EVALUATION OF INTAKE LIMITING AGENTS IN A SELF-FED DRIED DISTILLERS' SUPPLEMENT

A Thesis

by

JOEL DUSTIN SUGG

Submitted to the Office of Graduate Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

Chair of Committee,	Jason E. Sawyer
Committee Members,	Andy D. Herring
	William E. Pinchak
	Tryon A. Wickersham
Head of Department,	H. Russell Cross

August 2013

Major Subject: Animal Science

ABSTRACT

Response to range supplementation is in part driven by level of supplement consumed and amount of associated variation. In order to evaluate intake limiting agents in a self-fed dried distillers' grain supplement (DDG), heifers (n=59) in Trial 1 were offered an *ad libitum* amount of sorghum × sudangrass hay as well as DDG containing either no limiter (CON), monensin (185 mg/kg; MON), or one of six additional limiters alone or in combination with monensin (185 mg/kg, +M). Evaluated treatments and initial rates consisted of sodium chloride (NACL, 10%), urea (UREA, 2%), sodium bicarbonate (LIME, 1.68%), DL-malic acid (MLAC, 3%), calcium propionate (CAPR, 3%), and sodium bicarbonate plus urea (LIUR, 1.68% + 2%). Supplement intake was recorded daily and limiters were evaluated over three rates of inclusion, each for a duration of 14 d, on the basis of intake level, intake variation (cumulative stability), and rate of intake change over time (temporal stability). Data was analyzed as a 7 × 2 factorial initial 7 days of each period were removed to avoid acclimation influence. A baseline period was observed to ensure no inherent differences were detected. Within the initial rate period, limiter affected OM intake ($P = 0.02$) as consumption was reduced by NACL ($P < 0.01$) and tended to be lower when limited by MLAC ($P = 0.14$) and LIUR ($P = 0.11$). Neither monensin (P = 0.86) nor a limiter × monensin interaction were present. Cumulative stability was indicated that heifers consuming NACL ($P < 0.01$) and CAPR ($P < 0.01$) consumed supplement with greater regularity than did CONT. Monensin (P = 0.75) and monensin × limiter (P = 0.76) did not influence intake stability. Temporal stability was unaffected by limiter ($P = 0.43$), monensin ($P = 0.69$), or

monensin × limiter ($P = 0.93$). When rate of inclusion was 2 × initial rate, intake was affected by limiter ($P < 0.01$) with observations similar to the initial period. No monensin ($P = 0.49$) or interaction ($P = 0.27$) effect was present. Cumulative stability was unaffected by limiter ($P = 022$), monensin ($P = 0.39$), or interaction ($P = 0.86$). Temporal stability was increased with monensin (P 0.05) and an interaction resulted in an increased rate of supplement intake change in CONT when monensin was included. When supplement included limiters at 4 × the initial rate, Effects on intake and cumulative stability by limiter were the only significant responses. Intake of LIUR, NACL, and MLAC were reduced relative to CONT while NACL was consumed with greater regularity. Trial 2 was conducted to further compare sodium chloride and DL-malic acid as limiting agents. Each were included in a self-fed DDG supplement offered to steers (n=60, mean initial BW = 191 kg) at identical rates (8%, 16% 24%, and 32%) in addition to monensin (66 mg/kg). Within each rate, MLAC reduced supplement intake more effectively than NACL while cumulative stability and temporal stability measures were similar among limiter and only deviated from control levels at lower rates.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

CHAPTER I

INTRODUCTION AND REVIEW OF LITERATURE

Necessity for supplementation of livestock diets is driven by the magnitude of difference between nutritional value and quantity of available forage relative to nutrient level required to achieve production goals. Consumption of diets based on dormant warm season perennial grasses often generates a need for supplemental crude protein and, on occasion, energy to assist in both intake and digestibility of the forage base (DelCurto et al., 1990). In addition to protein and energy, vitamins and other nutrients may be offered at predetermined target amounts to mediate nutritional deficiencies. Similarly, supplemented feeds may be used as carriers for ionophores and/or antibiotics in which delivery of appropriate dosages are contingent upon an animal consuming at least a target amount of feed (Kunkel et al., 2000). Overconsumption will increase cost of supplementation and in the case of protein, animals are unable to efficiently utilize excess supply. Deviation from desired intake levels can lead to variance from the anticipated response to a supplemental regimen. Ideally, supplemented nutrients should be included and consumed in amounts only necessary to meet physiological demands within circumstance and without excess.

Intake variation

Supplement intake by grazing animals is often quantified by dividing supplement disappearance by number of animal days. As a result of evaluating intakes as a mean within a group, estimation of individual intake as well as intake variation are commonly unreported. This is common in a large portion of previously

published literature.

Individual intake variation has previously been reported as coefficient of variation (CV) for intake, proportion of animals in a group which consume minimal or no supplement (non-feeders), and proportion of animals within a group that consume a target amount of supplement (Bowman and Sowell, 1997). Intake variation in feedlot settings has commonly been assigned a residual value which is derived from the difference in daily intake relative to average daily feed intake over a period (Stock et al., 1995). Factors contributing to variation include form and palatability of supplement, mode of delivery, feed allowance, and multiple aspects of social interaction (Bowman and Sowell, 1997). To date, most studies estimating intake variation of free choice supplements to grazing animals have been conducted with molasses based blocks and liquid supplements.

Supplement form

Supplemental nutrients may be derived from a variety of sources which include ethanol by-products, oilseed by-products, animal by-products, forages, minerals, and grains. Diversity of supplement form includes meals, loose mixes, forages, cubes, blocks, or liquids. Physical form of feed has been identified as a factor influencing intake variation (Bowman and Sowell, 1997).

In an effort to describe differences in behavior and intake associated with form of supplemental protein, Garossino et al. (2003) assigned gestating beef cows either molasses blocks or liquid molasses. At least 70% of cows selected to receive liquid molasses consumed supplement on a daily basis whereas attendance to block supplement

ranged from 40 to 80%. No differences were detected in mean daily DM intake though standard deviation (SD) of daily intake was higher for the block treatment. Additionally, rate of consumption and was higher when supplement was in liquid form though duration of feeder attendance was reduced. These measures could directly relieve social dominance issues by increasing the proportion of time the bunk is unoccupied. Neither group of animals achieved the targeted mean daily intake. Before initiation of this trial, all cows were corralled with access to the block supplement which should eliminate novelty as a factor influencing variation of block intake in these observations.

Mulholland and Coombe (1979) provided wethers grazing wheat stubble with mineral blocks, urea-containing mineral blocks, liquid molasses, and urea-containing liquid molasses. Mean CV's were lowest for mineral blocks and highest for liquid molasses (44% vs. 64%) whereas values for the urea containing supplements were intermediate at 47% and 58% for blocks and liquid, respectively.

In multiple experiments evaluating intake and intake variation, Dixon et al. (2003) offered various supplements to grazing heifers. In Experiment 1, groups were assigned one of four treatments: 1) restricted amount of cottonseed meal (CSM), or *ad libitum* access to 2) liquid molasses containing 74 g/kg urea (M8U), 3) salt and urea-containing loose mineral mix (LMM), or 4) molasses blocks containing 62 and 99 g/kg, respectively, salt and urea (BLOCK). Intake variation expressed as CV among heifers within group after 10 weeks was highest for BLOCK (83%), intermediate for LMM (71%), and lowest for M8U and CSM (26% and 28%, respectively). Additionally, all heifers assigned CSM and M8U treatments consumed some supplement. Proportions of

non-feeders were 4% and 19% for LMM and BLOCK, respectively. These experiments suggest that both variation in supplement consumption and the proportion of non-consumers a herd are expected to be greater when supplements are offered in block form. In Experiment 2, heifers were offered *ad libitum* access to one of four liquid molasses supplements containing low amounts of urea (74 g/kg), high amounts of urea (107 g/kg), monensin (120-180 mg/kg), or meat meal. No significant differences in CV of supplement intake were detected among the four treatments. All heifers consumed supplement with exception of 30% of those assigned molasses containing meat meal.

Ducker et al. (1981) measured individual variation of block supplement intake of ewes from a wide range of environmental settings. Intake CV ranged from 46% to 231% among 15 flocks. The overall percentage of ewes not consuming any supplement was 19%. These authors attributed differences in intake to ewe age, alternative feed availability, and variation between flock locations.

Lobato and Pearce (1980) described influence of forage availability in relation to block supplement intake. Approximately 1200 total grazing ewes at 5 locations were offered molasses-urea blocks. After an initial 3 week period, 50% of ewes had consumed no supplement, and were removed and confined to yards where they were provided blocks and 350 g hay$\cdot$ewe$^{-1}\cdot$d^{-1}. After 3 weeks in confinement, percentage of non-feeders measured 19% and those were retained for an additional period ranging from 2 to 4 weeks during which 88% of ewes consumed supplement. Overall individual intake of supplement was highly variable on both pasture (100 to 400 g$\cdot$ewe$^{-1}\cdot$week^{-1}) and in confinement (100 to 500 g$\cdot$ewe$^{-1}\cdot$week^{-1}).

In a study of supplement and forage intake by ewes grazing native winter range, Taylor et al. (2002) fed supplemental protein in pellet (27% CP) and block (29.3% CP) form. Ewes receiving pelleted feed were group fed the wheat middling-soybean meal based supplement at 114 $g \cdot ewe^{-1} \cdot d^{-1}$ whereas ewes assigned to the block treatment were allowed *ad libitum* access. Mean consumption of the two treatments were 110 and 58 $g \cdot ewe^{-1} \cdot d^{-1}$ for the pellet and block supplements, respectively. Ewes receiving pelleted supplement had fewer non-feeders relative to those receiving block (2% vs. 35%), fewer individuals classified as having low-intake (5% vs. 10%), a higher proportion of animals consuming 51% to 150% of the mean intake (83% vs. 27%), fewer animals consuming excess supplement (10% vs. 28%), and a reduced CV of intake (32.0% vs. 99.5%).

Dove and Freer (1986) provided grazing lambs individually with sunflower meal (SFM) in loose meal or pelleted form. Mean intake of pelleted SFM was higher (388 vs. 335 $g \cdot lamb^{-1} \cdot d^{-1}$) and CV was reduced (9.9% vs. 21.2%). Similarly, Beck (1993) fed a self-limiting energy supplement to cattle grazing wheat pasture for four consecutive years. In years one and two, supplement was fed in pellet form and in meal form in years 3 and 4. Calculated CV's for individual supplement intake were 32% for years one and two (pellet) and 42.6% for years three and four (meal).

Intake variation of oats, hay, and urea-containing molasses blocks fed to grazing sheep was measured in multiple experiments by Lobato et al. (1980). Observed CV for oat grain intake was slightly lower than that of hay (23.4% vs. 30.5%). Measure for block intake was much higher at 143.6%. Mean intakes ($g\ DM \cdot hd^{-1} \cdot week^{-1}$) were highest for hay and lowest for molasses-urea block.

Tait and Fisher (1996) measured intake of a 1:1 salt-commercial mineral mix and found wide ranges of intake by grazing steers (50 to 300 $g \cdot hd^{-1} \cdot d^{-1}$) with a mean intake of 135 g/d and CV of 41%.

Collectively, these studies indicate that liquid supplements and dry feeds may deliver nutrients to grazing livestock with reduced variability relative to blocks.

Delivery method

Providing supplemental nutrients to grazing livestock can be achieved by delivering smaller amounts of feed intended for immediate consumption or by delivering feed in bulk that is designed or formulated to be consumed over extended periods. Hand-feeding a smaller portion allows for tighter control of daily intake but may increase operational costs associated with necessity of frequent delivery (Sawyer and Mathis, 2001). Due to feed supply in lesser amounts, hand-feeding my elevate level of competition and dominance issues leading to a higher proportion of non-feeders. These occurrences are influenced by numerous factors which include amount of accessible trough space, age of animals in the herd, and breed differences (Bowman and Sowell, 1997).

In contrast, self-feeding creates an opportunity to ameliorate costs by reducing frequency of travel and labor (Sawyer and Mathis, 2001). Energy and mineral supplements are especially suited for self-fed delivery since their efficacy is improved when offered daily. Benefits of providing protein in self-fed form are not as predictable due to flexibility in feeding frequency (Wallace and Parker, 1992; Huston et al., 1999). Additionally, manipulation of grazing distribution may be achieved by strategic

placement of stationary feeders (Bailey and Jensen, 2008). However, individual intake variation is expected to be higher in animals consuming supplement with this method (Bowman and Sowell, 1997).

Kendall et al. (1980) reported that heifers in confinement hand-fed a barley/SBM cubed supplement had individual supplement intake variation (CV) of 31%, compared to 57% in those fed blocks of the same formulation. These results were confirmed in a subsequent trial, where individual intake CV's were higher for blocks than for cubed supplement (82% vs. 55%, respectively).

Feed allowance

Feeding frequency may be drastically different depending on delivery method (hand-fed vs. self-fed) and, as a result, influence of feed allowance on intake variation must be considered. Schauer et al. (2005) reported no difference in CV values (28%) for protein supplement intake when cottonseed meal was offered to mature grazing cows daily and every sixth day (0.91 vs. 5.46 $kg \cdot hd^{-1} \cdot d^{-1}$). These data contradict observations of decreased variation when feed allowance is higher (Huston et al., 1999). When supplement is offered at identical daily rates, daily feedings will supply a smaller amount of feed per delivery. Less frequent delivery will increase feed availability for a period prior to depletion thereby decreasing competition and dominance issues. Schauer et al. (2005) measured intake and intake variation at a single point in time in this study and only a small portion of the population was observed. It is possible that the timing of data collection in this case did not provide a true indication of variability.

Foot et al. (1973) observed decreases in CV (36% to 16%) of intake by ewes when supplement allowance was increased from 100 to 453 $g \cdot hd^{-1} \cdot d^{-1}$. In evaluating block consumption by ewes, Ducker et al. (1981) identified an increase in proportion of non-feeders when mean flock consumption was lower and that the proportion decreased as mean consumption increased. Kendall et al. (1980) quantified intake variation of ewes provided low, intermediate, or high feed allowances offered in restricted, sufficient, or excess trough space and found that CV for supplement intake was inversely related to amount of supplement provided. Variability was further increased as trough space allowance was reduced.

Limitation of intake

Intake limiting agents

Diet selection by an animal is a function of nutritional demand and sensory acceptability of feed characteristics (Provenza, 1995, 1996 a,b). When provided with supplemental nutrients, unregulated consumption will often exceed amounts necessary to meet requirements. Supplemented nutrients generally make up a relatively small percentage of a ruminant's overall diet and level of necessary intake is likely reached well before satiety signals are received from gut hormones and central nervous system activity.

Sodium chloride is commonly used as a limiter of feed intake. Wide ranges (0.7%, Meyer et al., 1955; 45%, Judkins et al., 1985) have been included in feedstuffs with concentration adjustments occasionally needed due to increased tolerance (Kunkle et al., 2000). When salt is included as a limiter, a greater intake reduction per unit of salt

inclusion is expected with dry feeds relative to liquids. In addition, pelleted supplements diminish the effectiveness of salt as a limiter compared to loose mixes and feeds in meal form (Kunkle et al., 2000).

Gypsum ($CaSO_4$) has been successfully used to limit intake, and inclusion rates to meet targeted intakes may be lower than levels of salt required to achieve the same intake (Barrentine and Ruffin, 1958) though its use is less common due to considerations of high sulfur content and risks of toxicity or polioencephalomalacia. Similarly, calcium chloride has been used to effectively limit supplement intake when included at concentrations lower (2.5% to 5.0%) than those shown to be effective for salt. However, corrosive properties are a concern and excess calcium renders the ionic halide undesirable in feeds not high in phosphorous content (Kunkle et al., 2000).

Animal fat (yellow grease) included at a level of 10% in a supplement of No. 2 corn to grazing steers limited daily supplement intake to 0.79% BW (Wise et al., 1965). When salt was included in corn at levels to coincide with intakes of the fat-containing supplement, salt levels were similar and ranged from 7 to 10%. One advantage of fat inclusion is increased energy supply though difficulties associated with storage and handling in cold weather along with scouring and possible decreases in forage digestion are potential disadvantages. Wise et al. (1965) observed greater ADG and supplemental conversion over the six month trial when supplement was limited by fat.

Jensen (1979) measured the efficacy of numerous limiting agents in a ground corn supplement to cattle grazing irrigated pasture. In the first of multiple experiments, limiters (as a percentage of supplement) corn starch (50%), dehydrated potatoes (100%),

dicalcium phosphate (10%), and sodium tripolyphosphate (5%) were not effective at maintaining intake of supplement below a level of 1.82 $kg \cdot hd^{-1} \cdot d^{-1}$ after brief periods of adaptation. Another experiment utilized animal fat (7.5%, 10.0%, and 15.0%), sodium hydroxide (1.0%, 1.5%, and 2.0%), phosphoric acid (3.0%, 4.0%, 5.0%, 6.0%, and 7.0%), and phosphoric acid plus monensin (3.0%, 5.0%, 6.0%, and 7.0% in addition to 110 mg per kg) and again found no significant differences in overall intake among treatments ($P < 0.05$) with intakes generally increasing with animal adaptation. An additional experiment was conducted using sodium hydroxide (1.0%, 1.5%, and 2.0%), aluminum sulfate (4.0%, 6.0%, 8.0%, and 10.0%), ammonium chloride (2.0% and 6.0%), ammonium chloride plus ammonium sulfate (2.0% plus 6.0%), bone meal (8.0%), calcium chloride (4.0%), and corn gluten meal (60.0%) as intake modifiers. Sodium hydroxide at 2.0%, aluminum sulfate at 10.0%, and ammonium chloride at either 2% or 6% constrained supplement intakes to target levels. A comparison of aluminum sulfate plus monensin (5.0% and 10.0% plus 220 mg per kg), calcium carbonate plus monensin (5.0%, 10.0%, and 15% plus 220 mg per kg), and magnesium sulfate plus monensin (5.0% and 7.5% plus 220 mg per kg) suggested that all combinations successfully limited supplement intake although number of days observed were as few as three for some of these treatments. Measures of intake variation in this study are not reported.

Urea is included in most liquid feed products and its capacity to limit intake has been evaluated. Barker et al. (1988) fed a 3:1 urea/superphosphate blend in a grain supplement fed ad libitum to steers consuming low quality hay. Concentration of the

blend in the supplement was increased over a period of 20 weeks in 14-d intervals from 4% to 27%, resulting in a significant treatment effect on supplement intake with reductions as high as 70% compared to stable intake of supplement containing a constant inclusion of 2.67%.

Schauer et al. (2004) compared the effectiveness of three intake limiters in a wheat middling based supplement offered in consecutive years to steers grazing native prairie from June through October in North Dakota. Sodium chloride (16%), ammonium chloride (3%) plus ammonium sulfate (2.25%), and calcium hydroxide (7%) were included in the supplement which was also hand fed at a rate of .50% and 0.49% initial body weight (BW) during year 1 and 2, respectively. Treatments were offered in pellet (4.4 mm) form with the exception of the calcium hydroxide treatment which was fed in meal form. Data were analyzed in 28 d periods to monitor possible interaction with seasonal change. In year 1, no differences were detected in supplement intake which averaged 2.69 kg $DM \cdot hd^{-1} \cdot d^{-1}$ or 0.64% BW. In year 2, anionic salts failed to limit intake during the first two periods as intakes reached 2.05 and 2.55 $kg \cdot hd^{-1} \cdot d^{-1}$ compared to hand-fed rates of 1.64 and 1.63. Among these limiters, calcium hydroxide was most effective compared to other treatments during the first two periods. Steers assigned to control in this study were used to measure performance differences due to supplementation and did not receive supplement. Steers offered supplement without limiter were hand fed at a rate of 0.50% initial BW. Therefore, efficacy of limiters relative to unrestricted access of supplement is unknown. Intake of all limiter-containing supplements increased relative to hand-fed intake over the trial and no treatment effects

were present during the last two periods. As a likely indication of acclimation to the limiters, mean increase in supplement intake across both years from initial to final period was 221%, 161%, and 202% for sodium chloride, anionic salts, and calcium hydroxide, respectively.

Malic acid

Studies evaluating influence of the dicarboxylic organic acids fumaric acid and malic acid in ruminant diets to date have primarily pertained to their role in methanogenesis. Newbold et al. (2005) suggests that propionate precursors may function as electron sinks competing with methanogens for ruminal H_2. Published results have highly variable, ranging from no effect (Beauchemin and McGinn, 2006; McCourt et al., 2008) to reductions in methane production as high as 75% (Wallace et al., 2006). Use of these propionate precursors to reduce methanogenesis may also influence feed intake. Multiple studies present evidence of reduced CH_4 emissions/day but not per kg DMI, and attribute these findings to reductions in total DMI when diets contain various levels of organic acids (McGinn et al., 2004; Beauchemin and McGinn, 2006; Molano et al., 2008).

Foley et al. (2009) observed linear decreases in total DMI by including malic acid in a supplement fed to heifers ($P < 0.001$) and steers ($P = 0.002$). When heifers were subjected to 3 levels of malic acid inclusion (0%, 3.5%, and 7.5%), a linear decrease was also observed in supplement ($P < 0.001$) and silage ($P = 0.01$) intake. Malic acid inclusions of 0%, 2.5%, 5.0%, and 7.5% in supplement fed to steers generated quadratic reductions to silage consumption ($P = 0.003$) and total DMI ($P <$

0.001). A tendency for a quadratic effect ($P = 0.07$) was observed for supplement intake. In each case, DMI was affected by initial increment with no other reductions at subsequent levels.

Wallace et al. (2006) observed intake of an *ad libitum* concentrate diet by lambs of 0.3 kg and 0.2 kg lower relative to a control group ($P < 0.10$) when fed either 100g/kg concentrate of fumaric acid or 117g/kg of encapsulated fumaric acid, respectively. Similarly, Molano et al. (2008) observed reduced DMI when wethers were fed ground lucerne with fumaric acid inclusions of 4%, 6%, 8%, and 10% of DM. Reductions in DMI (kg/d) and CV's for DMI relative to control were 0.04 and 5.94%, 0.27 and 18.18%, 0.21 and 19.28%, and 0.22 and 17.07%, respectively. These results appear to be quadratic though polynomial contrasts were not reported.

Conversely, numerous studies have produced no evidence of malic acid influence on feed intake (Kung et al., 1982; Martin et al., 1999; Montano et al., 1999, Carro et al., 2006; Wang et al., 2009). However, these studies included organic acids at rates much lower relative to other studies where effects were observed. Of these studies finding no influence on voluntary intake, the highest inclusion rate was 2.66% DM (Montano et al., 1999).

Monensin

Manipulation of rumen metabolism has been the focus of extensive research due to possible performance increases associated with improved fermentation. Monensin sodium is a commonly used feed additive ionophore (Schelling, 1984). A primary advantage of feeding monensin is a shift in volatile fatty acid (VFA) production in which

propionate is increased at the expense of acetate (Richardson et al., 1976). As a result, energetic efficiency is improved due to the role of propionate in both gluconeogenesis as well as direct oxidation via the citric acid cycle. Schelling (1984) summarized monensin influence on ruminants and classified its biological effects into modification of VFA production, change in feed intake, change in gas production, modified digestibility, protein utilization, rumen passage, and other effects.

Muller et al. (1986) provided results of multiple trials in which monensin was included with a target consumption of 200 $mg \cdot hd^{-1} \cdot d^{-1}$ (actual 177 $mg \cdot hd^{-1} \cdot d^{-1}$) in self-fed energy supplements to grazing cattle on various types of pasture. Supplements were limited by salt and level of inclusion (5% to 36%) was adjusted to maintain desired intake levels in each trial. Pooled data indicate that cattle consuming monensin had higher average daily gain (ADG; $P < 0.01$) and consumed 18.2% less supplement than control cattle. In addition, salt levels were lower and less frequent adjustments to salt level in order to maintain intake were required in groups fed monensin. In the trials included, means both minimum and maximum levels of salt required to maintain targeted intakes were reduced by monensin inclusion by 49.6 and 30.4%, respectively. These data indicate that monensin may serve as a limiter of supplement intake without sacrificing gain performance.

Potter et al. (1986) observed similar effects when monensin (200 mg $hd^{-1} \cdot d^{-1}$) was fed in supplements to steers fed forage based diets in confinement. In trials where feed intake was measured, pooled values for cattle fed monensin were lower (3.1%, $P < 0.01$) than for control groups. Improvements in ADG and feed:gain were also realized (*P*

< 0.01). Paisley and Horn (1996) reported monensin (165 mg/kg) reduced intakes of a self-fed energy supplement by steers grazing wheat ($P < 0.001$; .64 and 2.29 $kg \cdot hd^{-1} \cdot d^{-1}$, respectively). Catsaounis et al. (1980) top-dressed the feed of bulls with a mineral supplement with or without monensin and observed reduced total daily feed intakes (8.9%) by bulls consuming monensin. Weight gain and feed conversion were increased (1.8% and 11.3%, respectively). Intake of corn-based supplement by finishing heifers on ryegrass was 25% less ($P < 0.05$) when the supplement contained 66 mg/kg monensin (Utley et al. 1978). No difference was observed in ADG between treatment groups. Jensen (1979) found that supplement intake was only temporarily held to target levels when monensin was included in a corn supplement at concentrations of 220 mg/kg, 330mg/kg, and 440 mg/kg. Intakes increased after cattle acclimated to treatments.

Dried distillers' grains

Supplements to grazed forage systems are provided to increase individual animal performance and their value is estimated by the extent of increased production as a result of improved nourishment. Due to relatively high crude protein and energy density levels, dried distillers' grains may be an ideal supplement to dormant warm season forages.

Roughly two-thirds of the protein content within DDG is undegradable (UIP, by-pass) by ruminal microbes (NRC, 1996). Though lower levels of animal production may be sustained from microbial crude protein alone, DDG as a supplement to grazing ruminants has the capacity to facilitate higher levels of production by contributing a considerable proportion of protein to the small intestine. MacDonald et al. (2006) measured daily gains when an alternate UIP source was supplemented at a level to

provide the amount of bypass protein obtained from DDG. Results indicated that gain response was only about 40% of that realized with DDG supplementation on growing forages. Implications are that about one third of gain increases observed with DDG supplementation may be due correcting microbial protein deficiencies.

As an added convenience, fat levels in DDG render the co-product a potential supplemental energy source supplying 120% the energy value of corn. The fiber in DDG is highly digestible and in combination with fat levels, increases value as a viable energy source (Lodge et al., 1997). Conventional grain-based energy supplements have reduced fiber digestibility of low and moderate quality forages when offered at levels up to 0.9% BW (Chase and Hibberd, 1987). Due to removal of starch during production of distillers' by-products, reduced competition between amolytic and cellulolytic microbes are expected, thus reducing the occurrence of negative associative effects.

Positive associative effects have been observed with use of supplemental DDG. Neither inclusion of supplemental UIP using corn gluten meal nor fat via corn oil in a supplement at levels contained within DDG resulted in observed ADG by heifers offered a DDG supplement on bromegrass pastures (MacDonald et al. 2007). This indicates that the combined attributes of proportional UIP of total crude protein and fat in DDG contribute the uniqueness of DDG and its potential to provide performance increases in grazing ruminants.

Numerous measures of performance increase and forage substitution are available. Heifers consuming various forage qualities supplemented with DDG up to 0.95% BW had increased overall ADG and consumed less forage per unit increase of

DDG intake (MacDonald and Klopfenstein, 2004; Morris et al., 2006). Luepp et al. (2009) fed increasing levels (up to 1.2% BW) of DDG to steers fed moderate quality forage and observed linear decreases in hay OMI and linear increases in both DDG and total OMI. Additionally, Loy et al. (2007) offered a DDG supplement both daily (0.4% BW) and on alternate days (0.8% BW) to heifers consuming grass hay (8.0% CP) and reported similar decreases in hay DMI and increases in total DMI.

From an economic perspective, value of DDG as a supplement can be derived from the sum of values attributed to increased performance and reduction in forage consumption. At the very least, DDG should be considered a viable option in selection of a highly valuable forage supplement. Allocation of DDG worth between forage replacement and animal performance is highly variable. In a trial by Morris et al. (2006), 89.1% of DDG value was attributed to improved animal performance with DDG supplement to low quality forages being 3.3 percentage units higher than the value on high quality forage (90.8% vs. 87.5%) when fed at identical rates. This result would be consistent with expectations due to greater potential for nutritional mediation in lower quality forage conditions. MacDonald and Klopfenstein (2005) grazed heifers on bromegrass and estimated that DDG supplement worth was primarily a factor of forage replacement (62.4%). In regards to DDG value partitioning, a portion of the discrepancies between these two studies may be attributed to inconsistencies in assigned value of grazed forage and higher rates of supplementation.

Market price of DDG is typically determined by the current price of conventional protein and energy feeds, soybean meal and corn. Common estimates of cost relative to

corn range from 70% to 90% on a DM basis. These prices are suspended by costs of processing that included drying but are lower than wet products due to reduced freight as a result of deliverable dry matter.

