# EFFECTS OF SODIUM BICARBONATE ON REDUCING ACIDOSIS IN CATTLE

by

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B.Sc., The University of Alberta, 1998

A THESIS SUBMITTED IN PARTIAL FULFILMENT OF
THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE

in

THE FACULTY OF GRADUATE STUDIES

(ANIMAL SCIENCE)

THE UNIVERSITY OF BRITISH COLUMBIA

July 2005

## ABSTRACT

A study was conducted to determine whether feeding sodium bicarbonate (SB) reduces the risk of subacute acidosis (SARA) in cattle receiving high concentrate diets. Twelve Hereford cross heifers (Exp.1) and six ruminally cannulated animals (Exp. 2; three Jersey steers and three Holstein cows), previously adapted to a high concentrate diet, were used in 3 × 3 Latin square designs to study the effects of SB on feed intake, ruminal pH, ruminal fermentation and blood characteristics. Animals were provided ad libitum access to a control diet containing steam-rolled barley, barley silage, and a supplement at 80, 12, and 8% (Exp. 1) and 81.2, 12.0 and 6.8% (Exp. 2) (DM basis), respectively. Treatments were: control, control with free choice access to a SB mixture offered as 30% dried molasses and 70% SB (free choice SB) and control diet supplemented with 0.7% SB (DM basis) (mix SB). Periods consisted of 21 d (Exp. 1) or 14 d with 11 d adaptation and 3 d of continuous ruminal pH measurements using indwelling electrodes (Exp. 2). Mean dry matter intake for Exp. 1 and 2 was not affected by treatment. Sodium bicarbonate intake differed when provided free choice verses when mixed into the diet ($P < 0.0001$) in Exp. 1 (2.1 vs. 55.3 g/d) and cows (17.4 vs. 57.8 g/d) and steers (129.1 vs. 56.1 g/d) in Exp. 2, respectively. Intakes of SB also tended to differ amongst animals ($P < 0.07$) in Exp. 1 and 2. Treatment had no affect on ruminal volatile fatty acids or blood variables (Exp. 2). Ruminal pH characteristics (mean, minimum, hours and area under a respective threshold pH < 5.8 or 5.5) were not affected by treatment (Exp. 2). Although neither form of SB supplementation eliminated SARA, the duration of bouts of severe SARA (pH < 5.5) tended ($P < 0.11$) to be reduced for animals fed free choice SB and mix SB compared to control. Of the two approaches of delivering SB, animals consuming free choice SB had shorter bouts of SARA ($P < 0.05$) and a tendency towards fewer of these bouts becoming severe compared to those animals consuming mix SB ($P = 0.09$). However, the intake of SB offered free choice was highly

variable, and even when intake of SB exceeded the recommended level, there was no correlation between SB intake and ruminal pH, indicating that other factors such as feed intake, primarily influenced ruminal pH. In conclusion, providing SB did not eliminate acidosis in cattle fed high grain diets, but offering SB free choice may have small, positive effects on reducing severity of acidosis.

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| | |
|---|---|
| **DMI** | Dry matter intake |
| **SB** | Sodium bicarbonate |
| **CP** | Crude protein |
| **SEM** | Standard error of the mean |
| **SD** | Standard deviation |
| **SARA** | Subacute ruminal acidosis |
| **VFA** | Volatile fatty acids |
| **BW** | Body weight |
| **TMR** | Total mixed ration |
| **pef** | Physically effective factor |
| **peNDF** | Physically effective fiber |
| **DM** | Dry matter |
| **OM** | Organic matter |
| **NDF** | Neutral detergent fiber |
| **ADF** | Acid detergent fiber |
| **DIFF** | Difference |
| **VAR** | Variation |
| **LDH** | Lactate dehydrogenase |
| **PCV** | Packed cell volume |
| **RFC** | Rapidly fermentable carbohydrates |
| **PEM** | Polioencephalomalacia |
| **ADG** | Average daily gain |
| **ME** | Metabolizable energy |

## ACKNOWLEDGEMENTS

I gratefully acknowledge the support of staff at the Agricultre and Agri-Food Canada Research Centers located in Kamloops, B.C. and Lethbridge, AB, in particular Bev Farr, Rena Wuerful and Lavona Liggins. I would like to thank the members of my supervisory committee at the UBC Animal Welfare Program, Drs. Marina von Keyserlingk and Dan Weary for their support, guidance, and encouragement. I would also like to thank Dr. Douglas Veira from Agriculture and Agri-Food Canada for all of his helpful comments and much appreciated feedback throughout this project. I am especially grateful to Dr. Karen Beauchemin from Agriculture and Agri-Food Canada for her patience, support, and insight during this thesis project. Financial support was provided by the British Columbia Cattlemen's Association and through a University of British Columbia Graduate Fellowship.

I owe great thanks to many of my fellow students in the Animal Welfare Program, in particular, Trevor deVries and Sophie Neveux for their help with the statistical analysis. I recognize team Point 05 big time for fighting for the truth and Nuni, Nuni, and Nuni for their assistance. I thank my family for their never-ending effort in trying to understand what my project was about. Lastly and most importantly, I thank my husband Eric Paton for keeping it real and of course my four legged friends, Henry, Rosie and particularly Chelsea, who will always make me smile.

## CO-AUTHORSHIP STATEMENT

The study was designed collaboratively by Laura Paton and Drs. Marina von Keyserlingk, Doug Veira and Karen Beauchemin. Laura Paton analyzed all data and prepared the manuscript under the guidance of Drs. Beauchemin, Veira, von Keyserlingk, and Weary.

