide (Iji et al., 2001). Feeding beef cows Bio-Mos in late gestation did not improve the transfer of passive immunity to the calf, nor did it improve calf growth performance. Other research reporting Ig concentrations in beef cows and calves as an indication of passive immunity is not available. Future experiments designed to evaluate IgG_1 concentration in the dam and newborn offspring as the primary indicator of passive immunity characteristics will need to consider the large number of experimental units required.

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Table 1. Effect of feeding Bio-Mos to beef cows on cow performance

	Supplement[1]			
Item;	Control	Bio-Mos	SE	*P*-value
Experiment initiation:				
No.	38	31		
Initial BW (2/10/2012), kg	568	571	9.96	0.79
Initial BCS	5.5	5.5	0.14	0.89
Initiation – calving[2]:				
BW at calving, kg	530	533	10.3	0.73
BW change initiation to calving,[3] kg	-86.1	-89.1	8.05	0.72
Calving – off-test[2]:				
BW at off-test, kg	517	532	9.64	0.14
BW change calving to off-test,[4] kg	6.68	22.39	9.41	0.10
BCS at off-test	4.8	5.0	0.12	0.24
BCS change initiation to off-test[4]	-0.24	-0.13	0.12	0.36
Off test – weaning[2]:				
No. at weaning	36	30		
BW at weaning, kg	537	539	9.78	0.82
BCS at weaning	4.7	5.0	0.22	0.14
Initiation – weaning[2]:				
BW change,[5] kg	-34.3	-30.4	6.91	0.58
BCS change[5]	-0.79	-0.44	0.18	0.05

[1]Supplements included (DM basis) 1) 1.36 kg/d during gestation of a 30% CP cottonseed meal based protein supplement and 1.81 kg/d during lactation of a 38% cottonseed meal based protein supplement (**Control**); 2) Control plus 10 g/d Bio-Mos (**Bio-Mos;** Alltech, Inc., Nicholasville, KY).
[2]Calving measurements were taken within 3-12 h from calving; Off-test is approximately 30 d after calving; weaning is approximately 180 d after calving; weaning is approximately 210 d after initiation.
[3]Change in BW from initial measurement to calving.
[4]Change in BW and BCS from calving to off-test.
[5]Change in BW and BCS from initiation to weaning.

Table 2. Effect of feeding Bio-Mos to beef cows on cow blood variables

Collection Period:	Initiation[1]				Parturition[2]			
	Supplement[3]				Supplement			
Item;	Control	Bio-Mos	SE	*P*-value	Control	Bio-Mos	SE	*P*-value
No.	38	31			38	31		
SPC,[4] g/dL	7.02	7.07	0.12	0.68	6.98	7.13	0.15	0.33
IgG_1, mg/dL	668	784	95.4	0.23	716	806	68.7	0.19
IgG_2, mg/dL	646	618	65.2	0.67	697	754	64.1	0.33
IgA, mg/dL	28	28	4.65	0.90	32	35	3.48	0.52
IgM, mg/dL	198	340	57.8	0.02	211	216	21.8	0.95

[1]February 10, 2012, which is approximately 30 d pre-average herd calving date.

[2]Blood collection took place within 3-12 h from calving.

[3]Supplements included (DM basis): 1) 1.36 kg/d during gestation of a 30% CP cottonseed meal based protein supplement and 1.81 kg/d during lactation of a 38% cottonseed meal based protein supplement (**Control**); 2) Control plus 10 g/d Bio-Mos (**Bio-Mos;** Alltech, Inc., Nicholasville, KY).

[4]Serum protein concentration.

Table 3. Effect of feeding Bio-Mos to beef cows on colostrum parameters collected within 3-12 h from birth

	Supplement[1]			
Item;	Control	Bio-Mos	SE	*P*-value
No.	38	31		
Colostrometer reading, mg/mL	42.7	33.8	8.58	0.30
IgG_1, mg/dL	909	1,402	450	0.28
IgG_2, mg/dL	46	52	5.18	0.26
IgA, mg/dL	635	794	315	0.61
IgM, mg/dL	304	282	69.5	0.76

[1]Supplements included (DM basis) 1) 1.36 kg/d during gestation of a 30% CP cottonseed meal based protein supplement and 1.81 kg/d during lactation of a 38% cottonseed meal based protein supplement (**Control**); 2) Control plus 10 g/d Bio-Mos (**Bio-Mos;** Alltech, Inc., Nicholasville, KY).

Table 4. Effect of feeding Bio-Mos to beef cows on calf blood variables

Collection Period:	Birth[1]				Off-Test[2]			
	Dam Supplement[3]				Dam Supplement			
Item;	Control	Bio-Mos	SE	*P*-value	Control	Bio-Mos	SE	*P*-value
No.	38	31			36	29		
SPC,[4] g/dL	5.80	5.65	0.26	0.55	6.08	6.01	0.10	0.47
IgG_1, mg/dL	909	988	200	0.70	716	692	109	0.83
IgG_2, mg/dL	66	81	11.4	0.19	101	121	32.6	0.65
IgA, mg/dL	422	438	112	0.88	6.11	5.72	0.79	0.63
IgM, mg/dL	78	75	16.5	0.88	61	48	13.4	0.02

[1]Blood collection took place within 3-12 h of birth.

[2]Blood collection took place when the dam was removed from the dietary supplement.

[3]Dam Supplements included (DM basis) 1) 1.36 kg/d during gestation of a 30% CP cottonseed meal based protein supplement and 1.81 kg/d during lactation of a 38% cottonseed meal based protein supplement (**Control**); 2) Control plus 10 g/d Bio-Mos (**Bio-Mos;** Alltech, Inc., Nicholasville, KY).

[4]Serum protein concentration.

Table 5. Effect of feeding Bio-Mos to beef cows on subsequent calf performance

	Dam Supplement[1]			
Item;	Control	Bio-Mos	SE	*P*-value
No. at birth	38	31		
Weight, kg				
Birth BW	34.1	34.4	1.37	0.80
BW at dam off-test[2]	65.9	66.0	2.16	0.96
BW at weaning[3]	194	189	6.93	0.47
Adjusted BW at weaning[4]	268	259	8.78	0.43
ADG birth to weaning, kg	0.92	0.88	0.04	0.29

[1]Dam supplements included (DM basis) 1) 1.36 kg/d during gestation of a 30% CP cottonseed meal based protein supplement and 1.81 kg/d during lactation of a 38% cottonseed meal based protein supplement (**Control**); 2) Control plus 10 g/d Bio-Mos (**Bio-Mos;** Alltech, Inc., Nicholasville, KY).

[2]Dam supplementation ended approximately 30 d post-calving.

[3]Weaning was at approximately 180 d of age.

[4]Adjust 205 d wean BW: ((BW at weaning – BW at birth)/calf age at weaning) x (205 + BW at birth + Age of dam adjusted).

CHAPTER IV

SUPPLEMENTATION OF MONENSIN AND SLOW RELEASE UREA TO BEEF COWS CONSUMING LOW QUALITY FORAGE DURING LATE GESTATION AND EARLY LACTATION

S.K. Linneen,* A.L. McGee,* J.R. Cole,* J.S. Jennings,† D.R. Stein,* G.W. Horn,* and D.L. Lalman*[1]

*Department of Animal Science, Oklahoma State University, Stillwater 74078; †Alltech, Inc., Nicholasville 40356

ABSTRACT

Two experiments were designed to investigate the effects of feeding monensin and/or slow release urea with a fibrolytic feed enzyme to beef cows on performance, milk production, calf growth performance, and cow blood metabolites. The same herd of spring-calving Angus and Angus x Hereford cows and heifers were used in a completely randomized design in both Exp. 1 (N = 84; initial BW = 534 ± 68 kg) and Exp. 2 (N = 107; initial BW = 508 ± 72 kg). Exp. 1 treatment supplements were formulated to meet the protein requirement of the cow and included 1) Cottonseed meal supplement with no monensin (**Control**); or 2) Monensin added to Control to supply 200 mg·head^{-1}·d^{-1}

[1] Corresponding author: david.lalman@okstate.edu

(**MON**). Supplements were individually fed at 1200 daily at a rate of 0.9 kg·head^{-1}·d^{-1} for duration of the 60-d study. Milk production was measured through weigh-suckle-weigh procedure on April 19 and May 10. In Exp. 2, treatment supplements were fed below the protein requirement of the cow and included 1) Cottonseed meal/wheat midd (**Control**), 2) Control plus soybean hulls, corn, and 61 g·cow^{-1}·d^{-1} slow-release urea with a fibrolytic feed enzyme (Optimase®; Alltech, Inc., Nicholasville, KY) (**SRU**), 3) Control plus soybean hulls, corn, and monensin to supply 200 mg·cow^{-1}·d^{-1} (Rumensin 90®; Elanco Animal Health; Greenfield, IN)(**MON**), and 4) SRU plus MON (**Combo**). Supplements were fed at a rate of 454 g·head^{-1}·d^{-1} during the 90-d study. Cows were managed as a contemporary group during both experiments with ad libitum access to prairie hay (4.5% CP; 55% TDN; DM basis). Data were analyzed using Mixed in SAS 9.3 with animal as the experimental unit. In Exp. 1, treatment did not affect cow BW or BCS performance ($P > 0.19$). Calf birth BW was not affected by dam dietary treatment ($P = 0.24$); however, calves from dams consuming MON weighed significantly more at d 25 and 60 of the study. Calves from dams fed MON also had greater ($P = 0.03$) ADG from birth to the end of the study. Milk production did not differ between cows on either treatment at any collection ($P > 0.26$), nor did MON ($P < 0.39$) affect pregnancy rate. In Exp. 2, treatment not affect ($P > 0.22$) cow BW and BCS from d 0 through d 54 of the study. On d 90 of the study, cows consuming monensin tended ($P = 0.07$) to have reduced BCS. Monensin also did not affect cow BW or BCS change during any period in the study ($P > 0.27$). Cows consuming Optimase® in the protein supplement tended ($P > 0.09$) to gain less weight from d 26 – 54 of the study. Cows fed the SRU supplement lost more BCS from d 0 – 54 ($P < 0.02$) and d 0 – 90 ($P < 0.03$) compared to cows consuming all other

treatments. Plasma glucose concentrations were unaffected by treatment ($P > 0.30$); however supplement treatment ($P < 0.01$) affected BUN concentration SRU inclusion in the supplement increased BUN. Monensin improved pre-weaning calf growth performance, making it an effective option for feeding beef cows during lactation to improve preweaning efficiency. Combining Optimase® and monensin did lesson the reduction in growth performance caused by Optimase® alone, but the efficacy of improved cow performance due to combining Optimase® and monensin needs to be further investigated.

Key words: calves, cows, glucose, monensin, weigh-suckle-weigh

INTRODUCTION

Monensin (Elanco Animal Health, Greenfield, IN) is an ionophore that improves energetic efficiency by altering VFA production in the rumen in grain-fed animals (Duffield et al., 2012; Sprott et al., 1988). Monensin debilitates gram positive bacteria in the rumen while gram negative bacteria are resistant, resulting in continual production of propionate from succinate (Ellis et al., 2012). Monensin also improves nitrogen metabolism and reduces proteolysis of degradable intake protein because of its protein sparing characteristics (Poos et al., 1979). Ionophores such as monensin have been shown to influence beef cow weight gain and feed efficiency without affecting fertility and milk production (Sprott et al., 1988). Lemenager et al. (1978a) reported that cows fed 200 mg/h/d monensin while grazing native range had decreased forage intake, but cow performance was unaffected by monensin. Feeding cows monensin in late gestation has been shown to significantly increase birth weight (Clanton et al., 1981) and calf ADG (Lemenager et al., 1978a).