Published intake ranges of DDG up to 1.0% BW are numerous; however, estimates of unregulated DDG intake are elusive. Though demonstrations of forage substitution have been reported at various supplement rates, limitations to dietary inclusion exist due to sulfur concentration and, in the case of cattle fed in confinement, issues with physically effective fiber (peNDF) and fat levels when used as the primary energy source. Published rates of supplemental inclusions are as high as 3.6 and 3.5 $kg \cdot hd^{-1} \cdot d^{-1}$ for pasture and pen-fed studies, respectively (Griffin et al., 2009). Due to difficulties associated with measuring forage consumption on pasture, estimates from pen studies are primarily used to quantify forage intake as a response to supplement intake.

Despite enhancement of supplement potential due to nutrient profile, wide variances in concentration of other nutrients have been observed. Belyea et al. (1989) stated that CP% of DDG can range from 27% to 35%. Mean concentrations and CV of CP% (30.2%, 6.4%) and crude fat (10.9%, 7.8%) published by Spiehs et al. (2002) were similar to estimates of concentration and variance reported by Belyea et al. (2004) who sampled DDG over a five year period. These wide ranges may contribute to difficulty in formulating supplements or estimating allowances to meet animal demands. Primary sources of nutrient variation have not been positively identified. Belyea et al. (2004) analyzed corn supply to an ethanol plant and reported no correlation between

components of corn and DDG thus suggesting that variation is likely not associated with variability of nutrient profiles in sourced corn but instead inconsistencies in ethanol processing.

Along with issues in nutrient variability, physical structure is a characteristic of concern that may influence the application in feeding systems. Analysis of DDG particle form identifies some properties of concern in effectively delivering the feed to grazing animals (Rosentrater, 2006). Issues of binding in storage bins and bulk feeders are less severe with dried products relative to wet feeds; however, residual moisture, fat concentrations, and shape and structure of particles contribute to occasional issues (Behnke, 2007). Coincidentally, attributes that yield DDG a desirable feedstuff also complicates its potential to conform as a consistent pellet. Two of the primary examples are reduced starch, which serves to bind particles, and a higher level of hydrophilic content due to oils that reduce bonding capability (Behnke and Beyer, 2002).

Metaphylaxis and implants

Metaphylaxis

Metaphylactic antimicrobial therapy is a mass application practice intended to mitigate risk and implications of disease outbreak within a group. Treatment is usually administered upon arrival as cattle typically break within the first few days. Despite costs of blanket treatments, mass drug application may be desirable due to possible occurrences of subclinical illness or caretaker failure to identify symptoms (Nickell and White, 2010). In a compilation of studies evaluating metaphylactic use in arriving feeder cattle, Wileman et al. (2009) reported improved ($P < 0.01$) morbidity treatment and

mortality rates of 47% and 53%, respectively. This same analysis also reported an improved ADG of 0.11 kg in cattle subjected to metaphylaxis compared to untreated cattle. Step et al. (2007) evaluated single or multiple dose administration and observed response to treatment was improved when cattle were dosed a single time but that bovine respiratory disease morbidity was less severe when cattle were treated once on arrival and a second time eight days later.

Implants

Anabolic growth promoting agents improve gain performance of stocker cattle in a consistent manner by 10 to 15% and have the potential to increase response to supplementation programs (Kuhl, 1997). McMurphy et al. (2009) measured influence on ADG by multiple implant types with multiple supplements by steers grazing summer pasture. Implantation generated increased ADG (7.7%, $P < 0.01$) over the entire 126-d grazing period with no differences observed among implant types. Interaction of implant and supplement type was not significant though steers consuming a dried distillers' grain supplement had higher ADG relative to steers consuming cottonseed meal due to increased energy utilization. Despite absence of this interaction in the presence of increasing plane of nutrition, other studies suggest expectations of performance as influenced by implantation should be expected to increase as quality and availability of basal diet improve (Selk et al., 2006).

Sufficient evidence that DDG is a viable option for range supplementation to grazing cattle is available in previous literature. However, there is uncertainty of effective delivery methods other than hand feeding. Further evaluation of intake control

agents with an emphasis on novel intake limiters and their combinations with monensin may lead to an increase in value of the by-product if its delivery to can be reliably predicted.

CHAPTER II

ASSESSING THE EFFECTIVENESS OF INTAKE CONTROL AGENTS IN A SELF-FED SUPPLEMENT BASED ON DRIED DISTILLERS' GRAINS

All animal care and use procedures described in this protocol were approved by the Texas A&M University Animal Care and Use Committee (AUP 2009-239). Research was conducted at the Texas A&M AgriLife McGregor Research Center outside of McGregor, TX during the winter of 2010/2011.

Introduction

Dried distillers' grains can be provided to grazing cattle as a source of both supplemental crude protein and energy from fat and highly digestible fiber (Lodge et al., 1997; MacDonald et al., 2007). Numerous studies have documented intake and animal performance responses using hand fed supplemental DDG at various consumption rates (MacDonald and Klopfenstein, 2004; Morris et al., 2006; Luepp et al., 2009); however, previous literature is void of efforts to quantify intake of supplemental DDG provided *ad libitum*. Method of supplement delivery is expected to influence precision with which nutrient mediation is achieved (Bowman and Sowell, 1997). Providing a supplement to grazing cattle free choice will often result in higher variation in supplement intake and thus diminish efficacy (Kendall et al., 1980). Therefore, value of DDG as a supplement may be enhanced if methods to accurately control intake are identified.

Traditionally, sodium chloride has been included in range supplements for purposes of intake restriction. Ranges of inclusion are wide and necessary formulations have been as high as 45% (Judkins et al., 1985). Kunkle et al. (2009) noted that frequent

formulation adjustments may also be needed due to acclimation. Exploration of alternative agents using criteria of intake and variation of intake are warranted. Muller et al. (1986) found that inclusion of monensin in an energy supplement offered to cattle not only served to limit intake, but also improved longevity of limiter effectiveness. With this reasoning, our objective was to evaluate potential intake limiting agents included at multiple rates both alone and in combination with monensin at in a self-fed DDG supplement. Criteria of evaluation included supplement intake, variation in daily supplement intake, and stability of supplement intake over time.

Materials and methods

Fifty-nine Angus-sired, weanling heifers (191 kg mean initial BW) were used to evaluate the effectiveness of potential intake limiting agents with and without monensin in a self-fed corn distillers' grains (DDG) supplement. Heifers were stratified by weaning weight and randomly assigned (2 heifers per pen) to pens equipped with four Calan gates (American Calan, Inc., Northwood, NH). Each heifer was fitted with keys enabling access to two Calan gate feed bunks, one containing chopped hay and the other containing supplement. Initially, heifers were provided 1 kg/d of DDG and *ad libitum* access to sorghum x sudangrass hay in the adjacent bunk until acclimated to the feeding system and bunk assignments.

Following acclimation, heifers were provided with *ad libitum* access to DDG and hay. Refusals of supplement were weighed each morning prior to feeding for four days preceding application of treatments to determine baseline intake of DDG and hay by each animal. Heifers were weighed on the first day that treatments were applied.

Treatments were randomly assigned to pen and bunk. Treatments were arranged as a 7 X 2 factorial with 6 limiting agents plus a negative control offered alone or in combination with monensin (185 $mg \cdot kg^{-1} \cdot suppl.^{-1}$). Limiting agents were: no limiter (**CONT**), salt (**NACL**), urea (**UREA**), limestone (**LIME**), malic acid (DL-malic acid, food grade, Baddley Chemicals, Inc., Baton Rouge, LA; **MLAC**), calcium propionate (NutroCal, Kemin AgriFoods North America, Des Moines, IA; **CAPR**), and limestone with urea (**LIUR**). Supplements were prepared in bulk each week by combining limiting agent and DDG in a portable rotary mixer for a duration of at least five minutes and until visual appraisal of consistent particle distribution was achieved. Treatments were sampled from mixer immediately after manufacturing of each 45 kg batch and composited within period.

Treatments were applied during three sequential experimental periods with increasing rates of limiter inclusion among periods (Table 1). Period durations were 14, 15, and 13 days for periods 1, 2, and 3, respectively. Animals and treatments were not re-randomized in subsequent periods to avoid the influence of novelty on consumption responses. Additionally, rates were not randomized among periods to avoid creation of aversions in earlier periods that would unduly influence responses in later periods. Thus, rate and period were purposefully confounded, and data from each period were analyzed separately to make comparisons among limiters within experimental periods.

Table 1. Limiting agent inclusion rate by period in a self-fed dried distillers' grain supplement fed to yearling heifers (% inclusion in supplement, as-fed basis)

	Treatment[1,2]						
Item	CONT	NACL	UREA	LIME	MLAC[3]	CAPR[4]	LIUR
Baseline	Control	0	0	0	0	0	0
Period 1	Control	10	2	1.68	3	3	1.68 + 2
Period 2	Control	20	4	3.36	6	6	3.36 + 4
Period 3	Control	40	8	6.72	12	12	6.72 + 8

[1]Treatments were salt (NACL), urea (UREA), calcium carbonate (LIME), malic acid (MLAC), calcium propionate (CAPR), and calcium carbonate plus urea (LIUR).
[2]Treatments were fed alone or in combination with monensin (+M; 2.5%)
[3]Kemin AgriFoods North America, Inc., Des Moines, IA.
[4]Baddley Chemicals, Inc., Baton Rouge, LA.

All animals had continuous, *ad libitum* access to chopped sorghum x sudangrass hay (Table 2). Hay availability was monitored daily during supplement feeding and supplied on an individual basis when necessary. During each experimental period, supplement refusals were weighed at 0700 daily. Supplement was then added to replace disappearance from previous day plus 0.91 kg of additional supplement to ensure *ad libitum* access. Every seventh day, accumulated refusals of both supplement and hay were sampled and discarded then replaced with fresh feed to mimic weekly replenishment of a bulk feeder in practice. Refusal samples collected every seventh day were composited by animal and within period for DM and OM measure.
Samples of supplements, hay, and refusals of both were placed in a forced-air oven at 60ºC for 96 h to determine DM. Samples were ground to pass through a 1 mm screen of a Wiley mill (Thomas Wiley, Laboratory Mill Model 4, Thomas Scientific Co., Philadelphia, PA) and analyzed for ash, NDF, ADF, and CP. Organic matter was calculated from ash content of a 0.5 g sample placed into a muffle furnace (500º C) for 8

hours. Fiber content was estimated using ANKOM 200 Fiber Analyzer (ANKOM Technologies, Inc., Macedon, NY). Crude protein values were derived using Dumas combustion (Rapid-N-Cube, Elementar America, Inc., Mt. Laurel, N.J.). Nutrient composition of treatments and hay by period are provided in Tables 2, 3, and 4.

Table 2. Initial rate period nutrient composition of treatment[1,2] in a self-fed dried distillers' grain supplement and sorghum x sudangrass hay.

			Nutrient		
Item	DM%	OM%	NDF%	ADF%	CP%
CONT	90.24	94.03	43.03	12.39	27.80
NACL	91.48	85.71	38.54	10.59	26.59
UREA	90.88	93.90	43.62	11.26	29.79
LIME	91.15	92.75	44.33	11.94	29.52
MLAC	91.19	94.82	43.64	12.39	27.61
CAPR	86.92	91.61	42.85	10.80	24.97
LIUR	90.36	93.27	46.18	13.70	28.74
CONT+M	90.90	94.06	43.54	10.44	27.80
NACL+M	91.34	85.21	41.07	9.99	27.78
UREA+M	90.11	94.25	41.63	11.56	23.90
LIME+M	89.84	91.89	42.94	12.47	31.30
MLAC+M	90.90	93.14	45.64	14.45	27.75
CAPR+M	91.34	92.58	39.41	12.02	27.22
LIUR+M	90.76	90.70	41.39	12.40	27.43
Hay	93.77	91.02	56.79	28.45	9.94

[1]Treatments were salt (NACL), urea (UREA), calcium carbonate (LIME), malic acid (MLAC), calcium propionate (CAPR), and calcium carbonate plus urea (LIUR).
[2]Treatments were fed alone or in combination with monensin (+M)

Table 3. 2 × initial rate nutrient composition of treatments[1,2] in a self-fed dried distillers' grain supplement and sorghum x sudangrass hay.

	Nutrient				
Item	DM%	OM%	NDF%	ADF%	CP%
CONT	89.10	94.44	43.65	13.57	26.25
NACL	91.09	73.68	37.55	9.77	22.75
UREA	90.80	94.49	43.02	11.92	32.92
LIME	90.92	88.79	39.98	12.02	24.08
MLAC	91.33	95.17	41.22	10.29	25.50
CAPR	91.44	90.70	39.58	10.77	27.10
LIUR	90.08	90.33	37.48	10.54	31.06
CONT+M	89.73	94.44	46.06	10.08	24.67
NACL+M	92.18	76.29	31.62	7.92	22.78
UREA+M	89.98	94.28	40.33	11.01	34.80
LIME+M	89.18	90.25	39.95	9.42	25.55
MLAC+M	92.12	94.14	37.12	12.11	26.79
CAPR+M	91.64	92.09	40.64	11.75	24.62
LIUR+M	88.88	90.73	41.92	10.76	29.87
Hay	95.95	90.51	65.55	39.66	10.78

[1]Treatments were salt (NACL), urea (UREA), calcium carbonate (LIME), malic acid (MLAC), calcium propionate (CAPR), and calcium carbonate plus urea (LIUR).
[2]Treatments were fed alone or in combination with monensin (+M)

Table 4. 4 × initial rate nutrient composition of treatments[1,2] in a self-fed dried distillers' grain supplement and sorghum x sudangrass hay.

	Nutrient				
Item	DM%	OM%	NDF%	ADF%	CP%
CONT	91.10	93.85	40.94	11.07	27.66
NACL	94.07	57.31	21.35	4.18	14.98
UREA	89.76	93.11	40.48	10.90	37.49
LIME	91.14	88.79	40.31	9.79	28.04
MLAC	90.39	94.16	39.87	9.63	25.85
CAPR	89.60	88.32	38.42	9.22	24.71
LIUR	89.89	85.65	38.83	8.24	35.80
CONT+M	90.78	93.79	38.17	9.10	27.36
NACL+M	94.90	45.74	22.16	4.61	13.03
UREA+M	89.15	93.98	42.43	40.48	36.13
LIME+M	91.64	87.22	37.45	8.95	26.97
MLAC+M	91.91	94.73	38.72	9.65	25.82
CAPR+M	91.98	88.48	36.36	8.62	22.42
LIUR+M	89.26	91.67	36.55	10.22	37.71
Hay	94.66	90.29	56.26	27.65	10.42

[1]Treatments were salt (NACL), urea (UREA), calcium carbonate (LIME), malic acid (MLAC), calcium propionate (CAPR), and calcium carbonate plus urea (LIUR).
[2]Treatments were fed alone or in combination with monensin (+M)

Within observation periods, calculated standard deviation of supplement intake (cumulative stability), rate of change in supplement intake (temporal stability), coefficient of variation of supplement intake, and daily hay intake were evaluated along with direct measure of supplement intake. Temporal stability, or the directional change in daily intake over time, was quantified by regressing individual intakes within period using day as a first order predictor. After initial analysis, second-order regressions of supplement intake on time were estimated for each heifer. The derivative of estimated equations were set to zero and solved to determine a critical value of the predictor. A secondary analysis was conducted of data including days after the mean critical value

among treatments, in order to remove days within each period potentially contributing variance as a result of acclimation. All regression parameters were estimated using regression procedures of SAS 9.2 (SAS Inst. Inc., Cary NC). Daily hay intake was calculated by dividing weekly disappearance by 7.

Within periods, response variables (mean supplement intake, mean hay intake, cumulative stability of supplement intake, and temporal stability of supplement intake) were analyzed as a completely randomized design with a 7 × 2 factorial treatment arrangement using the mixed models procedure of SAS 9.2. Limiter type, monensin inclusion, and their interaction were included as effects in the model. Mean responses for each limiter were compared to the control treatment using F-protected t-tests. Pairwise comparisons among limiters were not performed. Effects were considered statistically significant when $P < 0.05$. Effects are discussed as tendencies or trends when $0.05 < P < 0.15$.

The effect of inclusion rate of limiters on response variables was evaluated using regression procedures of SAS v 9.2, where inclusion rate and inclusion rate squared were included as predictor variables in the model.

Results and discussion

Limiting agents evaluated were selected based on either potential to compliment the nutritional profile of DDG, industry wide acceptance, or novelty. Limestone was chosen and included at an initial rate to achieve a 2:1 calcium to phosphorous ratio for the DDG-based supplement (NRC, 1996). Waller et al. (1980) observed increased protein utilization when urea was included in a DDG supplement due to complimentary

protein attributes of urea (DIP) relative to protein composition of DDG. A combination of limestone and urea was evaluated as an ideal complement to the composition of DDG. Sodium chloride is easily accessible with a well-documented ability to limit intake in self-fed applications. Calcium propionate salts may have a similar effect on palatability. Calcium propionate is sour (acidic), provides some calcium to the mixture and may serve as a source of additional energy in the supplement, in contrast to entirely inorganic ingredients. Finally, malic acid was evaluated due to its intake reduction characteristics in limited application (Foley et al., 2009) and its use in flavoring applications in the food industry. Monensin was included in the design based on prior observations of supplement intake reduction (Muller et al., 1986) and its effect on apparent energy value of diets.

Baseline

Supplement intake. Mean daily supplement DM and OM intakes during the acclimation period were 3.03 kg and 2.83 kg, respectively. There were no differences in OMI ($P = 0.56$) among heifers assigned to receive different limiter treatments, nor among heifers assigned to receive treatments with or without monensin ($P = 0.92$). Additionally, no limiter × monensin combinations ($P = 0.84$) were detected. These expected results confirm similarity among heifers at the initiation of the trial, and that subsequent differences in consumption were likely not the result of individual variation.

Cumulative stability. Measures of supplement OM intake variability were not different among treatments during the acclimation period ($P = 0.93$). Neither inclusion of monensin ($P = 0.81$) nor combinations of assigned limiter × monensin ($P = 0.91$)

affected intake variability. Mean supplement OMI variability was 0.86 kg. As expected, CV for OMI was not affected by limiter ($P = 0.95$) or monensin ($P = 0.83$). In the absence of agents, no limiter × interaction was detected ($P = 0.90$). Results of analysis on cumulative stability in this period indicate that differences in subsequent periods are not due to individual response to DDG.

Temporal stability. Changes in supplement OM intake over the first four days of collection were not different among treatment groups ($P = 0.60$). Numerically, OM intake decreased daily by 0.11 kg. Neither monensin ($P = 0.37$) nor combination of limiter × monensin ($P = 0.60$) affected temporal change.

Hay intake. Consumption of forage was not different among treatment assignments (OMI, $P = 0.56$) and no differences were detected between groups receiving treatments with or without monensin ($P = 0.92$). As expected, no limiter × monensin combination was present ($P = 0.84$). Baseline OM intake of hay among all treatment groups was 0.99 kg/d.

Treatment Period 1: Initial rate

Supplement intake. The degree by which voluntary consumption of a limiter-containing feed is reduced relative to that of an unadulterated feed of the same base is most indicative of a limiting agent's potential. When intake limiters in this study were included in supplement at the initial rates, OM intakes among treatment groups were affected ($P = 0.02$). Relative to CONT, NACL reduced supplement OMI ($P < 0.01$); MLAC ($P = 0.14$) and LIUR ($P = 0.11$) tended to reduce supplement OMI compared to DDG alone. As a percentage of BW, this level of salt intake (0.14%) is consistent with

typical inclusion levels of 0.05 to 0.15% BW when used as a limiter (Kunkle et al. 2000). Addition of monensin did not influence supplement OM intake ($P = 0.86$), and no limiter × monensin combination effects on supplement OM intake were observed ($P = 0.66$). Supplement intake means and variation measures for limiter treatments within the initial rate period are shown in Table 5.

Based on estimates of the critical value (days) required to achieve stable intake after introduction of the supplement, a separate analysis was conducted over the final 7 d of the measurement period. Using this truncated data, mean supplement OMI was diminished by NACL ($P = 0.02$) relative to CONT, with a similar response observed in heifers provided MLAC ($P = 0.05$). No other treatments effectively reduced supplement intake levels from those of heifers assigned CONT. Addition of monensin had minimal effect on supplement OMI ($P = 0.34$) during the last 7 d of the first experimental period and no limiter × monensin interaction was observed (OMI, $P = 0.60$).

Published reports of *ad libitum* intake levels of DDG are not available. Consumption levels observed in this experiment were much higher than expected. Over the final 7 d of the initial treatment period, and at an initial BW of 191 kg, heifers provided unregulated supplement consumed 2.5 % of their BW as DDG, which is substantially higher than provisions in previous research which typically peaked near 1.5% BW (Griffen et al., 2009). Based on these high levels of unrestricted consumption, effective means to control intake are essential to the use of DDG as a self-fed source of supplemental nutrients.

Table 5. Period 1 intake of hay and supplement, and supplement intake variance measures of a self-fed dried distillers' grain supplement fed to growing beef heifers

	Treatment[1]										
	CONT	CAPR	LIME	LIUR	MLAC	NACL	UREA	SE	L[2]	M[3]	L × M[4]
	DMI, kg/d										
14 d											
Hay Intake	1.50	1.97	2.00[a]	1.95	2.33[a]	2.66[a]	1.56	0.18	0.01	0.16	0.51
Supplement Intake	4.27	3.86	4.01	3.65	3.57	2.64[a]	4.11	0.34	0.03	0.87	0.55
Cumulative stability	1.14	1.20	1.35	1.29	1.04	1.41	0.97	0.14	0.21	0.19	0.17
Temporal stability	0.15	0.22	0.23[b]	0.20	0.05[b]	0.25[a]	0.11	0.04	0.01	0.04	0.08
last 7 d											
Hay Intake	1.12	1.54	1.60	1.67[a]	2.11[a]	2.05[a]	1.28	0.20	0.01	0.05	0.12
Supplement Intake	4.80	4.74	4.86	4.33	3.66[b]	3.63[a]	4.49	0.41	0.15	0.34	0.48
Cumulative stability	1.04	0.65[a]	1.20	1.17	0.97	0.69[a]	1.00	0.12	0.01	0.67	0.74
Temporal stability	0.26	0.12	0.31	0.28	0.22	0.19	0.15	0.07	0.45	0.63	0.92
	OMI, kg/d										
14 d											
Hay Intake	1.39	1.80	1.84[a]	1.78	2.13[a]	2.43[a]	1.43	0.16	0.01	0.16	0.49
Supplement Intake	4.09	3.60	3.75	3.40	3.43	2.52[a]	3.94	0.31	0.02	0.86	0.66
Cumulative stability	1.06	1.10	1.24	1.18	0.96	1.19	0.90	0.12	0.35	0.17	0.13
Temporal stability	0.14	0.20	0.22[b]	0.19	0.05[b]	0.22[b]	0.10	0.03	0.01	0.04	0.10
last 7 d											
Hay Intake	1.05	1.41	1.48	1.53[a]	1.93[a]	1.87[a]	1.18	0.18	0.01	0.05	0.11
Supplement Intake	4.61	4.42	4.55	4.03	3.55[a]	3.38[a]	4.32	0.37	0.12	0.34	0.60
Cumulative stability	0.96	0.59[a]	1.09	1.06	0.89	0.55[a]	0.92	0.11	0.01	0.75	0.76
Temporal stability	0.23	0.11	0.28	0.26	0.20	0.15	0.14	0.07	0.43	0.69	0.93

[1]Treatments were: control (CONT; no limiter); salt (NACL, 10%); urea (UREA, 2%); calcium carbonate (LIME, 1.68%); malic acid (MLAC; 3%), calcium propionate (CAPR, 3%), and calcium carbonate plus urea (LIUR, 1.68 + 2%)

[2]Limiter effect

[3]Monensin effect

[4]Limiter × monensin

[a]Within a row, means with a superscript differ from CONT at $P < 0.05$

[b]Within a row, means with a superscript differ from CONT at $P < 0.15$

Effects of malic acid on total dietary DMI in this study were similar to previous observations where included at a similar rate. Foley et al. (2009) observed a total DMI reduction of 4% when inclusion was 2.5% relative to a control. However, concentrate was pelleted and fed mixed with forage - both of which may have contributed to depressed efficacy of malic acid as a limiter. Comparisons of total DMI between these studies as opposed to supplement/concentrate alone are warranted due to the fact that, in the referenced study, feed was mixed when offered to achieve a targeted 60:40 concentrate to forage ratio and was then separated at weigh back to quantify fractions. In the current study, when malic acid was included at 3% supplement intake was reduced by 2.5%; whereas including 10% NACL reduced supplement intake by 4.1% relative to CONT. Foley et al. (2009) achieved a slightly greater reduction in total DMI likely due to the fact that the malic acid inclusion was 2.5% of the total diet whereas our inclusion rate was limited to DDG alone. Calculated inclusion of malic acid as a proportion of total DMI would have been 1.90% during the last 7 d of the initial period.

Although the objective of this study is to evaluate limiting agents independently against a control, it is notable that MLAC was more than two (2.02) times as effective as NACL at reducing total DMI per unit of inclusion.

Cumulative stability. Supplementation response may be influenced by variability in consumption. Therefore, discovering the extent to which intake limiters influence variation is important. The standard deviation of daily supplement OM consumption indicates the cumulative stability of supplement intake. Cumulative stability of supplemental OMI was not affected by treatment ($P = 0.35$) nor monensin ($P = 0.17$)

when supplements contained the initial rate of limiter. A trend for a limiter × monensin interaction on DDG OMI ($P = 0.13$) was observed where intake variability of CONT was reduced ($P = 0.03$) from 1.34 kg/d to 0.78 kg/d when monensin was included. In addition, a trend towards increased cumulative stability was observed with heifers fed LIUR ($P = 0.08$) as variation decreased by 0.41kg/d with the inclusion of monensin (Figure 1).

Comparisons of supplement OMI coefficient of variation (CV) are included because intake means varied among treatments. Calculated CV for DDG was affected by limiter ($P < 0.01$) during the initial treatment period; NACL was most variable (CV = 47%) and differed from CONT ($P < 0.01$). The lowest CV value for supplement OMI was observed with heifers offered UREA (CV = 24%) followed by CONT (CV = 26%). Monensin inclusion did not affect CV of supplement OMI ($P = 0.40$) though a tendency for limiter × monensin combination effect was detected ($P = 0.09$). When monensin was included in the supplement, OM intake CV was reduced among heifers consuming CONT ($P = 0.06$) and LIUR ($P < 0.01$) by 54% and by 57%, respectively. In addition, a numerical decrease in supplement OMI CV was observed when monensin was included in combination with sodium chloride. An apparent increase in supplement OMI CV was detected when monensin was included in MLAC and UREA.

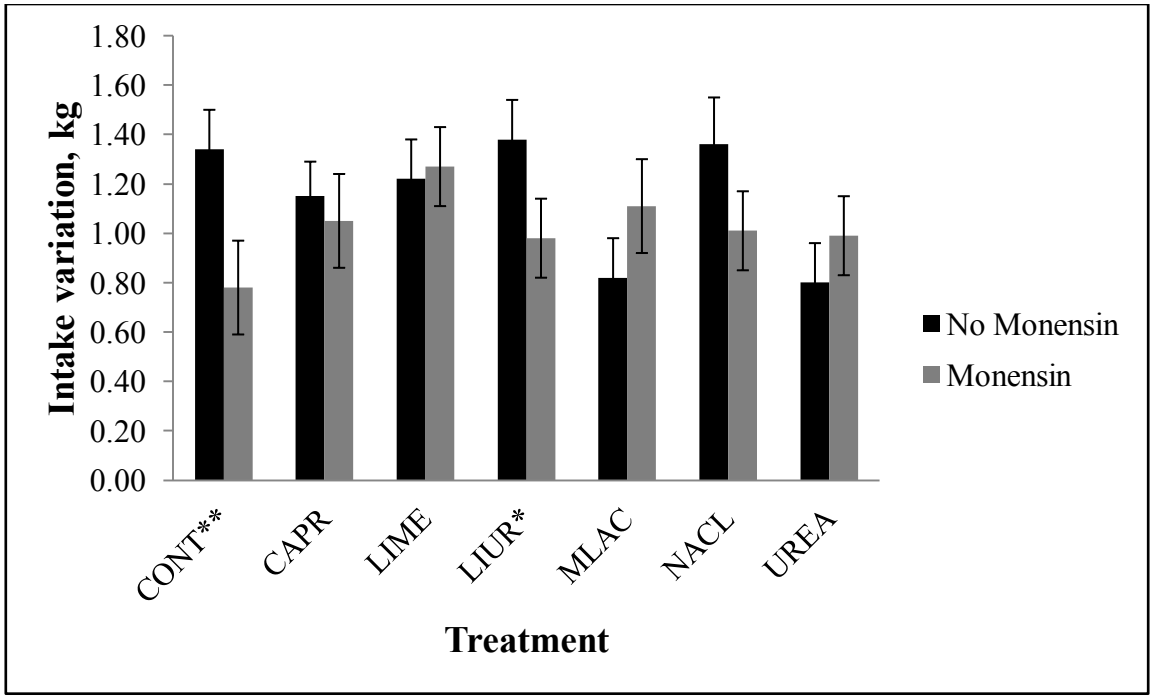

Figure 1. Cumulative stability of supplement OMI by treatment during the initial rate period. An "**" denotes significance at $P < 0.05$. An "*" denotes significance at $P < 0.15$.