# CHAPTER I - GENERAL INTRODUCTION

## Beef Feedlot Cattle and Acidosis

Cattle evolved as grazing animals consuming mainly forage based diets. However, in the past fifty years, the economics of the feedlot industry has favored finishing beef cattle on high grain diets, typically as high as 95% of the total mixed ration (TMR) (dry matter basis), in order to maximize cattle performance and ultimately profitability (Wheeler, 1980; Russell and Rychlik, 2001; Castillo et al., 2004). Providing a high energy diet that is economical due to low grain prices, combined with the ease of handling and mixing of grains in diets, as well as consumer demand for grain-fed beef has made feeding cattle grain diets a profitable alternative to feeding bulky, lower energy forages (Stock and Britton, 1993). However, the digestion of grains by ruminant animals creates distinct challenges since the ruminal ecosystem is susceptible to metabolic disturbances, which can lead to acidosis (Stock and Britton, 1993).

Acidosis is defined as a decrease in the alkali content (base exchange) in body fluids relative to the acid (hydrogen ion) content (Stedman, 1982 as cited in Owens et al., 1998). In ruminants, acidosis is defined as the biochemical and physiological stresses caused by rapid production and absorption of ruminal organic acids (volatile fatty acids (VFA) and lactic acid) that arise from the over consumption of readily fermentable carbohydrates (RFC) (Britton and Stock, 1986).

In North America, the feeding of cereal grains is the most common RFC source contributing to acidosis. Cereal grains vary in their starch fermentability and potential to cause acidosis (Dunlop, 1972; Huntington, 1988). For example, wheat, barley and high moisture corn are highly fermentable and therefore diets based on these ingredients are conducive to acidosis, while oats and sorghum grain based diets are less fermentable and the least likely to cause acidosis (Radostits, 1994). Heat and pressure processing of RFC such as steam flaking, rolling or popping, particle size reduction and high moisture storage can increase starch availability and

fermentability, which in turn increases the propensity for acidosis (Huntington, 1988; Radostits, 1994; Owens et al., 1998).

Ruminal acidosis is not one disease, but rather a continuum, with degree of severity often separated into acute and the more economically important, chronic or subacute ruminal acidosis (SARA) (Slyter, 1976; Britton and Stock, 1986).

**Acute Acidosis**

Cattle exhibit acute acidosis as an overt illness that commonly occurs when cattle are quickly transitioned from a forage diet to a concentrate based diet, at which time they are vulnerable to rapid overeating or over ingestion of grains (Owens et al., 1998). Feedlot cattle are typically adapted to high grain diets over a 2 to 4 week period using a series of transition diets that increase in proportion of grain (Owens et al., 1998). Acute acidosis symptoms are easily recognized by feedlot managers and can include bloat, depressed or listless appearance, founder, laminitis, cessation or abrupt reduction of feed intake and potentially polioencephalomalacia (PEM), which occurs from thiamine deficiency (Tremere et al, 1968; Koers et al. 1976; Owens et al., 1998). Cattle with PEM often wander aimlessly, stagger, or cannot stand and often give the impression of having "brain damage" (Stock and Britton, 2002; Stock and Britton, 1993). Other responses to rapid ingestion of high grain diets include a dramatic reduction of ruminal pH (5.2 or less) (Cooper and Klopfenstein, 1996), increased concentration of VFA and lactic acid in the rumen (Huntington, 1988) and a significant decline in total protozoa (Hristov et al., 2001).

In severely acute cases, death may occur within 24 to 72 hours following grain engorgement (Glock and DeGroot, 1998). Vogel and Parrot (1993) attributed 24% of mortalities in the Great Plains feedyards over a seven-year period to digestive disturbances, including acidosis. Radostits (1994) suggested that depending on the type of grain, the total amount of grain eaten and the previous experience of the animal, mortality rates in feedlot cattle are as high as 90%. Even when acidosis is treated, mortality rates can still range from 30-40% (Radostits,

1994). Fortunately, cattle that are acutely acidotic are readily identified and can be treated with proper management (Stock and Britton, 1993)

**Subacute Ruminal Acidosis (SARA)**

Most beef feedlot cattle typically experience SARA, which is much more difficult to identify than acute acidosis (Britton and Stock, 1986). Cattle exhibiting SARA commonly have a ruminal pH ranging from 5.8 to 5.2 (Ghorbani et al., 2002; Cooper and Klopfenstein, 1996) and reduced or variable feed intake accompanied by reduced performance (Nocek, 1997; Owens et al., 1998). Detecting animals with SARA is difficult as animals may not appear sick as with acute acidosis and often resume eating within a few days. In large feedlots where cattle are kept in pens of 100 or more, individuals with SARA may go unnoticed as feedlot managers monitor intake on a pen basis rather than on an individual basis (Huntington, 1988; Cooper and Klopfenstein, 1996; Owens et al., 1998). Thus, it is important for feedlot managers to be observant of secondary SARA associated symptoms including panting, lethargy, teeth grinding, eating dirt, diarrhea and belly kicking (Stock and Britton, 2002; Braun et al., 1992; Stock and Britton, 1993).

**Variable Animal Responses to Consuming High Grain Diets**

Individual animal responses to consumption of high concentrate diets vary dramatically. Some animals are resilient to these diets and maintain productivity while others become ill or die (Slyter, 1976, Schwartzkopf-Genswein et al., 2003). The type and amount of concentrate in the diet contributes to an animal's vulnerability to acidosis and degree of severity (Slyter, 1970; Slyter, 1976; Radostits, 1994). Moreover, individual animal differences including species, body condition and overall animal health, salivation rate, the amount of feed the animal is accustomed to eating and rate of ingestion, and the pre-existing microbial population will also contribute to an animal's response (Slyter, 1976; Radostits, 1994).

Unfortunately, evidence of an acidotic episode may only become apparent at slaughter, when abscessed livers reveal the damaging effects of acidosis (Huntington, 1988). The incidence of liver abscesses in cattle fed grain based diets can range from 1 or 2% to as high as 90 or 95% (Nagaraja and Chengappa, 1998). Brink et al. (1990) determined that liver abscesses were present in 161 of 566 (28%) of feedlot cattle while Kreikemeier et al. (1990) found higher incidences of condemned livers (54 to 71%) in steers fed steam rolled wheat based diets for 120 days. Cattle with hundreds of abscesses will seldom exhibit clinical signs, however, reduced feed intake and weight gain, decreased feed efficiency and lowered carcass dressing percentage have been observed in response to liver abscesses (Brink et al., 1990; Nagaraja and Chengappa, 1998).