Optimase® (Alltech, Nicholasville, KY) is a feed additive product for cattle that combines slow-release non-protein nitrogen (NPN) and fibrolytic enzyme technology. There is no previous research available evaluating the efficacy of Optimase® in beef cows. While having a similar N content as urea, Optigen® (slow release urea; Alltech, Nicholasville, KY) has been shown to effectively replace soybean meal in low quality forage diets (Kononoff et al., 2006). Akay et al., 2004 demonstrated that nitrogen disappearance of Optigen® over 30 h more closely matched disappearance of soybean meal than urea.

Considering the limited and dated library of research on feeding monensin to beef cows, Exp. 1 was designed to evaluate the impact of monensin on performance and milk production of spring-calving beef cows consuming low quality forage, as well as subsequent calf growth performance. Combining the two products, Optimase® and monensin, in beef cow winter protein supplements has the potential to reduce the cost of supplementation and the amount of forage required to maintain cows. Therefore, the objective of Exp. 2 is to evaluate the impact of Optimase®, monensin, and the combination of the two on gestating beef cow BW and BCS change, blood glucose, and blood urea nitrogen.

MATERIALS AND METHODS

These experiments were conducted in accordance with approved Oklahoma State University Animal Care and Use Committee protocols. They were both conducted at the Range Cow Research Center, North Range Unit located approximately 16 km west of Stillwater, OK using the same spring-calving cow herd.

Experiment 1

Animals. Spring-calving Angus and Angus x Hereford cows and heifers (N = 84; initial BW = 534 ± 68 kg; initial BCS = 5.27 ± 0.6; initial age = 4.8 ± 3 yr) were allotted to 1 of 2 treatment combinations in a completely randomized design. Cows were ranked by BW and age and randomly allocated so that BW and age were similar across treatments. Treatments included 1) Cottonseed meal supplement with no monensin (**Control**); or 2) Monensin added to Control to supply 200 mg·head^{-1}·d^{-1} (**MON**). Both

supplements were fed at a rate of 0.90 kg·head^{-1}·d^{-1} for the duration of the study. All supplements were fed as a 0.64-cm diameter pellet and formulated to contain 40% CP DM basis (Table 1). Supplements were balanced for Ca and P and formulated to meet the protein requirement of the cow according to the NRC (1996).

Prior to, during, and after the treatment period, cows were managed as a contemporary group. Cows had ad libitum access to prairie hay (CP, 5%; TDN, 55%; crude fat, 2.8%; DM basis) and mineral mixture (28.6% NaCl; 12.8% Ca; 8.5% P; 1.2% Mg; 1044 ppm Cu; 12 ppm Se; 3117 ppm Zn; DM basis). The supplementation period was initiated on March 11 and terminated on May 11 resulting in a 60 d treatment period.

Cows were fed individually at 1200 h daily in a barn containing 31 individual feeding stanchions to ensure that each cow received the assigned amount of feed. Each d the cows were gathered from a pasture adjacent to the feeding barn and placed into a feeding stall, restrained, and allowed approximately 20 min to consume their dietary supplement. During the trial, there were 14 refusals of the Control supplement and 8 refusals of the MON supplement with an average amount of refusal of approximately 0.2 kg per feeding (data not shown).

Individual cow BW and BCS were determined at study initiation (March 11) and conclusion (May 11). Body condition scores (scale 1 through 9; Wagner et al., 1988) were determined by the same 2 evaluators throughout the experiment. Cow BW was also recorded approximately every 2 weeks after initiation (March 29, April 12, and April 26), and at parturition. Cows and calves were weighed within 24 h of birth, and a subsequent BW was collected on the calf every 2 weeks until trial conclusion. Calf weaning BW

was obtained on September 6 and reported as a 205-d adjusted BW (Beef Improvement Federation and Guidelines, 2002).

Milk production was measured through weigh-suckle-weigh (WSW) procedure. This procedure was conducted twice during the experiment (April 19 and May 10), each time using cows that were at least 30 d postpartum. A total of 23 cows were used in the 1st collection and a total of 55 cows, including the 23 used in the 1st collection, were used in the 2nd collection. The evening preceding the data collection, calves were separated from cows at 2300 h. Calves were weighed at 0645 h the following morning and then reunited with their dam to nurse until satiated. After nursing, the calf was immediately weighed and isolated from the dam until 1445 h. At this time calves were weighed and the process began again for a total of 3 collections to determine milk production over a 24 h period. Cows were exposed to bulls June through August for natural service breeding. Conception rate was determine by transrectal ultrasonography 50 d (July 30) and 90 d (September 8) after the bulls were removed from the cow herd.

Experiment 2

Animals. Spring calving Angus and Angus x Hereford gestating cows and heifers (N = 107; initial BW = 508 ± 72 kg; initial BCS = 4.7 ± 0.8; initial age = 4.2 ± 3 yr) were allotted to 1 of 4 treatment combinations in a completely randomized design. Cows were sorted by BW and age and randomly allocated so that BW and age were similar across treatments. Treatment supplements included 1) Cottonseed meal/wheat midd (**Control**), 2) Control plus soybean hulls, corn, and 61 $g \cdot cow^{-1} \cdot d^{-1}$ slow-release urea with a fibrolytic feed enzyme (Optimase®; Alltech, Inc., Nicholasville, KY) (**SRU**), 3) Control plus soybean hulls, corn, and monensin to supply 200 $mg \cdot cow^{-1} \cdot d^{-1}$ (Rumensin 90®; Elanco

Animal Health; Greenfield, IN)(**MON**), and 4) SRU plus MON (**Combo**). All supplements were fed at a rate of 454 g·head^{-1}·d^{-1} during the study in a 40% CP (DM basis) pellet (Table 2). Steers were fed at protein deficient rate intended to determine differences among treatments; therefore, diets were formulated to provide 471 g/d DIP (DIP balance of -49%) calculated using cow intake rate at 2% of BW and supplement TDN = 56%. All supplements were also balanced for Ca and P to meet NRC (1996) requirements.

Prior to, during, and after the treatment period, cows were managed as a contemporary group with ad libitum access to prairie hay (4.5% CP; 55% TDN; DM basis). A mineral supplement was provided free choice (28.6% NaCl; 12.8% Ca; 8.5% P; 1.2% Mg; 1044 ppm Cu; 12 ppm Se; 3117 ppm Zn; DM basis). The experiment was initiated on September 21 and terminated on December 19 resulting in a 90 d experimental supplementation period.

Cows were fed individually at 1130 h daily to ensure that each cow received the assigned amount of feed. The feeding barn contained 31 individual feeding stanchions and cows were brought in to the barn in groups until each cow was fed. Each d the cows were gathered from a pasture adjacent to the feeding barn and placed into a feeding stall, restrained, and allowed approximately 20 min to consume their dietary supplement. Supplement refusals were documented throughout the study.

Individual cow BW was determined at study initiation (d-0; September 20), d-26, d-54, and at trial termination (d-90; December 19). Body condition scores (scale 1 through 9; Wagner et al., 1988) were determined by the same evaluator on d-0, d-54, and d-90. Blood was also collected on d-0, d-26, d-54, and d-90 at approximately 2 h after

feeding, at 1330 h. Blood was collected via coccygeal venipuncture into vacuum tubes (BD Vacutainer) to determine blood urea nitrogen (BUN) and glucose. Samples were placed in tubes that contained Ethylenediaminetetraacetic acid and heparin for BUN and glucose, respectively. Samples were placed on ice, followed by refrigeration for < 24 h, and then centrifuged at 3,800 X g for 20 min at 4°C. Serum was removed and stored at -20°C until analysis.

Approximately 2 ml of sample was shipped cool to the University of Kentucky for BUN analysis in duplicate using BUN assay from Marsh et al. (1965). Glucose concentration was determined with the remaining sample at the Oklahoma State University Animal Science Analytical Laboratory according to procedures outlined by Camacho et al. (2012). A glucometer (OneTouch UltraMini, Life Scan, Johnson and Johnson, Milpitas, CA) was used by exposing each strip to approximately 10 µL of plasma in duplicate.

Statistical Analysis. All data in both experiments were analyzed using the MIXED procedure of SAS (SAS Inc., Cary, NC) with animal as the experimental unit. In Exp 1., for the cow and calf performance data, the model included treatment and cow age. The model included treatment for milk production data analysis. Conception rates were analyzed using the GLIMMIX procedure of SAS, assuming a binomial distribution and treatment as a fixed effect. Calf age, which also indicated days in milk, was used as a covariate where applicable. For significant effects, treatment means were compared using least significant difference multiple comparisons. In Exp. 2, data were analyzed using a model that contained treatment and cow age as fixed effects. Preplanned single-degree-of-freedom contrasts were used to determine the effect of: 1. Control and SRU vs

Mon and Combo (C_1), 2. Control and MON vs SRU and Combo (C_2), and 3. MON and SRU vs Combo (C_3). If the treatment *P*-value was > 0.10, the contrast results were not reported. No differences were seen in the BUN data when it was analyzed by month of collection, so data were pooled by treatment across time. In both experiments, the alpha level to determine statistical significances was set to $\alpha = 0.05$, and tendencies were reported at $0.05 < P\text{-value} < 0.10$.

RESULTS

Experiment 1. There were no significant differences ($P > 0.33$; Table 3) in cow BW or BCS at any time in the study. There were also no differences ($P > 0.19$) in cow BW or BCS change from d 0 to calving, calving to d 60, or d 0-60. Calf birth BW was not affected by dam dietary treatment ($P = 0.24$; Table 4); however, calves from dams consuming monensin weighed more at d 45 ($P = 0.02$) and d 60 ($P = 0.04$) of the study. Calves from dams fed monensin also had greater ($P = 0.04$) ADG from birth to the end of the study. Although there was a 6% increase in milk produced when cows consumed monensin, this response was not significant ($P > 0.26$; Table 5). Monensin did not affect conception rate either 40-d prior to weaning or at weaning ($P > 0.38$; Table 6).

Experiment 2. Treatment did not affect ($P > 0.22$) cow BW and BCS from d 0 through d 54 of the study (Table 7). On d 90 of the study, cows consuming monensin tended ($P = 0.07$) to have reduced BCS, although BW among cows across the 4 treatments was not different ($P = 0.59$) at this time. Monensin also did not affect cow BW or BCS change during any period in the study ($P > 0.27$). From d 26 to 54 and d 0 to 54, cows tended ($P > 0.09$) to lose more BW when consuming SRU, and BCS change was significantly reduced by SRU from d 0 to 54 ($P = 0.02$). From d 0 to 90, cows

consuming MON or Control supplements gained more BCS ($P = 0.03$) than cows consuming SRU or Combo supplements. Supplement intake was similar for cows across treatments ($P > 0.17$; data not shown). Plasma glucose concentrations were unaffected by treatment at all 4 collections ($P > 0.16$; Table 8), but cows consuming a supplement containing SRU had greater ($P < 0.01$) BUN concentration.