When intake variation associated with treatment acclimation was removed, differences in OMI variability among treatments were magnified ($P < 0.01$). Heifers provided NACL ($P < 0.01$) and CAPR ($P < 0.01$) had lower supplement OM intake variation measures than those offered CONT. No other treatments differed from CONT in cumulative intake stability. In summation, treatments (MLAC and NACL) that effectively reduce DDG intake at these inclusion rates would be expected to do so without increasing the intake variability relative to that observed in heifers offered CONT.

During the last 7 d of the experimental period, monensin did not affect cumulative stability of supplemental OMI ($P = 0.76$) and no limiter × monensin

interaction was detected ($P = 0.76$). Absence of a monensin effect on variation is contradictory of some previous findings (Burrin et al., 1988; Stock et al., 1995; Paisley and Horn, 1996).

As a result of cumulative stability differences in the latter half of the period, it appears that acclimation to supplement during the initial days may have masked some influence of treatment. Regardless of monensin inclusion, measures of cumulative stability of NACL and LIUR during the terminal portion of the period were similar to those of during the entire period when supplement contained the ionophore. This suggests that although no overall treatment effect exists, monensin may aid in reducing variation in supplement intake resulting from initial exposure to sodium chloride or a combination of limestone and urea.

Temporal stability. A supplemental regimen to deliver nutrients or increase carrying capacity may be either short- or long-term. In either case, the ability of a self-fed supplement to be consumed at a stable rate over length of time - withstanding possible acclimation and/or aversion - is a valuable attribute. Supplements consumed with greater consistency over time, i.e., exhibiting temporal stability, will require less frequent adjustment to formulation and/or allowance to match targeted intakes. Mean rates of change in daily supplement OMI (slope of regression of intake on days) were used as indexes of the temporal stability of supplement intakes.

When limiters were included in supplements at the initial rates, temporal stability of supplement OMI was affected by limiter ($P < 0.01$) as well as monensin ($P = 0.04$). In addition, an interaction of limiter × monensin tended to influence temporal stability of

supplement OM consumption ($P = 0.10$). Supplement OMI tended to increase at a greater rate by heifers assigned NACL ($P = 0.010$) and LIME ($P = 0.10$) relative to CONT. In contrast, supplement OMI of MLAC tended to increase over time ($P = 0.06$), but at a more modest rate than intake of OM from CONT. Increasing intake of all treatments over time suggest that heifers became acclimated to DDG and all limiting agents, although to different degrees.

Inclusion of monensin improved temporal stability by reducing daily OMI change by 0.05 kg/d when included in the supplement. Interaction of limiter × monensin resulted in more modest increases in daily supplement OM intake by heifers offered CONT ($P < 0.02$) and NACL ($P < 0.02$) when monensin was included. Daily consumption was reduced by 0.16 kg OM/d in both treatments. In addition, temporal stability of supplement OMI tended to improve ($P = 0.14$) by 0.09 kg OM/d with the inclusion of monensin in heifers fed LIUR. Enhancements in temporal stability of supplement intake, generally and in combination with salt, are consistent with earlier reports of fewer required changes to limiter inclusion when formulated to include monensin (Muller, 1986).

Subsequent to removing data preceding the point of intake stability, analysis indicated that neither limiter ($P = 0.43$) nor monensin ($P = 0.69$) affected temporal stability of supplement OM intake. In addition, removal of the initial 7 d in the period removed the tendencies for a limiter × monensin interaction ($P = 0.93$).

Over time, cattle may develop an aversion and reduce daily consumption possibly due to post-ingestive feedback (Provenza, 1995), become acclimated to the

limiter and consume more supplement, or sustain a constant level of intake. Within this set of observations, heifers consuming CONT, UREA, LIME, MLAC, and LIUR all consumed more supplement during the latter portion of the initial rate period relative to the overall mean. However, none of the limiter containing treatments differed from CONT in their degree of daily supplemental OMI change. During the truncated portion of the initial rate period, OMI of CONT increased by 0.23 kg/d. Therefore, from a temporal stability standpoint, a limiter would be effective if its absolute value rate of change was 0.23 kg/d or less. No treatments resulted in daily intake changes different from CONT and only LIME (0.28 kg) and LIUR (0.26 kg) had daily changes in supplement OM intake numerically greater than CONT.

Mean daily supplement OMI change by heifers not fed monensin during the entire period was +0.18 kg/d. After days were removed to allow for acclimation, mean DDG consumption by heifers offered monensin increased by 0.21 kg/d. This data suggests that improved temporal stability by CONT and NACL treatment groups during the entire period was likely a result of a palatability response to monensin and that an acclimation occurred. From this, it appears that including monensin at the rate included here and in combination with the other agents at their respective levels will not influence temporal stability of supplement intake.

Hay intake. Encompassing the entire 14-d initial treatment period, hay OMI was affected by limiter ($P < 0.01$) with UREA being the only treatment group to consume a similar amount to CONT ($P = 0.84$). Heifers fed NACL, MLAC, and LIME ($P = 0.04$) consumed more hay OM ($P < 0.01$) than those offered CONT, whereas heifers

consuming CAPR ($P = 0.07$) and LIUR (P = 0.08) tended to consume more. Monensin did not influence OMI of hay ($P = 0.16$), though consumption was numerically decreased by 0.17 kg/d. No limiter × monensin interaction was detected for hay OMI ($P = 0.49$). MacDonald and Klopfenstein (2004) estimated that hay DMI would decrease 0.78 kg per kg of supplement consumed up to 1.91 kg/d supplement. Morris et al. (2005) estimated forage intake by beef heifers would decrease at a more modest rate of 0.24 or 0.15 kg/d with high quality and low quality forages, respectively, when supplements were offered at rates more comparable to those observed in our study (2.72 kg/d). When supplement intakes from the initial treatment period were included in the linear prediction model of Morris et al. (2005), actual hay consumption was 52% of the amount predicted with all treatment groups having an actual to estimated ratio less than 1. Heifers consuming CONT consumed the most supplement and least amount of hay (34%) relative to the predicted hay DMI of 3.57. Heifers assigned NACL had the highest actual hay consumption relative to predicted amount (60%).

Hay OMI during the last 7 days of the initial period was significantly affected by limiter ($P < 0.01$). Heifers fed LIUR, NACL and MLAC consumed more hay than those offered CONT ($P < 0.05$). Heifers consuming LIME ($P = 0.09$) and CAPR ($P = 0.15$) tended to have increased hay intake. It would appear that decreases in hay intake relative to control were result of forage substitution.

Adding monensin to the supplements resulted in a decrease in daily hay OMI by 15.81% ($P < 0.05$). These reductions are similar to those observed in cows on winter range (19.6% decrease; Lemenager et al., 1978). A tendency for a limiter × monensin

interaction was present for daily hay OMI ($P = 0.11$). Inclusion of monensin in the MLAC treatment reduced daily hay intake by 1.11 kg/d ($P < 0.01$), and tended to reduce hay OMI when heifers were fed UREA ($P = 0.12$), but had minimal influence on hay OMI for other treatments. Figure 2 contains measures of limiter × monensin effect on hay OMI.

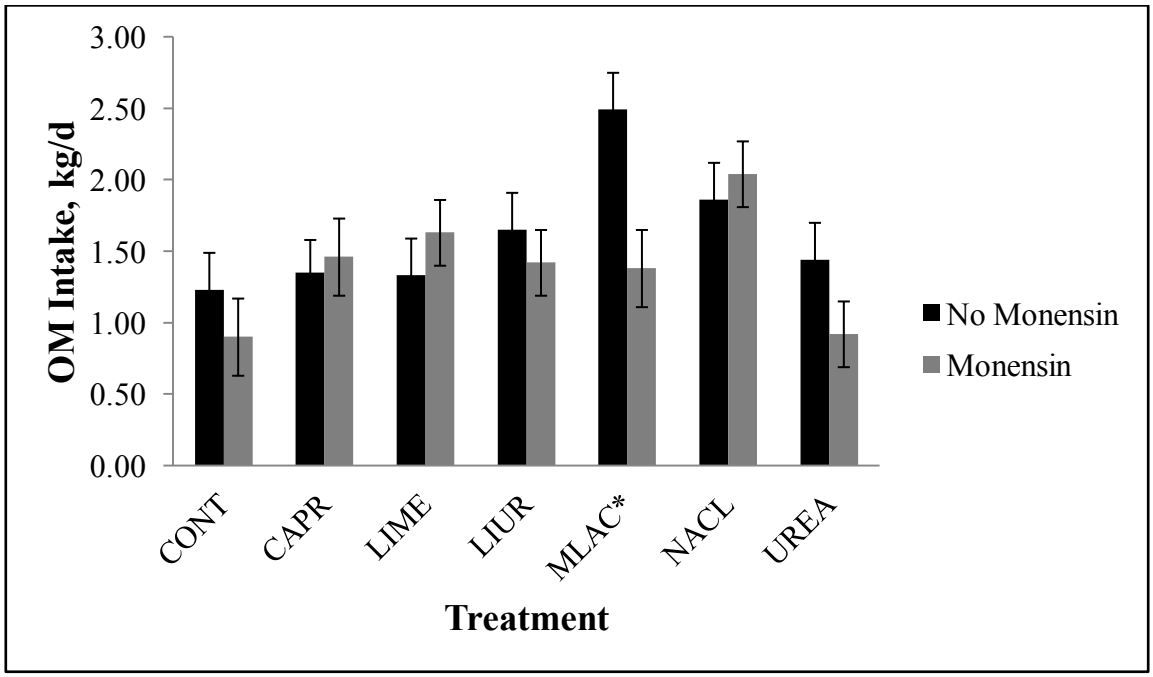

Figure 2. Hay OMI by treatment during the final 7 d of the initial rate period. An "*" denotes significance at P < 0.01.

Predicted hay intake based on the model of Morris et al. (2005) for the last 7 d of the initial supplementation period was slightly less than for the entire period due to increased supplement intake. Observed hay intake during the final 7 days were only a fraction (46%) of modeled values. Supplement DM intakes of CONT, CAPR, and LIME

were similar though hay intake by heifers consuming CONT was 34%lower than predicted values, whereas hay intake by heifers consuming CAPR and LIME were 46% and 45% of predicted values, respectively. By contrast, DDG intake was lowest by heifers offered MLAC and NACL. Forage intake was similar among these treatment groups with actual:estimated ratios of 0.54:1 and 0.55:1. Figure 3 shows supplement and hay DMI during the last 7 d of the period. The consistency by which hay was consumed in much lower amounts by all treatment groups suggests that the reduction is not related at these rates to specific treatment feedback but, instead, likely due in some part to the fact that supplement was consumed at a much higher rate in this study than in others (Morris et al., 2005). Additionally, use of linear prediction models for hay intake that were developed with lower levels of supplement intake than observed in this study may not be appropriate for estimating forage intake in this experiment.

Due to the objective of this study, no treatment group was assigned forage alone; thus, a precise measure of forage replacement by DDG is not available. Lower levels of actual hay consumed relative to modeled predictions may be due to higher supplement intakes. Supplement consumption during the final portion of the initial rate period was 1.97% BW. This level approaches a two-fold increase in the highest DDG level (kg/d) for forage-fed cattle reported in the literature (Klopefenstein et al., 2007). Mean total DMI (hay + supplement) across all treatments during the final 7 d of this period was 3.14% BW. Heifers fed LIME consumed the most total dietary DM per unit of BW (3.38%) whereas those provided MLAC, NACL, and UREA consumed the least (3.02).

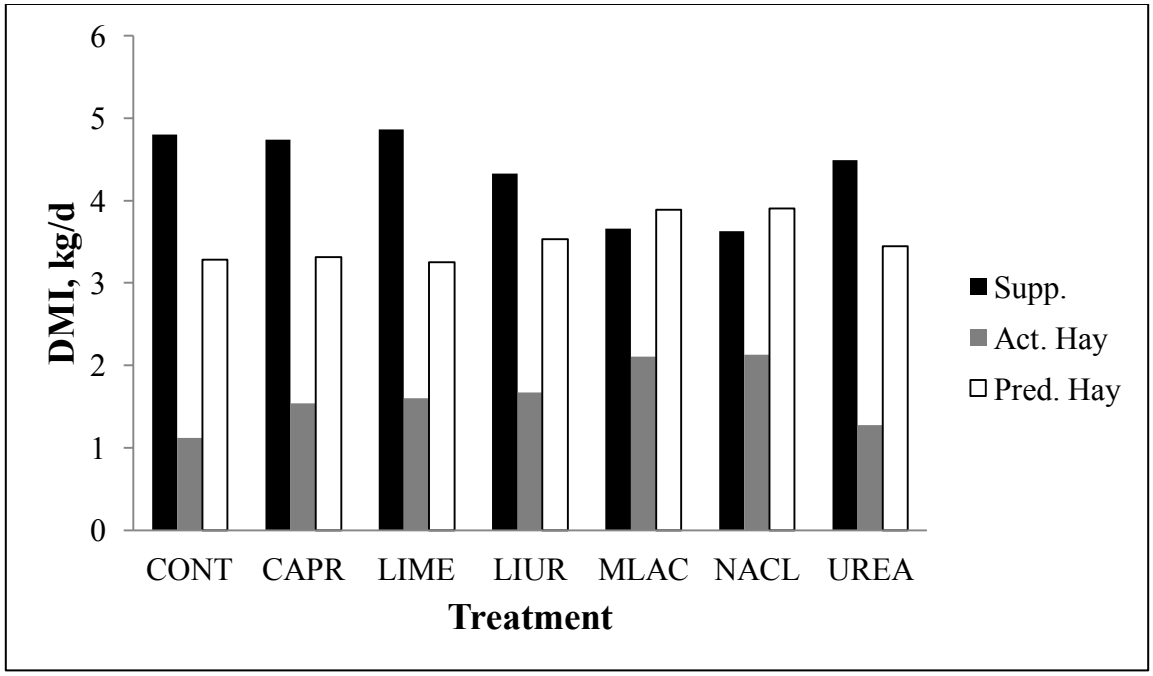

Figure 3. Supplement DMI by treatment during the final 7 d of the initial rate period. Predicted hay estimates as a result of supplement intake are derived from: y = -0.5312x + 12.864 (Morris et al., 2005).

Treatment Period 2: 2X initial rate

Supplement intake. When limiting agents were included at double the initial rate, supplement OM intake was affected by limiter ($P < 0.01$, Table 6.) Daily supplement OM consumption of NACL and MLAC was lower than CONT ($P < 0.01$) whereas OMI of LIUR tended ($P = 0.08$) to be consumed at a reduced rate. No effects on supplement OMI by inclusion of monensin ($P = 0.47$) or the interaction of limiter × monensin ($P = 0.62$) were detected.

As with data in the initial treatment period, the first seven days of the second treatment period were removed and data from the final 7 d of the observation period were analyzed in order to reduce intake variability related to supplement acclimation.

During the final 7 d of the observation period, supplement OM intake was effected by limiter ($P < 0.01$) as intakes of NACL and MLAC were both reduced ($P < 0.01$) relative to CONT with respective means only 45% and 60% of CONT. Heifers fed LIUR consumed supplement OM at levels that tended to be lower ($P = 0.12$) than CONT. Neither monensin ($P = 0.49$) nor a combination of limiter × monensin ($P = 0.27$) produced an effect on DDG intake. Similar supplement OM intakes of LIME ($P < 0.60$) and UREA ($P < 0.81$) relative to CONT suggest that the tendency observed with LIUR resulted from either a combination effect of the agents or was due to their cumulative proportion in the supplement.

Consumption of supplement limited by salt at this level is approximate to the 2.97 kg DM/d reported by Schauer et al. (2004) when formulated inclusion was 16%. Inclusion of malic acid as a percentage of total OMI during this period was 2.63%

Table 6. Period 2 intake of hay and supplement, and supplement intake variance measures of a self-fed dried distillers' grain supplement fed to growing beef heifers

	Treatment[1]										
	CONT	CAPR	LIME	LIUR	MLAC	NACL	UREA	SE	L[2]	M[3]	L × M[4]
	DMI kg/d										
14 d											
Hay Intake	1.82	2.42[a]	1.66	2.18	3.49[a]	3.90[a]	1.71	0.22	0.01	0.01	0.01
Supplement Intake	4.92	4.60	5.20	4.27	2.64[a]	2.20[a]	4.99	0.37	0.01	0.58	0.49
Cumulative stability	0.96	0.75	0.86	0.69	0.88	0.83	0.90	0.11	0.63	0.62	0.18
Temporal stability	0.01	0.07	-0.02	0.05	0.07	0.05	0.04	0.03	0.28	0.48	0.24
last 7 d											
Hay Intake	2.06	2.49	1.97	2.57	4.09[a]	4.50[a]	1.73	0.29	0.01	0.03	0.02
Supplement Intake	4.96	4.82	5.03	4.42	2.88[a]	2.39[a]	5.04	0.36	0.01	0.59	0.20
Cumulative stability	0.81	0.67	0.93	0.53	0.71	0.97	1.01	0.15	0.20	0.36	0.86
Temporal stability	-0.06	0.12[a]	0.12[a]	0.09[b]	0.16[a]	0.16[a]	0.15[a]	0.06	0.13	0.05	0.03
	OMI kg/d										
14 d											
Hay Intake	1.66	2.20[a]	1.52	1.99	3.17[a]	3.54[a]	1.56	0.19	0.01	0.01	0.01
Supplement Intake	4.69	4.21	4.64	3.89[b]	2.59[a]	2.00[a]	4.79	0.33	0.01	0.47	0.62
Cumulative stability	0.88	0.69	0.78	0.62[a]	0.82	0.58	0.83	0.10	0.22	0.51	0.15
Temporal stability	0.01	0.06	-0.02	0.04	0.07	0.04	0.04	0.03	0.30	0.47	0.23
last 7 d											
Hay Intake	1.88	2.26	1.79	2.34	3.71[a]	4.08[a]	1.58	0.26	0.01	0.03	0.02
Supplement Intake	4.72	4.41	4.49	4.02[b]	2.81[a]	2.14[a]	4.83	0.33	0.01	0.49	0.27
Cumulative stability	0.75	0.61	0.84	0.48	0.66	0.68	0.92	0.13	0.22	0.39	0.86
Temporal stability	-0.05	0.11[a]	0.11[a]	0.08[b]	0.15[a]	0.11[a]	0.14[a]	0.05	0.13	0.05	0.02

[1]Treatments were: control (CONT; no limiter); salt (NACL, 20%); urea (UREA, 4%); limestone (LIME, 3.36%); malic acid (MLAC; 6%), calcium propionate (CAPR, 6%), and limestone plus urea (LIUR, 3.36 + 4%)

[2]Limiter effect

[3]Monensin effect

[4]Limiter × monensin

[a]Within a row, means with a superscript differ from CONT at $P < 0.05$

[b]Within a row, means with a superscript differ from CONT at $P < 0.15$

whereas sodium chloride comprised 6.88% of intake. This level of malic acid as a percentage of total diet is similar to that of Foley et al. (2009) who reported a reduction in total intake of 4%. Greater reductions of total feed intake in this study may be the result of separate hay feeding. Hay intakes provided later depict compensation of supplement consumption by increased forage intake. In comparison of supplement OM intake reduction per unit of limiter inclusion, MLAC limited intake 5 percentage units with each increment included whereas sodium chloride reduced intake roughly 2.7 percent for every unit formulated. Therefore, malic acid reduced supplement OMI roughly 1.85 times more effectively than sodium chloride at these levels.

Cumulative stability. Variation in supplement OMI over the course of the second treatment period was not affected by limiter ($P = 0.22$) nor monensin inclusion ($P = 0.51$). A limiter × monensin interaction tended to influence cumulative stability of supplement OMI ($P = 0.15$) as variation associated with MLAC increased by 0.34 kg/d with the addition of monensin ($P = 0.07$; Figure 4). Numerical differences in cumulative stability among treatments indicated that none of the limiting agents applied at their respective rate will improve or disrupt the cumulative stability of DDG fed alone. No monensin effect ($P = 0.85$) or limiter × monensin interaction ($P = 0.68$) was detected.

Variation as a proportion of supplement OMI (CV) was different among limiters ($P < 0.01$) where heifers fed NACL (28%) and MLAC (32%) produced a greater degree of intake variation relative to CONT (19%, $P = 0.01$). No other treatments were significantly or numerically more variable than cattle offered unadulterated DDG.

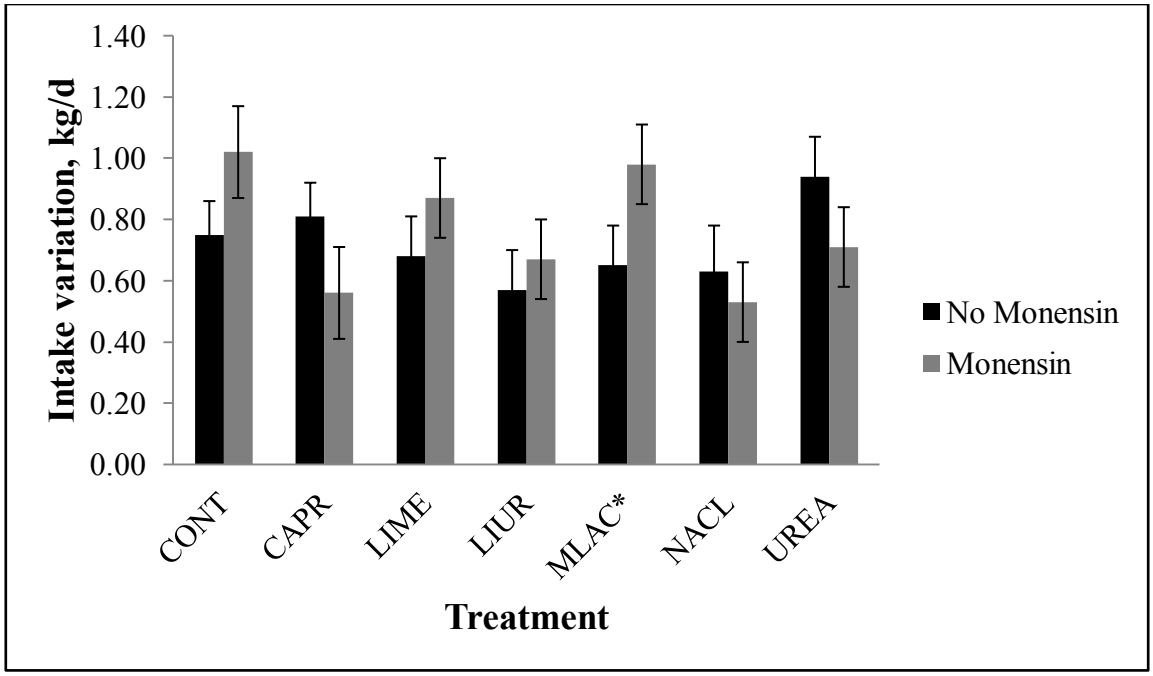

Figure 4. Cumulative stability of OMI by treatment during the intermediate rate period. An "*" denotes significance at $P = 0.07$.

Cumulative stability measures of supplement OM intake during the last 7 days of the treatment period were not different among limiters ($P = 0.22$) and no monensin effect was detected ($P = 0.39$). Additionally, no limiter × monensin interaction was detected ($P = 0.86$). Mean cumulative stability measures of supplement OMI across treatments decreased from the last 7 days of the initial treatment period (0.87 kg) to the last 7 days of the second treatment period (0.71 kg). Only NACL resulted in a noticeably decreased cumulative stability of supplement OMI (0.55 kg to 0.68 kg). All other treatments either approached replication or reduced intake volatility which may suggest that heifers were still adjusting to the supplement. This reasoning is not refuted by the fact measures of temporal stability in the second half of the initial treatment period indicated that supplement OMI was increasing for all treatments.

Coefficient of variation for supplement OMI over the final 7 d of the period was affected by limiter ($P = 0.01$) and tended to be influenced by monensin ($P = 0.13$). Supplement OM intake CV of NACL (31%, $P < 0.01$) and MLAC (27%, $P < 0.04$) were greater than CONT (16%). Supplement intake variation expressed as CV of supplement OMI was reduced numerically from 22% to 18% when monensin was included. Combination of limiter x monensin had no affect ($P = 0.39$) on CV of supplement OMI. Mean CV of supplement OMI among all treatments during the last 7 d of the second period was 2.14 percentage points lower than the last 7 d of the initial rate period indicating that cumulative stability was improving a faster rate than supplement intakes declined. This value was depressed by NACL which was the only treatment not to decrease CV of supplement OMI. Coefficient of variation of NACL OM intake increased from 22% in the initial period to 31% in the intermediate rate period.

Temporal stability. Inclusion of limiting agents at twice the initial rate produced no differences in temporal stability of supplement OMI due to limiter ($P = 0.30$) or monensin ($P = 0.47$) In addition, no effect of a limiter × monensin combination was detected ($P = 0.23$).

Using abridged data from the second treatment period, temporal stability of supplement OMI tended to differ by limiter ($P = 0.13$) with all treatments excluding CONT being consumed at a higher rate each day. All treatments differences from CONT ($P < 0.05$) were significant with the exception of LIUR, which tended to increase ($P = 0.07$) consumption but at a lower rate. Inclusion of monensin in the supplement tended to reduce ($P = 0.06$) temporal variation of supplement OMI from +0.13 kg/d to +0.05

kg/d. A limiter × monensin interaction on supplement OMI was detected ($P = 0.02$) resulting in reduced consumption consistency of CONT ($P = 0.02$) from +0.06 kg OM/d to -0.18 kg OM/d with the addition of monensin whereas temporal variation in heifers offered UREA improved ($P < 0.01$) from +0.29 kg OM/d to 0.00 kg OM/d. Heifers offered LIUR tended to consume supplement OM with more temporal variation when the supplement contained monensin ($P = 0.06$). Figure 5 contains measures of interaction influence on OMI temporal stability.

Results of CONT and NACL responses when combined with monensin are in agreement with those of Muller et al. (1986) though previous literature appears to be vacant of effects on temporal stability due to limestone, urea, or a combination with monensin. Interactions of limiter × monensin in this period suggest that limiting agents may not have been incorporated at high enough rates in the initial treatment period where no response was not detected. That is to say that since rate of monensin inclusion was not different between periods, increasing proportion of limiter was responsible for provoking the combination effect. However, one could oppose certainty of this notion by referencing the conflicting results on monensin effect which produced no difference in temporal stability of supplement intake during the same time frame in the initial period but tended to reduced temporal variation here when formulated inclusion was unchanged. These results are difficult to explain and, although results of monensin inclusion in this period were deemed significant via statistical analysis, the nature of inconsistent results among treatment periods generates doubt in application to improve

temporal intake stability alone or in unison with any limiting agent other than sodium chloride.

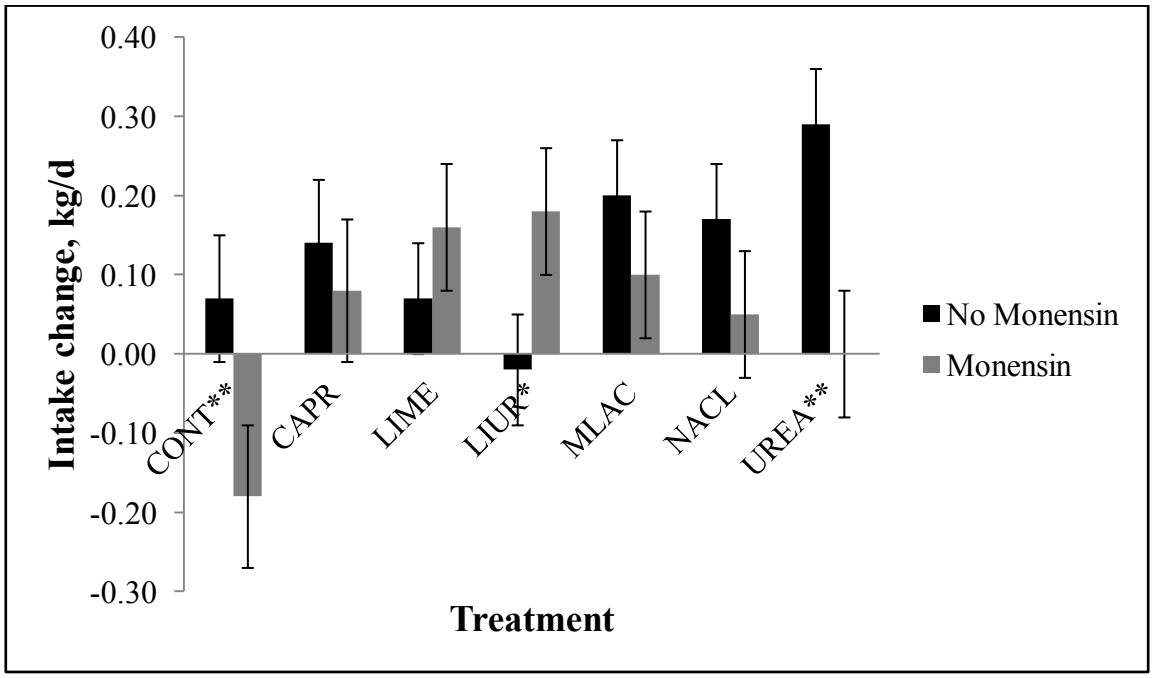

Figure 5. Temporal stability of supplement OMI by treatment during the final 7 d of the intermediate rate period. An "**" denotes significance at $P < 0.05$. An "*" denotes significance at $P < 0.15$.