**Economics of Acidosis**

Acidosis is the greatest nutritional problem for feedlot producers and is linked to substantial economic losses arising directly from the treatment of liver abscesses, lameness, morbidity, and indirectly from secondary associated symptoms including variable feed intake and reduced performance (Britton and Stock, 1993; Nocek, 1997; Owens et al., 1998). Stock and Britton (1993) estimated that feeding cattle a dry rolled wheat diet that resulted in SARA had an associated cost of approximately $9.40 per steer due to reduced feed intake, gain and efficiency. Schwartzkopf-Genswein et al. (2003) estimated that erratic feeding behavior and reduced performance resulting from SARA incurs a loss of $15 to $20 per animal. Stock and Britton (2002) estimated that liver abscesses could potentially reduce daily gain by 11 percent and feed efficiency by 9 percent, culminating in a total loss of $3 per head. Liver condemnation also results in significant economic losses to producers and meat packers and translates into lower market prices (Nagaraja and Chengappa, 1998). Thus, it is important for feedlot producers to have a reliable treatment for acidosis or to implement a prevention strategy in order to curb production losses related to acidosis.

## Acidosis: A Brief Etiology

There are two major phases involved in the etiology of acidosis: 1) abrupt increase in the ingestion of RFC accompanied by altered ruminal microbial population profile and subsequent accelerated ruminal fermentation to acids and, 2) absorption of acids into the blood stream leading to systemic and metabolic acidosis (Huntington, 1988).

### Phase one: Ingestion of RFC, ruminal fermentation to acids and alteration of the ruminal microbial population profile

In forage fed animals, ruminal pH is generally near neutrality (pH 7.0) and is well buffered by bicarbonate and phosphate from saliva, proteins, forage cell walls and VFA (Wheeler, 1980; Russell and Hino, 1985). Normally, when grain is added to ruminant diets, ruminal protozoa adapt by increasing their numbers and adequately removing the additional starch (Mackie et al., 1978). However, if the grain levels ingested exceed protozoal capacity to remove starch, protozoa populations will decline (Hristov et al., 2001), a drastic increase in bacterial counts will occur, fermentation to VFA and lactic acid will proceed and ruminal pH will decline (Mackie et al., 1978).

Excess starch in the rumen causes glucose fermenting bacteria to proliferate and liberate glucose from starch granules (Radostits, 1994; Owens et al., 1998). Normally, ruminal glucose concentrations are extremely low; however, following grain engorgement of cattle, glucose concentrations have been reported to exceed 160 mg/100ml (Horn et al., 1979). Amylolytic or sugar-using bacteria, such as Streptoccus bovis compete for free glucose substrate in the rumen and proliferate, producing acetate, formate and low concentrations of L (+) lactate as fermentation end products which lowers ruminal pH below 6.0 (Mackie and Gilchrist, 1979; Schwartzkopf-Genswein et al., 2003). Below pH 6.0, further declines in populations of ruminal protozoa (Radostits, 1994; Owens et al., 1998; Goad et al., 1998), fibrolytic (Braun et al., 1992; Radostits, 1994, Calsamiglia et al., 2002) and cellulolytic bacteria (Kreikemeier et al., 1990;

Radostits, 1994; Russell and Wilson, 1996) occur due to the presence of volatile fatty acids and lack of dietary fiber. Despite the production of lactate in the rumen from S. bovis, lactate accumulation is normally prevented by lactate utilizing protozoa and bacteria such as Selenomonas spp and Megasphaera elsdenii that metabolize the lactic acid. Thus, a balance is achieved between lactate producing and lactate utilizing microbes (Mackie and Gilchrist, 1979; Schwartzkopf-Genswein et al., 2003).

Acidosis can become problematic when RFC are consumed rapidly or at high levels. The availability of starch substrate results in unrestricted growth of S. bovis and increased L (+) lactate production causing further declines in rumen pH (Russell and Hino, 1985). As the ruminal environment becomes increasingly acidic and approaches pH 5.0, growth of S. bovis gradually declines (Slyter, 1976; Russell and Hino, 1985). A niche is created for other lactate producing bacterial populations, such as Lactobacillus spp., ultimately reducing ruminal pH below 5.0 and leading to systemic and metabolic acidosis (Braun et al., 1992; Radostits, 1994; Owens et al., 1998).

Normally, ruminal organic acids are passed with ingesta from the rumen through the omasal orifice, provide substrate for lactolytic bacteria or are absorbed across the rumen wall (Huntington, 1988; Allen, 1997). If the compensatory mechanisms of the body (plasma bicarbonate buffering system) are able to maintain blood pH so that the entry of organic acids into the body fluids is not too rapid, cattle will be ill for a few days but will recover, as with SARA (Radostitis, 1994). However, if acid entry into the body fluids is too rapid as occurs with acute acidosis, the compensatory mechanisms become exhausted and systemic acidosis is likely to preside (Radostits, 1994; Goad et al., 1998).

**Phase two: Systemic Acidosis**

The systemic impact of acidosis can have several physiological implications including decreased ruminal motility and complete ruminal stasis occurring at pH of 5.0 or less (Huber,

1976; Radostits, 1994), hyperkeratosis (thickened intestinal tract tissue) (Krehbiel et al., 1995), liver abscesses (Nagaraja and Chengappa, 1998), rumenitis and laminitis (Nocek, 1997).