DISCUSSION

Feeding monensin to grazing beef cows did not affect cow performance in these studies. In previous literature, cows consuming monensin during gestation had decreased forage intake, accompanied by either an improvement (Barnett et al., 1982; Sexten et al., 2011) or no change in cow gain (Walker et al., 1980; Moseley et al., 1977). Bretschneider et al. (2008) reported an increase in ADG of 12.1% for grazing cattle consuming monensin when comparing 46 experiments in a review on the effects of feeding grazing cattle monensin. Perhaps in this study monensin simply did not improve the energetic efficiency in large enough magnitude to elicit a gain response. The rumen microbial environment of cows fed low-quality forage is dominated by cellulolytic and protozoa microbes. Monensin is selecting against gram negative microbes, causing reduced proteolysis and increased propionate production in the rumen (Owens and Goetsch, 1988; Russell and Strobel, 1989; van der Merwe et al., 2001). This increase in energy may not directly translate to an improvement in animal performance, and is affected by individual animal variation in gain, especially when the trial period includes parturition.

Forage intake was not measured in the current study. Although Lemenager et al. (1978) reported reductions in cow DMI when cows consumed monensin during lactation,

other grazing studies did not measure intake (Hixon et al., 1982; Bailey et al., 2008). Measuring DMI in grazing cattle is difficult resulting in very few monensin studies reporting this response. Bretschneider et al. (2008) summarized 13 experiments where grazing cattle were fed monensin and concluded that monensin did not affect DMI. These authors recognized that forage quality and type of supplement (energy or protein) used to deliver monensin may differ among studies, and that the majority of the studies in the review used growing cattle. In a meta-analysis on feedlot cattle receiving monensin, DMI was significantly reduced by 3.1% for the 151 experiments summarized (Duffield et al., 2012).

In this study feeding cows monensin resulted in improved calf performance during the feeding period of the study. The cows were fed supplements daily in an individual stanchion after being separated from the calf, so there was no opportunity for calves to consume monensin. Clanton et al. (1981) reported a significant increase in calf birth weight among calves born to cows consuming monensin and Lemenager et al. (1978a) reported an improvement in ADG for calves from cows consuming monensin, although the authors acknowledged that the calves had access to the supplement containing monensin. Conversely, Turner et al. (1980) and Walker et al. (1980) found no improvement in calf gain when cows consumed monensin, although forage was fed at 90% of the control allotment in these studies. There were no differences ($P = 0.33$) in 205-d adjusted calf wean weight among, which was expected since this weight was collected 120 d after the feeding period of the trial was terminated.

Only cows at least 30 d post parturition were included in the WSW procedure to measure milk yield, which may have affected the power associated with this response

because of limited experimental units. In a review of the published experiments on feeding dairy cows monensin, Duffield et al. (2008b), reported a 2.3% increase in milk yield when monensin was included in the total mixed ration among 71 experiments. These authors recognize that the response in milk yield to monensin is consistent, but often not in large enough magnitude to be significant. Duboc et al. (2010) reported that monensin increased milk yield in dairy cows less than 150 d in milk, but greater than 150 d in milk it had no effect. This suggests that monensin may be most beneficial in early lactation, when the cow is in a negative energy balance. In beef cows, few studies have measured milk yield, and those indicate no change in milk yield due to monensin (Lemenager et al., 1978a; Hixon et al., 1982; Grings and Males, 1988). The inconsistent response to monensin between dairy and beef cows may be due to breed differences, time of milk collection relative to parturition, or simply a function of not enough research on this topic in beef cows (Sprott et al., 1988).

The dairy industry has commonly reported milk production efficiency as a response when testing the effects of monensin inclusion in the TMR. Milk production efficiency is maximized when monensin decreases feed intake, but does not decrease milk yield. Ramanzin et al. (1997) reported an improvement in milk production efficiency due to feeding monensin to dairy cows. A decrease in forage DMI, as seen in the results by Lemenager et al. (1978a), accompanied by no change in milk yield resulted in improved milk production efficiency in beef cows.

Interestingly, the response of cow BW change shows a pattern for less BW loss from trial initiation to calving for cows consuming monensin. From calving to the end of the feeding period, the same cows demonstrated numerically greater BW loss. Perhaps

the increased energy available from feeding monensin is being partitioned toward lactation, as evidenced by an increase in calf gain. Data from Ørskov (1977) suggests that increasing nonglucogenic VFAs will decrease efficiency of metabolizable energy utilization above maintenance for dairy cows. In relation to this study, increasing propionate production may improve efficiency of metabolizable energy utilization above maintenance, specifically for milk production. In a meta-analysis review on feeding dairy cows monensin, Duffield et al. (2008a) determined that precalving glucose concentrations in the blood of the cow were lower when they consumed monensin indicating a portioning of glucose toward fetal development. Considering the previously discussed increase in milk yield due to monensin, it is reasonable to speculate that the increased energy from monensin is being partitioned for lactation after parturition.

The second experiment investigated the effect of SRU in the form of Optimase® on cow performance. Similar studies feeding NPN to beef cows consuming forage have reported an increase in cow performance when urea or slow-release urea is fed compared to a negative control (unsupplemented) group of cows (Rush et al., 1976; Currier et al., 2004). Waterman et al. (2007) also reported that cows consuming urea along with wheat straw and alfalfa hay had similar DMI to those consuming a diet without urea. When compared to cows consuming a positive control (natural protein), both urea and slow-release urea consumption reduces cow BW and BCS (Lemenager et al., 1978b; Forero et al., 1980; Farmer et al., 2004). This was seen in the current study when cows consuming the SRU treatment had reduced performance compared to cows fed the positive control. In contrast, other studies reported no change in cow performance when Optigen® was fed to cows compared to cows consuming either a negative control (Wahrmund et al., 2007)

or a positive control containing soybean meal (Kononoff et al., 2006). Regardless, the appeal of feeding NPN in partial replacement of natural protein has been in balancing the magnitude of reduction in animal performance with cost savings associated with a less expensive form of crude protein that NPN offers.

Combining monensin and Optimase® in a single supplement (contrast C_3) did appear to lessen cow BCS loss induced by SRU. Monensin is debilitating gram positive bacteria in the rumen while gram negative bacteria are resistant, resulting in continual production of succinate leading to propionate production (Ellis et al., 2012). Propionate is used more efficiently than acetate by the tissues (Schelling, 1984), in addition to being used for gluconeogenesis. That leads to improved energetic efficiency by the animal, even among those consuming low quality forage (Lemenager et al., 1978a; Walker et al., 1980; Schelling, 1984), which may have compensated for the performance reductions induced by SRU.

Feeding cattle urea has historically reduced intake due to palatability concerns (Forero et al., 1980), although it is less of a problem with low inclusion rates and in coated urea products intended to slow ammonia release rate. Both Farmer et al. (2004) and Kononoff et al. (2007) reported no reduction in cattle intake when urea or slow-release urea were consumed. As mentioned previously, feeding monensin has caused a reduction in forage intake among beef cows (Lemenager et al., 1978a); however, forage intake was not measured in the current study so this response could not be evaluated.

Multiple studies corroborate the glucose results from this study. Wahrmund and Hersom, 2007, Wahrmund et al., 2007, and Holder, 2012 reported no improvement in glucose when cows consumed Optigen II® in comparison to cattle consuming urea or a

control supplement. Few other studies have looked at the effects of feeding monensin to grazing cattle on glucose concentrations. Grings and Males (1988) reported a numeric reduction in plasma glucose among cows consuming monensin in gestation. This effect was reversed after calving when monensin significantly improved plasma glucose. In a meta-analysis review on feeding dairy cows monensin, Duffield et al. (2008a) determined that precalving glucose concentrations in the blood of the cow were lower when they consumed monensin, and this may be indicative of the fetus receiving the glucose generated from monensin. Therefore, the increased proportion of propionate that results from feeding monensin may have been going directly to the fetus resulting in low plasma glucose concentration in the cow. The current study was conducted in mid-to-late gestation using protein deficient diets, which may have contributed to low glucose concentrations among all cows. Glucose concentration of lactating dairy cows is consistently increased by monensin, but it is a sensitive measurement that often is insignificant among treatments because of a lack of statistical power (Duffield et al., 2008a).

The concentration of BUN across all 4 treatments is lower than expected, reflecting poor forage quality and supplements deficient in DIP. Sampling time of 2 h post-feeding may have been too soon for collection, affecting the results of this response. Adding SRU to the supplement increased BUN, which is similar to previous research. Both Currier et al. (2004) and Wahrmund et al. (2007) reported a significant increase in cow BUN when slow-release urea was fed compared to unsupplemented cows (negative control). Conversely, Ammerman et al. (1972) reported no significant improvement in plasma urea nitrogen (PUN) when wethers consumed urea conmpared to wethers

consuming cottonseed meal. With respect to the effects of monensin on BUN, cows on the MON treatment had increased BUN compared to cows on the Control supplement. This is consistent with previous literature from Poos et al. (1979) and van der Merwe (2001), as well as a meta analysis by Duffield et al. (2008a) when they reported a 6.2% increase in PUN when monensin is fed to dairy cows. Given the previously discussed monensin mechanisms of action, it is plausible that cows consuming monensin had increased efficiency of nitrogen utilization compared to cows consuming the Control treatment.

Including monensin in the winter beef cow supplement did not influence cow BW or BCS change throughout the trials, although there is a numeric pattern for less BW loss before parturition and more BW loss after parturition among cows consuming monensin in Exp. 1. The increased energy derived from the increased propionate produced by monensin may have been portioned toward lactation, verified by the increase in calf gain and 6% numeric increase in milk yield. Feeding monensin to spring-calving beef cows improved pre-weaning calf growth performance without changing milk production or cow performance, making it an effective option for feeding beef cows during lactation to improve preweaning efficiency. Feeding monensin to beef cows in late gestation had no affect on cow performance or blood glucose concentration two hours after feeding. Increased glucose from feeding monensin may have been used for fetal development resulting in similar glucose levels among cows. Including Optimase® in the cow supplement reduced cow gain midway through the study, and reduced BCS of cows over the duration of the experiment. Combining Optimase® and monensin did lesson the reduction in performance caused by Optimase® alone. The efficacy of combining

monensin with a slow release urea to improve cow performance needs to be further investigated.

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Table 1. Supplement composition and amount of nutrients supplied to beef cows during a 90-d study (Exp. 1)

	Supplement[1]	
	Control	MON
Item (DM basis);	% of DM	
Cottonseed meal	91	92
Cane molasses	5.50	5.50
Rumensin 90®	-	0.09
Vitamin A 30,000 IU/g[2]	0.22	0.22
Vitamin E 50%	0.10	0.10
Mineral mixture[3]	2.20	2.20
Nutrients supplied:	Nutrients supplied, kg/d	
DM	0.79	0.79
CP	0.38	0.38
TDN	0.69	0.69
Crude fat	0.01	0.01
Chemical composition:	Chemical composition, %	
DM	88.3	88.3
CP	42.0	42.0
TDN	76.5	76.4

[1]Supplements included: 1) Cottonseed meal supplement with no monensin (**Control)**; 2) Monensin added to Control to supply 200 $mg \cdot hd^{-1} \cdot d^{-1}$ (**MON**; Rumensin 90®, Elanco Animal Health, Greenfield, IN). Supplements were fed at a rate of 0.90 $kg \cdot hd^{-1} \cdot d^{-1}$ for duration of the study.

[2]Provided 32,960 IU of Vitamin A per kg of supplement DM.

[3]Supplied calcium carbonate, copper sulfate, zinc sulfate, cobalt carbonate, and selenium 600.