Hay intake. Analysis of hay OM intakes over the course of the entire second period produced a significant a response to limiter ($P < 0.01$), monensin ($P < 0.01$), and limiter × monensin interaction ($P < 0.01$). Relative to CONT, NACL ($P < 0.01$), MLAC ($P < 0.01$), and CAPR ($P < 0.03$) consumed more hay OM daily. No other treatments differed. Inclusion of monensin decreased daily hay OMI by 0.40 kg. Heifers consuming MLAC consumed less hay OM ($P < 0.01$) when the supplement contained monensin and

were the only treatment group exhibiting a significant response to a limiter × monensin interaction though NACL tended ($P = 0.07$) to have the same effect.

Among treatment groups, mean hay DMI for the overall period was closer to predicted levels of 66% using the linear model of Morris et al. (2005). Consumption of supplement limited by sodium chloride was lowest among treatments and resulted in most accurate hay DMI relative to prediction (86%), whereas heifers fed LIME consumed the most supplement and consumed only 54% of expected forage

Analysis of data after removing the initial 7 d of the second treatment period continued to detect effects by limiter ($P < 0.01$) and monensin ($P = 0.03$) on hay OMI. In addition, an interaction of limiter × monensin also persisted ($P < 0.02$). Heifers assigned supplements containing NACL and MLAC both consumed more hay OM ($P < 0.01$) relative to CONT. Including monensin in the supplement decreased hay OMI by 0.45 kg/d. As a product of limiter × monensin interaction, hay OM intakes by heifers consuming MLAC ($P < 0.01$) and NACL ($P = 0.04$) were reduced by respective measures of 2.15 kg/d and 1.18 kg/d when monensin was included. Figure 7 depicts OMI values relative to the interaction over the final 7 d of the period.

As with observations from the initial treatment period, supplement intakes were generally higher in the last 7 d relative to the entire period. However, hay intakes during the current period increased along with supplement consumption which is trend reversal from the initial rate period. This suggests that although supplement intake increased, limiters may have limited intake to an extent and that heifers relied on forage to reach satiety. Forage intakes during the final 7 d of the second treatment period were 75% of

predicted by the Morris et al. (2005) model. Hay DMI by heifers consuming NACL was higher than predicted (1.06:1) with all others being lower than projected (Figure 8). If the assumption is made that lower hay intake during the initial rate period was attributed to the level of supplement consumption, then results here - where mean supplement intake was higher and actual hay consumption approached predicted levels - may indicate that an increase in heifer growth and gut capacity over the 14 day span was enough to accommodate intakes.

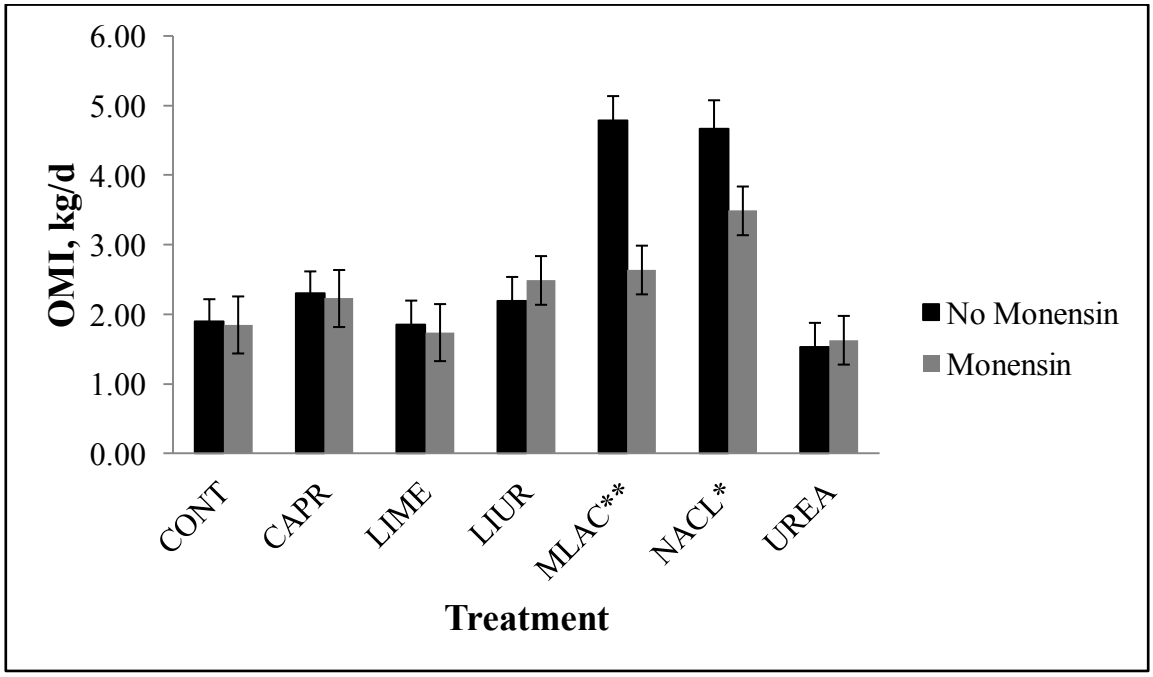

Figure 6. Hay OMI by treatment during the final 7 d of the intermediate rate period. An "**" denotes significance at $P < 0.01$. An "*" denotes significance at $P = 0.04$.

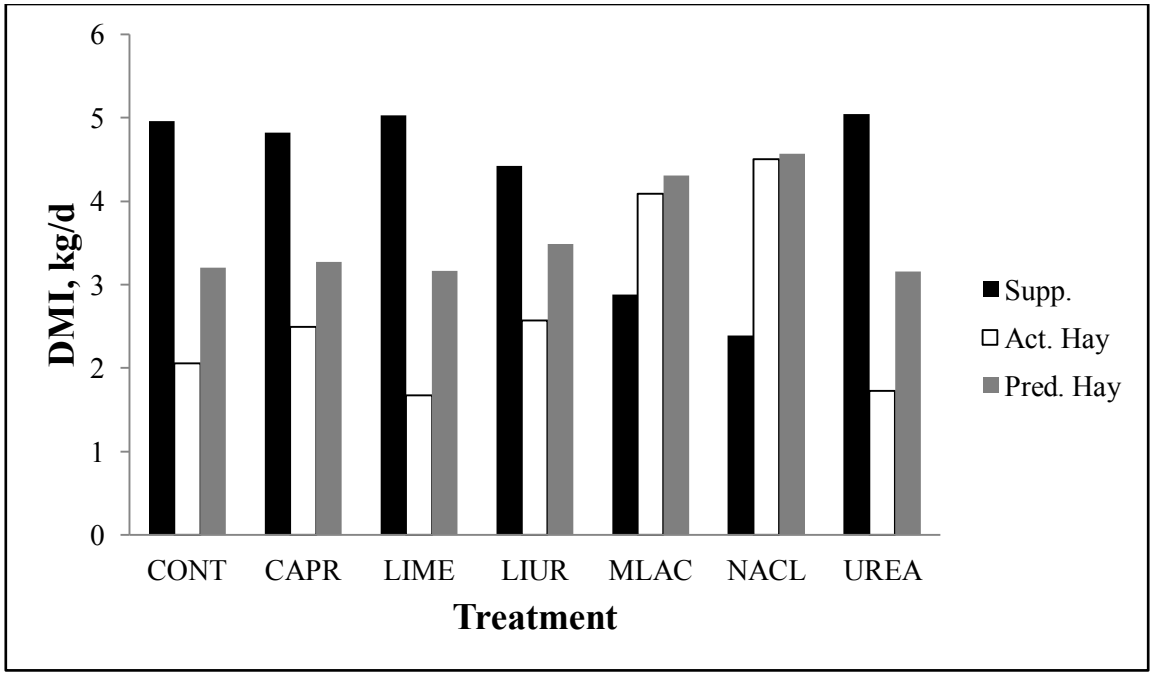

Figure 7. DMI by treatment during the final 7 d of the intermediate rate period. Predicted hay estimates as a result of supplement intake are derived from: y = -0.5312x + 12.864 (Morris et al., 2005)

Treatment Period 3: 4X initial rate

Supplement Intake. When treatments contained the highest level of limiter (4 x initial rate), supplement OMI over the entire period differed among treatment ($P < 0.01$) but was not affected by monensin ($P = 0.41$). Supplement OM intakes of NACL ($P < 0.01$), MLAC ($P < 0.01$), and LIUR ($P = 0.02$) were lower relative to CONT. A limiter × monensin interaction ($P = 0.10$) tended to reduce OM consumption of MLAC by 1.80 kg/d ($P < 0.01$) when monensin was included. Table 7 provides intakes and intake variation measures for the final treatment period.

Over the final 7 d of treatment period 3, intake of supplement OM continued to be affected by limiter ($P < 0.01$) and consumption of NACL, MLAC, and LIUR

Table 7. Period 3 intake of hay and supplement, and supplement intake variance measures of a self-fed dried distillers' grain supplement fed to growing beef heifers

	Treatment[1]										
	CONT	CAPR	LIME	LIUR	MLAC	NACL	UREA	SE	L[2]	M[3]	L × M[4]
DMI											
14 d											
Hay Intake	2.12	2.82[a]	2.20	2.88[a]	3.44[a]	3.97[a]	1.67	0.20	0.01	0.01	0.01
Supplement Intake	4.74	5.72[b]	4.98	4.01	2.69[a]	0.96[a]	5.02	0.40	0.01	0.21	0.38
Cumulative stability	0.64	1.98[a]	0.59	0.65	0.73	0.34[b]	0.71	0.12	0.01	0.26	0.19
Temporal stability	0.01	-0.37[a]	-0.01	-0.07[b]	-0.09[a]	-0.03	-0.02	0.03	0.01	0.25	0.36
last 7 d											
Hay Intake	2.09	2.73[a]	2.48	2.91[a]	3.61[a]	3.93[a]	1.73	0.31	0.01	0.27	0.06
Supplement Intake	4.87	4.57	5.00	3.74[b]	2.33[a]	0.86[a]	5.03	0.44	0.01	0.17	0.39
Cumulative stability	0.51	0.57	0.49	0.48	0.48	0.27	0.69	0.11	0.27	0.98	0.10
Temporal stability	-0.07	-0.02	-0.01	-0.02	-0.14	-0.01	-0.15	0.07	0.51	0.72	0.51
OMI											
14 d											
Hay Intake	1.93	2.56[a]	2.01	2.61[a]	3.13[a]	3.62[a]	1.52	0.17	0.01	0.01	0.01
Supplement Intake	4.52	5.04	4.45	3.41[a]	2.63[a]	0.77[a]	4.75	0.35	0.01	0.41	0.10
Cumulative stability	0.59	1.73[a]	0.51	0.57	0.68	0.16[a]	0.66	0.10	0.01	0.28	0.17
Temporal stability	0.01	-0.33	-0.01	-0.07	-0.09	-0.01	-0.02	0.03	0.01	0.25	0.36
last 7 d											
Hay Intake	1.90	2.48[a]	2.26	2.65[a]	3.28[a]	3.59[a]	1.58	0.28	0.01	0.26	0.06
Supplement Intake	4.64	4.03	4.47	3.16[a]	2.30[a]	0.73[a]	4.76	0.40	0.01	0.32	0.14
Cumulative stability	0.47	0.50	0.42	0.42	0.45	0.13[a]	0.64	0.10	0.04	0.96	0.09
Temporal stability	-0.07	-0.02	-0.01	-0.02	-0.13	-0.01	-0.14	0.06	0.48	0.66	0.54

[1]Treatments were: control (CONT; no limiter); salt (NACL, 40%); urea (UREA, 8%); limestone (LIME, 6.72%); malic acid (MLAC; 12%), calcium propionate (CAPR, 12%), and limestone plus urea (LIUR, 6.72 + 8%)
[2]Limiter effect
[3]Monensin effect
[4]Limiter × monensin
[a]Within a row, means with a superscript differ from CONT at $P < 0.05$
[b]Within a row, means with a superscript differ from CONT at $P < 0.15$

continued to be consumed at a lower rate ($P < 0.01$) than CONT. No significant response to monensin was detected on supplement OMI ($P = 0.32$). A tendency for a limiter × monensin effect on supplement OMI ($P = 0.14$, Figure 9) persisted as consumption of MLAC decreased ($P = 0.02$) by 1.84 kg OM/d and LIUR ($P = 0.14$) tended to increase by 1.11 kg OM/d when monensin was incorporated in the supplement.

Further intake reductions of supplement limited by sodium chloride, malic acid, and a combination of limestone and urea enhance confidence in their efficacy to restrict intake of DDG. A lack of response to monensin on supplement intake during this treatment period and throughout is surprising considering previous findings (Muller et al., 1986). Results of a limiter × monensin interaction in this set of observations are not supported by data in the previous treatment periods and are either by chance or a response to respective limiting agents at specific inclusion rates.

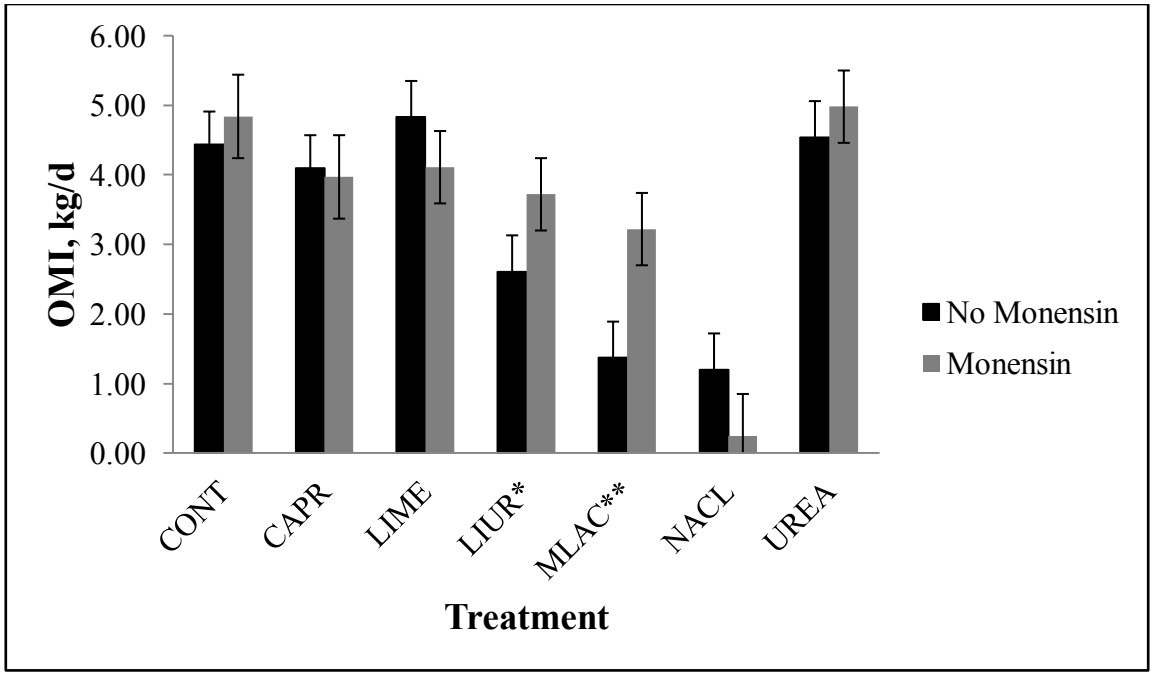

Figure 8. Supplement OMI by treatment during the final 7 d of the final rate period. An "**" denotes significance at $P = 0.02$. An "*" denotes significance at $P = 0.14$.

Cumulative stability. Supplement OMI variability differed among treatments ($P < 0.01$) over the duration of the final period. Cumulative stability of CAPR was reduced ($P < 0.01$) relative to CONT whereas variation associated with NACL organic matter intake decreased ($P < 0.01$). Monensin ($P = 0.28$) did not affect variation of supplement OMI and no limiter × monensin interaction was detected ($P = 0.17$). Variation of OMI expressed as CV was affected by limiter ($P < 0.01$). Supplements containing CAPR (36%), MLAC (30%), and NACL (33%) had CVs higher ($P < 0.01$) than CONT (14%). No monensin ($P = 0.82$) effect or limiter × monensin interaction was detected ($P = 0.74$).

Analysis of data after removal of supplement adaptation days continued to show a treatment effect ($P = 0.05$) on cumulative stability of supplement OMI as variation of

NACL intake was reduced ($P = 0.02$). No other treatments differed from the level of variation of CONT. Inclusion of monensin had no effect on cumulative stability of supplement OMI ($P = 0.96$) though a tendency for a limiter × monensin interaction ($P = 0.09$) resulted in a decreased variation of 0.47 kg OM/d ($P = 0.02$) by heifers offered UREA. In addition, adding monensin to CAPR tended to increase ($P = 0.15$) daily supplement OMI variation (Figure 10). Coefficient of variation of supplement OMI was different ($P < 0.01$) among limiters as both MLAC (26%) and NACL (30%) were higher than CONT (10%, $P < 0.01$). Monensin had minimal influence on the CV of supplement intake ($P = 0.16$) though a limiter × monensin combination effect ($P < 0.01$) increased supplement OMI variation of NACL from 11% to 49% when monensin was added ($P < 0.01$). A tendency for a reversed response due to monensin was observed with heifers offered UREA ($P = 0.14$) and LIUR ($P = 0.07$) as respective CV values were reduced by 10% and 13% when supplement included monensin.

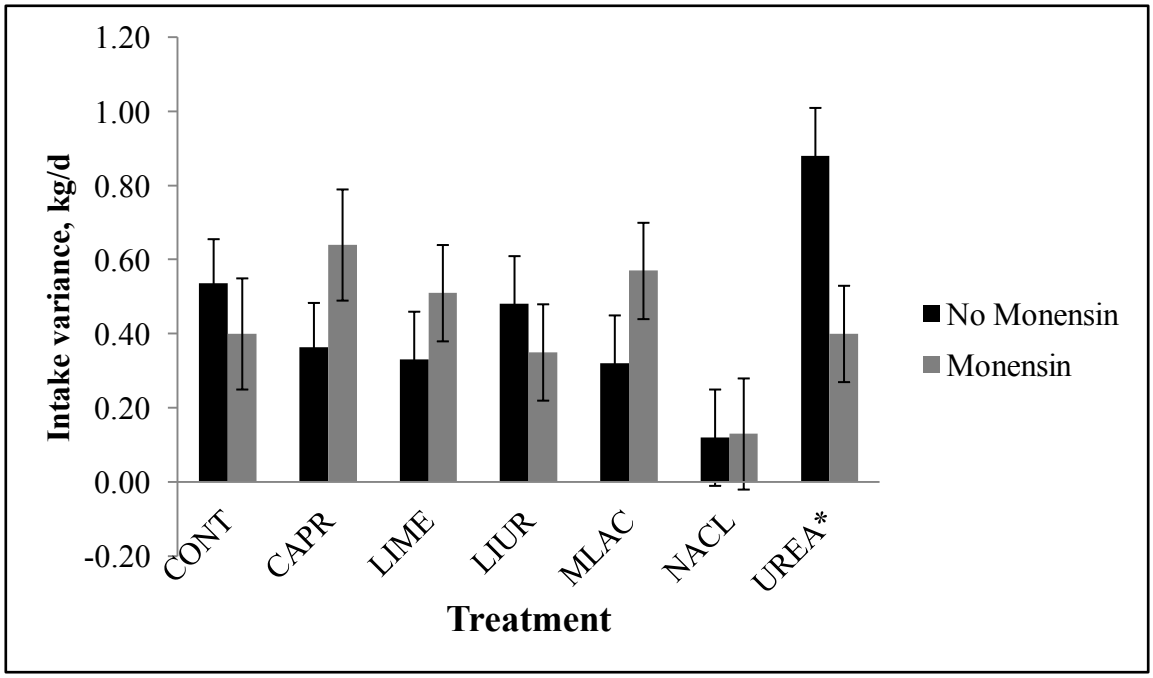

Figure 9. Cumulative stability of supplement OMI by treatment during the final 7 d of the final treatment period. An "*" denotes significance at $P = 0.02$.

Temporal stability. Daily change in rate of supplement OM intake change over the entire 14 d of treatment period 3 differed by limiter ($P < 0.01$) as MLAC ($P = 0.02$) and CAPR ($P < 0.01$) consumption decreased daily relative to CONT which was the only treatment to be consumed at an increasing rate. Daily supplement OMI of LIUR tended to decrease ($P = 0.06$). No monensin affect ($P = 0.25$) or limiter × monensin interaction ($P = 0.36$) were detected.

During the last 7 d of the period, temporal stability of daily supplement OM intake was not affected by limiter ($P = 0.48$) or monensin ($P = 0.66$). Additionally, no limiter × monensin interaction was present ($P = 0.54$). Temporal stability measures, both over the duration of treatment period 3 and during the last 7 d alone, indicate that

supplement consumption ceased to increase. Heifers offered CONT had daily supplement intake changes greater zero (0.01 kg) during the entire period but consumed less supplement (-0.01 kg/d) when the last 7 days were analyzed. Therefore, a couple of conclusions can be drawn. First, heifers no longer had intakes that were influenced by acclimation of DDG. In fact, consumption of CONT during the last 7 d of treatment period 3 was 0.09 kg less than consumption of CONT during the last 7 d of the previous period. Also, declining intakes would suggest that the window of limiter of inclusion rates of all respective limiters used here would likely accommodate accurate intake level prediction by regression procedures.

Hay intake. During the final treatment period, OM hay consumption was affected by limiter ($P < 0.01$) and monensin ($P < 0.01$). Forage consumption by heifers fed NACL , MLAC , CAPR , and LIUR increased ($P < 0.01$) relative to CONT whereas heifers assigned UREA tended to have decreased hay OM intake ($P = 0.09$) relative to CONT. When monensin was added to DDG, hay OMI decreased 0.35 kg/d. These reductions are similar to the decreases in forage intake during treatment period 2. Mean supplement intake during the second treatment period was 4.12 kg and mean intakes during this period are 4.01 kg. Therefore, monensin consumption would be essentially equal and confirms similarities in response across rates. A limiter × monensin interaction on hay OMI ($P < 0.01$) was also present in the final period where inclusion of monensin in MLAC resulted in a reduction of 1.71 kg. Including monensin in LIUR tended also to reduce hay OMI by 0.61 kg/d ($P < 0.06$). Across treatments, intakes were 74% of predictions using linear regressions (Morris et al., 1995). Heifers offered supplement

limited by salt consumed the least amount of DDG and consumed the most hay, though intake was still 1.63 kg less than predicted. Heifers receiving CAPR consumed the most supplement and were the only treatment group to match actual (2.82 kg) and predicted (2.80 kg) hay intakes.

Hay OM intakes during the last 7 d of the final period continued to be affected by treatment ($P < 0.01$) but not monensin ($P = 0.26$). Consumption of CONT was lower ($P < 0.01$) than both NACL and MLAC. Heifers assigned LIME had higher OM hay intakes ($P = 0.04$) whereas CAPR ($P = 0.07$) and LIUR ($P = 0.07$) tended to consume more hay relative to CONT. A slight combination effect of limiter × monensin was observed on hay OMI ($P = 0.06$) as heifers offered MLAC consumed less hay ($P < 0.01$) when supplement contained monensin. Intake reductions due to MLAC × monensin were 1.94 kg OM/d (Figure 11).

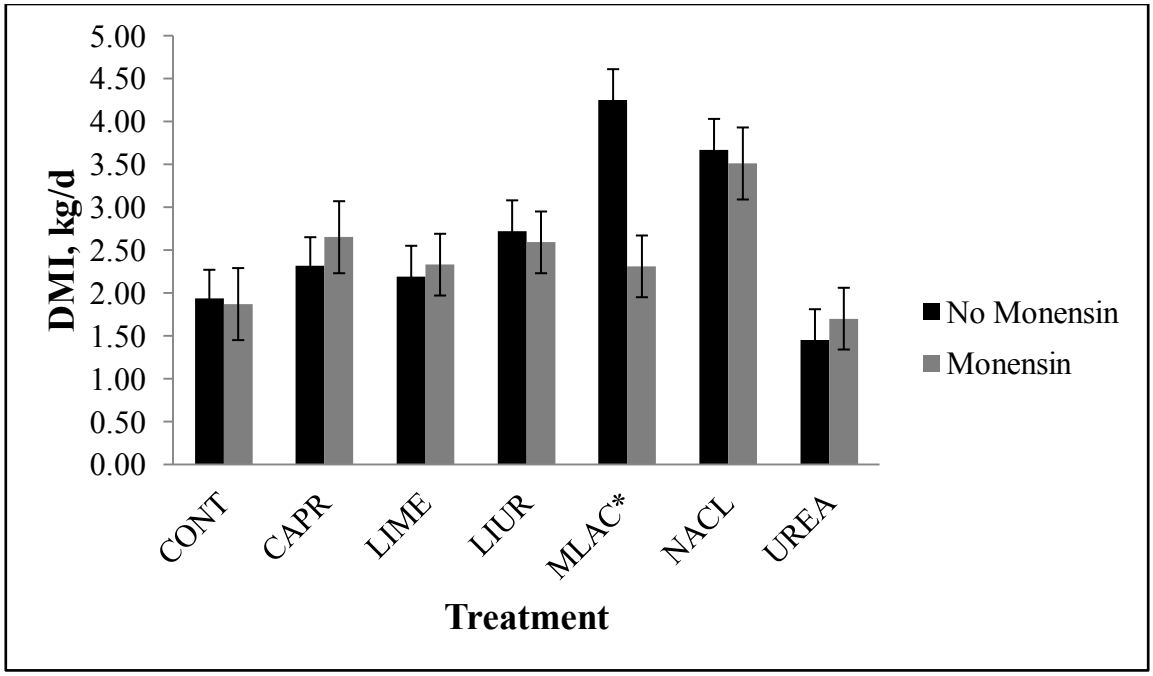

Figure 10. Temporal stability of hay OMI by treatment during the final 7 d of the final treatment period. An "*" denotes significance at $P = 0.01$.

Across rate analysis

In order to evaluate limiter inclusion rate as a predictor of response variables in a DDG supplement, results within limiter treatment were regressed across periods (rates of inclusion) and subjected to statistical analysis to determine whether limiting agents alone or in combination with monensin projected differently than DDG alone. Comparisons were made using CONT as an independent treatment to ensure effects on response variables were a result of limiter and rate. In addition, data was evaluated with CONT serving as an index value by which all other treatments were compared.

Supplement intake. Intercepts of supplement OM intake were different than zero for all treatments ($P < 0.01$). When regressed across rate, results indicated that

Table 8. Regression coefficients[1] of OMI values over entire 14 d periods.

Limiter[2]	β_0			β_1			β_2		
CAPR	3.023*	±	0.3711	0.8621*	±	0.3892	-0.0842	±	0.1033
CAPR + M	2.246*	±	0.4791	1.171*	±	0.5025	-0.124	±	0.1333
CONT	2.794*	±	0.3711	1.435*	±	0.3892	-0.258*	±	0.1033
CONT + M	3.207*	±	0.4791	1.24*	±	0.5025	-0.2216	±	0.1333
LIME	2.331*	±	0.4149	1.833*	±	0.4352	-0.3082*	±	0.1154
LIME + M	1.965*	±	0.4149	2.026*	±	0.4352	-0.3685*	±	0.1154
LIUR	2.383*	±	0.4149	1.29*	±	0.4352	-0.2759*	±	0.1154
LIUR + M	2.629*	±	0.4149	0.999*	±	0.4352	-0.1832	±	0.1154
MLAC	3.443*	±	0.4149	-0.6529	±	0.4352	0.052	±	0.1154
MLAC + M	2.904*	±	0.4149	0.3256	±	0.4352	-0.0435	±	0.1154
NACL	3.154*	±	0.4149	-0.5937	±	0.4352	0.0299	±	0.1154
NACL + M	3.12*	±	0.4149	-0.9662*	±	0.4352	0.2035	±	0.1154
UREA	2.244*	±	0.4149	1.886*	±	0.4352	-0.3343*	±	0.1154
UREA + M	3.325*	±	0.4149	1.038*	±	0.4352	-0.1498	±	0.1154

*Coefficients differ from zero, $P < 0.05$

[1]β_0 = intercept, β_1 = rate, β_2 = rate*rate

[2]Treatments were: control (CONT; no limiter); salt (NACL); urea (UREA); calcium carbonate (LIME); malic acid (MLAC), calcium propionate (CAPR), and limestone plus urea (LIUR), plus monensin (+ M)

supplement OMI of NACL (β_1, $P = 0.01$; β_2, $P = 0.03$) and MLAC (β_1, $P = 0.02$; β_2, $P = 0.06$) could be predicted by rate of inclusion. No other treatment had rate of rate^2 coefficients that differed from zero. Table 8 contains regression parameters of OM supplement intake over the full 14 day periods.