During acute acidosis, blood acidosis occurs in response to the highly acidic ruminal environment resulting from excessive production or insufficient removal of acids that subsequently results in high rumen osmolarity (Owens et al., 1998). The high ruminal osmotic pressure causes water to be rapidly drawn from the blood inward through the rumen wall in attempts to neutralize the osmotic pressure and increase ruminal pH by removing hydrogen ions (Allen, 1997; Owens et al., 1998). The loss of fluid in the plasma and the presence of acids in the bloodstream results in increased blood osmolality, lactate concentrations and packed cell volume (PCV) or hematocrit from dehydration (Huntington and Britton, 1979; Owens et al., 1998; Goad et al., 1998). High ruminal osmolality also reduces the concentration of bicarbonate in the blood and reduces plasma and serum minerals, in particular Ca, often resulting in increased drinking behavior (Huntington and Britton, 1979; Owens et al., 1998; Goad et al., 1998). Moreover, the influx of water into the rumen can cause the ruminal papillae to swell and eventually, the internal surface layers of the rumen wall can be stripped and patches of rumen epithelium are pulled into the gasrtointestinal tract, resulting in abscesses in both the rumen and small intestine (Owens et al., 1998).

The high osmolality of ruminal contents lowers the rate of absorption of acids and contributes to acid accumulation in the rumen (Tabaru et al., 1990 as cited by Owens et al., 1998). Acid accumulation in the rumen can provoke tissue inflammation in the small intestine and reduce blood flow to the gastro-intestinal tract thereby reducing the absorption of all organic acids from the rumen (Stock and Britton, 1993). Absorption of VFA may be inhibited for months or years following the initial damage as repaired tissues of the digestive tract will be thickened due to prolonged exposure to the high acid concentrations (hyperkeratosis or parakeratosis), which could potentially culminate in reduced average daily gain (ADG) and feed efficiency in

ruminants due to loss of total metabolizable energy (ME) normally supplied by VFA absorption (Krehbiel et al., 1995). Also, the removal of hydrogen ions in the rumen by VFA absorption is dramatically reduced (Allen, 1997).

The damaged ruminal wall allows for the potential systemic invasion of lactate utilizing bacteria such as Fusobacterium necrophorus, which are primarily responsible for liver abscesses (Nagaraja and Chengappa, 1998). As well, alteration of intestinal bacterial populations can occur, with proliferation of coliforms and clostridial species that may be associated with the production of enterotoxins contributing to diarrhea and loss of electrolytes and other toxic factors such as histamine, tyramine, tryptamine, ethnol and endotoxins (Allison et al., 1975; Huntington, 1988). In severe cases, acidosis can interfere with renal function and oxygen transport and may result in the rupturing of peripheral arterioles, particularly in the extremities resulting in laminitis or founder (Huber, 1976; Huntington, 1988; Nocek, 1997).

**Systemic Acidosis and SARA**

Changes in systemic acid-base status are minimal during SARA (Horn et al., 1979; Goad et al., 1998; Brown et al., 2000). However, SARA can still lead to tissue damage, unstable microbial and bacterial populations and reduced protozoal numbers (Huntington, 1988). Prolonged effects can occur from one single bout of acidosis, attributing to cattle being "chronics" or "poor-doers", where they have reduced feed intake, weight gain and feed efficiency (Huntington, 1988, Stock and Britton, 1993; Krehbiel et al., 1995).

**Induced Acidosis in Metabolism Research Trials**

Scientists have attempted to mimic acidosis to study potential solutions to this disease. To imitate SARA, researchers usually abruptly replace a portion of the forage component of the TMR with a grain source and compare the response to the control diet (original forage based TMR). Acute acidosis may be induced by withholding a high concentrate diet for 12 to 24 h and then allowing the animal unrestricted access thereby resulting in over ingestion of the RFC due

to hunger (Owens et al., 1998). Metabolism research trials often define acidosis severity in terms of DMI and ruminal pH. These types of studies often report several physiological parameters such as ruminal fermentation and osmolality and blood pH, acid/base status, metabolites and osmolarity. These measures are discussed below as they pertain to feedlot cattle exhibiting SARA conditions, as this was the focus of the research undertaken for this thesis.

**Feed Intake**

Feed intake patterns of cattle consuming primarily forage diets are governed by ruminal fill or a combination of ruminal fill and chemostats (energy sensing mechanisms coordinated in the brain) (Stock and Britton, 1993). However, when cattle are transitioned to and maintained on high grain diets, ruminal microbes must adapt to varying substrates and feed intake control mechanisms are governed primarily by chemostatic regulation (Stock and Britton, 1993). This causes instability in the rumen resulting in varying degrees of acidosis. Cattle will compensate for low ruminal pH by adjusting daily feed intake as well as their intake patterns, often resulting in reduced voluntary food intake (Bergen, 1972; Stock and Britton, 1993; Phy and Provenza, 1998; Beauchemin, K. A., unpublished data as cited by Schwartzkopf – Genswein et al., 2003).

Erratic feed intake patterns, described as cyclical or "yo-yoing", have been used as a primary indicator of cattle experiencing SARA (Galyean et al., 1992; Stock and Britton, 1993; Cooper et al., 1999). Intake patterns of cattle suffering from SARA were characterized in a study by Fulton et al. (1979a) where cattle were adapted in a step-wise manner to diets of either dry-rolled corn, which is more slowly fermented in the rumen, or highly fermentable wheat. Observations indicated that at each inclusion rate of wheat in the diet, fluctuations in intake were observed. Moreover, cattle developed a cyclic feeding pattern such that when they over ate and thus experienced acidosis, they subsequently reduced their intake for a few days supposedly to recover after which they would begin the cycle again by over eating. These researchers also observed that these cattle ate several smaller meals throughout the day rather than eating a few

large meals, thus partitioning their feed over a longer period of time and reducing the acid load in the rumen. In contrast, animals consuming the corn diet only became problematic at the 90% level at which time they exhibited the same cyclical feeding pattern as was observed for cattle consuming wheat (Fulton et al. 1979a).