Table 2. Supplement composition and amount of nutrients supplied to beef cows during a 90-d study (Exp. 2)

	Treatment[1]			
	Control	SRU	MON	Combo
Item;	% of DM			
Cottonseed meal	87.8	1.25	87.7	1.25
Wheat midds	4.50	68.5	4.50	68.3
Soybean hulls	-	14.1	-	14.1
Limestone	2.00	1.50	2.00	1.50
Cane molasses	5.50	5.50	5.50	5.50
Optimase®	-	9.00	-	9.00
Rumensin 90®	-	-	0.17	0.17
Vitamin A, 30,000 IU/g[2]	0.11	0.11	0.11	0.11
Vitamin E 50%	0.05	0.05	0.05	0.05
	Nutrients supplied, kg/d			
DM	0.41	0.41	0.41	0.41
CP	0.18	0.18	0.18	0.18
TDN	0.34	0.31	0.34	0.31
Fat	0.01	0.02	0.01	0.02
	Chemical composition, %			
DM	90.2	89.6	90.2	89.6
CP	40.0	40.5	40.0	40.5
TDN	74.8	69.1	74.6	68.9

[1]Treatments included: 1) Cottonseed meal/wheat midd (**Control**), 2) Control plus soybean hulls, corn, and 61 g·cow-1·d-1 SRU (Optimase®; Alltech, Inc., Nicholasville, KY) (**SRU**), 3) Control plus soybean hulls, corn, and monensin to supply 200 mg·cow^{-1}·d^{-1} (Rumensin 90®; Elanco Animal Health; Greenfield, IN)(**MON**), and 4) SRU plus MON (**Combo**). Diets were formulated to provide 471 DIP g/d (-49% DIP balance).

[2]Provided 32,960 IU of Vitamin A per kg of diet DM.

Table 3. Effects of feeding monensin to beef cows on cow performance (Exp. 1)

	Treatment[1]			
Item;	Control	MON	SEM	*P*-value
No. of cows	42	42		
BW, kg				
D0	580	572	10.8	0.45
D17	601	595	12.2	0.62
Parturition[2]	555	554	11.8	0.98
D31	578	570	12.4	0.54
D45	581	572	11.7	0.45
D60	536	529	11.1	0.52
D0 BCS	5.4	5.2	0.14	0.33
D60 BCS	4.9	4.8	0.19	0.59
D0 – parturition BW change, kg	-26	-20	4.55	0.19
Calving – D60 BW change, kg	-18	-26	5.62	0.21
D0 – 60 BW change, kg	-44	-44	5.18	0.99
D0 – 60 BCS change	-0.51	-0.51	0.14	0.97

[1]Supplements included: 1) Cottonseed meal supplement with no monensin (**Control)**; 2) Monensin added to control to supply 200 mg·hd^{-1}·d^{-1} (**MON;** Rumensin 90®, Elanco Animal Health, Greenfield, IN). Supplements were fed at a rate of 0.90 kg·hd^{-1}·d^{-1} for duration of the study.

[2]Weight was taken within 24 h of parturition.

Table 4. Effects of feeding monensin to beef cows on calf growth performance (Exp. 1)[1]

	Treatment[2]			
Item;	Control	MON	SEM	*P*-value
No. of calves	42	42		
BW, kg				
Birth	38.4	39.6	1.04	0.24
D45	59.9	64.5	4.39	0.02
D60	70.9	75.7	2.25	0.04
D0-60 ADG, kg	0.55	0.60	0.03	0.04
205 d adj WW, kg	222	228	6.54	0.33

[1]Dams consumed dietary treatments from late gestation through early lactation, for a total of 60 d.

[2]Supplements included: 1) Cottonseed meal supplement with no monensin (**Control)**; 2) Monensin added to Control to supply 200 $mg \cdot hd^{-1} \cdot d^{-1}$ (**MON;** Rumensin 90®, Elanco Animal Health, Greenfield, IN). Supplements were fed at a rate of 0.90 $kg \cdot hd^{-1} \cdot d^{-1}$ for duration of the study.

Table 5. Effects of feeding monensin to beef cows on cow milk production (Exp. 1)[1]

	Treatment[2]			
Item;	Control	MON	SEM	*P*-value
D 41				
No. of cows	13	19		
Milk production, kg	14.1	15.0	1.22	0.47
D 60				
No. of cows	24	31		
Milk production, kg	10.7	11.4	0.80	0.41

[1]Milk production was determined on D 41 (April 19) and D 60 (May 10) by the weigh-suckle-weigh procedure.

[2]Supplements included: 1) Cottonseed meal supplement with no monensin (**Control)**; 2) Monensin added to Control to supply 200 $mg \cdot hd^{-1} \cdot d^{-1}$ (**MON;** Rumensin 90®, Elanco Animal Health, Greenfield, IN). Supplements were fed at a rate of 0.90 $kg \cdot hd^{-1} \cdot d^{-1}$ for duration of the study.

Table 6. Effects of feeding monensin to beef cows on conception rate in (Exp 1)[1]

	Treatment[2]			
Item;	Control	MON	SEM	*P*-value
Pregnancy rate, %				
D 40 pre-weaning	77	78	0.06	0.94
Weaning	81	89	0.06	0.38

[1]Conception rate was determined by transrectal ultrasonography.
[2]Supplements included: 1) Cottonseed meal supplement with no monensin (**Control)**; 2) Monensin added to Control to supply 200 mg·hd^{-1}·d^{-1} (**MON;** Rumensin 90®, Elanco Animal Health, Greenfield, IN). Supplements were fed at a rate of 0.90 kg·hd^{-1}·d^{-1} for duration of the study.

Table 7. Effects of feeding monensin and a slow release urea with a fibrolytic feed enzyme to beef cows on cow performance (Exp. 2)

	Treatment[1]					Probability,[2] $P <$			
	Control	SRU	MON	Combo	SEM	Trt	C_1	C_2	C_3
No.[3]	27	27	27	26					
D0 weight, kg	549	550	541	536	9.20	0.51	-	-	-
D0 BCS	4.6	4.8	4.6	4.5	0.16	0.53	-	-	-
D26 weight, kg	548	549	540	536	9.64	0.64	-	-	-
D54 weight, kg	556	545	543	539	9.74	0.54	-	-	-
D54 BCS	5.3	4.9	5.0	4.8	0.17	0.22	-	-	-
D90 weight, kg	572	564	557	557	10.28	0.59	-	-	-
D90 BCS	5.6	5.2	5.2	5.2	0.16	0.07	0.07	0.18	0.95
D0 - D26 BW change, kg	5.27	5.39	6.33	6.50	3.14	0.98	-	-	-
D26 - D54 BW change, kg	7.91	1.84	3.45	4.56	2.95	0.05	0.69	0.09	0.22
D0 - D54 BW change, kg	13.1	1.18	8.53	9.79	3.80	0.07	0.55	0.11	0.23
D0 - D54 BCS change	0.76	0.04	0.39	0.33	0.18	0.02	0.83	0.02	0.55
D54 - D90 BW change, kg	15.5	13.9	11.2	14.8	3.34	0.73	-	-	-
D54 - D90 BCS change	0.49	0.31	0.05	0.33	0.19	0.31	-	-	-
D0 - D90 BW change, kg	28.7	16.4	20.4	25.3	4.84	0.17	-	-	-
D0 - D90 BCS change	0.93	0.19	0.33	0.51	0.14	< 0.01	0.27	0.03	0.11
Supplement intake, total kg	40.7	40.5	40.7	40.8	0.08	0.08	0.17	0.21	0.13

[1]Treatments included: 1) Cottonseed meal/wheat midd (**Control**), 2) Control plus soybean hulls, corn, and 61 g·cow-1·d-1 SRU (Optimase®; Alltech, Inc., Nicholasville, KY) (**SRU**), 3) Control plus soybean hulls, corn, and monensin to supply 200 mg·cow^{-1}·d^{-1} (Rumensin 90®; Elanco Animal Health; Greenfield, IN)(**MON**), and 4) SRU plus MON (**Combo**).

[2] C_1 = Control and SRU vs MON and Combo; C_2 = Control and MON vs SRU and Combo; C_3 = MON and SRU vs Combo.

[3]Spring calving Angus and Angus x Hereford gestating cows and heifers (N = 107; initial BW = 508 ± 72 kg; initial BCS = 4.7 ± 0.8; initial age = 4.2 ± 3 yr) were allotted randomly to one of four treatment combinations in a completely randomized design.

Table 8. Effects of feeding monensin and a slow release urea with a fibrolytic feed enzyme to beef cows on cow blood glucose and blood urea nitrogen (BUN; Exp. 2)

	Treatment[1]					Probability,[2] $P <$			
	Control	SRU	MON	Combo	SEM	Trt	C_1	C_2	C_3
No.[3]	27	27	27	26					
Glucose, mg/dL	41.5	42.1	38.8	43.2	1.69	0.30	-	-	-
BUN, mmol/L	1.09	1.59	1.44	1.69	0.10	< 0.01	< 0.01	< 0.01	0.05

[1]Treatments included: 1) Cottonseed meal/wheat midd (Control), 2) Control plus soybean hulls, corn, and 61 g·cow-1·d-1 SRU (Optimase®; Alltech, Inc., Nicholasville, KY) (SRU), 3) Control plus soybean hulls, corn, and monensin to supply 200 mg·cow-1·d-1 (Rumensin 90®; Elanco Animal Health; Greenfield, IN)(MON), and 4) SRU plus MON (Combo).

[2] C_1 = Control and SRU vs MON and Combo; C_2 = Control and MON vs SRU and Combo; C_3 = MON and SRU vs Combo.

[3]Spring calving Angus and Angus x Hereford gestating cows and heifers (N = 107; initial BW = 508 ± 72 kg; initial BCS = 4.7 ± 0.8; initial age = 4.2 ± 3 yr) were allotted randomly to one of four treatment combinations in a completely randomized design.

CHAPTER V

IN SITU RUMINAL DEGRADATION CHARACTERISTICS AND APPARENT DIGESTIBILITY OF LOW QUALITY PRAIRIE HAY FOR STEERS CONSUMING MONENSIN AND SLOW RELEASE UREA

S.K. Linneen,* A.R. Harding,* M.T. Smallwood,* G.W. Horn,* J.S. Jennings,† C.L. Goad,‡ and D.L. Lalman*

*Department of Animal Science, Oklahoma State University, Stillwater 74078; †Alltech, Inc., Nicholasville 40356; and ‡Department of Statistics, Oklahoma State University, Stillwater 74078

ABSTRACT

Seven ruminally cannulated crossbred steers (BW = 758 ± 62 kg) were used in a randomized crossover design (4 periods each 16 d) to evaluate in situ NDF and DM degradation characteristics of low quality prairie hay along with change in rumen VFA concentration and pH over a 24-h time period. Steers were allowed ad libitum access to prairie hay (4.8% CP and 55% TDN) and were provided one of four dietary cottonseed meal based 38% CP supplements at 0800 daily. Treatments included: 1) Cottonseed meal/wheat midd (**CONTROL**), 2) Control plus soybean hulls, corn, and 34 $g \cdot steer^{-1} \cdot d^{-1}$ slow-release urea with a fibrolytic feed enzyme (Optimase®; Alltech, Inc.,

Nicholasville, KY) (**SRU**), 3) CONTROL plus soybean hulls, corn, and 0.25 g·steer^{-1}·d^{-1} Rumensin (Rumensin 90®; Elanco Animal Health; Greenfield, IN)(**MON**), and 4) SRU plus MON (**COMBO**). Steers were adapted to diets for 12 d prior to incubation of prairie hay. On d 9 of the adaptation period, dry matter intake (DMI) was measured and fecal samples were collected twice daily for 5 d to determine apparent digestibility. Duplicate forage samples were incubated for 0, 2, 4, 6, 8, 12, 16, 24, 36, 48, 72, and 96 h in steers consuming the same forage. On the final d of the in-situ procedure, rumen fluid was collected 10 times over a 24 h period. Acid detergent insoluble ash (ADIA) was used as an internal marker to estimate particulate passage rate (Kp). Dry matter intake was not affected by treatment ($P = 0.13$). Steers consuming MON had lower ($P < 0.01$) rumen pH than cows consuming all other treatments and had a lower ($P = 0.01$) acetate as a percentage of total VFA than steers fed all other treatments. A time x treatment ($P < 0.01$) interaction was observed for rumen ammonia N due to a rapid increase followed by a quick decline in NH_3-N by steers consuming SRU and COMBO that was not observed by steers consuming all other treatments. Slow release urea did not affect apparent digestibility of forage; however, DM, NDF, and ADF apparent digestibility were increased by monensin. Combining monensin with SRU improved forage utilization among steers consuming low quality prairie hay. Including monensin in the supplement offers a means of improving efficiency by increasing forage digestion due to an increase in propionate and a reduction in feed intake. Replacing a portion of the cottonseed meal with SRU did not reduce animal performance, or digestibility, making it a valid replacement for true protein in the winter cow supplementation program.