When initial days in each period were removed to suppress variation at least partially due to novelty, intercepts of supplement OMI were significant for all treatments ($P < 0.01$) excluding CONT+M ($P = 0.03$). Table 9 contains results of regression analysis of supplement intake during terminal portions of each period. Among all treatments, only supplement intake of NACL and MLAC could be projected by rate of inclusion. With each bifold increase in rate of limiter, supplement was reduced by NACL ($P = 0.01$) and MLAC ($P = 0.02$). When squared rate of limiter was incorporated into the model, NACL ($P = 0.03$) and MLAC ($P = 0.06$) were again the only treatments exhibiting a significant response. In each case, response to squared inclusion rate tempered the decrease in OMI associated with inclusion rate increases. Collectively, these figures demonstrate that the most drastic response to NACL and MLAC in a DDG supplement would be at the initial inclusion with intake reduction becoming less severe as supplement contained larger proportions of limiter. This is intuitive assuming that a limiter will negatively influence intake to a point that consumption levels approach zero and reductions associated with subsequent increases in limiter are increasingly minute.

Intake by index. Due to the confounded structure of the trial, supplement intake, cumulative stability, and temporal stability measures were divided by the mean CONT response of each respective variable to provide a refined indication of treatment affect

Table 9. Regression coefficients[1] of OMI values over last 7 d of periods.

Limiter[2]	β_0			β_1			β_2		
CAPR	4.157*	±	0.868	-0.3241	±	0.8629	0.0258	±	0.1523
CAPR + M	3.088*	±	1.121	0.6294	±	1.068	-0.1216	±	0.1966
CONT	5.69*	±	0.868	-0.2589	±	0.8269	0.0296	±	0.1523
CONT + M	2.406*	±	1.121	1.859	±	1.068	-0.2657	±	0.1966
LIME	4.989*	±	1.121	-0.125	±	1.068	-0.0052	±	0.1966
LIME + M	4.454*	±	0.9705	-0.5665	±	0.9245	0.0804	±	0.1703
LIUR	4.277*	±	0.9705	-0.3755	±	0.9245	-0.0017	±	0.1703
LIUR + M	4.627*	±	0.9705	0.1643	±	0.9245	-0.0568	±	0.1703
MLAC	5.421*	±	1.121	-2.571*	±	1.068	0.3803	±	0.1966
MLAC + M	4.216*	±	0.9705	-0.2303	±	0.9245	-0.0148	±	0.1703
NACL	6.201*	±	0.9705	-2.426*	±	0.9245	0.3836*	±	0.1703
NACL + M	5.385*	±	1.121	-0.8165	±	1.068	0.0006	±	0.1966
UREA	3.856*	±	0.9705	0.8103	±	0.9245	-0.2206	±	0.1703
UREA + M	3.232*	±	0.9705	0.8274	±	0.9245	-0.1699	±	0.1703

*Coefficients differ from zero, $P < 0.05$

[1]β_0 = intercept, β_1 = rate, β_2 = rate*rate

[2]Treatments were: control (CONT; no limiter); salt (NACL); urea (UREA); calcium carbonate (LIME); malic acid (MLAC), calcium propionate (CAPR), and calcium carbonate plus urea (LIUR), plus monensin (+ M)

within each period. As with the non-indexed data, within period observations were regressed on rate and rate^2 to evaluate inclusion rate as a predictor of response variables.

As expected, OM intake intercepts resulting from regressed 14-d indexed values were differed from zero for all treatments ($P < 0.01$). Among all treatments, reliable OMI prediction equations based on rate of limiter were produced for only LIUR+M (β_1, $P = 0.04$; β_2, $P = 0.08$), MLAC (β_1 and β_2, $P < 0.01$), NACL (β_1, $P < 0.01$, β_2, $P = 0.02$), and NACL+M (β_1, $P < 0.01$; β_2, $P = 0.08$). Table 10 contains results of indexed supplement OMI regressions over entire observation periods.

Indexed OMI regressions inclusive of only the terminal seven days in each period again resulted in an intercept of 1.00 for CONT. Results of regressing the abridged data indicated that only supplement intake of UREA (DM; β_1, $P = 0.03$; β_2, $P = 0.04$) could be predicted by rate of limiter inclusion (Table 11).

Table 10. Regression coefficients[1] of indexed OMI values over entire 14 d periods.

Limiter	β_0			β_1			β_2		
CAPR	0.9271*	±	0.0776	-01074	±	0.0932	0.0342	±	0.0211
CAPR + M	0.996*	±	0.1687	-0.0347	±	0.2142	0.0245	±	0.0497
CONT	1.000*	±	0.0938	0.000	±	0.1192	0.000	±	0.0278
CONT + M	1.157*	±	0.0822	-0.0894	±	0.1044	0.0156	±	0.0242
LIME	0.8794*	±	0.0823	0.116	±	0.0988	-0.017	±	0.0223
LIME + M	0.8186*	±	0.101	0.134	±	0.1212	-0.0255	±	0.0274
LIUR	0.8685*	±	0.1201	0.0078	±	0.1525	-0.0119	±	0.0354
LIUR + M	1.138*	±	0.0801	-0.2527*	±	0.0962	0.0448	±	0.0217
MLAC	1.183*	±	0.1037	-0.503*	±	0.1316	0.0864*	±	0.0305
MLAC + M	1.038*	±	0.1505	-0.2451	±	0.1911	0.0462	±	0.0446
NACL	1.12*	±	0.0569	-0.4909*	±	0.0762	0.0709*	±	0.0177
NACL + M	1.099*	±	0.1052	-0.4718*	±	0.1343	0.0541	±	0.0318
UREA	0.8212*	±	0.0864	0.1596	±	0.1096	-0.0284	±	0.0254
UREA + M	1.231*	±	0.1312	-0.0398	±	0.1666	0.0032	±	0.0386

*Coefficients differ from zero, $P < 0.05$

[1]β_0 = intercept, β_1 = rate, β_2 = rate*rate

[2]Treatments were: control (CONT; no limiter); salt (NACL); urea (UREA); limestone (LIME); malic acid (MLAC), calcium propionate (CAPR), and limestone plus urea (LIUR), plus monensin (+ M)

Table 11. Regression coefficients[1] of indexed OMI values over entire last 7 d of periods.

Limiter	β_0			β_1			β_2		
CAPR	0.7656*	±	0.3216	0.1214	±	0.3111	-0.0299	±	0.0599
CAPR + M	0.9311	±	0.5635	0.0718	±	0.545	-0.0104	±	0.1049
CONT	1.000*	±	0.2272	0.000	±	0.2198	0.000	±	0.0423
CONT + M	0.6004*	±	0.2032	0.3258	±	0.1966	-0.0508	±	0.0378
LIME	0.9818*	±	0.238	-0.008	±	0.2302	0.0087	±	0.0443
LIME + M	0.762*	±	0.2589	0.1429	±	0.2504	-0.0254	±	0.0482
LIUR	0.6102	±	0.3668	0.2967	±	0.3548	-0.0756	±	0.0683
LIUR + M	0.7803*	±	0.2128	0.0485	±	0.2058	-0.0085	±	0.0396
MLAC	1.125*	±	0.4257	-0.5028	±	0.4118	0.0869	±	0.0792
MLAC + M	0.6606	±	0.7183	0.1182	±	0.6702	-0.0255	±	0.1272
NACL	1.08*	±	0.2246	-0.434	±	0.2248	0.058	±	0.0436
NACL + M	0.6612	±	0.3342	-0.0273	±	0.324	-0.031	±	0.0629
UREA	0.3269	±	0.2251	0.5777*	±	0.2178	-0.1008*	±	0.0491
UREA + M	0.8377*	±	0.3465	0.248	±	0.3352	-0.0475	±	0.0645

*Coefficients differ from zero, $P < 0.05$

[1]β_0 = intercept, β_1 = rate, β_2 = rate*rate

[2]Treatments were: control (CONT; no limiter); salt (NACL); urea (UREA); calcium carbonate (LIME); malic acid (MLAC), calcium propionate (CAPR), and calcium carbonate plus urea (LIUR), plus monensin (+ M)

Cumulative stability. Regressed measures of OM intake cumulative stability over 14 d durations indicated the presence of variation among all treatments. However, only NACL (β_1, $P = 0.30$; β_2, $P = 0.03$) and CAPR (β_1, $P = 0.13$; β_2, $P < 0.01$) yielded equations that contained a coefficient deviating from zero and thus could be predicted by rate (Table 12).

Removing the initial 7 d of each period resulted in no treatment with statistically significant parameter estimates indicating that cumulative stability could not be estimated by rate of limiter (Table 13).

Cumulative stability by index. When cumulative stability values of the full periods were indexed against the mean of the CONT and regressed on rate and rate squared, β_2 coefficients of CAPR ($P < 0.01$) and CAPR + M ($P < 0.01$) were substantially greater than CONT (Table 14).

When the initial seven days of each period were removed, regressions identified LIUR as the sole treatment with predictable measures of supplement OMI cumulative stability as a function of limiter inclusion rate (Table 15). With each rate increase, supplement OM intake cumulative stability would be expected to decrease ($P = 0.04$) and do so at a decreasing rate ($P = 0.05$).

Table 12. Regression coefficients[1] OMI cumulative stability values over entire 14 d periods.

Limiter	β_0			β_1			β_2		
CAPR	1.17*	±	0.2623	-0.3729	±	0.2426	0.1379*	±	0.047
CAPR + M	0.7427*	±	0.3386	-0.1105	±	0.3131	0.0752	±	0.0607
CONT	0.9471*	±	0.2623	0.0889	±	0.2426	-0.0444	±	0.047
CONT + M	1.142*	±	0.3386	-0.1447	±	0.3131	0.0013	±	0.0607
LIME	1.38*	±	0.2932	-0.3363	±	0.2712	0.0216	±	0.0525
LIME + M	0.9695*	±	0.2932	0.1525	±	0.2712	-0.061	±	0.0525
LIUR	1.04*	±	0.2932	-0.0638	±	0.2712	-0.0089	±	0.0525
LIUR + M	1.13*	±	0.2932	-0.234	±	0.2712	0.0152	±	0.0525
MLAC	1.038*	±	0.2932	-0.2606	±	0.2712	0.0347	±	0.0525
MLAC + M	1.034*	±	0.2932	0.0457	±	0.2712	-0.026	±	0.0525
NACL	0.8723*	±	0.2932	0.2846	±	0.2712	-0.1154*	±	0.0525
NACL + M	0.7862*	±	0.2932	0.0657	±	0.2712	-0.0563	±	0.0525
UREA	0.6614*	±	0.2932	0.2192	±	0.2712	-0.0453	±	0.0525
UREA + M	0.8765*	±	0.2932	0.0199	±	0.2712	-0.029	±	0.0525

*Coefficients differ from zero, $P < 0.05$

[1]β_0 = intercept, β_1 = rate, β_2 = rate*rate

[2]Treatments were: control (CONT; no limiter); salt (NACL); urea (UREA); calcium carbonate (LIME); malic acid (MLAC), calcium propionate (CAPR), and limestone plus urea (LIUR), plus monensin (+ M)

Table 13. Regression coefficients[1] OMI cumulative stability values over last 7 d of periods.

Limiter	β_0			β_1			β_2		
CAPR	0.677	±	0.4859	0.0549	±	0.4703	-0.0362	±	0.0866
CAPR + M	0.9748	±	0.6273	-0.3159	±	0.6071	0.068	±	0.118
CONT	1.277*	±	0.4859	-0.4212	±	0.4703	0.052	±	0.0866
CONT + M	1.761*	±	0.6273	-0.8707	±	0.6071	0.1336	±	0.1118
LIME	1.556*	±	0.6273	-0.4974	±	0.6071	0.0507	±	0.1118
LIME + M	0.994	±	0.5432	0.0794	±	0.5258	-0.0522	±	0.0969
LIUR	1.598*	±	0.5432	-0.6	±	0.5258	0.0712	±	0.0969
LIUR + M	1.635*	±	0.5432	-0.6028	±	0.5258	0.0751	±	0.0969
MLAC	0.6348	±	0.6273	-0.0279	±	0.6071	-0.0129	±	0.1118
MLAC + M	1.068	±	0.5432	-0.1286	±	0.5258	-0.0066	±	0.0969
NACL	0.3781	±	0.5432	0.408	±	0.5258	-0.1038	±	0.0969
NACL + M	0.3072	±	0.6273	0.6073	±	0.6071	-0.1526	±	0.1118
UREA	0.8769	±	0.5432	0.1253	±	0.5258	-0.0409	±	0.0969
UREA + M	1.253*	±	0.5432	-0.505	±	0.5258	0.0808	±	0.0969

*Coefficients differ from zero, $P < 0.05$

[1]β_0 = intercept, β_1 = rate, β_2 = rate*rate

[2]Treatments were: control (CONT; no limiter); salt (NACL); urea (UREA); calcium carbonate (LIME); malic acid (MLAC), calcium propionate (CAPR), and calcium carbonate plus urea (LIUR), plus monensin (+ M)

Table 14. Regression coefficients[1] of indexed OMI cumulative stability over 14 d periods.

Limiter	β_0			β_1			β_2		
CAPR	0.9282*	±	0.2706	-0.43	±	0.325	0.2523*	±	0.0734
CAPR + M	1.061*	±	0.209	-0.4681	±	0.2653	0.202*	±	0.0615
CONT	1.000*	±	0.2153	0.000	±	0.2735	0.000	±	0.0637
CONT + M	1.254*	±	0.2982	-0.195	±	0.3786	0.0299	±	0.0878
LIME	0.9874*	±	0.098	-0.0244	±	0.1176	-0.0155	±	0.0266
LIME + M	0.8114*	±	0.2335	0.2546	±	0.2804	-0.051	±	0.0634
LIUR	1.09*	±	0.2562	-0.2425	±	0.3252	0.0609	±	0.0754
LIUR + M	0.9062*	±	0.1381	-0.055	±	0.1658	0.0026	±	0.0375
MLAC	1.214*	±	0.1925	-0.4454	±	0.2443	0.0925	±	0.0567
MLAC + M	1.116*	±	0.2226	-0.0145	±	0.2828	0.0161	±	0.066
NACL	0.924*	±	0.2548	0.0857	±	0.3415	-0.0597	±	0.0792
NACL + M	0.8181*	±	0.219	0.0179	±	0.2794	-0.0431	±	0.0662
UREA	0.6895*	±	0.2228	0.1998	±	0.2828	-0.01	±	0.0656
UREA + M	0.7294*	±	0.1744	0.1533	±	0.2214	-0.0344	±	0.0513

*Coefficients differ from zero, $P < 0.05$

[1]β_0 = intercept, β_1 = rate, β_2 = rate*rate

[2]Treatments were: control (CONT; no limiter); salt (NACL); urea (UREA); calcium carbonate (LIME); malic acid (MLAC), calcium propionate (CAPR), and calcium carbonate plus urea (LIUR), plus monensin (+ M)

Table 15. Regression coefficients[1] of indexed OMI cumulative stability over last 7 d of periods.

Limiter	β_0			β_1			β_2		
CAPR	0.3119	±	0.5701	0.3569	±	0.5515	-0.0658	±	0.1061
CAPR + M	0.4074	±	0.6298	0.2374	±	0.6092	-0.0185	±	0.1172
CONT	1.000	±	0.564	0.000	±	0.5456	0.000	±	0.105
CONT + M	1.193	±	0.7864	-0.2119	±	0.7607	0.025	±	0.1464
LIME	1.244*	±	0.53	-0.1268	±	0.5127	-0.007	±	0.0987
LIME + M	1.066	±	0.8254	0.102	±	0.7984	-0.0325	±	0.1536
LIUR	2.486*	±	0.6745	-1.556*	±	0.6524	0.29	±	0.1256
LIUR + M	1.447*	±	0.448	-0.5811	±	0.4333	0.0961	±	0.0834
MLAC	0.7983	±	0.5334	0.0722	±	0.5159	-0.0317	±	0.0993
MLAC + M	1.675*	±	0.6239	-0.7681	±	0.5822	0.1539	±	0.1105
NACL	-0.1232	±	0.6442	0.9924	±	0.6449	-0.226	±	0.1249
NACL + M	0.2238	±	0.5362	0.521	±	0.5199	-0.1291	±	0.1008
UREA	0.3482	±	1.34	0.6815	±	1.296	-0.0901	±	0.2495
UREA + M	0.4824	±	0.7974	0.5418	±	0.7713	-0.113	±	0.1484

*Coefficients differ from zero, $P < 0.05$

[1]β_0 = intercept, β_1 = rate, β_2 = rate*rate

[2]Treatments were: control (CONT; no limiter); salt (NACL); urea (UREA); calcium carbonate (LIME); malic acid (MLAC), calcium propionate (CAPR), and calcium carbonate plus urea (LIUR), plus monensin (+ M)

Temporal stability. Inclusive of entire 14 d periods, regressions of supplement OMI temporal stability across rates resulted in no differences at the intercepts (Table 16). Rate of limiter inclusion coefficients appeared indicate effects of CONT (β_1, P = 0.05; β_2, P = 0.03), UREA (β_1, P = 0.05; β_2, P = 0.04), MLAC + M (β_1, P = 0.06; β_2, P = 0.03), and LIUR (β_1, P = 0.10; β_2, P = 0.03).

A repeat of the analysis using only the terminal 7 d of each treatment period removed the influence on each of the treatments. Intercepts of all treatments excluding LIUR + M ($P < 0.01$) were essentially zero. In addition, LIUR + M was the only treatment to have an influence of rate (β_1, P = 0.04; β_2, P = 0.11) on OMI temporal stability (Table 17).

Temporal stability by index. Change in OM supplement intake over time across full periods regressed on rate and rate^2 resulted in no intercepts that differed from zero. Among all treatments, rate of limiter inclusion of CAPR could reliably predict temporal stability (P = 0.01) and was the sole treatment to indicate a significant response. When rate^2 was applied as a variable, CAPR ($P < 0.01$), CAPR+M (P = 0.01), and UREA+M (P = 0.03) indicated a predictable effect on temporal stability change. Table 18 contains results of entire period regressions.

When the abridged version of the data was analyzed to evaluate trends across rates, CONT+M was the sole treatment producing a significant intercept (P = 0.03), response to rate (P = 0.02), and effect of rate^2 (P = 0.03) on supplement OMI temporal stability relative to CONT. Values of intercepts and coefficients are contained in Table 19.

Table 16. Regression coefficients[1] of OMI temporal stability values over 14 d periods.

Limiter	β_0			β_1			β_2		
CAPR	0.1229	±	0.1740	0.1012	±	0.1665	-0.0557	±	0.0313
CAPR + M	-0.0322	±	0.2246	0.2174	±	0.215	-0.0714	±	0.0404
CONT	-0.2881*	±	0.174	0.3435*	±	0.1665	-0.0689*	±	0.0313
CONT + M	0.266	±	0.2246	-0.2315	±	0.215	0.0431	±	0.0404
LIME	0.2131	±	0.1945	-0.1015	±	0.1862	0.0121	±	0.035
LIME + M	0.147	±	0.1945	-0.0392	±	0.1862	-0.002	±	0.035
LIUR	-0.1523	±	0.1945	0.3095	±	0.1862	-0.0773	±	0.035
LIUR + M	0.085	±	0.1945	0.0119	±	0.1862	-0.0086	±	0.035
MLAC	-0.1333	±	0.1945	0.1712	±	0.1862	-0.0409	±	0.035
MLAC + M	-0.2714*	±	0.1945	0.3652*	±	0.1862	-0.0795*	±	0.035
NACL	-0.0931	±	0.1945	0.2272	±	0.1862	-0.0535	±	0.035
NACL + M	-0.1718	±	0.1945	0.2589	±	0.1862	-0.0554	±	0.035
UREA	-0.2842*	±	0.1945	0.3733*	±	0.1862	-0.076*	±	0.035
UREA + M	0.0359	±	0.1945	0.0172	±	0.1862	-0.0096	±	0.035

*Coefficients differ from zero, $P < 0.05$

[1]β_0 = intercept, β_1 = rate, β_2 = rate*rate

[2]Treatments were: control (CONT; no limiter); salt (NACL); urea (UREA); calcium carbonate (LIME); malic acid (MLAC), calcium propionate (CAPR), and calcium carbonate plus urea (LIUR), plus monensin (+ M)

Table 17. Regression coefficients[1] of OMI temporal stability values over last 7 d of periods.

Limiter	β_0			β_1			β_2		
CAPR	0.0101	±	0.2614	0.1575	±	0.251	-0.0376	±	0.048
CAPR + M	0.2338	±	0.3375	-0.0341	±	0.324	-0.0107	±	0.062
CONT	0.4847	±	0.2614	-0.2894	±	0.251	0.0373	±	0.048
CONT + M	0.6071	±	0.3375	-0.5783	±	0.324	0.1027	±	0.062
LIME	-0.0301	±	0.3375	0.0858	±	0.324	-0.0205	±	0.062
LIME + M	0.5613	±	0.2923	-0.2512	±	0.2806	0.0228	±	0.0537
LIUR	0.3708	±	0.2923	-0.1985	±	0.2806	0.0299	±	0.0537
LIUR + M	0.8848*	±	0.2923	-0.5984*	±	0.2806	0.0877	±	0.0537
MLAC	0.1531	±	0.3375	0.0475	±	0.324	-0.026	±	0.062
MLAC + M	0.3547	±	0.2923	-0.0187	±	0.2806	-0.0265	±	0.0537
NACL	0.0999	±	0.2923	0.0613	±	0.2806	-0.0191	±	0.0537
NACL + M	0.1985	±	0.3375	-0.1156	±	0.324	0.0167	±	0.062
UREA	0.1596	±	0.2923	0.0928	±	0.2806	-0.0436	±	0.0537
UREA + M	0.3964	±	0.2923	-0.2484	±	0.2806	0.0362	±	0.0537

*Coefficients differ from zero, $P < 0.05$

[1]β_0 = intercept, β_1 = rate, β_2 = rate*rate

[2]Treatments were: control (CONT; no limiter); salt (NACL); urea (UREA); calcium carbonate (LIME); malic acid (MLAC), calcium propionate (CAPR), and calcium carbonate plus urea (LIUR), plus monensin (+ M)

Table 18. Regression coefficients[1] of indexed OMI temporal stability over 14 d periods.

Limiter	β_0			β_1			β_2		
CAPR	-50.86	±	88.77	312.8*	±	106.6	-161.6*	±	24.09
CAPR + M	-25.79	±	112.8	207.9	±	143.2	-110.2*	±	33.21
CONT	1.000	±	43.09	0.000	±	54.73	0.000	±	12.75
CONT + M	1.976	±	65.26	-19.87	±	82.84	10.73	±	19.21
LIME	1.699	±	53.01	-9.025	±	63.67	4.705	±	14.39
LIME + M	-3.08	±	58.29	20.95	±	70.01	-11.05	±	15.82
LIUR	-12.27	±	90.15	101.2	±	114.4	-53.57	±	26.53
LIUR + M	0.2108	±	48.92	-0.0056	±	58.75	0.3123	±	13.27
MLAC	-7.723	±	53.1	63.05	±	67.41	-33.37	±	15.63
MLAC + M	-5.999	±	120.3	63.01	±	152.8	-32.35	±	35.65
NACL	-1.481	±	31.16	16.38	±	41.75	-8.799	±	9.684
NACL + M	-0.1647	±	7.875	5.659	±	10.05	-2.76	±	2.379
UREA	0.6306	±	21.82	-0.0033	±	27.69	0.612	±	6.421
UREA + M	-4.188	±	23.73	33.69	±	30.12	-18.3*	±	6.985

*Coefficients differ from zero, $P < 0.05$

[1]β_0 = intercept, β_1 = rate, β_2 = rate*rate

[2]Treatments were: control (CONT; no limiter); salt (NACL); urea (UREA); calcium carbonate (LIME); malic acid (MLAC), calcium propionate (CAPR), and calcium carbonate plus urea (LIUR), plus monensin (+ M)

Table 19. Regression coefficients[1] of indexed OMI temporal stability over last 7 d of periods.

Limiter	β_0			β_1			β_2		
CAPR	-2.289	±	4.405	3.685	±	4.261	-0.906	±	0.82
CAPR + M	-2.125	±	5.418	3.107	±	5.241	-0.542	±	1.009
CONT	1.000	±	4.085	0.000	±	3.952	0.000	±	0.761
CONT + M	9.117*	±	3.267	-10.09*	±	3.16	2.13*	±	0.6082
LIME	1.059	±	3.147	-0.1133	±	3.045	0.0517	±	0.5859
LIME + M	-0.9616	±	4.293	3.147	±	4.152	-0.7712	±	0.7991
LIUR	4.001	±	4.258	-3.557	±	4.118	0.7107	±	0.7926
LIUR + M	-2.386	±	3.118	4.362	±	3.016	-0.9468	±	0.5805
MLAC	-2.756	±	2.083	4.069	±	2.015	-0.7094	±	0.3877
MLAC + M	1.399	±	6.549	-0.9934	±	6.111	0.507	±	1.159
NACL	-2.801	±	2.354	4.553	±	2.356	-0.943	±	0.4565
NACL + M	0.7029	±	1.594	0.1736	±	1.545	-0.0685	±	0.2997
UREA	-4.217	±	13.01	5.887	±	12.59	-0.8559	±	2.423
UREA + M	3.124	±	4.707	-3.307	±	4.553	0.774	±	0.8762

*Coefficients differ from zero, $P < 0.05$

[1]β_0 = intercept, β_1 = rate, β_2 = rate*rate

[2]Treatments were: control (CONT; no limiter); salt (NACL); urea (UREA); calcium carbonate (LIME); malic acid (MLAC), calcium propionate (CAPR), and calcium carbonate plus urea (LIUR), plus monensin (+ M)

Hay intake. When mean OM intakes of all days within periods were regressed by treatment, intercepts of CAPR ($P < 0.01$), CONT+M ($P = 0.03$), MLAC+M ($P = 0.05$), and UREA+M ($P < 0.01$) were different from zero (Table 20). Additionally, coefficients associated with CAPR (β_1, $P < 0.01$; β_2, $P = 0.02$) indicated significance by treatment inclusion. Organic matter hay intake was also influenced by the inclusion of UREA+M (β_1, $P = 0.05$; β_2, $P = 0.07$). Influence of rate on NACL+M intake was detected ($P = 0.05$) though intercept only tended to differentiate from zero ($P = 0.09$).

When indexed, forage OM intake intercepts of CAPR+M ($P < 0.01$), LIME ($P = 0.05$), LIME+M ($P = 0.04$), LIUR ($P < 0.01$), and UREA ($P = 0.05$) differed from CONT. Organic matter coefficients are contained in Table 21. Only consumption of CAPR+M appeared to be influenced by limiter inclusion (β_2, $P = 0.01$).

Conclusion

Intake of supplements containing MLAC and NACL were consistently lower than CONT over the course of the trial. Cumulative stability of intake was improved with each treatment when initial days were removed to allow for acclimation and decreased with most treatments as limiter inclusion was elevated. After removal of initial days, intake cumulative stability of all limiter-containing treatments were essentially equal to that of heifers offered CONT. Rates of intake change over time were less predictable by individual period. All treatments were consumed at an increasing rate relative to CONT during the intermediate period but did not differ during the final period. All limiting agents served to influence supplement intake and hay intake seemed to increase as limiters restricted supplement consumption.

Table 20. Regression coefficients[1] of sorghum x sudangrass OMI by treatment.

Limiter	β_0			β_1			β_2		
CAPR	6.37*	±	0.101	7.05*	±	0.129	-1.05*	±	0.03
CAPR + M	5.56	±	1.54	6.87	±	1.96	-0.941	±	0.454
CONT	6.67	±	1.70	3.45	±	2.16	-0.432	±	0.501
CONT + M	5.70*	±	0.30	2.90	±	0.38	-0.317	±	0.088
LIME	6.83	±	2.52	4.88	±	3.20	-0.836	±	0.741
LIME + M	8.46	±	2.83	3.83	±	3.60	-0.647	±	0.834
LIUR	7.14	±	1.75	4.53	±	2.22	-0.214	±	0.515
LIUR + M	7.84	±	0.88	4.05	±	1.12	-0.463	±	0.259
MLAC	7.05	±	2.40	14.43	±	3.05	-2.29	±	0.707
MLAC + M	8.69*	±	0.711	5.31	±	0.902	-0.871	±	0.209
NACL	6.87	±	1.71	14.68	±	2.17	-2.52	±	0.503
NACL + M	5.65	±	0.761	13.63*	±	0.966	-2.37	±	0.224
UREA	7.34	±	0.806	3.52	±	1.02	-0.714	±	0.237
UREA + M	6.14*	±	0.214	3.54*	±	0.271	-0.569	±	0.063

*Coefficients differ from zero, $P < 0.05$

[1]β_0 = intercept, β_1 = rate, β_2 = rate*rate

[2]Treatments were: control (CONT; no limiter); salt (NACL); urea (UREA); limestone (LIME); malic acid (MLAC), calcium propionate (CAPR), and limestone plus urea (LIUR), plus monensin (+ M)

Table 21. Regression coefficients[1] of indexed sorghum x sudangrass OMI by treatment.