The cyclical feeding patterns adopted by cattle during SARA have been attributed to large amounts of VFA in the rumen, as lactate is present at very low levels during SARA (Koers et al., 1976; Slyter, 1976; Owens et al., 1998). Koers et al. (1976) and Tremere et al. (1968) reported that sheep and dairy heifers, respectively, were acutely acidotic as indicated by high ruminal lactic acid levels and low ruminal pH that caused animals in both studies to go off feed. However just prior to going off feed, VFA concentrations were highest, lactate levels were low and dairy heifers continued to feed (Tremere et al., 1968). This suggests that lactic acid is a strong inhibitor of feed intake and as long as lactic acid does not accumulate, cattle with SARA will continue to feed and regulate acid load by adopting a cyclic feeding pattern (Britton and Stock, 1986).

Variable feed intake has been negatively correlated with animal performance in some studies (Galyean et al., 1992; Cooper et al., 1999), but not in others (Zinn, 1994; Soto-Navarro et al., 2000; Hickman, et al., 2002; Schwartzkopf – Genswein et al., 2004). Further, imposed day-to-day feed delivery variations have been shown to increase the risk of SARA in a study by Cooper et al. (1999), but not in the study by Schwartzkopf – Genswein et al. (2004). Discrepancies also exist as to whether feed intake variation causes SARA or SARA causes feed intake variation (Cooper and Klopfenstein, 1996; Cooper et al., 1999).

**Physiological measures**

**Ruminal pH**

Clinical diagnosis of acidosis severity uses ruminal pH thresholds of 5.8 or less for SARA and 5.2 or less for acute acidosis (Cooper and Klopfenstein, 1996; Owens et al., 1998;

Ghorbani et al., 2002). However, ruminal pH thresholds should be used as general guidelines as the actual ruminal pH where SARA becomes acute is difficult to determine as cattle vary in their ability to cope with dietary changes that induce acidosis and characterization of acidosis severity should be based more on symptoms (Stock and Britton, 1993, Schwartzkopf – Genswein et al., 2003).

Accumulation of VFA in the rumen, rather than lactic acid, is responsible for the decline in ruminal pH during SARA. Lactic acid is present in the rumen at very low concentrations during SARA (Mackie and Gilchrist, 1979; Britton and Stock, 1986; Owens et al., 1998; Ghorbani et al., 2002). Burrin and Britton (1986) reported steers fed a high moisture corn diet had gradual increases in VFA concentrations to levels of 130 mM, which were highly correlated with reductions in ruminal pH. Similarly, Soto-Navarro et al. (2000) reported steers fed once per day had highest VFA concentrations 9-h after feeding which corresponded with lowest ruminal pH. Therefore, when determining declines in ruminal pH, total acid load and not just lactic acid should be considered, particularly with SARA (Slyter, 1976; Mackie et al., 1978; Huntington, 1988, Goad et al., 1998; Owens et al., 1998).

Mean ruminal pH for cattle fed high-grain diets usually ranges from 5.6 to 6.2 (Schwartzkopf – Genswein et al., 2003). For instance, Beauchemin et al. (2001) reported mean ruminal pH values that ranged from 5.79 to 6.06 and minimum ruminal pH values ranging from 5.21 to 5.66 for cattle fed high grain barley diets depending on degree of grain processing. Similar ruminal pH values for cattle fed barley based diets were reported by Ghorbani et al. (2002), where mean pH for control fed steers was 5.72 and minimum pH was 5.14. Most feedlot cattle experience wide ranges in pH within a day with lowest ruminal pH (nadir) occurring 8 to 16-h after feeding (Keunen et al., 2002; Ghorbani et al., 2002; Schwartzkopf – Genswein et al., 2003). Numerous researchers have reported that cattle fed in the morning exhibit ruminal pH profiles that typically decreased gradually after feeding, then increased or recovered overnight

and peaked prior to the next feeding (e.g. Cooper and Klopfenstein, 1996; Beauchemin et al., 2001; Nocek et al., 2002; Keunen et al., 2002; Ghorbani et al., 2002; Schwartzkopf – Genswein et al., 2004). As well, Nocek et al. (2002) reported that the degree and rate of ruminal pH decline after feeding and the duration of lowest pH increased accordingly as the percentage of grain in the diet increased. Therefore, reporting the average daily ruminal pH does not adequately reflect the diurnal fluctuations in ruminal pH (Nocek, 1997, Schwartzkopf – Genswein et al., 2003).

Mackie and Gilchrist (1979) suggested using time that pH was below the optimal ruminal pH (they used pH 6.0) and the magnitude of the deviation from this optimal pH or area (unit of time × pH units below pH thresholds) as an index of variation in ruminal pH. Measuring area and time below particular ruminal pH thresholds provides a clearer indication of acidosis severity. For example, Cooper and Klopfenstein (1996) reported for steers adapted to a finisher diet of 82% dry rolled corn (DM basis) and fed ad libitum, area below pH 5.6 averaged 2.3 pH units × h for the first five days and decreased further for the second five days to 4.4 pH units × h. This provides a better description of pH patterns rather than solely reporting mean values. Similarly, Ghorbani et al. (2002) summarized ruminal pH data of steers fed a barley diet using pH thresholds of 5.8 (mild SARA) and 5.5 (severe SARA). In that study, time and area below pH 5.8 averaged 11.75 h and 6.46 pH units × h, respectively and time and area below pH 5.5 averaged 8.56 h and 3.45 pH units × h, respectively.

Fluctuations or ranges in rumen pH as an indication of acidosis severity have previously been quantified. For example, Cooper and Klopfenstein (1996) used the daily variance of ruminal pH (pHVAR) and the daily magnitude of ruminal pH change called pHDIFF, calculated as the maximum ruminal pH minus minimum ruminal pH per day per animal to reflect ruminal pH fluctuations with varying treatments. Similarly, Hristov et al. (2001) reported that pH variability was more pronounced in steers fed a high barley diet verses a medium barley diet over 30 d as evidenced by the range in ruminal pH.