Key Words: In situ, monensin, steer, urea

INTRODUCTION

In the Southern Great Plains, supplements for beef cattle grazing dormant winter forage typically contain degradable intake protein (DIP) sources such as cottonseed meal or soybean meal. Nonprotein nitrogen (NPN) is recognized as a readily degradable source of DIP, and is a less expensive source of crude protein than cottonseed meal and soybean meal per unit N. Nonprotein-N has an undesirably rapid release of ammonia in the rumen, causing asynchrony between protein and carbohydrate fermentation (Huntington and Archibeque, 1990). Slow release urea products, such as biuret, Starea®, or more recently, Optigen® (encapsulated urea; Alltech, Inc., Nicholasville, KY) have been developed to decrease the rate of ammonia production. Utilizing NPN to replace a portion of protein from oilseed is advantageous because bacteria that ferment structural carbohydrates prefer ammonia over AA or peptide, and increased ammonia availability in the rumen has been shown to increase OM digestion (Huntington and Archibeque, 1990; Köster et al., 2002).

Optimase® (Alltech, Inc., Nicholasville, KY; **SRU**) is a product that combines a slow-release NPN with a fibrolytic feed enzyme (FFE) and has been shown to have similar N release rate as soybean meal (Akay et al., 2004). Kononoff et al. (2006) indicated that the slow release technology of Optimase® can effectively replace soybean meal in a dairy ration containing high forage without compromising cow performance. Although there is no published research on the efficacy of feeding beef cows a FFE, combining the slow-release NPN with a FFE is a novel characteristic of this product, compared to other slow-release NPN products.

Monensin is an ionophore commonly fed to feedlot cattle to improve efficiency due to reducing DMI or improving average daily gain (Bretschneider et al., 2008). It is well recognized that monensin alters VFA production in favor of propionate, at the expense of acetate and somewhat butyrate (Russell and Strobel, 1989; McGuffey et al., 2001; Ellis et al., 2012). Additionally, monensin is thought to have protein sparing characteristics by sparing amino acids that would otherwise be used for gluconeogensis (Muntifering et al., 1980; Owens et al., 1980). Furthermore, Turner et al. (1988) reported that cows maintained more BW through calving and early lactation when they consumed 90% of the protein requirement plus monensin compared to cows fed 100% of the protein requirement with no monensin. This suggests monensin caused increased nutrient utilization by cows consuming forage. To the authors knowledge, the only previous study looking at the effects of feeding beef cows monensin, urea, and a combination of the two in a winter supplement along with low-quality prairie hay was completed by Lemenager et al. (1978c). The authors found no differences in cow performance regardless of whether the cows received monensin, urea or both, and they found the addition of monensin to reduce rumen ammonia concentrations. The reduction in rumen ammonia among cattle consuming monensin was verified by Muntifering et al. (1980) and Yang and Russell (1993).

Including monensin in supplements for grazing animals has the potential to improve forage utilization, which has broader implications for grazing systems, such as increased stocking rate. Combining this advantage with a slow release urea, such as Optimase®, offers an alternative source of protein for winter supplementation programs. The objective of this study was to evaluate the effects of monensin, SRU, and a

monensin/SRU combination on in situ DM and NDF degradation of forage in steers serving as a model for a winter cow beef supplementation program.

MATERIALS AND METHODS

This experiment was conducted in accordance with an approved Oklahoma State University (OSU) Animal Care and Use Committee protocol.

Animals. This experiment was conducted at the Range Cow Research Center, North Range Unit located approximately 16 km west of Stillwater, OK. Seven ruminally cannulated crossbred steers (BW = 758 ± 62 kg) were used in a randomized crossover design (4 periods each 16 d) to evaluate change in rumen VFA concentration and pH over a 24-h time period and in situ N, NDF, and DM degradation characteristics of low quality prairie hay. Steers were allowed ad libitum access to prairie hay (4.8% CP and 52% TDN) and provided of one of four supplements (38% CP each) daily. Steers were fed at protein deficient rate intended to determine differences among treatments; therefore, diets were formulated to contain 647 DIP g/d (DIP balance of -2%), which was calculated based off steer intake of 2% of BW and supplement TDN = 56% (Table 1). All supplements were also balanced for Ca and P to meet NRC (1996) requirements. Treatments included: 1) Cottonseed meal/wheat midd (**CONTROL**), 2) Control plus soybean hulls, corn, and 34 g·steer^{-1}·d^{-1} slow-release urea with a fibrolytic feed enzyme (Optimase®; Alltech, Inc., Nicholasville, KY) (**SRU**), 3) CONTROL plus soybean hulls, corn, and 1.26 g·steer^{-1}·d^{-1} Rumensin (supplying 250 mg·steer^{-1}·d^{-1} Rumensin 90®; Elanco Animal Health; Greenfield, IN)(**MON**), and 4) SRU plus MON (**COMBO**). Soybean hulls and corn were added to SRU and Combo treatments to make the supplements isonitrogenous. Dietary supplements were fed to provide an equal mg/kg

per BW basis of CP (38%). The BW used to determine feeding amount was collected at the onset of each period after a 12 h withdrawal from feed and water. Chemical composition of the dietary supplements is show in Table 1. Steers were adapted to this diet 12 d prior to initiating the in situ experiment each period.

In Situ Procedure. The procedures used in this experiment were adapted from Vanzant et al. (1998). Bag weight was recorded after Dacron bags (Ankom Technology, Macedon, NY; 10 x 20 cm, 53 ± 15 µm pore size) were labeled with a waterproof permanent marker. All samples were ground in a Wiley Mill (Model 4, Thomas Scientific, Sweedesboro, NJ) to pass a 2-mm screen before being weighed into the Dacron bags. Five grams (as-fed) of low quality prairie hay (94.5% DM; 4.7% CP, 55% TDN; DM basis) were weighed in to duplicate Dacron bags and sealed using a rubber band around a #6 rubber stopper. Before insertion into the rumen, bags were preincubated in tepid water (39°C) for 20 min to remove water soluble fractions and reduce wetting lag time. After the preincubation, all bags (except 0 h) were inserted under the rumen mat in the ventral rumen in a mesh laundry bag in reverse order. Across the 96-h incubation period, bags were inserted at 1900 h on d 1; 1900 h on d 2; 1900 h on d 3; 0700 and 1900 on d 4; and 0300, 0700, 1100, 1300, 1500, and 1700 h on d 5. These times correspond to incubation times of 96, 72, 46, 36, 24, 16, 12, 8, 6, 4, and 2 h. After removal from the rumen, bags were washed per individual steer in a washing machine on the delicate setting for a 1-min rinse and 2-min spin cycle, with 10 replicates. Bags were oven dried after rinsing at 50°C for 72 h and allowed to equilibrate to atmospheric conditions for 60 min at room temperature prior to weighing to determine DM. Duplicate forage residue samples from each incubation time were composited within individual

steer prior to further analysis. Forage and forage residue samples were analyzed for NDF content using an Ankom Fiber Analyzer (Ankom Technology, Macedon, NY) at the OSU Animal Science Nutrition Lab and for N content using a Leco CN-628 N Analyzer (Leco Corporation, St. Joseph, MI) at the OSU Soil, Water, and Forage Analytical Laboratory.

Total NDF and DM were partitioned into three fractions (A, B and C) based on susceptibility to ruminal degradation. The A fraction was considered to be immediately soluble while the C fraction was deemed unavailable to rumen degradation and the B fraction was the portion that was degraded at a measurable rate (Coblentz et al., 2002). Regression was used to determine degradation kinetics of the percentage of DM and NDF remaining on incubation time. Data were fitted to the nonlinear regression model described by Mertens and Loften (1980). The A and B fractions, lag time and the fractional rate constant (K_d) were determined directly from the nonlinear model. The C fraction was determined experimentally and equals the residual in the 96-h bags. The effective rumen degradability (RD) was calculated according to Ørskov and McDonald (1979) using the equation: Extent = $A + [(B \times K_d)/(K_d + K_p)]$ where K_d = rate of degradation of B fraction and K_p is passage rate from the rumen, as described below.

Approximately 24 h after the in situ procedure was completed passage rate was determined by procedures described by Coblentz et al. (1999). Manual evacuation of rumen contents was conducted for each steer before feeding (0 h) and four h post feeding. Total rumen contents were weighed, mixed, and subsampled in triplicate, then returned to the rumen. The samples were subsequently dried for 48 h at 50°C in a forced air oven prior to grinding through a 2-mm screen with a Wiley Mill. Hay and orts were collected during each period per steer and used to determine ADF and ADIA, as described below

in apparent digestibility. Fractional passage rate of ADIA (kp) was determined by dividing the mean ADIA intake (grams per h) by the mean (from the 0- and 4-h ruminal evacuations) ruminal mass of ADIA (Waldo et al., 1972). The hourly intake of ADIA for each steer was calculated by dividing total daily intake of ADIA by 24 h.

Rumen Fluid Collection. Beginning on d 15 of the 16 d period, rumen fluid was collected from each steer at 10 timepoints post-feeding across 24 h. Feeding time was moved up 1 h on this d to accommodate all the necessary collections. Collection times post-feeding included: 30 min (0730), 1 h (0800), 2 h (0900), 4 h (1100), 6 h (1300),7.5 h (1430), 11.5 h (1830), 16.5 h (2330), 20.5 h (0330), and 24.5 h (0730). Steers were fed the following d (d 16) at 0700 again, 30 minutes prior to the final rumen fluid collection. Rumen fluid was strained through four layers of cheese cloth to ensure that approximately 100 ml of fluid was collected at each sampling. A small portion of the sample was placed into a disposable beaker to determine pH using a pH electrode (Oakton pH 6+, Oakton Instruments, Vernon Hills, IL). The pH reading was read in duplicate per steer per collection and reported as an average. The remaining sample was placed into duplicate (2 tubes/steer) sterile 50 ml falcon tubes (BD, Franklin Lakes, NJ) containing 5 ml of HCl per 50 ml tube to terminate microbial growth. Samples were placed on ice and later stored at -20°C until analysis for VFAs and rumen ammonia (NH_3-N).