Limiter	β_0			β_1			β_2		
CAPR	0.981	±	0.16	0.338	±	0.203	-0.064	±	0.047
CAPR + M	0.826*	±	0.004	0.397*	±	0.006	-0.068*	±	0.001
CONT	1.00	±	0.00	0.00	±	0.00	0.00	±	0.00
CONT + M	0.888	±	0.171	-0.034	±	0.217	0.009	±	0.05
LIME	1.01*	±	0.075	0.134	±	0.096	-0.037	±	0.022
LIME + M	1.26*	±	0.073	-0.054	±	0.093	-0.003	±	0.02
LIUR	1.07*	±	0.022	0.106	±	0.028	0.007	±	0.006
LIUR + M	1.20	±	0.11	-0.018	±	0.139	0.006	±	0.032
MLAC	1.13	±	0.443	0.973	±	0.562	-0.184	±	0.13
MLAC + M	1.36	±	0.281	0.015	±	0.357	-0.015	±	0.083
NACL	1.09	±	0.367	1.01	±	0.466	-0.203	±	0.108
NACL + M	0.88	±	0.244	0.989	±	0.309	-0.2	±	0.071
UREA	1.12*	±	0.094	-0.065	±	0.119	-0.008	±	0.028
UREA + M	0.947	±	0.129	0.004	±	0.164	-0.008	±	0.038

*Coefficients differ from zero, $P < 0.05$

[1]β_0 = intercept, β_1 = rate, β_2 = rate*rate

[2]Treatments were: control (CONT; no limiter); salt (NACL); urea (UREA); calcium carbonate (LIME); malic acid (MLAC), calcium propionate (CAPR), and calcium carbonate plus urea (LIUR), plus monensin (+ M)

Monensin inclusion did not affect supplement intake or cumulative stability of intake. Influence of monensin on temporal stability seemed to vary by days observed and rate of limiter. Rate of supplement intake change was only affected by monensin during the latter half of the intermediate rate period. Interactions of limiter × monensin were not detected on supplement intake or cumulative stability and influence on temporal stability paralleled monensin influence. Monensin did, however, consistently decrease hay intake of heifers assigned MLAC.

CHAPTER III

COMPARISON OF SODIUM CHLORIDE AND DL-MALIC ACID AS INTAKE LIMITING AGENTS IN A SELF-FED DRIED DISTILLERS' GRAIN SUPPLEMENT

All animal care and use procedures described in this protocol were approved by the Texas A&M University Animal Care and Use Committee (AUP 2009-239). Research was conducted at the Texas AgriLife Research Center outside of McGregor, TX during late winter and spring of 2011.

Materials and methods

Sixty angus-sired, weanling steers (257 kg mean initial BW) were used to compare the efficacy of a novel intake limiting agent relative to sodium chloride when included at identical rates in a self-fed, monensin-containing corn distillers' grains (DDG) supplement. Steers were stratified by weight and randomly assigned (2 steers per pen) to pens equipped with four Calan gates (American Calan, Inc., Northwood, NH). Each steer was fitted with keys enabling access to two Calan gate feed bunks, one containing chopped hay and the other containing supplement. Initially, heifers were provided daily with 1 kg of DDG and *ad libitum* access to sorghum x sudangrass hay in the adjacent bunk until acclimated to the feeding system and bunk assignments. Steers were weighed on the first day that treatments were applied.

Treatments were randomly assigned to pen and bunk. Limiting agents were included in treatments at 4 identical rates. Each treatment, excluding negative control, contained 66.1mg·kg^{-1}·suppl.$^{-1}$ of monensin. Treatments were: negative control, no

limiter **(CON)**; monensin **(RUM)**; sodium chloride (NACL) included at 8%, 16%, 24%, and 32% **(8NACL, 16NACL, 24NACL,** and **32NACL)**; and malic acid (MLAC, Baddley Chemicals, Inc., Baton Rouge, LA) included at 8%, 16%, 24%, and 32% **(8MLAC, 16MLAC, 24MLAC,** and **32MLAC)**. Treatments were prepared in bulk every week by combining components in a portable rotary mixer for a duration of at least five minutes and until visual appraisal of consistency was achieved. Treatments were sampled from mixer immediately after manufacturing of batch and composited within week.

All animals had continuous, ad libitum access to chopped sorghum x sudangrass hay. Hay availability was monitored daily during supplement feeding and supplied on an individual basis when necessary. Supplement was initially fed at predetermined amounts that were dependent upon limiter and inclusion rate after review of previous trials. Modifications of daily supply to both conserve feed and ensure *ad libitum* access were made on an individual basis. Supplement refusals were weighed daily at 0700 and disappearance from previous day was added. Modifications to delivery amount were applied on an individual basis throughout the trial to ensure availability and minimize waste. Every seventh day, accumulated refusals of both supplement and hay were sampled and discarded then replaced with fresh feed to mimic weekly replenishment of a bulk feeder in practice. Supplement refusal samples were composited by treatment each week whereas hay refusal sample were retained for individual steers.

Samples of manufactured treatments, unfed hay, and refusals of both were placed in a forced-air oven at 60ºC for 96 h to determine DM content. Samples were ground to pass

through a 1 mm screen of a Wiley mill (Thomas Wiley, Laboratory Mill Model 4, Thomas Scientific Co., Philadelphia, PA) and analyzed for ash, NDF, ADF, and CP. Organic matter was calculated from ash content of a 0.5 g sample placed into a muffle furnace (500º C) for 8 hours. Fiber content was estimated using ANKOM 200 Fiber Analyzer (ANKOM Technologies, Inc., Macedon, NY). Crude protein values were derived using Dumas combustion (Rapid-N-Cube, Elementar America, Inc., Mt. Laurel, N.J.). Nutrient composition of treatments and hay are provided in Table 22.

Within periods, response variables (mean supplement intake, mean hay intake, cumulative stability of supplement intake, and temporal stability of supplement intake) were analyzed as a completely randomized design with a 4 × 2 factorial treatment arrangement using the mixed model procedure of SAS 9.2. Limiter type, percentage of inclusion, and their interaction were included as effects in the model. Mean responses for each limiter were compared to the control treatment using t-tests. Pairwise comparisons among limiters were not performed. Significance of all statistical analysis was declared where $P < 0.05$. Reports of trends in data are limited to results where $P < 0.15$.

Table 22. Nutrient composition of treatments[1] and sorghum x sudangrass hay by week.

	Nutrient, %				
Item	DM	OM	NDF	ADF	CP
Week 1					
8MLAC	88.93	93.34	29.48	7.94	23.88
16MLAC	88.29	93.26	25.07	6.61	21.17
24MLAC	87.87	93.98	23.36	5.72	19.35
32MLAC	92.82	94.92	22.18	5.50	17.27
8NACL	89.75	85.60	28.18	7.58	24.03
16NACL	90.82	75.96	26.04	6.87	21.27
24NACL	91.45	70.21	23.87	6.22	18.84
32NACL	87.06	57.30	19.77	5.11	17.68
CON	89.41	92.44	32.09	8.97	26.11
RUM	89.11	92.16	31.43	8.66	25.81
Hay	91.42	90.68	58.96	33.67	10.92
Week 2					
8MLAC	92.88	92.06	31.01	8.09	25.17
16MLAC	92.14	93.32	29.18	7.83	22.87
24MLAC	91.54	94.41	25.01	6.56	20.59
32MLAC	92.82	94.92	22.18	5.50	17.27
8NACL	93.10	85.32	30.71	8.15	25.59
16NACL	93.00	86.44	31.49	8.45	25.03
24NACL	93.47	72.33	26.00	6.86	20.76
32NACL	94.82	60.19	21.36	5.71	17.47
CON	89.46	92.21	30.18	9.22	27.40
RUM	93.36	92.45	31.93	9.01	25.92
Hay	90.35	90.06	56.14	29.35	10.40
Week 3					
8MLAC	92.90	91.40	33.44	9.41	25.42
16MLAC	91.37	92.14	29.41	8.32	23.35
24MLAC	91.54	94.41	25.01	6.56	18.59
32MLAC	89.76	93.96	25.65	6.71	16.25
8NACL	93.45	88.11	36.84	11.78	25.09
16NACL	93.25	80.69	34.00	11.29	25.03
24NACL	93.34	73.07	29.67	9.20	20.81
32NACL	94.16	62.52	26.05	8.18	18.56
CON	89.00	92.22	31.89	8.46	26.51
RUM	92.89	92.31	38.48	12.28	26.83
Hay	92.13	89.92	57.40	30.13	10.41

continued on next page

Table 22. (continuted)

	Nutrient, %				
Item	DM	OM	NDF	ADF	CP
Week 4					
8MLAC	92.83	93.80	42.30	15.41	23.79
16MLAC	90.69	93.02	35.73	12.84	20.86
24MLAC	89.11	95.01	32.09	11.60	18.59
32MLAC	89.14	95.40	27.78	9.56	16.09
8NACL	92.80	84.52	39.85	14.86	25.18
16NACL	92.89	79.12	36.86	13.64	22.50
24NACL	93.19	73.94	35.44	13.15	20.29
32NACL	94.66	60.19	30.63	11.41	17.49
CON	92.64	92.43	33.41	9.31	25.91
RUM	92.73	94.23	46.50	17.74	26.72
Hay	92.03	90.64	58.82	30.33	10.11
Week 5					
8MLAC	93.01	94.27	42.06	16.89	24.38
16MLAC	91.45	95.51	38.11	13.89	21.84
24MLAC	89.05	95.40	29.43	10.85	18.08
32MLAC	89.54	96.19	28.32	10.18	16.46
8NACL	92.62	89.03	44.71	17.42	25.18
16NACL	92.46	78.53	38.60	15.03	22.60
24NACL	93.60	66.24	32.30	11.67	19.19
32NACL	93.95	57.60	28.35	10.43	16.70
CON	92.62	94.29	29.88	8.48	27.17
RUM	93.04	94.67	47.68	17.92	26.65
Hay	91.85	90.31	57.56	29.10	10.18
Week 6					
8MLAC	92.34	93.88	34.91	11.43	24.46
16MLAC	90.15	93.74	28.75	9.24	21.98
24MLAC	89.05	95.40	29.43	10.85	18.08
32MLAC	89.77	96.35	26.42	8.59	16.22
8NACL	93.11	86.54	34.12	11.65	26.08
16NACL	93.13	79.49	34.42	11.71	23.43
24NACL	94.55	68.82	27.83	9.26	20.62
32NACL	93.39	60.74	24.30	14.11	18.13
CON	92.87	94.79	31.82	9.04	26.82
RUM	89.56	93.97	46.03	16.11	27.40
Hay	91.85	90.70	59.21	29.86	9.74

[1]Control (CON), malic acid at 8% (8MLAC), 16% (16MLAC), 24% (24MLAC), 32% (32MLAC), sodium chloride at 8% (8NACL), 16% (16NACL), 24% (24NACL), 32% (32NACL), and monensin (RUM). All except CON contained 66.1 mg/kg monensin.

The effect of increasing percentage of limiters in supplement on response variables was evaluated using regression procedures of SAS v 9.2, where inclusion rate and inclusion rate squared were included as predictor variables in the model.

Results and discussion

Based on the results of the initial screening trial, limiting agents malic acid (MLAC) and sodium chloride (NACL) were employed at identical rates to compare efficacy in limiting supplement intake. As in the initial trial, measures included intake level to identify agent ability to decrease consumption, cumulative stability as an indication of the regularity of nutrient delivery within a herd at a given inclusion rate, and temporal stability as a measure of effective duration (Table 23).

Supplement intake

Comparison of supplement intake by steers fed CON and RUM indicated that the supplement intake was not affected by monensin (DM, $P = 0.66$; OM, $P = 0.62$). As a result, combined means of CON and RUM are used to contrast the influence of MLAC and NACL. Intake of supplement limited by MLAC was consistently lower compared to supplement containing NACL at identical rates. Consumption of supplement containing 8% limiter on an as-fed basis differed (DM and OM, $P < 0.01$) between limiter-containing treatments and was reduced relative to unlimited feed (4.87 kg, DM). Relative to supplement containing no limiter, DMI was 66.5 percentage units lower when limited by MLAC (1.66 kg, DM). Similarly, intake was only 70.7% of unlimited supplement when NACL (3.45 kg, DM) was included. Response to malic acid at this rate was greater than predicted based on the findings in Trial 1 where a similar inclusion rate

of 6% resulted in a 42 percentage point reduction in DMI. However, response to NACL in this study was comparable to the reduction in Trial 1 a formulated inclusion of 10% inclusion in supplement resulted in DMI of 3.63 kg. In direct comparison of limiters in this trial alone, MLAC was 2.11 times more effective at limiting intake at this rate than was NACL on a DM basis. This response measure is almost identical to that observed in the initial period of the first trial (2.02). When supplement in this trial contained an incremental rate increase of limiter, consumption was 15.8% and 31.7% of unlimited supplement on a DM basis when agent was 16MLAC and 16NACL, respectively. Differences in intake between limiters (DM and OM, $P < 0.01$) at this rate were again twice as pronounced (2.01) on a DM basis when 16MLAC (0.77 kg) was used compared to 16NACL (1.55 kg).

Supplement containing 24MLAC was consumed at a lower rate ($P = 0.01$) on a DM basis and tended to differ ($P = 0.10$) from 24NACL when expressed as OMI. Reduction in DMI measured 4.37 kg when 24MLAC (0.50 kg) was included whereas 24NACL (1.16 kg) in supplement resulted in a decrease of 3.71 kg relative to unregulated DDG. Due to the inverse relationship of organic matter content and sodium chloride inclusion, it is likely that decreases in OMI resulted in intake values not statistically different among treatments though DMI continued to be more heavily altered by use of MLAC. In comparison of treatments formulated to include 24% limiter on an as-fed basis, MLAC reduced intake 2.34 times as much as NACL. Differences in supplement intake on a DM basis were 0.67 kg whereas supplement OMI measured only 0.35 kg.

Table 23. Intake, cumulative stability, and temporal stability of supplement intake by limiter and inclusion rate.

	Intake				Cumulative stability				Temporal stability			
Rate[1]	MLAC	NACL	SE	*P*	MLAC	NACL	SE	*P*	MLAC	NACL	SE	*P*
8%												
DM	1.64	3.45	0.233	0.01	1.45	1.91	0.171	0.01	-0.276	0.529	0.202	0.01
OM	1.55	3.09	0.19	0.01	1.37	1.65	0.145	0.06	-0.247	0.585	0.172	0.01
16%												
DM	0.77	1.55	0.242	0.01	0.62	0.94	0.178	0.08	-0.08	0.331	0.210	0.06
OM	0.70	1.41	0.197	0.01	0.60	0.73	0.150	0.42	-0.118	0.58	0.179	0.01
24%												
DM	0.50	1.16	0.253	0.01	0.36	0.65	0.186	0.12	0.246	0.34	0.22	0.67
OM	0.55	0.90	0.207	0.10	0.35	0.44	0.157	0.57	0.499	0.645	0.188	0.44
32%												
DM	0.18	0.86	0.253	0.01	0.11	0.49	0.186	0.05	0.107	0.212	0.22	0.64
OM	0.17	0.58	0.207	0.05	0.11	0.25	0.157	0.37	0.077	0.351	0.188	0.15

[1]Inclusion rate on an as-fed basis.

Finally, supplement intake at 32% limiter inclusion differed (DM, $P = 0.01$; OM, $P = 0.05$) between limiting agents used. Supplement intake was mediated to a greater degree by 32MLAC (0.18 kg, DM) relative to 32NACL (0.85 kg, DM). At this inclusion rate, MLAC reduced intake 4.65 times greater on a DM basis than did NACL. Due to the extent that consumption was reduced by MLAC, differences in OMI between the two treatments were significant despite the level of sodium chloride.

Results indicating a two-fold intake reduction when supplement is limited by MLAC relative to NACL are supported by factorial analysis. Dry matter consumption of 8MLAC was only numerically different ($P = 0.71$) than that of 16NACL though differences within treatments were significant (MLAC and NACL, $P < 0.01$) at these rates. Similarly, supplement DMI by steers offered DDG formulated to included 16% MLAC was not different ($P = 0.73$) than those fed supplement composed of 32% NACL. Level of DMI at inclusion rates of 16% and 32% differed within treatment (MLAC, $P = 0.02$; NACL, $P < 0.01$).

In regards to overall intake, steers assigned CON and RUM consumed a mean 9.05 kg (DM). Across the range of limiter inclusions, steers fed supplement containing MLAC had similar total DMI values ranging from 67.98% to 76.92% of unlimited supplement. By contrast, steers fed supplement limited by NACL expressed a total DMI range of 69.64% to 93.53% of supplement containing no limiter. These results suggest that an initial, lower inclusion of MLAC may lead to a relatively substantial decrease in total DMI with progressive increments having a diminished effect (appear quadratic). On

the other hand, total DMI reductions by steers consuming NACL appear to roughly align with inclusion rate (appear linear).

Reliability of inclusion rate as a predictor of supplement intake was derived by regressing mean intakes within treatment. Table 24 contains regression coefficients for all dependent variables. Intakes across all percentage rates resulted in intercepts that were different from zero ($P < 0.01$) on both a DM and OM basis for both treatments. In addition, significant differences from zero at both inclusion rate (DM and OM, $P < 0.01$) and inclusion rate squared (DM and OM, $P < 0.01$) indicate a predictable response of both MLAC and NACL on supplement intake. Each formulated inclusion percentage of MLAC and NACL in supplement decreased DMI by 0.37 and 0.26 kg, respectively. Response to inclusion was reduced to a degree of 0.007 when MLAC was included and by 0.004 kg when NACL was used.

Cumulative stability

No differences in cumulative stability were detected (DM, $P = 0.26$; OM, $P = 0.25$) among steers consuming CON and RUM. Therefore, means used in comparison to MLAC and NACL treatments are inclusive of both.

Table 24. Regression coefficients[1] for intake, intake variance, and time stability.

Item[2]	β0		SE	β1		SE	β2		SE
MLAC[3]									
DMI	4.067*	±	0.2997	-0.3675*	±	0.0449	0.0074*	±	0.0013
OMI	4.337*	±	0.283	-0.3445*	±	0.0424	0.0069*	±	0.0013
DMCS	2.607*	±	0.1289	-0.1641*	±	0.0193	0.0027*	±	0.0006
OMCS	2.466*	±	0.1213	-0.1546*	±	0.0182	0.0026*	±	0.0005
DMTS	0.7439*	±	0.3035	-0.1013*	±	0.0455	0.0027*	±	0.0014
OMTS	0.6541*	±	0.2902	-0.0839	±	0.0435	0.0023	±	0.0013
NACL[4]									
DMI	4.980*	±	0.3073	-0.2632*	±	0.046	0.0042*	±	0.0014
OMI	4.666*	±	0.2665	-0.2544*	±	0.0399	0.004*	±	0.0021
DMCS	2.687*	±	0.1258	-0.1331*	±	0.0188	0.002*	±	0.0006
OMCS	2.526*	±	0.0991	-0.1405*	±	0.0148	0.0022*	±	0.0005
DMTS	0.9196*	±	0.2952	-0.0495	±	0.0442	0.0009	±	0.0013
OMTS	0.815*	±	0.2655	-0.0152	±	0.0397	0.0001	±	0.0012

**P*-values differ at 0.05

[1]β0 = intercept, β1 = percent limiter, β2 = percent*percent

[2]DMI = dry matter intake, OMI = organic matter intake, DMCS = dry matter cumulative stability, OMCS = organic matter cumulative stability, DMTS = dry matter temporal stability, OMTS = organic matter temporal stability

[3]Malic acid (MLAC; 0%, 8%, 16%, 24%, 32%). All levels formulated to include 66.1 mg/kg monensin.

[4] Salt (NACL; 0%, 8%, 16%, 24%, 32%). All levels formulated to include 66.1 mg/kg monensin.

Cumulative stability of supplement intake was improved over unlimited supplement (2.63 kg, DM) by both limiters at all rates of inclusion. In comparison of limiting agents, inclusion at the lowest and highest rate resulted in significant intake stability improvements when MLAC was used relative to NACL. Intermediate rates of limiter in supplement here tended to induce the same result on a DM basis. Both improvement over unregulated supplement and consistency in limiter influence oppose the erratic findings in Trial 1. Comparison of mean intake variation between treatments formulated to contain 8% limiter differed (DM, $P < 0.01$; OM, $P = 0.06$) resulting in cumulative stability that was reduced by 0.46 kg/d (24%) when NACL was included as opposed to MLAC. At formulated inclusion of 16% ($P = 0.08$) and 24% ($P = 0.12$), MLAC was more effective at stabilizing DMI by measures of 0.32 kg and 0.29 kg, respectively. Cumulative stability of supplement intake by steers consuming 32MLAC and 32NACL differed ($P = 0.05$) on a DM basis but was similar ($P = 0.37$) on an OM basis. When limiting agent composed 32% of supplement, DMI variation within treatment was decreased 0.38 kg/d (78%) with use of MLAC relative to NACL.

Analysis of cumulative stability regression values indicate that percentage and squared percentage rate of limiter inclusion are of use in predicting cumulative stability of both MLAC and NACL in a DDG supplement. A similar decline in cumulative variance is predicted for both limiters (DM and OM, $P < 0.01$) when included at like rates. By percentage of inclusion, MLAC tempered cumulative variance of supplement DMI by 0.16 kg whereas NACL improved stability by 0.13 kg. Additionally, significance of the β_2 coefficient (DM and OM, $P < 0.01$) with both limiters suggests

that response to inclusion percentage will dissipate at increased levels. Improvements in supplement DMI cumulative stability were dissipated by 0.002 kg with each incremental inclusion of both agents. A visual reference to cumulative stability by rate in this study is provided in Figure 11.

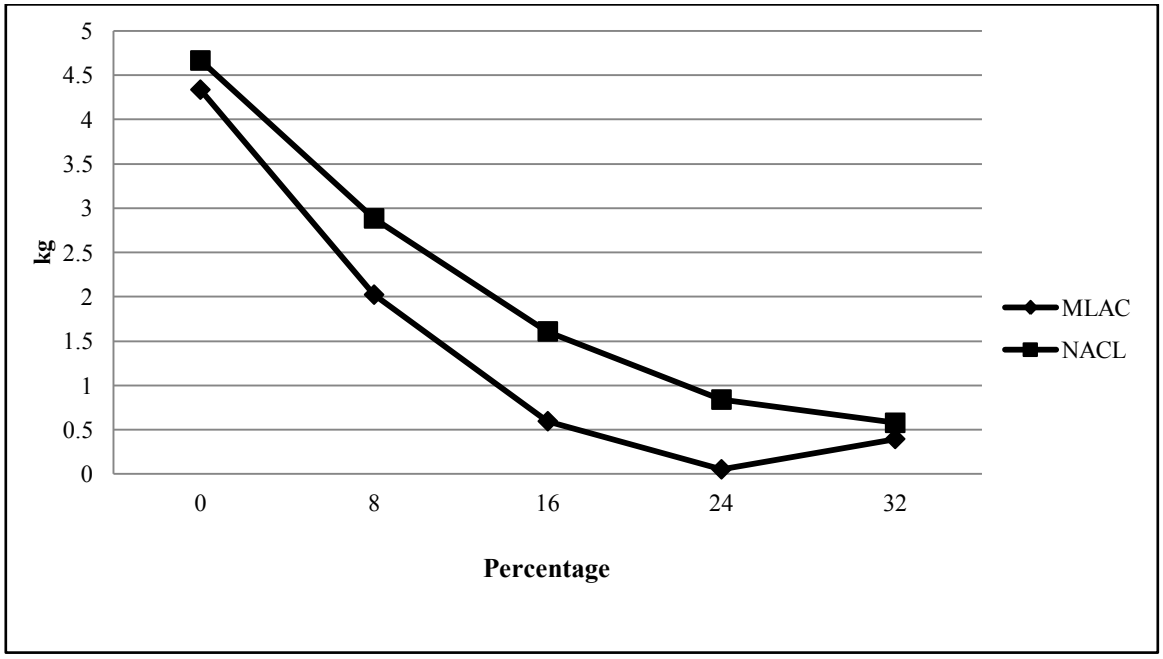

Figure 11. Regressed DMI cumulative stability by percentage of limiter inclusion.

Temporal stability

As with intake and cumulative stability, monensin had no affect on temporal stability. As a collective mean, daily intake change of both CON and RUM over the course of the trial was +0.95 kg/d.

Daily changes in supplement consumption were significantly different between limiter-containing treatments when included at 8% (DM and OM, $P < 0.01$) and 16% (DM, $P = 0.06$; OM, $P = 0.05$). At 8% inclusion, DMI decreased 0.28 kg/d when limited

by MLAC and increased 0.53 kg/d when NACL was included. An intake reduction by steers consuming this level of MLAC contradicts the slight temporal increase (0.16 kg) observed in Trial 1 when supplement contained 6%. Heifers consuming DDG containing 10% salt during Trial 1 increased their daily intake to a lesser degree (0.16 kg vs. 0.53 kg) relative to steers in this trial consuming 8NACL. Organic matter intake of supplement formulated to include 8% limiter decreased 0.25 kg/d and increased 0.59 kg/d when containing MLAC and NACL, respectively. When 16MLAC and 16NACL were provided, DM temporal stability measures were -0.08 kg and 0.33 kg, respectively. Organic matter of supplement including 16% limiter was consumed at a decreasing rate (-0.11 kg/d) when limiter was MLAC and at an increasing rate (0.58 kg/d) when tempered by NACL. Differences in temporal stability at inclusion levels of 24% (DM, $P = 0.67$; OM, $P = 0.44$) and 32% (DM, $P = 0.64$; OM, $P = 0.15$) were undetected.

When daily mean temporal stability measures within treatment were regressed, analysis indicated that only temporal intake change of MLAC could be reliably predicted by inclusion rate. Intercept (0.74 kg, DM) of supplement temporal stability associated with MLAC (DM, $P = 0.02$; OM, $P = 0.03$) as well as coefficients of percentage (-0.37 kg, DM, $P = 0.03$; OM, $P = 0.06$) and percentage squared (0.01 kg, DM, $P = 0.05$; OM, $P = 0.09$) suggest that increasing concentration will decrease temporal variance at a decreasing rate with progressive inclusions. On the other hand, regression of NACL temporal shifts resulted in an intercept that differed significantly from zero (0.92 kg, DM and OM, $P = 0.01$) but neither a response to percentage (-0.05, DM, $P = 0.27$; OM, $P = 0.71$) nor percentage squared (0.00 kg, DM, $P = 0.51$; OM, $P = 0.94$) proved to be useful

in temporal predictability. Figure 14 contains a visual comparison of regressed temporal stability.

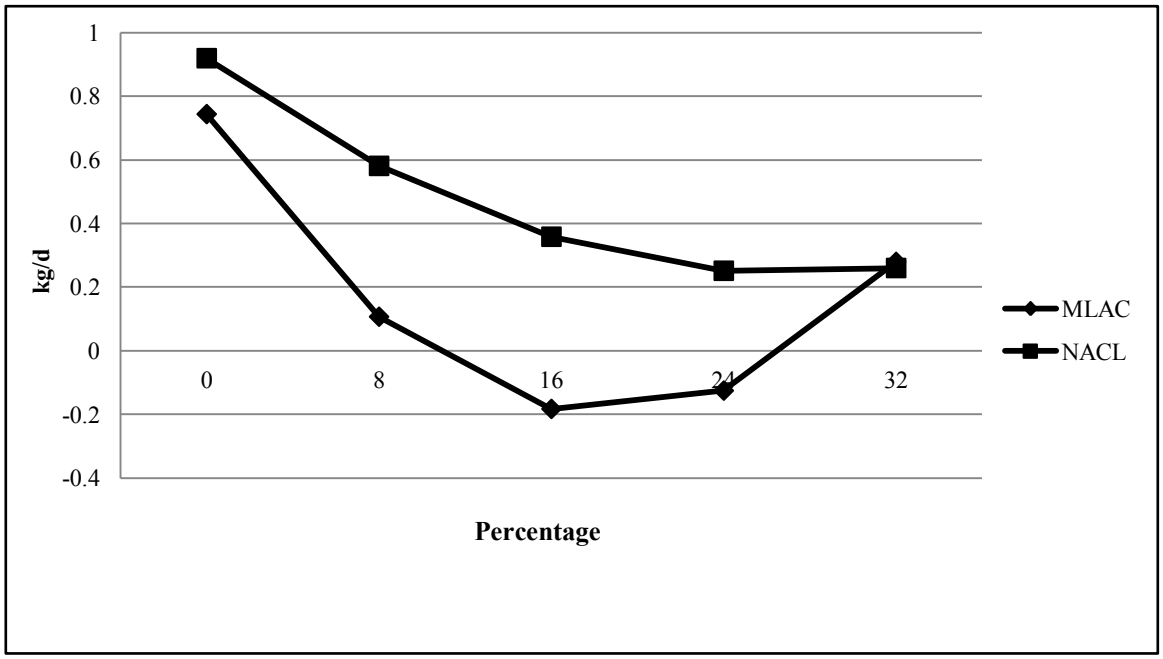

Figure 12. Regressed DMI temporal stability by percentage of limiter inclusion.

Hay intake. Overall, hay intake was unaffected by monensin (DM, $P = 0.56$; OM, $P = 0.53$) or limiter type (DM, $P = 0.30$; OM, $P = 0.29$). Hay intakes by percentage of limiter inclusion are provided in Table 25.

Table 25. Mean daily hay intakes (kg) by percentage of limiter inclusion.