Ruminal pH must be depressed for prolonged periods of time in order for SARA effects to occur (Plaizer et al., 1999; Keunen et al., 2002). Ruminal pH that is depressed for a brief period of time below optimal ruminal pH (pH 6.3) is unlikely to affect the health of the animal (de Veth and Kolver, 2001). The duration that ruminal pH must be depressed below pH thresholds before negative effects are demonstrated is unclear (Nocek, 1997). Calsamiglia et al. (2002) suggested that when rumen pH was decreased below pH 6.0 for less than 4 h, the effects on rumen fermentation were negligible. However, the range that defines "below pH 6.0" is unclear and it is uncertain if the pH were lower, for example 5.5, whether the time required to cause negative impacts on ruminal fermentation would be less than 4 h.

In acidosis studies, the effects of SARA on ruminal pH are usually presented as the total time ruminal pH was depressed below a chosen pH threshold in a 24 h period (Cooper and Klopfenstein, 1996; Ghorbani et al., 2002). For example, Schwartzkopf – Genswein et al. (2003) considered SARA to exist when ruminal pH fell below 5.8 for greater than a total of 12 h/d. Plaizer et al. (1999) reported dairy cows experienced SARA when ruminal pH was reduced below pH 6.0 and 5.6 for a substantial part of the day (5 and 1 h, respectively). Although these studies give an indication of the effects of varying treatments on SARA in terms of time and area below designated ruminal pH thresholds in a 24 h period, a clearer understanding of the duration that would typify a single episode or bout of SARA within a 24 h period is required.

Correlations between DMI and mean rumen pH or lowest daily ruminal pH have been identified, where DMI is reduced in response to low or lowest mean ruminal pH of the previous day (Fulton et al., 1979ab; K. A. Beauchemin, unpublished data as cited by Schwartzkopf – Genswein et al., 2003; Brown et al., 2000). Schwartzkopf – Genswein et al. (2004) reported that daily ruminal pH and daily DMI were moderately negatively correlated ($r = 0.57$) indicating that reduced feed intake was associated with increased ruminal pH because less substrate was in the

rumen for fermentation. In contrast, Ghorbani et al. (2002) found that day-to-day variations in ruminal pH did not correspond directly to changes in daily DMI.

**Blood: Acid/Base Status and Metabolites**

Clinical diagnosis of acidosis relies on blood pH levels equal to or less than 7.35 (Owens et al., 1998). Changes in systemic acid-base status are minimal during SARA (Horn et al., 1979; Goad et al., 1998; Brown et al., 2000). However, when ruminal lactate accumulation occurs, there is an elevated risk of metabolic acidosis whereby plasma lactate concentrations rise and blood pH is lowered (< 7.35) (Telle and Preston, 1971; Burrin and Britton 1986; Krehbiel et al., 1995; Owens et al., 1998; Brown et al., 2000). As well, concentrations of blood bicarbonate are reduced and hematocrit increases due to the efflux of water from the blood into the rumen resulting in high concentrations of blood $CO_2$ (Telle and Preston, 1971; Huntington and Britton, 1979; Burrin and Britton 1986; Krehbiel et al., 1995; Bigner et al., 1997; Owens et al., 1998; Brown et al., 2000). Aerobic metabolism is greatly reduced when lactic acid accumulates (Huber, 1976) suggesting exhaustion of the compensatory mechanisms (Burrin and Britton, 1986).

High ruminal osmolality causes reductions in concentrations of plasma or serum minerals, in particular Ca, and increased concentrations of blood lactate dehydrogenate (LDH) reflecting the need to metabolize lactate (Owens et al., 1998, Ghorbani et al., 2002). Steers diagnosed with SARA had blood metabolic profiles as follows: plasma lactate concentration was 0.46 mM, plasma glucose 78 mg/dL, serum Na 141 mEq/L, serum P 3.9 mEq/L, serum Ca 4.8 mEq/L and serum Cl 102 mEq/L (Brown et al., 2000). Similarly, Bigner et al. (1997) reported concentrations of plasma Cl was > 107 meq/L, Na was > 136 meq/L, K was < 4.2 meq/L and plasma glucose was < 75 mg/100ml.

**Ruminal Fermentation**

With forage fed animals, VFA do not accumulate in the rumen at sufficient concentrations to reduce ruminal pH as they are usually absorbed across the rumen wall (Allen, 1997), however, feeding concentrate diets increases substrate availability and production of VFA (Mackie and Gilchrist, 1979). For example, VFA concentrations in cows fed 90% concentrate were 83.9 mM compared to 68.3 mM in cows fed forage (Russell et al., 1998). Lyle et al. (1981) reported total VFA concentrations increased for steers adapted to a shelled corn diet from 70.6 mM initially to 167.1 mM. Total VFA concentrations are correlated with increased grain proportions as shown by Hristov et al. (2001), where total VFA were higher for steers fed a high verses medium barley grain diet over 30 d (125 vs. 109 mM). As well, Tremere et al. (1968) reported that highest VFA concentrations were detected the day before heifers went off feed (160 mM).

For ruminants gradually adapted to high grain diets, trace levels of lactate, in particular the L (+) lactate isomer, have been reported at very low concentrations not exceeding 10mM (e.g. Slyter, 1976; Mackie et al., 1978; Huntington, 1988, Goad et al., 1998; Owens et al., 1998; Hristov et al., 2001; Ghorbani et al., 2002). Fulton et al. (1979ab) suggested that low lactate levels are indicative of microbial adaptation over time to high grain diets. During transition to high-grain diets, ruminants are susceptible to rapid or abrupt consumption of grains generally causing increased lactate production, predominantly the D (-) lactate isomer. D (-) lactate is utilized less rapidly than L (+)- lactate and therefore more likely to accumulate in the rumen, with concentrations occasionally reaching 100 mM (Fulton et al., 1979a; Radostits, 1994; Owens et al., 1998). Lactic acid production following engorgement of RFC has been reported to be as high as 3 g/100 ml, causing ruminal pH to decline to 5.0 and gradually reducing the production of VFA (Crichlow and Chaplin, 1985; Dirksen, 1985 as cited by Braun et al., 1992; Krehbiel et al., 1995). Ruminal pH is lowered more drastically in response to lactic acid accumulation

compared to VFA accumulation because lactic acid is ten times stronger in acidity compared to VFA and its pK (the pH point of maximum buffering) is 3.8 compared to 4.6 for VFA (Slyter, 1976; Owens et al., 1998). Thus, lactate easily exceeds the buffering capacity of rumen fluid (Slyter 1976; Russell and Hino, 1985; Owens et al., 1998).