Samples were analyzed for VFAs and NH_3-N at the Langston University E (kiki) de la Garza American Institute for Goat Research Analytical Laboratory. Concentrations of VFA were measured using gas chromatography (Hewlett-Packard 6890 gas chromatography, 183 X 0.635 cm column, Supelco SP 1200 packing, N2 carrier at 30

mL/min, flame ionization detector at 250°C). Rumen ammonia N was determined by automated analysis (Bran Luebbe™ AutoAnalyzer 3, SEAL Analytical, Mequon, WI).

Apparent Digestibility. Individual steer hay intake was recorded from d 8 to d 12 of the 16 d collection period. Fecal grab samples were also collected twice daily at 0800 and 1700 h per steer to estimate fecal output from acid detergent insoluble ash (ADIA) concentration. Subsamples of the supplement, hay, and orts were dried at 100°C for 48 h to determine DM. Supplement, hay, orts, and feces were dried at 50°C and ground in a Wiley mill (Model 4, Thomas Scientific, Sweedesboro, NJ) through either a 2-mm (supplement, hay, and orts) or a 1-mm (feces) screen before analysis. After grinding, samples were composited by steer within period. Composited samples were analyzed for CP, NDF, ADF, and ADIA. Neutral detergent fiber and ADF content were determined using an Ankom Fiber Analyzer (Ankom Technology, Macedon, NY) according to the manufacturer's instructions. Samples were analyzed for N content using a Leco CN-628 N Analyzer (Leco Corporation, St. Joseph, MI) and ADIA was determined as what remained after complete combustion of the residue (Van Soest et al., 1991).

Statistical Analysis. Steer performance, degradation characteristics, and apparent digestibility were analyzed using the PROC MIXED procedure of SAS (SAS Inst. Inc., Cary, NC) and the Satterthwaite approximation for degrees of freedom. For steer performance, the model included supplement treatment, steer, sequence, and carryover as independent variables. If carryover was not significant, it was removed from the model. Period by steer within sequence was used as a random effect. Rumen fluid characteristics (NH_3-N, VFA, pH) were analyzed as repeated measures using the PROC GLIMMIX procedure of SAS. Fixed effects included supplement treatment, sequence, period, time,

and time by treatment. Random subject effects included period by steer within sequence. Preplanned single-degree-of-freedom contrasts were used to determine the effect of: 1. Control and SRU vs Mon and Combo (C_1), 2. Control and MON vs SRU and Combo (C_2), and 3. MON and SRU vs Combo (C_3). If the treatment effect was not significant, the contrast results were not reported. For all analyses, when the *P*-value for the F-Statistic was ≤ 0.05, least square means were separated and reported. Tendencies were reported at $0.05 < P\text{-value} < 0.15$.

RESULTS

Animal performance. Inclusion of SRU or monensin in the supplement did not affect ($P > 0.32$; Table 2) final steer BW. Dry matter intake was also not affected ($P = 0.21$) by SRU, but monensin tended ($P = 0.07$) to reduce DMI.

Rumen environment characteristics. There was a significant treatment by time interaction for pH ($P = 0.02$; data not shown) because pH declined over time after feeding for all treatments, but at different magnitudes. Inclusion of SRU in the supplement in the current study increased ($P < 0.01$; Table 3) mean pH over the 24 h collection. Feeding steers SRU did not affect ($P > 0.36$) acetate, butyrate, propionate, acetate:propionate ratio, or total VFA production. Adding monensin to the dietary supplement increased ($P = 0.01$) the amount of propionate as a percentage of total VFA production, resulting in lower acetate and butyrate ($P = 0.01$ and $P = 0.02$, respectively) being produced as a percent of the total. Monensin also reduced the acetate:propionate ratio ($P = 0.01$). Combining MON and SRU did not influence VFA concentration ($P > 0.21$).

A significant treatment by time interaction ($P = 0.01$; Figure 1) for rumen NH_3-N was present. Rumen ammonia concentrations were highest ($P = 0.01$) for steers consuming the Combo supplement compared to steers consuming all other supplements, until 6 h post feeding, when the NH_3-N levels were similar among steers on all four treatments for the remainder of the 24 h. Rumen ammonia concentrations for steers consuming SRU were similar ($P = 0.14$) to those from steers receiving the Control and MON supplements 30 min post feeding. From 1 to 6 h post feeding, steers consuming the SRU treatment had greater ($P < 0.05$) NH_3-N concentrations than steers receiving the Control and MON supplements. Finally, the NH_3-N level among steers consuming either Control or MON was not different ($P > 0.17$) for the duration of the 24 h collection.

Degradation characteristics. There were no differences in rumen fill, % ADIA of rumen contents, or passage rate ($P > 0.48$; Table 2) due to dietary treatment. Dry matter or NDF degradation, lag time, or rumen degradable DM were not affected by supplement treatment ($P > 0.20$; Table 4).

Apparent digestibility. Apparent digestibility of DM, NDF, ADF, and CP were not affected by the inclusion of SRU in the dietary supplement ($P > 0.25$; Table 5). Inclusion of monensin in the supplement improved DM, NDF, and ADF apparent digestibility ($P \leq 0.05$); however, it did not improve CP apparent digestibility ($P = 0.47$). Combining MON and SRU increased ($P < 0.01$) digestibility of DM, NDF, and ADF, compared to feeding MON or SRU alone.

DISCUSSION

In a meta-analysis on feedlot cattle receiving monensin, DMI was significantly reduced by 3.1% among the 151 experiments summarized (Duffield et al., 2012). Measuring DMI in grazing animals consuming monensin is much more difficult resulting in very few studies reporting this response. Bretschneider et al. (2008) summarized 13 experiments in a review on feeding monensin to grazing cattle and concluded that monensin did not significantly improve DMI. These authors recognized that forage quality and type of supplement (energy or protein) used to deliver monensin may differ among studies, and that the majority of the studies in the review used growing cattle. Bretschneider et al. (2008) also reported an increase in ADG of 12.1% among grazing cattle consuming monensin when comparing 46 experiments. Sexten et al. (2011) supported this claim when they reported a 45% increase in cow gain among those consuming monensin with no change in DMI. Lemenager et al. (1978b) reported an improvement in cow efficiency as well, but this was derived from no differences in cow BW change and a significant reduction of 20% in forage intake among cows consuming monensin. The authors estimated intake from esophageal samples, which is different than most other studies that measured true cattle forage intake.

In the second experiment completed by Lemenager et al. (1978b), they estimated DMI based on grazing time and found that time spent grazing was similar during winter dormancy, but when grass became green cows consuming monensin spent 22% less time grazing. This suggests that forage quality influences animal response to monensin, which was also recognized by Bretschneider et al. (2008). Monensin tended to decrease forage DMI in the current study, which is consistent with the intake results during dormancy

reported by Lemenager et al. (1978b) since the forage used in this study was very poor quality. Inconsistencies in the response of grazing cattle to monensin supplementation described in the literature may be due to differences in experimental design, forage type and quality, or simply because few grazing studies have measured forage intake. Regardless, it appears that the intake response to monensin by cattle is more pronounced among feedlot animals, when monensin is included in diets with sufficient metabolizable protein and available energy.

A treatment by time interaction was expected for pH, as the response changes over time relative to feeding (Rumsey et al., 1970). The authors of this study felt the overall treatment means, regardless of time, were most important. Monensin did not affect pH, which is consistent with results of Dinius et al. (1976) who reported no change in pH when grazing animals were supplemented with monensin. Feeding monensin to grain-fed cattle typically increases ruminal pH (Vagoni et al., 1995) by moderating lactate production (Russell and Strobel, 1989). Monensin has also been shown to decrease propensity for digestive disorders in feedlot animals (Cooper et al., 1997) by reducing daily ruminal pH variation. In contrast, others have suggested that pH will mirror the response of the acetate:propionate ratio (Lana and Russell, 1997; Lana et al., 1998). This relationship between VFA ratio and pH may have more application in studies using low quality forage, rather than cereal grains plentiful in carbohydrates.

This study supports previous observations that feeding urea increases ruminal pH, especially immediately after feeding (Chalupa, 1968; Zinn et al., 2003). Due to inconsistencies in sample time relative to feeding, there is a great deal of variation in results among comparable studies. Wahrmund and Hersom (2007) reported that urea did

not affect pH; however, slow-release urea in the form of Optigen® (Alltech, Inc., Nicholasville, KY) reduced mean pH among cows consuming bahiagrass hay. Other studies indicated that pH was unaffected when urea and high fiber diets were fed (Köster et al., 2002; Farmer et al., 2004). The current study measured pH in rumen fluid immediately after collection at 10 timepoints over 24 h after feeding, which the authors felt was a representative and accurate profile of treatment influence on ruminal pH of steers consuming low quality hay.

Urea, and slow-release NPN products, are not generally thought to impact VFA concentrations (Lemenager et al., 1978c; Farmer et al., 2004; Taylor-Edwards, 2009). Although ruminal pH was not statistically influenced by monensin, the numeric decrease in pH among steers consuming monensin matches the pattern of reduction in acetate:propionate. Increasing propionate at a cost of acetate and butyrate is a hallmark response of feeding monensin regardless of whether the diet is high concentrate or high forage (Lemenager et al., 1978a; Lana and Russell, 1997; Ellis et al., 2012). These results indicate that even when steers are in a negative DIP balance, monensin shifts VFA production in favor of propionate. Monensin acts on gram positive bacteria in the rumen while gram negative bacteria are resistant, resulting in sustained production of succinate leading to increased propionate production (Ellis et al., 2012). Propionate is used more efficiently than acetate by the tissues (Schelling, 1984), in addition to being used for gluconeogenesis. That leads to improved energetic efficiency by the animal, even among those consuming low quality forage (Lemenager et al., 1978c; Walker et al., 1980; Schelling, 1984).

Rumen ammonia concentration indicates microbial proteolysis of protein, with lower values indicating less deamination of true protein that is influenced by energy intake (Rémond et al., 1993). Low rumen ammonia concentrations also reflect dietary N deficiency or limited endogenous nitrogen supplied from recycled urea. Rumen ammonia concentration in this study, especially among steers consuming the Control and MON supplements, were lower than expected for the diets that contained no dietary urea. According to Satter and Slyter (1974), level in which maximum microbial protein synthesis occurs is 5 mg NH_3-N· 100 ml^{-1}, with 2 mg NH_3-N· 100 ml^{-1} limiting microbial protein synthesis. Diet composition can affect ruminal NH_3-N levels resulting in lower rumen NH_3-N concentration in high fiber diets, ranging from 2-3 m*M* (Yang and Russell, 1993; Vagnoni et al., 1995; Lana and Russell, 1997). The average rumen NH_3-N concentration in this trial for Control fed steers was 1.06 mg NH_3-N·100 ml^{-1}. This may have been due to supplement feeding frequency, poor forage quality (4.7% CP, DM basis), or because supplements were formulated below the DIP requirement of the steers. Infrequent protein supplementation encourages diurnal variation in rumen NH_3-N concentration, allowing it to decline to levels not conducive to microbial protein synthesis depending on the source of forage. The concentration of NH_3-N among steers in all treatments in the current study returned to a baseline level around 6 h. García-González et al. (2007), Males et al. (1979), and Forero et al. (1980) also reported that NH_3-N levels returned to a similar baseline level 4 to 6 h after feeding.