	MLAC		NACL	
Percentage	DM	OM	DM	OM
0%	4.18	3.80	4.18	3.80
8%	5.33	4.88	5.02	4.57
16%	5.40	5.04	5.71	5.25
24%	5.67	5.21	5.49	5.01
32%	5.97	5.47	5.45	4.99

When regressed across percentage rates of limiter inclusion (Table 26), hay intake intercepts of both limiters differed from zero on both a DM and OM basis ($P <$ 0.01). Percentage of limiter in supplement affected hay intake of steers fed both MLAC (DM and OM, $P < 0.01$) and NACL (DM and OM, $P < 0.01$). With each percentage increase of limiting agent incorporated into DDG, hay intake increased by 0.12 kg when limited by MLAC and 0.13 kg when NACL was used. Squaring the percentage of limiter in supplement resulted in only a slight response with both agents. Response to MLAC in terms of hay DMI was relatively stable (-0.002 kg, $P = 0.07$) across the range of limiter inclusion though the effect on hay DMI by steers fed NACL was more pronounced (-0.003 kg, $P = 0.03$).

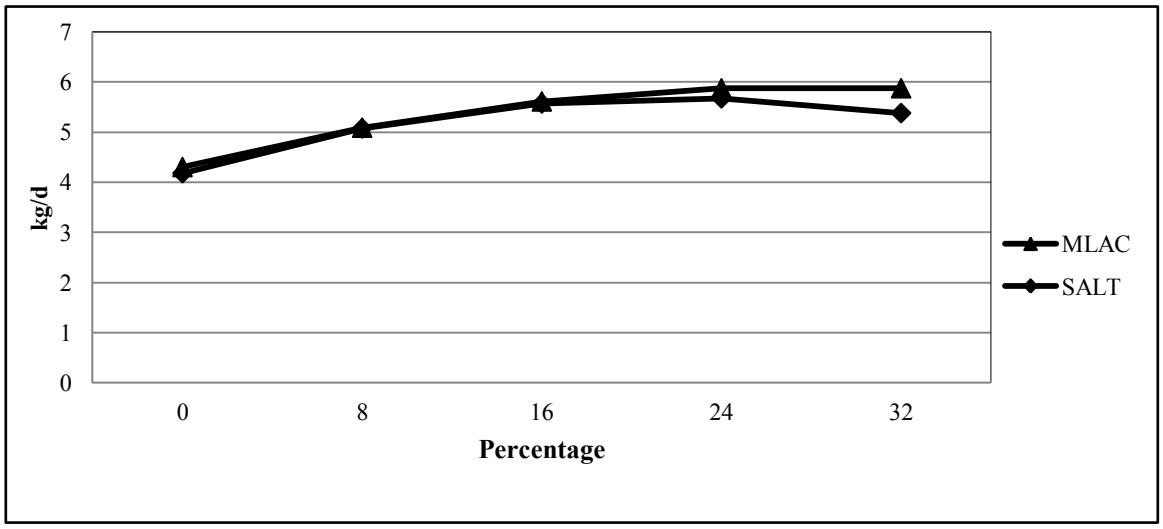

Figure 13. Regressed DMI of sorghum x sudangrass hay by percentage of limiter inclusion.

Table 26. Regression coefficients[1] of indexed DM and OM intakes.

Item[2]	β0		SE	β1		SE	β2		SE
MLAC[3]									
DMI	4.296*	±	0.2657	0.1207*	±	0.0398	-0.0022	±	0.0012
OMI	3.9145*	±	0.2466	0.1134*	±	0.037	-0.0021	±	0.0011
NACL[4]									
DMI	4.1812*	±	0.2730	0.1291*	±	0.0408	-0.0029*	±	0.0012
OMI	3.8038*	±	0.2567	0.1203*	±	0.0384	-0.0027*	±	0.0012

**P*-values < 0.05

[1]β0 = intercept, β1 = percent limiter, β2 = percent*percent

[2]DMI = dry matter intake, OMI = organic matter intake, DM SD = dry matter standard deviation, OM SD = organic matter standard deviation, DM Slope = daily change in DMI, OM Slope = daily change in OMI

[3]Malic acid (MLAC; 0%, 8%, 16%, 24%, 32%). All levels included 66 mg/kg monensin.

[4] Salt (NACL; 0%, 8%, 16%, 24%, 32%). All levels included 66 mg/kg monensin.

Conclusion

Intake of MLAC was consistently lower than NACL at each level of inclusion. Cumulative stability of MLAC was improved relative to that of NACL at 8% inclusion but differences diminished as inclusion was increased. Daily intake reductions were more pronounced with MLAC up to 16% inclusion. Hay intake was reduced relative to RUM by limiter inclusion up to 8% MLAC and 16% NACL.

CHAPTER IV

EVALUATION OF STACKED PRODUCTION TECHNOLOGIES ON ADG OF CALVES GRAZING SMALL GRAIN WINTER PASTURES

All animal care and use procedures described in this protocol were approved by the Texas A&M University Animal Care and Use Committee (AUP 229-239). Research was conducted from January through April 2011 at the Beef Research Unit at Texas A&M University, College Station, TX.

Materials and methods

One hundred crossbred steers (240 kg mean initial BW) were used in a split-plot design to evaluate the influence of beef production technologies on ADG over the course of a 98-d grazing period on yearling cattle grazing irrigated winter oats (*Avena sativa L.*) pastures. Steers were weighed and at weaning and received a multivalent killed viral vaccine (Vira Shield 6+VL5, Novartis Animal Health US, Inc., Greensboro, NC) and clostridial vaccinine (Clostri Shield 7, Novartis Animal Health US, Greensboro, NC). Body weights were obtained again 23 days prior to the study to obtain sorting weights. All steers originated from a single location (Texas AgriLife Research Center, McGregor, TX) and were relocated to study site and fed hay in a drylot until prior to trial initiation.

Sub plot treatments were randomly assigned to experimental units (steers). Whole plot treatments were hand-fed energy supplement (Table 27) fed at a rate of 0.91 $kg \cdot hd^{-1} \cdot d^{-1}$ with (**RUM**; 135 $mg \cdot hd^{-1} \cdot d^{-1}$) or without **(CON)** monensin (Rumensin 90; Elanco Division, Eli Lilly and Co., Indianapolis, IN). Sub plot treatments were arranged in a 2 x 2 factorial and consisted of administration of a metaphylactic (**MM**; Micotil 300,

Elanco Division, Eli Lilly and Co., Indianapolis, IN) dosed at 1.5 ml/cwt of initial BW or no metaphylaxis **(PT)** and a single dose implant (**I**; TEG with Tylan, Elanco Division, Eli Lilly and Co., Indianapolis, IN) given at initial day of study in the middle third of the right ear or no implant **(0)**. Four whole plot experimental units (paddocks) consisted of four replicates containing equal numbers (n=3) of each sub plot treatment experimental units whereas remaining paddocks contained on additional, randomly assigned sub plot experimental unit. Thus, four paddocks contained 12 steers and four paddocks contained 13 steers. Paddocks were approximately 6.22 ha equating to stocking rates of 1.93 hd per ha. Sort weights were used to allocate steers to whole plot replicates. Figure 48 depicts layout of paddocks and treatment assignments.

Shrunk body weights were collected in the morning of d 28, 56, 84, and 105. On the preceding days, steers were gathered in the afternoon and held overnight without access to feed or water. Manual palpation of ear was performed to confirm presence of implant. After weighing, steers were immediately resorted into returned to assigned paddocks.

Table 27. Composition of hand-fed energy supplement fed to steers grazing oat pastures (% as-fed)

	Treatment			
	monensin		control	
Item	batch 1[1]	batch 2[2]	batch 1[1]	batch 2[2]
Dried distillers' grains	97.35%	94.51%	97.50%	94.70%
Commercial mineral	2.50%	2.43%	2.50%	2.43%
Molasses	-	2.91%	-	2.87%
Rumensin 90[3,4]	0.150%	0.146%	-	-

[1]Batch 1 was fed d 1 through 34

[2]Batch 2 was fed d 35 through 105

[3]135 and 131 $mg \cdot hd^{-1} \cdot d^{-1}$ of active ingredient in Batch 1 and 2, respectively

[4]Elanco Division, Eli Lilly and Co., Indianapolis, IN

Supplement was provided daily in metal troughs (3.05 meters) placed in relative proximity to water. Every seventh day, remaining supplement was gathered prior to feed delivery and weighed in order to calculate supplement intake·hd^{-1}·d^{-1}. Due to low intakes of monensin-containing supplement, molasses (3%) was added to both diets and fed beginning d 35. In order tom minimize effects of supplement intake variation between dietary treatments, supplement allowance was reduced to 0.45 kg·hd^{-1}·d^{-1} beginning on d 42. A timeline is presented in figure #. Samples from each batch of formulated supplement were collected weekly. Samples were dried in a forced air oven (60º C) for 96 h for DM determination. Samples were ground to pass through a 1 mm screen of a Wiley mill (Thomas Wiley, Laboratory Mill Model 4, Thomas Scientific Co., Philadelphia, PA) and analyzed for ash, NDF, ADF, and CP. Organic matter was calculated from ash content of a 0.5 g sample placed in an ashing oven (500º C) for 8 h. Fiber content was estimated using an ANKOM 200 Fiber Analyzer (ANKOM Technologies, Inc., Macedon, NY). Crude protein values were derived using Dumas combustion (Rapid-N-Cube, Elementar America, Inc., Mt. Laurel, N.J.).

Procurement of forage samples to estimate availability and nutrient content coincided with weigh dates. Within each paddock, 8 samples of forage clipped to approximately 2.5 cm were obtained by tossing at random a 0.1 sq meter quadrant. Each sample was placed into a pre-weighed paper bag and dried in a forced air oven (60º C) for 96 h to determine mean DM content. Mean sample weights were multiplied by 94512.38 to convert m^2 to forage mass per ha. With each collection, samples were composited by paddock and ground to pass through a 1 mm screen of a Wiley mill

(Thomas Wiley, Laboratory Mill Model 4, Thomas Scientific Co., Philadelphia, PA) and analyzed for ash, NDF, ADF, and CP. Organic matter was calculated from ash content of a 0.5 g sample placed in an ashing oven (500º C) for 8 h. Fiber content was estimated using an ANKOM 200 Fiber Analyzer (ANKOM Technologies, Inc., Macedon, NY). Crude protein values were derived using Dumas combustion (Rapid-N-Cube, Elementar America, Inc., Mt. Laurel, N.J.).

Calves were monitored once daily for health status using a 5-point clinical illness score. Morbidity signs including depression, ocular and nasal discharges, and respiratory issues were pulled for temperature reading. A rectal temperature ≥ 40º C warranted treatment by antibiotic according to label instructions and visually severe symptoms resulted in treatment regardless of temperature. Description of clinical illness score criteria is provided in Table 28. Steers exhibiting morbidity were treated initially with entrofloxacin (Baytril 100, Bayer Corporation, Shawnee, MI) at a rate of 10 mg/kg BW. Steers requiring a follow up treatment were administered tulathromycin (Draxxin, Pfizer Animal Health, Madison, NJ). Bloat was quantified with a 4-point scoring system which followed Paisley and Horn (1998). Values were: 0 = normal, 1 = slight distention, 2 = distention with rumen elevated toward backline, and 3 = severe distention with rumen at or above back level.

Table 28. Descriptive criteria for subjective health evaluation

Score	Description
0	Normal. No clinical sign of illness.
1	Mildly abnormal respiration. Dyspnea with minor depression, gauntness, nasal and/or ocular discharges.
2	Moderatley abnormal respiration. Dyspnea with noticeable depression gauntness, nasal and/or ocular discharges.
3	Severely abnormal respiration. Pronounced dyspnea, depression, gauntness, nasal and/or ocular discharges.
4	Moribound – immobile.

Weight and ADG were analyzed using the mixed model (PROC MIXED) procedure in SAS (SAS Inst. Inc., Cary, NC). Monensin, metaphylaxis, and implant served as fixed effects with paddock replicate as a random effect. Forage availability and nutrient content were analyzed using repeated measures with compound symmetry as the covariance structure.

Supplement intake was used in the 1996 NRC model to estimate forage intake. Body weights used in the model were means of d 0 and d 98 for each dietary treatment group. Measures of cumulative ADG for diet × implant were used with mean intakes of both diets to calculate differences in energy associated with dietary treatment by adjusting forage intake to meet ADG. Difference in estimated NEg by removal of supplement intake was attributed to supplement.

Results and discussion

Initial weight

A by-treatment summary of initial BW, final BW, cumulative ADG, and ADG within each period is provided in Table 29. Initial BW (d 0) was not affected by dietary treatment ($P < 0.28$) or either of the subplot treatments (implant, $P < 0.68$; metaphylaxis, $P < 0.38$). Additionally, no interactions were significant with measures of initial BW (diet × implant, $P < 0.25$; diet × metaphylaxis, $P < 0.98$; implant × metaphylaxis, $P < 0.86$; diet × implant × metaphylaxis, $P < 0.61$). Interactions are summarized in Table 30.

ADG within period

Weight gain during the first 28 d period was influenced by both implant ($P < 0.02$) and metaphylaxis ($P < 0.05$). Daily gains were increased by 20.46% in steers receiving implant and 16.41% in steers given metaphylactic treatment. No difference was attributed to dietary treatment or any treatment interactions.

From d 29 through 56, implant use was effective ($P < 0.01$) in raising ADG. Metaphylactic application did not significantly increase gains ($P < 0.71$) though a slight interaction was observed between dietary treatment and metaphylaxis ($P < 0.08$). Diet alone did not influence gains nor were did any other interactions.

Gains during d 57 through 84 were higher in implanted cattle ($P < 0.01$) and an interaction between diet and implant was also detected ($P < 0.01$). Diet alone tended to influence gains ($P < 0.11$) with cattle receiving RUM having 9.33% higher gains. Metaphylaxis and other interactions did not significantly affect ADG.

Table 29. Mean BW and ADG by treatment of steers grazing winter oats

	Diet				Implant				Metaphylaxis			
Item[1]	CON[2]	RUM[3]	SEM	*P*-value	0[4]	I[5]	SEM	*P*-value	PT[6]	MM[7]	SEM	*P*-value
BW0	233.96	245.50	6.81	.28	240.67	238.80	5.31	.68	237.78	241.69	5.31	.38
BW98	373.00	378.10	6.69	.61	368.64	382.46	5.68	.03	372.00	379.09	5.68	.26
ADG28	0.98	0.91	0.08	.55	0.86	1.04	0.07	.02	0.88	1.02	0.07	.05
ADG56	1.49	1.44	0.10	.72	1.39	1.54	0.08	.01	1.48	1.45	0.08	.71
ADG84	1.36	1.49	0.05	.11	1.33	1.52	0.04	.01	1.39	1.45	0.04	.32
ADG98	1.60	1.20	0.07	.01	1.32	1.48	0.06	.03	1.41	1.57	0.09	.74
CUM56	1.24	1.17	0.03	.22	1.12	1.29	0.03	.01	1.18	1.24	0.03	.19
CUM84	1.28	1.28	0.03	.99	1.19	1.37	0.03	.01	1.25	1.31	0.03	.09
CUM98	1.34	1.26	0.03	.13	1.22	1.38	0.03	.01	1.28	1.32	0.03	.15

[1]BW0 = initial BW, BW98 = final BW, ADG28 = ADG d 1-28, ADG56 = ADG d 29-56, ADG84 = ADG d 57-84, ADG98 = ADG d 85-98, CUM56 = ADG d 1-56, CUM84 = ADG d 1-84, CUM98 = ADG d 1-98

[2,3]CON = Control, RUM = Rumensin containing (135 mg·hd^{-1}·d^{-1})

[4,5]PT = pull and treat (no metaphylaxis), MM = metaphylaxis (Micotil 300, 1.5 ml/100 kg BW)

[6,7]0 = no implant, I = single dose implant (TEG + Tylan)

Table 30. Mean BW and ADG by treatment interactions of steers grazing winter oats (kg)

	Diet[1]									
	CON				RUM					
	PT[2]		MM[3]		PT		MM			
Item[6]	0[4]	I[5]	0	I	0	I	0	I	SEM	*P*, D*M*I[7]
BW0	231.83	232.09	232.77	239.17	246.38	240.81	251.71	243.13	8.82	.68
BW98	359.12	379.81	368.39	384.68	369.75	379.33	377.29	386.01	10.25	.89
ADG28	0.71	1.03	1.12	1.09	0.77	1.00	0.85	1.03	0.12	.32
ADG56	1.60	1.51	1.31	1.54	1.29	1.50	1.35	1.61	0.12	.28
ADG84	1.10	1.51	1.26	1.57	1.52	1.45	1.43	1.54	0.09	.20
ADG98	1.50	1.63	1.54	1.74	1.11	1.33	1.15	1.23	0.11	.43
CUM56	1.15	1.27	1.21	1.32	1.03	1.25	1.10	1.32	0.07	.94
CUM84	1.13	1.35	1.23	1.40	1.19	1.31	1.21	1.39	0.05	.46
CUM98	1.21	1.41	1.29	1.44	1.18	1.32	1.20	1.36	0.05	.62

[1]CON = Control, RUM = Rumensin containing (149 mg/kg d 1-35, 144 mg/kg d 36-105)

[2,3]PT = pull and treat (no metaphylaxis), MM = metaphylaxis (Micotil 300, 1.5 ml/100 kg BW)

[4,5]0 = no implant, I = single dose implant (TEG + Tylan)

[6]BW0 = initial BW, BW98 = final BW, ADG28 = ADG d 1-28, ADG56 = ADG d 29-56, ADG84 = ADG d 57-84, ADG98 = ADG d 85-98, CUM56 = ADG d 1-56, CUM84 = ADG d 1-84, CUM98 = ADG d 1-98

[7]D*M*I = Diet × metaphylaxis × implant

Day 85 to 98 ADG were influenced by diet ($P < 0.01$) and implant ($P < 0.03$). Steers consuming CON gained 33.28% more than steers fed monensin whereas implanted steers had higher gains by 49.14%.

Cumulative ADG by period

Through d 56, significant differences in ADG were again attributed to use of implant ($P < 0.01$) where steers receiving treatment had gains 14.65% higher than those not receiving implant. Influence of metaphylaxis was not detected ($P < 0.19$) though treated steers gained 5.28% more weight. Again, no differences in ADG in the period were attributed to dietary treatment or treatment interactions.

At d 84, cumulative rate of gain was again improved by implant ($P < 0.01$). Steers receiving the treatment gained 1.365 kg/d as opposed to 1.192 kg/d. Metaphylaxis tended to increase ADG ($P < 0.09$) by increasing gains 4.88%. No differences were observed as a result of supplement or interactions of treatments.

Encompassing the entire trial, use of implants improved ADG by 13.46% ($P < 0.01$). Gains were slightly affected ($P < 0.13$) by dietary treatment as steers assigned CON supplement gained 28.08% more weight. Metaphylactic usage also increased gains ($P < 0.15$; 16.65%). No treatment interactions were detected. Gains of cattle receiving metaphylactic were 16% higher during the first 28 days on pasture and tended to be slightly higher during days 0 to 84 and 0 to 98. Initial gain differences are nearly identical to those provided in a summary by Wileman et al., 2009. Galyean et al. (1995) observed no difference in ADG among steers treated with tilmicosin or a control in an initial 56 day feeding period; however, a significant reduction in cattle pulled for bovine

respiratory disease was achieved. Herd health in our study was excellent and void of any necessary treatment associated with internal abnormalities. Part of this stability may be attributed to common origin of the cattle. Influence on ADG during the later periods are somewhat unexpected in that most problems associated with bovine respiratory disease and associated performance reductions are manifested within the first 45 of arrival (Edwards, 1996). However, reviewed temporal patterns of outbreaks in feedlots suggest that occurrence may be delayed (Babcock et al., 2009). Despite occasional late occurrences, treatment is most effective as it relates to ADG when provided on arrival as opposed to delay (Kreikemeier et al., 1996).

Implanting steers provided the most consistent impact among treatments in this trial. Final body weight as well as all ADG measures both within period and cumulative were significantly increased as a result of implantation. Mean increases in ADG among all measures in this study were 0.17 kg/d. These results are substantially higher than approximate reported ranges of cattle on small grains (0.08 kg/d, McCollum, 2006). Gains relative to control from this study were identical to steers fed a distillers' grain supplement in a split-plot design (mean = 0.17 kg/d, McMurphy et al., 2009) evaluating implant type though gain response in regards to implant assignment was undefined. Interactions in this study were largely insignificant as expected due to unrelated modes of action among between treatments.

Supplement intake

Mean DMI of dietary treatments were different overall ($P < 0.01$) with steers assigned RUM consuming less than CON (0.26 vs. 0.56 $kg \cdot hd^{-1} \cdot d^{-1}$, respectively).

Within dietary treatments, supplement DMI was not different among paddocks (RUM, $P = 0.28$; CON, $P = 0.91$). Overall, supplement containing monensin in this study was consumed at a considerably reduced rate relative to a control. Intakes were reduced 54% by an inclusion rate similar to that of Paisley and Horn (1996) who observed intake reductions of 43%. As an expected result of reduced means, coefficient of variation for weekly supplement intake was much higher in this study for supplement containing monensin compared to a control diet. Though intakes of a monensin-containing supplement were slightly higher, calculated coefficient of variation was much lower in the data published by Paisley and Horn (1996) and much closer to control diet variation measures.

Final weight

Ending BW (d 98) was significantly influenced by use of implant ($P < 0.03$). Body weights were increased by a mean of 13.82 kg relative to non-implanted steers. Neither diet ($P < 0.61$) nor metaphylaxis ($P < 0.26$) impacted final weights and no interactions were detected.

Response to supplement

Estimation of forage intake to meet observed gains using the 1996 NRC model indicated that supplement containing monensin resulted in an 8.45% increase in NEg yield (CON = 1.42 Mcal/kg, RUM = 1.54 Mcal/kg). Estimated forage intake to meet actual gain in the absence of supplement was lowest for RUM -0 (4.43 kg/d) group and highest for CON-I (5.10 kg/d). Intake was the same for CON-0 and RUM-I (4.75 kg/d). Neither initial nor final BW in this study was significantly influenced by dietary

treatment. This suggests that steers assigned monensin either compensated by consuming more forage or were provided sufficient energy despite intake level to facilitate gains similar to other steers consuming twice as much. Average daily gains were only significantly influenced by diet from day 84 to day 98; however, cumulative ADG tended to higher in steers not consuming monensin. Typically, monensin would be expected to increase ADG (Horn et al., 1981). Therefore, atypical results of this study are attributed to low level of supplement intake. Supplemental energy will increase ADG on small grains pastures due to increased microbial growth and protein synthesis (McCollum and Horn, 1990; Horn et al., 1995). Therefore, reduced intake associated with monensin inclusion would be expected to reduce ADG response relative to steers consuming a higher level of supplement.

Standing crop

Over the course of the trial, forage availability was unaffected by dietary treatment ($P = 0.52$) and no dietary treatment × day interaction occurred ($P = 0.26$). Standing crop decreased from d 0 to 56 then increased through d 98 where forage mass peaked (6324 ± 366 kg DM/ha) resulting in a day effect ($P < 0.01$, Figure 16).

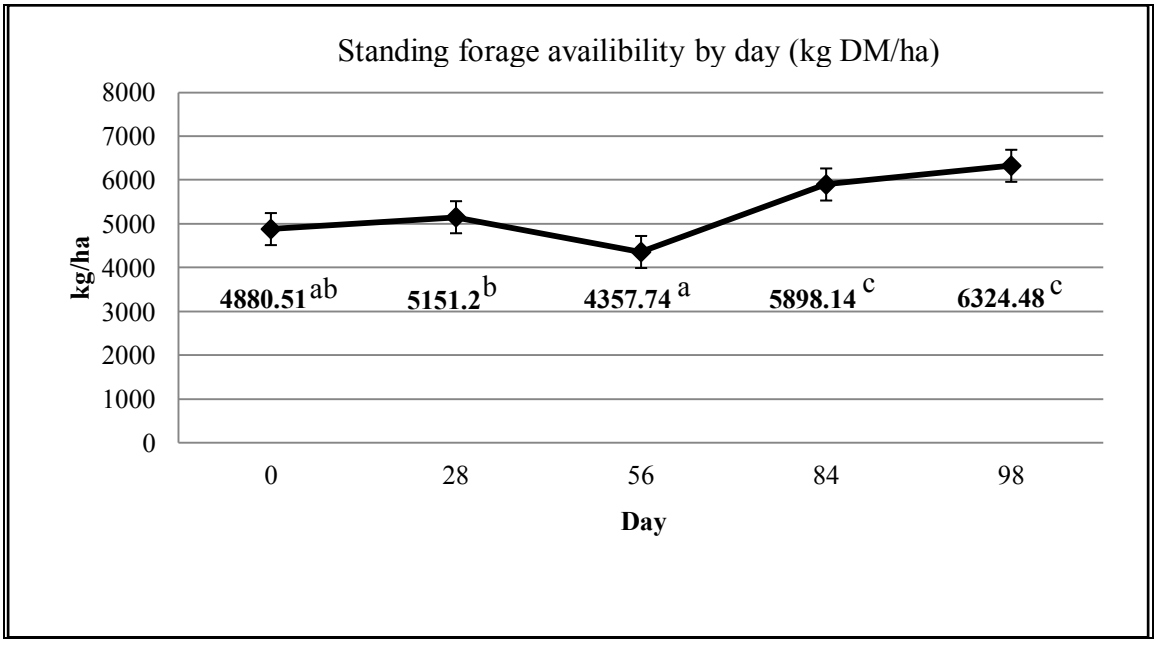

Figure 14. Standing forage (kg DM/ha) in paddocks expressed as means within collection date.

Forage mass per kg BW

Available forage per kg BW was unaffected by diet (P = 0.38) and no interaction of diet × collection day was observed (P > 0.50). Forage allowance per kg BW was highest at beginning to trial in paddocks where RUM was fed whereas forage availability increased between d 0 and 56 in paddocks assigned to CON supplement. Figure 17 depicts trend in forage availability per kg BW for both diets.

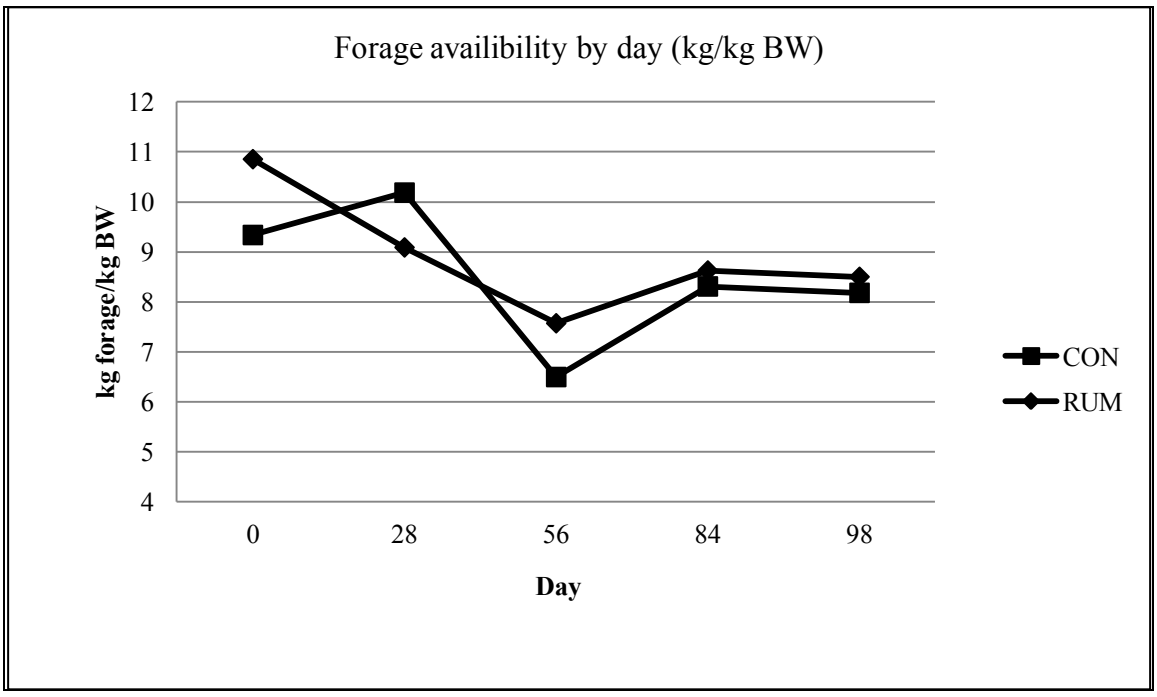

Figure 15. Standing forage mass relative to mean BW of steers by dietary treatment.

When forage availability is expressed on a DM basis per 100 kg of BW, diet tended to influence herbage mass ($P < 0.18$) whereas day ($P < 0.01$) and diet × day ($P < 0.02$) both had a significant impact on availability. Again, trend in forage mass availability from d 0 and d 28 was antithetical between dietary treatments. Figure 18 provides measures at each date. Density was sufficient to support maximum gain throughout the trial. Collectively, Pinchak et al. (1996) and Redmon et al. (1995) stated that ranges of herbage allowance to fully support ADG potential be maintained between 20.0 and 27.3 kg DM/100 kg BW. At a minimum, forage density in this trial was calculated to exceed 160.0 kg DM/100 kg BW.