Changes in molar proportions of acids occur during acidosis and are reflective of defaunation (reduction or eradication of protozoal populations) (Hristov et al., 2001). In ruminants gradually adapted to grain diets, increased propionate and butyrate proportions relative to acetate have been observed, (Goad et al., 1998; Russell, 1998; Soto-Navarro et al., 2000) thus, the acetate to propionate ratio is reduced (Mackie and Gilchrist, 1979). When ruminal pH approaches 5.0 (as with acute acidosis cases), increasing acetic acid and decreasing fractions of propionic and butyric acids have been documented, and the acetate to propionate ratio is broadened (Tremere et al., 1968; Fulton et al., 1979a; Krehbiel et al., 1995; Russell, 1998). Acetic acid has a greater effect on reducing ruminal pH than propionic or butyric acid and is also more easily absorbed (Allen, 1997). Leedle (1993) also suggested that feed efficiency may be compromised when propionate levels are reduced.

**Ruminal Osmolality**

Osmolality in the rumen is usually constant near 240 to 265 mOsm/L with forage diets and 280 to 300 mOsm/L with concentrate based diets (Garza et al., 1989 as cited by Owens et al., 1989; Owens et al., 1998). Horn et al. (1979) reported tonicity in rumen contents in steers fed a high corn diet increased by greater than 50% 1-h post feeding. Similarly, Bergen (1972) reported that osmolalities peaked 2 h post feeding in sheep to 370 mOsm, and then declined. Brown et al. (2000) reported the range in ruminal osmolality in steers with SARA was from 305 to 330 mOsm/kg. High osmolality has been correlated directly with lactic acid levels as shown by Crichlow and Chaplin (1985). In that study, sheep that engorged on wheat exhibited rumen fluid

osmolality of 247.0 mmol/kg just before feeding and this steadily increased to 454.0 mmol/kg 12 h after engorgement, at which time highest lactate levels were reported (148.35 mmol/L).

High rumen osmotic pressure (> 300 mOsm) has also been suggested to contribute to fluctuations in feed intake as osmoreceptors in the rumen may limit voluntary feed consumption (Bergen, 1972; Owens et al., 1998; Carter and Grovum, 1990). As well, high rumen osmolarity causes the inhibition of bacterial digestion of starch and reduces gut motility, which in turn slows intestinal ingesta turnover, leads to stagnant ruminal contents and the inhibition of acid outflow (Slyter, 1976; Huber, 1976).

**Welfare Implications**

Behavioral signs can be used to suggest that the welfare of feedlot cattle is compromised to some extent from acute and subacute acidosis. Cattle that are examined clinically a few hours after grain engorgement have been observed to be kicking at the belly, a sign indicative of abdominal pain (Radostits, 1994). Other behaviors displayed by feedlot cattle are panting, lethargy, dull appearance, diarrhea, eating dirt and reduced feed intake (Braun et al., 1992; Phy and Provenza, 1998; Stock and Britton, 2002). Braun et al. (1992) observed acidotic sheep to frequently grind their teeth. Telle and Preston (1971) reported that when ewes were infused with D-L lactic acid, founder became apparent within 10-h after infusion, as well as loss of appetite, marked lameness and a mucoid nasal discharge occurred. As well, an autposy of one ewe that died 24-h after infusion revealed massive rumen hemorrhages and swollen hoofs. Ewes euthanized and autopsied 28-d after infusion had blood clots throughout the abdominal cavity, hemorrhaged rumens and laminitis, culminating in the necropsy diagnosis being founder. According to Hood et al. (2001), laminitis in horses is a painful condition and causes shifts in the weight distribution between the front and hind feet in attempts to mediate discomfort. Huntington et al. (1977) reported that for 450 lambs fed a typical high concentrate feedlot diet, 33 developed urinary calculi as evidenced by urinary blockage or the presence of calculi in the

urinary bladder or kidneys, 9 died from PEM, 5 developed severe synovitis, an arthritic condition, while numerous others were afflicted to a lesser degree, and 10 died from undetermined causes. Huntington and Britton (1979) reported postmortem examination of a lamb after receiving a 90% concentrate diet revealed severe damage to the rumen epithelium with large areas of sloughed papillae, and the abomasum and part of the duodenum were inflamed.

From this brief review, feedlot animals experience various degrees of illness from consuming high concentrate diets. Although not as detrimental as acute acidosis, SARA is a welfare issue because of the secondary symptoms that occur in response to the digestive upsets associated with the consumption of RFC sources. Thus, acidosis is not strictly a production and economic concern in the feedlot industry, but also an important welfare issue.

**Prevention and Treatment of Acidosis**

The most reliable prevention of acidosis is through management techniques, including close feed bunk monitoring which entails altering feeding practices such that cattle consume feed in a uniform manner incorporating smaller and more frequent meals (Schwarkopf-Genswein et al., 2003). As well, gradual adaptation to high grain diets over several weeks is necessary to achieve stability of the microbial population and to avoid accumulation of ruminal acids (Slyter, 1976; Huntington, 1988; Radostits, 1994).