Steers voluntarily consumed only 1% of BW of forage per day, which is below the expected consumption rate of 2% of BW, potentially causing more urea recycling. Low intake of fermentable carbohydrates can affect fiber digestion and urea utilization.

Specifically, cellulose may have been hydrolyzed and fermented too slowly for bacteria to optimize use of microbial protein (Galo et al., 2003). More simply, low intake may have supplied inadequate energy to rumen microorganisms for either microbial growth or converting ammonia to urea (Huntington and Archibeque, 1990; van der Merwe et al., 2001). The low NH_3-N concentrations, seen especially from 6 to 24 h post feeding, indicates reduced protein degradation and fiber digestion (Vagnoni et al., 1995), although this is not reflected in reduced steer performance. Supplying sufficient levels of UIP is also a concern when feeding NPN, especially in the circumstances of this experiment since diets were formulated to be deficient in DIP. It is possible that low intake of both UIP and DIP in the current study decreased metabolizable protein supply potentially not meeting the metabolizable protein requirement of the animal. Furthermore, the metabolizable protein requirement of the microorganisms may have been challenged because of a deficiency in branched chain amino acids required by some microorganisms (Farmer et al., 2004; Köster et al., 1997; Köster et al., 2002).

Monensin has been shown to reduce rumen NH_3-N concentration by potentially reducing ammonia producing microorganisms (Yang and Russell, 1993; Vagnoni et al., 1995) or defaunating the rumen (Huntington and Archibeque, 1990). In this study rumen NH_3-N was not different between steers consuming the Control and MON supplements; however, combining monensin with SRU significantly increased NH_3-N. Lemenager et al. (1978c) and Vagnoni et al. (1995) found that combining monensin with urea lowered rumen NH_3-N concentrations compared to urea alone in cattle consuming low quality native range hay and Bermudagrass, respectively. Considering protein sparing characteristics of monensin, it was hypothesized in the current study that combining

monensin with urea would reduce rumen ammonia levels to a more optimum release rate, causing less ammonia to be wasted and presumably more microbial protein production. The cause of higher rumen NH_3-N concentration for steers fed the COMBO treatment compared to steers consuming the SRU treatment remains unclear. In other literature, rumen NH_3-N levels were not affected by monensin (Walker et al. 1980; Faulkner et al., 1985; Lana and Russell, 1997), magnifying the inconsistent response of rumen NH_3-N concentration to monensin.

Monensin acts on gram positive microorganisms, although some of them are resistant to monensin, such as small and large species of *Entodinia* (van der Merwe, 2001) and *Streptococcus bovis* (Dawson and Boling, 1987). Yang and Russell (1993) have hypothesized that gram positive microorganisms resistant to monensin are active ammonia producing microorganisms, which was verified by Huntington and Archibeque (1990). Many gram positive microorganisms use amino acids and peptides as energy resulting in protein being spared when they are reduced by monensin. Additionally, research indicates that monensin reduces protozoa population in the rumen (Ankrah et al., 1990; van der Merwe et al., 2001), resulting in an increase in propionate, bacterial protein, and bypass protein, as well as a decrease in pH (Lana and Russell, 1997). Apparent digestibility of fiber was improved and pH was not influenced by monensin in the current study, which may simply be a result of incomplete defaunatinzation (eliminating protozoa from the rumen) compared to other research. The quantity of protozoa was not measured in this study, so it is not possible to determine the degree of defaunatinzation in the rumen. Defaunating the rumen may not be entirely favorable

anyway because protozoa are beneficial for degrading fibrous constituents when cattle consume high fiber rations such as fed in this experiment.

Rumen fill as a percent of BW was approximately 1.6% lower in steers consuming supplements containing monensin (11 vs. 9.3%), which reflects only a tendency in differences in DMI among steers. These results are corroborated by Yang and Russell (1993) and Vagnoni et al. (1995), who also found no differences in passage rate due to monensin. Faulkner et al. (1985) reported an increase in particle passage rate, but not liquid passage rate, among cattle consuming high fiber diets with monensin. Both Ellis et al. (1984) and Bretschneider et al. (2008) suggested that rumen fill regulating dry matter intake may limit the response of monensin for cattle consuming forages. Considering the forage quality of hay used in this study, physical bulk of the forage may have limited DMI resulting in similarities in passage rate among treatments.

Apparent digestibility was unaffected by SRU, which is reflected in similarities in DMI among steers when they consumed each supplement. Feeding urea typically increases digestibility of OM and fiber compared to unsupplemented cattle (Owens et al., 1980; Lee et al., 1987; Currier et al., 2004). It does not improve digestibility compared to a control treatment receiving a supplement with a natural protein (Swingle et al., 1977), varying levels of urea (Farmer et al., 2004), or when comparing urea to slow-release NPN (Ammerman et al., 1972; Forero et al., 1980). In the current study, consideration was given to using a negative control, unsupplemented group of cattle, rather than the positive control that was ultimately used. The intention of this study was to use a control treatment that closely matched current production practices in the Southern Great Plains to determine differences among treatments in application to producers. Unlike dated

literature, the inclusion of SRU did not diminish forage utilization compared to a positive control, indicating that SRU is a reasonable replacement for an oilseed meal.

The effect of monensin on fiber digestion in previous literature is very inconsistent. Monensin has been shown to either improve (Faulkner et al., 1985; Sexten et al., 2011), reduce (Poos et al., 1979) or not affect (Dinius et al., 1976; Lemenager et al., 1978a) fiber digestibility. Interestingly, in Sexten et al. (2011), improvements in DM, NDF, and ADF digestibility were also observed, yet only a tendency for improved CP digestibility was reported. Bypass protein is increased if monensin spares protein in the rumen, which may result in decreased proteolysis, but not a large enough magnitude to affect CP digestibility. Improved digestibility from feeding monensin has often been attributed to a reduction in feed intake causing reduced passage rate (Owens, 1980). Passage rate was not impacted in the current study, similar to observations of Faulkner et al. (1985). Ellis et al. (1984) suggested that monensin can affect digestibility, even when passage rate is unchanged. Other mechanisms must be contributing to increased digestibility, such as increased energy availability for fiber digestion due to a shift in the VFA profile in favor of propionate.

A novel characteristic of this study is the slow-release urea product used, Optimase® (Alltech, Inc., Nicholasville, KY), in combination with a fibrolytic feed enzyme (FFE). Fibrolytic feed enzymes are included in the diet to improve fiber digestion by stimulating microorganisms in the rumen, and often increasing passage rate (Murillo et al., 2000; Beauchemin et al., 2004). Rode et al. (1999) and Hristov et al. (2007) verified the improvement in fiber digestion when FFE in the form of xylanase was fed; however, multiple other studies have found no impact of FFE on digestibility (Pinos-

Rodríguez et al., 2002; Avellaneda et al., 2007; Giraldo et al., 2008). Fibrolytic feed enzymes are substrate specific resulting in limited enzymatic activity if the forage substrate does not match the enzyme (White et al., 1993). This may cause inconsistencies in results across studies. Additionally, Adesogan et al. (2014) identified multiple challenges to maximizing enzyme effectiveness, including rumen pH. Xylanase activity is most optimal at a pH of 5 (Adesogan et al., 2014), which would suggest that the rumen pH (μ = 6.8) of steers in this study limited xylanase activity. The contrast statements used in this study were intended to determine the effect of monensin, SRU, or the combination of the two. Due to this treatment structure it is not possible to determine whether the FFE in the SRU product directly influenced forage digestibility in this study.

The combination of monensin and SRU (COMBO) was included in this study to determine if the potential benefits of the two products may be additive. Combining the two products has the potential to provide producers an alternative protein source to use in the winter cow supplementation program in the Southern Great Plains and potentially reduce the amount of forage required to maintain cows. Combining the two products reduced forage intake, increased rumen ammonia nitrogen availability up to 6 h post feeding, and improved DM, NDF, and ADF digestibility. Combining monensin with SRU improved forage utilization among steers consuming low quality prairie hay. The only other studies that combined monensin and urea in a high fiber diet were those by Davis and Erhart (1976), Lemenager et al. (1978c), Poos et al. (1979), and Vagnoni et al. (1995). Both Davis and Erhart (1976) and Poos et al. (1979) used high concentrate diets fed to steers and lambs, respectively. Poos et al. (1979) reported many results in contrast to the ones derived from this study. Both Lemenager et al. (1978c) and Vagnoni et al.

(1995) found that combining monensin and urea reduced rumen ammonia concentration compared to a treatment only containing urea. As mentioned previously, it remains unclear as to why the Combo treatment in the current study increased rumen ammonia concentration above that of urea, especially since FFE has been shown to have no impact on rumen ammonia concentration (Avellaneda et al., 2008; Giraldo et al., 2008; Hristov et al., 2008). The report of Lemenager et al. (1978c) is perhaps the most similar published experiment to the current study, as it was conducted in the Southern Great Plains using cows grazing native range. Although the study used cows, they also reported a 15% increase in propionate when monensin was fed in a 30% CP supplement in the first study. This did not correspond to an improvement (less) in cow weight loss in that study, so any improvement in efficiency would be seen by reduced intake, which was not measured.

IMPLICATIONS

The objective of this study was to evaluate in situ DM and NDF degradation characteristics of the forage due to influence of monensin, SRU, and the combination of the two in steers serving as a model for beef cows on a winter supplementation program. Including monensin in the supplement offers a means of improving efficiency by increasing propionate, resulting in reduced feed intake and increased forage digestibility. Replacing a portion of the cottonseed meal with SRU did not reduce animal performance, or digestibility, making it a valid replacement for true protein in the winter cow supplementation program.

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Table 1. Supplement composition and amount of nutrients supplied daily to steers

	Treatment[1]			
	Control	SRU	MON	Combo
Item;	% of DM			
Cottonseed meal	86.3	42.5	86.3	42.5
Wheat midds	8.09	31.8	7.90	31.7
Cane molasses	5.50	5.50	5.50	5.50
Soybean hulls	-	10.0	-	10.0
Corn	-	5.00	-	5.00
Optimase®	-	5.00	-	5.00
Rumensin 90®	-	-	0.19	0.19
Vitamin A, 30,000 IU/g[2]	0.11	0.11	0.11	0.11
Vitamin E 50%	0.05	0.05	0.05	0.05
	Nutrient supplied, kg/d[3]			
DM	0.71	0.75	0.76	0.77
CP	0.33	0.34	0.34	0.35
TDN	0.63	0.62	0.65	0.63
Crude fat	0.01	0.02	0.01	0.02
	Chemical composition, %			
DM	90.0	89.7	90.0	89.7
CP	40.1	40.8	40.1	40.8
TDN	76.2	74.0	76.0	74.0

[1]Treatments included: 1) Cottonseed meal/wheat midd (**Control**), 2) Control plus soybean hulls, corn, and 34 g·steer^{-1}·d^{-1} SRU (Optimase®; Alltech, Inc., Nicholasville, KY) (**SRU**), 3) Control plus soybean hulls, corn, and monensin added to supply 250 mg·steer^{-1}·d^{-1} (Rumensin 90®; Elanco Animal Health; Greenfield, IN)(**MON**), and 4) SRU plus MON (**Combo**).

[2]Provided 32,960 IU of Vitamin A per kg of supplement DM.

[3]Calculated based on average steer daily feed allotment per treatment for all periods.