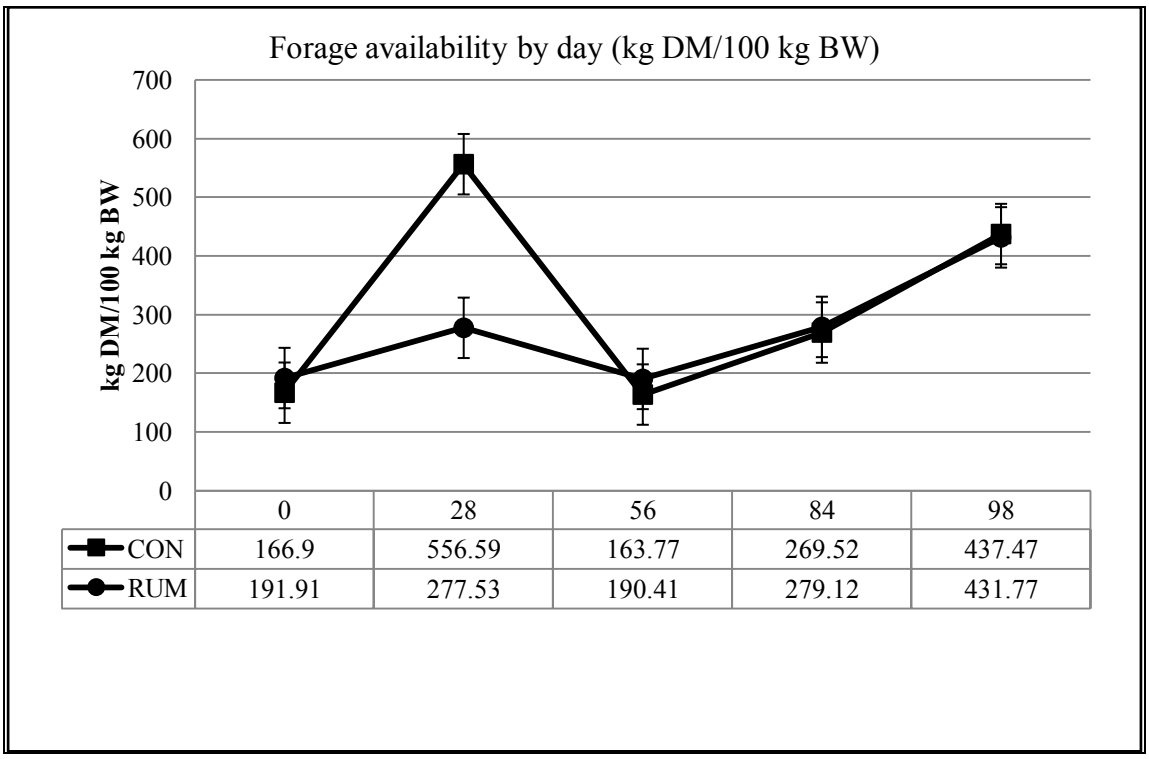

Figure 16. Available forage by diet and day expressed in kg DM/100 kg BW

Nutrient composition

A visual reference to nutrient trend is provided in figure 19. Nutrient content of paddocks are expressed as means within each collection date due an absence of diet effect or diet × day interaction for all measures (Table 31).

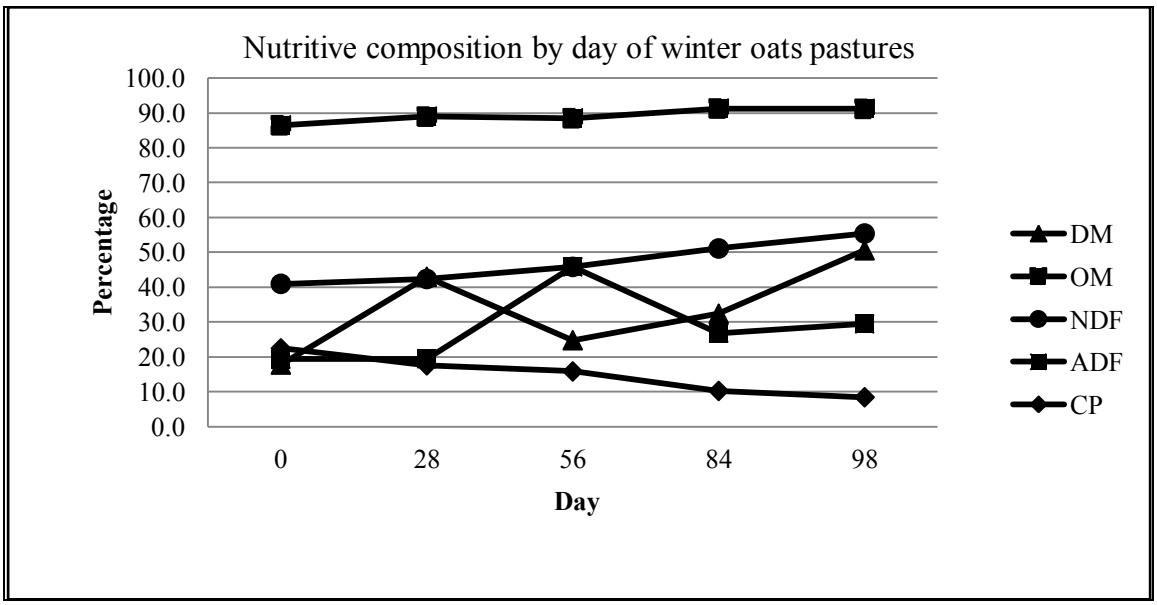

Figure 17. Nutritive values of paddock forage expressed as means within collection date.

Dry matter, OM, NDF, ADF, and CP were all affected by day ($P < 0.01$). Crude protein decreased from 22.7% to 8.4% over the course of the trial and was unaffected by diet ($P = 0.99$) or day × diet ($P = 0.79$). Proportion of fiber components increased during the study with no diet effect (P = 0.26 NDF, P = 0.28 ADF) or diet × day interaction (P = 0.40 NDF, P = 0.63 ADF).

Table 31. Nutrient composition by day of winter oat pastures

	d						*P*-value		
Item, %	0	28	56	84	98	SE	Day	Diet	Day × Diet
DM	17.9	44.9	24.6	32.3	50.7	3.28	.01	.34	.34
OM	86.3	89.0	88.5	91.3	91.2	0.35	.01	.89	.43
NDF	40.8	42.4	45.5	51.3	55.2	0.84	.01	.86	.40
ADF	19.3	19.4	23.6	27.2	29.4	0.45	.01	.28	.63
CP	22.7	17.5	15.9	10.1	8.4	0.93	.01	.99	.79

Both dietary treatments had lower ADG in the first period relative to the last period. This is contrary to expectations that ADG would decline as crude protein declines concurrent to increased neutral and acid detergent fiber levels. However, previous findings have indicated similar responses due to an initial period of reduced forage DMI associated with inability to process high levels of soluble N (Chalupa et al., 1964; Phillips, 1986; Phillips and Horn, 2008). Climatic data collected with exception of wind are provided in Table 32.

Table 32. Weather data expressed as means within collection period

	d			
Item	1 – 28	29 – 56	57 – 84	85 - 98
Min temp, ºC	2.8	7.2	13.3	15.9
Max temp, ºC	15.0	21.1	25.6	29.5
Humidity, %	65.3	63.9	63.8	59.6
Precipitation, cm[1]	7.9	0.8	1.8	0.0

[1]Precipitation expressed as total accumulation within collection period.

Conclusion

Response to an energy supplement containing monensin did not significantly influence final BW or cumulative ADG of steers grazing small grains. At the only

significant observation, supplement void of ionophore resulted in increased gains. These results are not supported in a consistent manner by previous study and are attributed here to an inadequate level of monensin-containing supplement intake. However, modeled forage intake to meet gains did indicate an increased energy effect by which forage level necessary to achieve results in the absence of supplement was reduced. Use of metaphylaxis in steers at initiation of the trial improved ADG for the first 28 days of grazing relative to untreated steers. Use of a single dose implant was effective at increasing weight gain for the entire 98 day grazing period. Combinations of the technologies did not increase performance.

LITERATURE CITED

Babcock, A. H., B. J. White, S. S. Dritz, D. U. Thomson, and D. G. Renter. 2009. Feedlot health and performance effects associated with the timing of respiratory disease treatment. J. Anim. Sci. 87:314-327.

Bailey, D. W., and D. Jensen. 2008. Method of supplementation may affect cattle grazing patterns. J. Range Manage. 61:131-135.

Barker, D. J., P. J. May, and W. M. Jones. 1988. Controlling the intake of grain supplements by cattle using urea/superphosphate. Pages 146-149 in Proc. Australian Society of Animal Production, Sydney, Australia.

Barrentine, B. F., and B. G. Ruffin. 1958. Salt and gypsum as regulators of cottonseed meal intake. Agric. Exp. Sta. Information Sheet 587:1. Mississippi State Univ., Starkeville.

Beauchemin, K. A., and S. M. McGinn. 2006. Methane emissions from beef cattle: Effects of fumaric acid, essential oil, and canola oil. J. Anim. Sci. 84:1489–1496.

Beck, P. A. 1993. Development of a self-limited monensin-containing energy supplement for growing cattle on wheat pasture. M.S. Thesis, Oklahoma State Univ., Stillwater.

Behnke, K. C. 2007. Feed manufacturing considerations for using DDGs in poultry and livestock diets. In: Proc. 5th Mid-Atlantic Nutrition Conference, College Park, MD.

Behnke, K.C., and R. S. Beyer. 2002. Effect of feed processing on broiler performance. In: Proc. 8th International Seminar on Poultry Production and Pathology. Santiago, Chile.

Belyea, R. L., B. J. Steevens, R. J. Restrepo, and A. P. Clubb. 1989. Variation in composition of by-product feeds. J. Dairy Sci. 72:2339.

Belyea, R. L., K. D. Rausch, and M. E. Tumbleson. 2004. Composition of corn and distillers' dried grains with solubles from dry grind ethanol processing. Bioresour. Technol. 94:293–298.

Bowman, J.G.P, and B. F. Sowell. 1997. Delivery method and supplement consumption by grazing ruminants: a review. J. Anim. Sci. 75:543-550.

Burrin, D. G., R. A. Stock, and R. A. Britton. 1988. Monensin level during grain adaptation and finishing performance in cattle. J. Anim. Sci. 66:513.

Carro, M. D., M. J. Ranilla, F. J. Giraldez, and A. R. Mantecon. 2006. Effects of malate on diet digestibility, microbial protein synthesis, plasma metabolites, and performance of growing lambs fed a high-concentrate diet. J. Anim. Sci. 84:405–410.

Catsaounis, N., D. Zygoyiannis, S. Kyriaks, C. Tsaltas. 1980. Deltion tes Hellenikes Kteniatrikes Hetereias (Bulletin of the Hellenic Veterinary Medical Society). 31:253-258.

Chalupa, W., J. L. Evans, and M. C. Stillions. 1964. Metabolic aspects of urea utilization by ruminant animals. J. Nutr. 84:77–82.

Chase, C. C. and C. A. Hibberd. 1987. Utilization of low-quality native grass hay by beef cows fed increasing quantities of corn grain. J. Anim. Sci. 65:557-566.

DelCurto, T. R., R. C. Cochran, L. R. Corah, A. A. Beharka, E. S. Vanzant, and D. E. Johnson. 1990. Supplementation of dormant tallgrass-prairie forage: II. Performance and forage utilization characteristics in grazing beef cattle receiving supplements of different protein concentrations. J. Anim. Sci. 68: 532-542.

Dixon, R. M., A. White, P. Fry, and J. C. Petherick. 2003. Effects of supplement type and previous experience on variability in intake of supplements by heifers. Aust. J. Agric. Res. 54:529-540.

Dove, H., and M. Freer. 1986. The use of tritiated gypsum for estimating individual feed intakes of pelleted or unpelleted supplement by lambs fed individually or in groups. Aust. J. Exp. Agric. 26:19-22.

Ducker, M. J., P. T. Kendall, R. G. Hemingway, and T. H. McClelland. 1981. An evaluation of feedblocks as a means of providing supplementary nutrients to ewes grazing upland/hill pastures. Anim. Prod. 33:51.

Edwards, A. 1996. Respiratory diseases of feedlot cattle in central USA. Bov. Pract. 30:5-7.

Foley, P. A., D. A. Kenny, J. J. Callan, T.M. Boland, and F. P. O'Mara. 2009. Effect of DL-malic acid supplementation on feed intake, methane emission, and rumen fermentation in beef cattle. J. Anim. Sci. 87:1048-1057.

Foot, J. Z., A. J. F. Russel, T. J. Maxwell, and P. Morris. 1973. Variation in intake among group-fed pregnant Scottish Blackface ewes given restricted amount of food. Anim. Prod. 17:169.

Galyean, M. L., S. A. Gunter, and K. J. Malcom-Callis. 1995. Effects of arrival medication with tilmicosin phosphate on health and performance of newly received beef cattle. J. Anim. Sci. 73:1219–1226.

Garossino, K. C., B. J. Ralston, T. A. McAllister, and M. E. Olson. 2003. Measuring individual free-choice protein supplement consumption by wintering beef cattle. Can. J. Anim. Sci. 83 (1):21-27.

Griffin, W. A., V. R. Bermer, T. J. Klopfenstein, L. A. Stalker, L. W. Lomas, J. L. Moyer, and G. E. Erickson. 2009. Summary of grazing trials using dried distillers' grains supplementation. Nebr. Beef Rep. (Lincoln) MP92:37.

Horn, G. W., M. D. Cravey, F. T. McCollum, C. A. Strasia, E. G. Krenzer, Jr., and P. L. Claypool. 1995. Influence of high-starch vs high-fiber energy supplements on performance of stocker cattle grazing wheat pasture and subsequent feedlot performance. J. Anim. Sci. 73:45-54.

Horn, G. W., T. L. Mader, S. L. Armbruster and R. R. Frahm. 1981. Effect of monensin on ruminal fermentation, forage intake and weight gains of wheat pasture stocker cattle. J. Anim. Sci. 52:447.

Huston, F. E., H. Lippke, T. D. A. Forbes, J. W. Holloway, and R. V. Machen. 1999. Effects of supplemental feeding interval on adult cows in western Texas. J. Anim. Sci. 77:3057-3067.

Jensen, J. E. 1979. Restricting intake of energy supplements fed to yearling steers grazing irrigated pasture. M.S. thesis. Univ. of Nebraska, North Platte.

Judkins, M. B., L. J. Krysl, J. D. Wallace, M. L. Galyean, K. D. Jones, and E. E. Parker. 1985. Intake and diet selection by protein supplemented grazing steers. J. Range Manage. 38:210-382.

Kendall, P. T., R. G. Hemingway, and M. J. Ducker. 1980. Variation in probable intake of ewes given concentrates with varying trough-space allowance or self-help feedblocks. Proc. Nutr. Soc. 30:16A.

Klopfenstein, T. J., L. Lomas, D. Blasi, D. C. Adams, W. H. Schacht, S. E. Morris, K. H. Gustad, M. A. Greenquist, R. N. Funston, J. C. MacDonald, and M. Epp. 2007. Summary analysis of grazing yearling response to distillers' grains. Nebraska Beef Cattle Rep. MP90:10.

Köster, H. H., R. C. Cochran, E. C. Titgemeyer, E. S. Vanzant, T. G. Nagaraja, K. K. Kreikemeier, and G. St. Jean. 1997. Effect of increasing proportion of supplemental nitrogen from urea on intake and utilization of low-quality tallgrassprairie forage by beef steers. J. Anim. Sci. 75:1393-1399.

Kreikemeier, K., G. Stokka, and T. Marston. 1996. Influence of delayed processing and mass medication with either chlortetracycline or tilmicosin phosphate on health and growth of highly stressed calves. Kansas State Univ. Southwest Res. Ext. Cent. Rep. Kansas State Univ., Manhattan.

Kuhl, G. L. 1997. Stocker cattle responses to implants. Symposium: Impact of implants on performance and carcass value of beef cattle. Okla. Agric. Exp. Sta. P-957:51-62.

Kung, L., Jr., J. T. Huber, J. D. Krummrey, L. Allison, and R. M. Cook. 1982. Influence of adding malic acid to dairy cattle rations on milk production, rumen volatile acids, digestibility, and nitrogen utilization. J. Dairy Sci. 65:1170-1174.

Kunkle, W. E., J. T. Johns, M. H. Poore, and D. B. Herd. 2000. Designing supplementation programs for beef cattle fed forage-based diets. Proc. Am. Anim. Sci., 1999. Available at: http://www.asas.org/jas/symposia/proceedings/0912.pdf. Accessed July 22, 2011.

Lane, M. A., M. H. Ralphs, J. D. Olsen, F. D. Provenza, and J. A. Pfister. 1990. Conditioned taste aversion: potential for reducing cattle losses to larkspur. J. Range Manage. 43:127-131.

Lemenager, R. P., F. N. Owens, K. S. Lusby and Robert Totusek. 1978. Monensin, forage intake and lactation of range beef cows. J. Anim. Sci. 47:247

Leupp, J. L., G. P. Lardy, K. K. Karges, M. L. Gibson, and J.S. Canton. 2009. Effects of increasing level of corn distillers' dried grains with solubles on intake, digestion, and ruminal fermentation in steers fed 70% concentrate diets. J. Anim. Sci. 87:2906–2912.

Lobato, J.F.P., and G. R. Pearce. 1980. Responses to molasses-urea blocks of grazing sheep and sheep in yards. 1980. Aust. J. Exp. Agric. Anim. Husb. 20:417-421.

Lobato, J.F.P., G. R. Pearce, and R. G. Beilharz. 1980. Effect of early familiarization with dietary supplements on the subsequent ingestion of molasses-urea blocks by sheep. App. Anim. Eth. 6:149-161.

Lodge, S. L., R. A. Stock, T. J. Klopfenstein, D. H. Shain, and D.W. Herold. 1997. Evaluation of corn and sorghum distillers' byproducts. J. Anim. Sci. 75:37–43.

Loy, T. W., J. C. MacDonald, T. J. Klopfenstein, and G. E. Erickson. 2007. Effect of distillers' grains or corn supplementation frequency on forage intake and digestibility. J. Anim. Sci. 85:2625–2630.

MacDonald, J.C., and T.J. Klopfenstein. 2004. Dried distillers' grains as a grazed forage supplement. Nebr. Beef Cattle Rep. MP 80-A: 25-27.

Martin, S. A., M. N. Streeter, D. J. Nisbet, G. M. Hill, and S. E. Williams. 1999. Effects of dl-malate on ruminal metabolism and performance of cattle fed a high-concentrate diet. J. Anim. Sci. 77:1008–1015.

McCollum, F. T. III, and G. W. Horn. 1990. Protein supplementation of grazing livestock: A review. Prof. Anim. Sci. 6:1–16.

McCollum, F. T. III. 1996. Grazing stocker cattle on small grains. Page 37 in Proc. Southwest Beef Management Symposium, Tucumcari, New Mexico.

McCourt, A. R., T. Yan, S. Mayne, and J. Wallace. 2008. Effect of dietary inclusion of encapsulated fumaric acid on methane production from grazing dairy cows. Page 64 in Proc. Br. Soc. Anim. Sci. Annu. Conf., Scarborough, UK. Br. Soc. Anim. Sci., Midlothian, Scotland.

McGinn, S. M., K. A. Beauchemin, T. Coates, and D. Colombatto. 2004. Methane emissions from beef cattle: Effects of monensin, sunflower oil, enzymes, yeast, and furmaric acid. J. Anim. Sci. 82:3346–3356.

McMurphy, C. P., E. D. Sharman, D. A. Cox, G. W. Horn, and D. L. Lalman. 2010. Effects of implant type and protein source on growth of steers grazing summer pasture. Proc. West. Sect. Am. Soc. Anim. Sci. 61:100-105.

Meyer, J. H., W. C. Weir, N. R. Ittner, and J. D. Smith. 1955. The influence of high sodium chloride intakes by fattening sheep and cattle. J. Anim. Sci. 14:412-418.

Molano, G., T. W. Knight, and H. Clark. 2008. Fumaric acid supplements have no effect on methane emissions per unit of feed intake in wether lambs. Aust. J. Exp. Agric. Sci. 48:165–168.

Montano, M. F., W. Chai, T. E. Zinn-Ware, and R. A. Zinn. 1999.Influence of malic acid supplementation on ruminal pH, lactic acid utilization, and digestive function in steers fed high concentrate finishing diets. J. Anim. Sci. 77:780–784.

Morris, S. E., J. C. MacDonald, D. C. Adams, T. J. Klopfenstein, R. L. Davis, and J. R. Teichert. 2006. Effects of supplanting dried distillers' grains to steers grazing summer sandhill range. Nebr. Beef Cattle Rep. MP 88A:30–32.

Morris, S. E., T. J. Klopfenstein, D. C. Adams, G. E. Erickson, and K. J. Vander Pol. 2005. The effects of dried distillers' grains on heifers consuming low or high-quality forage. Nebraska Beef Cattle Report. MP 83-A:18–20.

Mulholland, J. G., and J. B. Coombe. 1979. Supplementation of sheep grazing wheat stubble with urea, molasses and minerals: quality of diet, intake of supplements and animal response. Aust. J. Exp. Agric. Anim. Husb. 19:23-31.

Muller, R. D., E. L. Potter, M. I. Wray, L. F. Richardson, and H. P. Grueter. 1986. Administration of monensin in self-fed (salt limiting) dry supplements or on an alternate-day feeding schedule. J. Anim. Sci. 62:593-600.

Newbold, C. J., S. Lopez, N. Nelson, J. O. Ouda, R. J. Wallace, and A. R. Moss. 2005. Propionate precursors and other metabolic intermediates as possible alternative electron acceptors to methanogenesis in ruminal fermentation in vitro. Br. J. Nutr. 94:27–35.

Nickell, J. S. and B. J. White. 2010. Metaphylactic antimicrobial therapy for bovine respiratory disease in stocker and feedlot cattle. Veterinary Clinics of North America: Food Animal Practice. 26:285-301.

NRC. 1996. Nutrient Requirements of Beef Cattle. 7th rev. ed. Natl. Acad. Press, Washington, DC.

Oba, M., and M. S. Allen. 2003. Intraruminal infusion of propionate alters feeding behavior and decreases energy intake of lactating dairy cows. J. Nutr. 133:1094–1099.

Paisley, S. I., and G. W. Horn. 1996. Effects of monensin on intake of a self-limited energy supplement for growing steers grazing winter wheat pasture. J. Anim. Sci. 74(Suppl. 1):37.

Phillips, W. A. 1986. Adaptation of stocker calves to non-protein nitrogen diets as a result of grazing wheat forage. J. Anim. Sci. 62:464–472.

Phillips, W. A., and G. W. Horn. 2008. Intake and digestion of wheat forage by stocker calves and lambs. J. Anim. Sci. 86:2424-2429.

Pinchak, W. E., W. D. Worrall, S. P. Caldwell, L. J. Hunt, N. J. Worrall, and M. Conoly. 1996. Interrelationships of forage and steer growth dynamics on wheat pasture. J. Range Manage. 49:126–130.

Potter, E. L., R. D. Muller, M. I. Wray, L. H. Carroll, and R. M. Meyer. 1986. Effect of monensin on the performance of cattle on pasture or fed harvested forages in confinement. J. Anim. Sci. 62:583-592.

Provenza, F. D. 1995. Postingestive feedback as an elemental determinant of food preference and intake in ruminants. J. Range Manage. 48:2.

Provenza, F. D. 1996a. Acquired aversions as the basis for varied diets of ruminants foraging on rangelands. J. Anim. Sci. 74:2010.

Provenza, F. D. 1996b. A functional explanation for palatability. In N.E. West (Ed.) Proc. Fifth Int. Rangeland Cong. Soc. Range Manage., Denver, CO (In press).

Provenza, F. D., C. B. Scott, T. S. Phy, and J. J. Lynch. 1996. Preferences of sheep for foods varying in flavors and nutrients. J. Anim. Sci. 74:2355–2361.

Redmon, L. A., F. T. McCollum III, G. W. Horn, M. D. Cravey, S. A. Gunter, P. A. Beck, J. M. Mieres, andR. San Julian. 1995. Forage intake by beef steers grazing winter wheat with varied herbage allowances. J. Range Manage. 48:198–201.

Richardson, L. F., A. P. Raun, E. L. Potter, C. O. Cooley and R. P. Rathmacher. 1976. Effect of monensin on rumen fermentation *in vitro* and *in vivo*. J. Anita. Sci. 43:657.

Rosentater, K. A. 2006. Physical properties of dried distillers' grains with soluble (DDGS). In: Proc. American Society of Agricultural and Biological Engineers Annual International Meeting, Portland, OR.

Sawyer, J. E., and C. P. Mathis. 2001. Supplement delivery systems. Cooperative Extension Service – New Mexico State University. Circular 571. Available at: http://www.cahe.nmsu.edu/pubs/-circulars/Cr-571.pdf. Accessed August 15, 2011

Schauer, C. S., D. W. Bohnert, D. C. Ganskopp, C. J. Richards, and S. J. Falck. 2005. Influence of protein supplementation frequency on cows consuming low quality forage: Performance, grazing behaviour and variation in supplement intake. J. Anim. Sci. 83:1715–1725.

Schauer, C. S., G. P. Lardy, W. D. Slanger, M. L. Bauer, and K. K. Sedivec. 2004. Self-limiting supplements fed to cattle grazing native mixed-grass prairie in the northern Great Plains. J. Anim. Sci. 82:298-306.

Schelling, G. T. 1984. Monensin mode of action in the rumen. J. Anim. Sci. 58:1518–1527.

Selk, G. E., R. R. Reuter, and G. L. Kuhl. 2006. Using growth-promoting implants in stocker cattle. Veterinary Clinics of North America: Food Animal Practice. 22:435-449.

Spiehs, M. J., M. H. Whitney, and G. C. Shurson. 2002. Nutrient data for distiller's dried grains with solubles produced from new ethanol plants in Minnesota and South Dakota. J. Anim. Sci. 80:2639–2645.

Step, D. L., T. Engelken, C. Romano, B. Holland, C. Krehbiel, J. C. Johnson, W. L. Bryson, C. M. Tucker, and E. J. Robb. 2007. Evaluation of three antimicrobial regimens used as metaphylaxis in stocker calves at high risk of developing bovine respiratory disease. Vet. Ther. 8:136-147.

Stock, R., T. Klopfenstein and D. Shain. 1995. Feed intake variation. Proc. Symp: Feed Intake by Feedlot Cattle. Okla. State Univ., Stillwater. P-942:56–59.

Stock, R. A., S. B. Laudert, W. W. Stroup, E. M. Larson, J. C. Parrott, and R. A. Britton. 1995. Effect of monensin and monensin tylosin combination of feed intake variation of feedlot steers. J. Anim. Sci. 73:39-44.

Tait, R. M., and L. J. Fisher. 1996. Variability in individual animal's intake of minerals offered free-choice to grazing ruminants. Anim. Feed Sci. and Tech. 62:69-76.

Taylor, N., P. G. Hatfield, B. F. Sowell, J.G.P. Bowman, J. S. Drouillard, and D. V. Dhuyvetter. 2002. Pellet and block supplements for grazing ewes. Anim. Feed Sci. and Tech. 96:193-201.

Utley, P. R., G. L. Newton, R. J. Ritter III and W. C. McCormick. 1976. Effects of feeding monensin in combination with zeranol and testosterone estradiol implants for growing and finishing heifers. J. Anim. Sci. 42:754.

Wallace, L. R., and O. F. Parker. 1966. Comparison of three systems of feeding silage after calving. Pages 148–158 in Proc. Ruakura Farmers' Conference, Ruakura, New Zealand.

Wallace, R. J., T. A. Wood, A. Rowe, J. Price, D. R. Yanez, S.P. Williams, and C. J. Newbold. 2006. Encapsulated fumaric acid as a means of decreasing ruminal methane emissions. Int. Congr. Ser. 1293:148–151.

Waller, J., T. Klopfenstein, and M. Poos. 1980. Distillers' feeds as protein sources for growing ruminants. J. Anim. Sci. 51:1154.

Wang, C., Q. Liu, W. Z. Yang, Q. Dong, X. M. Yang, D. C. He, K. H. Dong, and Y. X. Huang. 2009. Effects of malic acid on feed intake, milk yield, milk components and metabolites in early lactation Holstein dairy cows. Livest. Prod. Sci. 124:182-188

Waterman, R. C., J. E. Sawyer, C. P. Mathis, D. E. Hawkins, G. B. Donart, and M. K. Petersen. 2006. Effects of supplements that contain increasing amounts of metabolizable protein with or without Ca-propionate salt on postpartum interval and nutrient partitioning in young beef cows. J. Anim. Sci. 84:433–446.

Wileman, B. W., D. U. Thomson, C. D. Reinhardt, and D. G. Renter. 2009. Analysis of modern technologies commonly used in beef cattle production: Conventional beef production versus nonconventional production using meta-analysis. J. Anim Sci. 87:3418-3426.

Wise, M. B., E. R. Barrick, and T. N. Blumer. 1965. Finishing steers with grain on pasture. Agric. Exp. Sta. Bull. 425. North Carolina Stat