Table 2. Effects of feeding monensin and a slow release urea with a fibrolytic feed enzyme on steer BW and DMI

	Treatment[1]					Probability, $P <$			
Item	Control	SRU	MON	Combo	SE	Trt	C_1	C_2	C_3
No. of animals	9	10	9	10					
Average BW, kg	711	715	727	728	15.9	0.32	-	-	-
Forage DMI, kg	7.69	8.73	7.50	7.46	0.43	0.13	0.07	0.21	0.18
Rumen contents									
Fill, kg	80.9	76.3	67.4	68.7	15.3	0.92	-	-	-
ADIA, %	3.20	3.37	3.38	3.39	0.11	0.66	-	-	-
Passage rate, %/h	0.37	0.62	0.55	0.58	0.95	0.48	-	-	-

[1]Treatments included: 1) Cottonseed meal/wheat midd (**Control**), 2) Control plus soybean hulls, corn, and 34 g·steer^{-1}·d^{-1} SRU (Optimase®; Alltech, Inc., Nicholasville, KY) (**SRU**), 3) Control plus soybean hulls, corn, and monensin added to supply 250 mg·steer^{-1}·d^{-1} (Rumensin 90®; Elanco Animal Health; Greenfield, IN)(**MON**), and 4) SRU plus MON (**Combo**).

[2] C_1 = Control and SRU vs MON and Combo; C_2 = Control and MON vs SRU and Combo; C_3 = MON and SRU vs Combo.

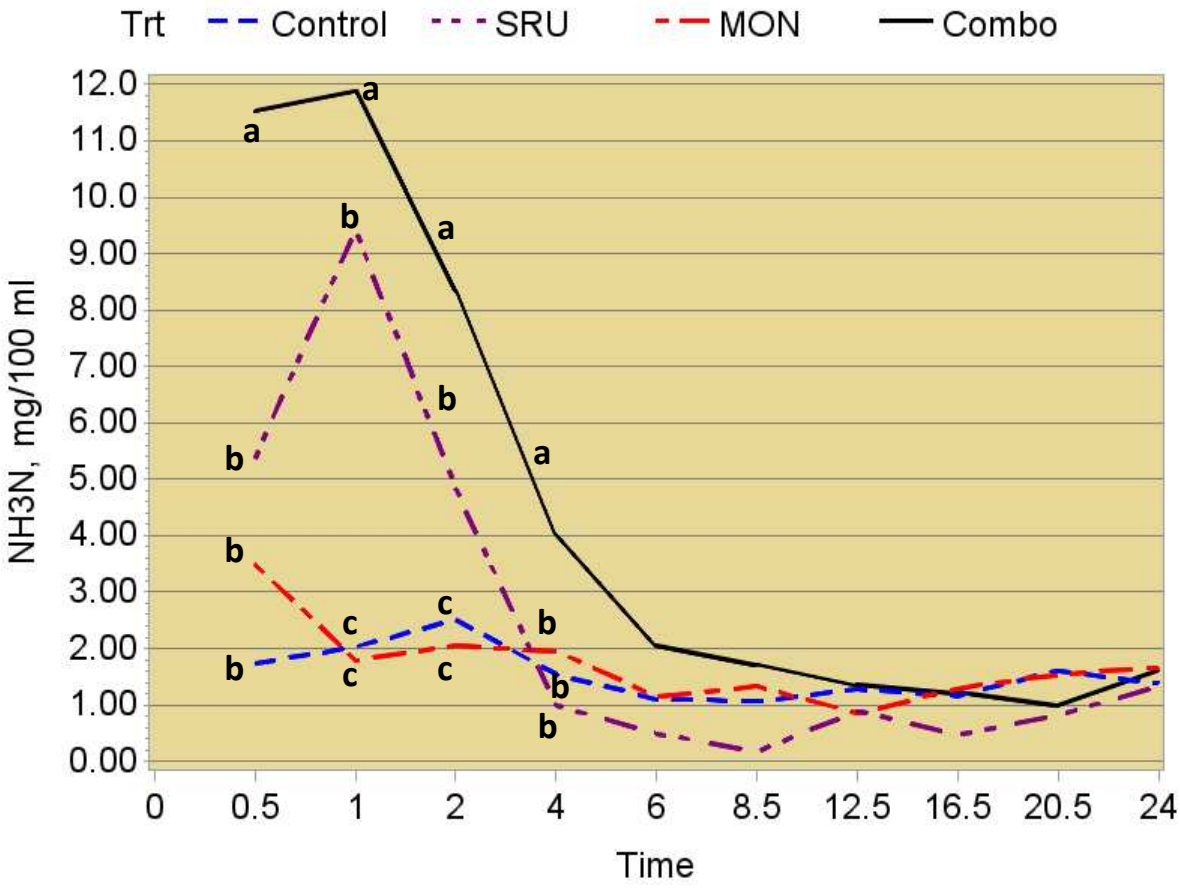
Table 3. Effects of feeding monensin and a slow release urea with a fibrolytic feed enzyme on rumen pH and VFAs

	Treatment[1]					Probability, $P <$			
Item;	Control	SRU	MON	Combo	SE	Trt	C_1	C_2	C_3
Rumen pH	6.87	6.88	6.75	6.96	0.04	< 0.01	0.41	0.01	< 0.01
VFAs, % of total									
Acetate	74.4	74.2	72.4	73.3	0.58	0.05	0.01	0.50	0.53
Propionate	15.8	16.0	18.6	18.0	0.79	0.08	0.01	0.84	0.48
Butyrate	9.6	9.8	9.2	9.3	0.24	0.11	0.02	0.36	0.21
Acetate:Propionate	4.85	4.86	3.95	4.07	0.24	0.05	0.01	0.76	0.28
Total VFA, mg/dL	70.4	78.5	66.9	71.3	3.09	0.23	-	-	-

[1]Treatments included: 1) Cottonseed meal/wheat midd (**Control**), 2) Control plus soybean hulls, corn, and 34 g·steer^{-1}·d^{-1} SRU (Optimase®; Alltech, Inc., Nicholasville, KY) (**SRU**), 3) Control plus soybean hulls, corn, and monensin added to supply 250 mg·steer^{-1}·d^{-1} (Rumensin 90®; Elanco Animal Health; Greenfield, IN)(**MON**), and 4) SRU plus MON (**Combo**).

[2]C_1 = Control and SRU vs MON and Combo; C_2 = Control and MON vs SRU and Combo; C_3 =MON and SRU vs Combo.

Figure 1. Effect of feeding monensin and a slow release urea with a fibrolytic feed enzyme on ruminal fluid ammonia concentration of steers over a 24 h period[1]



[1]Treatments included: 1) Cottonseed meal/wheat midd (**Control**), 2) Control plus soybean hulls, corn, and 34 g·steer^{-1}·d^{-1} SRU (Optimase®; Alltech, Inc., Nicholasville, KY) (**SRU**), 3) CONTROL plus soybean hulls, corn, and monensin added to supply 250 mg·steer^{-1}·d^{-1} (Rumensin 90®; Elanco Animal Health; reenfield, IN)(**MON**), and 4) SRU plus MON (**Combo**). Time is relative to feeding at time 0.

[abc]Means within time with different superscripts differ ($P < 0.05$).

Table 4. Effects of feeding monensin and a slow release urea with a fibrolytic feed enzyme on DM and NDF degradation

	Treatment[1]					Probability, *P* <
Item;	Control	SRU	MON	Combo	SE	Trt
DM degradation;						
A fraction, %	13.7	15.5	13.8	13.8	0.72	0.25
B fraction, %	43.8	42.5	38	39.7	4.08	0.64
C fraction, %	42.5	42.0	48.2	46.5	3.86	0.44
Lag, h	5.65	4.74	1.36	5.69	1.87	0.27
Rate of B degradation, % h^{-1}	3.27	2.69	3.56	4.19	0.51	0.25
Rumen degradable DM, %	52.3	48.9	41.5	47.9	4.19	0.20
NDF degradation;						
A fraction, %	13.8	15.0	14.6	13.7	0.69	0.47
B fraction, %	42.5	39.9	43.2	42.3	2.78	0.84
C fraction, %	43.8	45.1	42.1	44.0	2.71	0.91
Lag, h	4.12	6.37	2.47	4.91	1.28	0.25
Rate of B degradation, % h^{-1}	3.96	3.20	3.36	3.40	0.54	0.77
Rumen degradable DM, %	51.7	47.7	51.0	49.6	2.43	0.60

[1]Treatments included: 1) Cottonseed meal/wheat midd (**Control**), 2) Control plus soybean hulls, corn, and 34 g·$steer^{-1}$·d^{-1} SRU (Optimase®; Alltech, Inc., Nicholasville, KY) (**SRU**), 3) Control plus soybean hulls, corn, and monensin added to supply 250 mg·$steer^{-1}$·d^{-1} (Rumensin 90®; Elanco Animal Health; Greenfield, IN)(**MON**), and 4) SRU plus MON (**Combo**).

Table 5. Effects of feeding monensin and a slow release urea with a fibrolytic feed enzyme on apparent digestibility of DM, NDF, ADF, and CP

	Treatment[1]					Probability, $P <$			
Item;	Control	SRU	MON	Combo	SE	Trt	C_1	C_2	C_3
Apparent digestibility, %									
DM	63.0	60.5	62.0	68.2	1.48	0.01	0.04	0.25	< 0.01
NDF	64.5	60.9	63.3	69.8	1.87	0.03	0.05	0.45	< 0.01
ADF	57.5	46.2	53.4	60.5	1.79	< 0.01	0.01	0.26	< 0.01
CP	52.9	53.1	51.3	58.9	3.72	0.47	-	-	-

[1]Treatments included: 1) Cottonseed meal/wheat midd (**Control**), 2) Control plus soybean hulls, corn, and 34 g·steer^{-1}·d^{-1} SRU (Optimase®; Alltech, Inc., Nicholasville, KY) (**SRU**), 3) CONTROL plus soybean hulls, corn, and monensin added to supply 250 mg·steer^{-1}·d^{-1} (Rumensin 90®; Elanco Animal Health; Greenfield, IN)(**MON**), and 4) SRU plus MON (**Combo**).

[2]C_1 = Control and SRU vs MON and Combo; C_2 = Control and MON vs SRU and Combo; C_3 = MON and SRU vs Combo.

VITA

Sara Koren Linneen

Candidate for the Degree of

Doctor of Philosophy

Thesis: APPLICATION OF FEED ADDITIVE TECHNOLOGIES TO ENHANCE HEALTH AND PERFORMANCE OF FORAGE-FED CATTLE

Major Field: Animal Nutrition

Biographical:

Education:

Completed the requirements for the Doctor of Philosophy in Animal Science at Oklahoma State University, Stillwater, Oklahoma in July, 2014.

Completed the requirements for the Master of Science in Animal Science at Kansas State University, Manhattan, Kansas in 2007.

Completed the requirements for the Bachelor of Science in Animal Science at The University of Arizona, Tucson, Arizona in 2005.

Experience:

- Graduate research assistant, Oklahoma State University, August 2011 – Present
- Agriculture instructor, Western Oklahoma State College, August 2009 – June 2011
- Sow research coordinator, Swine Graphics Enterprises, May 2007 – June 2009
- Graduate research assistant, Kansas State University, August 2007 – March 2009

Professional Memberships:

- Oklahoma State University Graduate Student Association, August 2011 – Present
- Member of the American Society of Animal Science
- Certified in Beef Quality Assurance
- Certified in Pork Quality Assurance
- Certified in Masters of Beef Advocacy (Beef Checkoff)