Several feed additives are commonly used in conjunction with good management practices and include antibiotics that have either systemic effects or ruminal effects and dietary buffers designed to neutralize acidic conditions in the rumen (Huntington, 1988). Antibiotics that have systemic effects, such as chlortetracycline and in particular tylosin, are useful in managing the incidence of liver abscesses and improve feedlot cattle performance (Huntington, 1988; Nagaraja and Chengappa, 1998). Antibiotics that have rumen effects include ionophores such as monensin and lascalocid. Ionophores have been shown to affect feed intake patterns by

reducing intake depressions and altering ruminal fermentation patterns to facilitate microbial adaptation to increased concentrate intake (Huntington, 1988; Stock and Britton, 1993; Cooper and Klopfenstein, 1996). However, the use of antibiotics has come under public scrutiny as the prevalence of antibiotic-resistant bacteria has increased in recent years (Salyers, 1995 as cited by Russell and Rychlik, 2001). In 2002, the European commission proposed a ban on antibiotic growth promotants by 2006, which will control (or limit) the import of meat from countries where antibiotic growth promotants are allowed in animal feeds (Carro and Ranilla, 2002 as cited by Castillo et al., 2004). There is an obvious need for antibiotic alternatives as a means to manage feedlot acidosis from an economic as well as a health and welfare perspective.

Organic acids, (e.g. malate and fumarate), have been considered as an alternative to monensin, however, further research is required to determine their effect on beef cattle performance (Castillo et al., 2004). Other potential alternatives are dietary buffers (e.g. bicarbonates, hydroxides and silicates) that neutralize ruminal acidity, increase ruminal pH and stimulate the utilization of lactate (Owens et al., 1998). The concept of adding buffers to the diets of sheep and cattle has been of interest to ruminant nutritionists since the 1950's (Wheeler, 1980). Responses to buffers in high concentrate diets are inconsistent largely due to the variation in type and quantities of buffers fed, level and type of grain and forage content in the diet, and levels of preformed acids in the diet (Russell et al., 1980; Wheeler, 1980). As well, it has been suggested that buffer additions may only be useful during the phase of early adaptation to high concentrate diets and lack of a sustained response is indicative of adjustment to the bicarbonate load by the animal (Huntington et al., 1977; Russell et al., 1980; Wheeler, 1980; Ha et al., 1983).

Ruminants experiencing SARA have been shown to prefer feed containing sodium bicarbonate (SB). For example, Phy and Provenza (1998) demonstrated that sheep fed barley pellets containing SB or sodium chloride would actively select and preferred the pellet supplemented with SB. The authors concluded that the sheep selected the pellets containing SB

in attempts to self-attenuate ruminal acidosis. However, due to the large number of sheep, ruminal pH was not monitored and sheep exhibiting SARA were diagnosed based on observable signs such as diarrhea and reduced feed intake. Cooper et al. (1996) demonstrated that sheep fed high energy density feeds (barley based or sugar beet/barley based) supplemented with SB (0, 1, 2 or 4% [wt/wt]) would actively select feed supplemented with SB in preference to equivalent unsupplemented feeds. As well, it was reported that preference for SB supplemented diets was not related to level of supplementation. According to the authors, sheep preferred SB supplemented diets in attempts to attenuate low ruminal pH and stimulate the intake of high energy density foods. However, as with Phy and Provenza (1998), measurements of ruminal conditions were not made and therefore, it was not known if sheep were actually experiencing SARA conditions. Cumby et al. (2001) fed dairy cattle a corn based diet and then switched to feeding a wheat barley grain pellet to induce SARA. Ruminal pH was measured in that study and averaged 5.78 and 5.95 for the weeks that the grain pellets were fed, indicating that cows were subacute. During the feeding of the grain pellet, either 4.0% SB pellets or 4.5% sodium chloride pellets were offered and the preference for each pellet was monitored. Cows increased their preference for the SB pellets over time and the authors suggested that the cows developed a learned response that SB minimizes acidosis. However, in that study, the authors acknowledged that the strength of the preference for the SB pellets may have been aided by the aversion to the sodium chloride pellets.

Buffer supplementation of high concentrate diets has been shown to improve or stabilize feed intake and increase animal performance in some studies (Fulton et al., 1979b; Ha et al., 1983; Solorzano et al., 1989; Zinn, 1991), but not in others (Bergen, 1972; Colling et al., 1979; Russell et al., 1980; Hart and Polan, 1984; Coppock et al., 1986, Ghorbani et al., 1989). Increases in mean ruminal pH have also been documented when concentrate diets were supplemented with buffers (Fulton et al., 1979b; Horn et al., 1979; Ha et al., 1983; Ghorbani et

al., 1989; Zinn 1991); however these findings have not been consistent (Colling et al., 1979; Russell et al. 1980; Hart and Polan, 1984).

The effect of buffer supplementation on ruminal fermentation is variable. Some studies reported that buffers had no effect on total VFA (Horn et al., 1979; Fulton et al., 1979b; Ha et al., 1983; Solorzano et al., 1989), while others have reported that supplementation with buffers increased VFA concentrations (Hart and Polan, 1984). In some studies, molar proportions of acids have been shown to remain unchanged in response to buffers (Horn et al., 1979; Ha et al., 1983; Hart and Polan, 1984). However, other studies have shown increased acetate, valerate and butyrate concentrations (Fulton et al., 1979b; Colling et al., 1979; Russell et al., 1980), reductions in propionic acid (Colling et al., 1979; Russell et al., 1980; Coppock et al., 1986; Zinn, 1991) and a wider acetate to propionate ratio (Coppock et al., 1986). Horn et al. (1979) and Bigner et al. (1997) reported blood pH and blood bicarbonate were higher in steers and cows fed or administered buffers compared to control animals (non buffered). Cows fed buffers were also reported to have increased concentrations of plasma Na, Cl and K but not glucose compared to pretreatment concentrations (Bigner et al., 1997). Serum Ca levels were lowered and Mg and P levels raised by the addition of buffers (Huntington et al., 1977). Lastly, Ha et al. (1983) reported buffers did not affect blood lactate concentrations or packed cell volume (PCV).

Buffers have been shown to increase rumen osmolality, suggesting that they are osmotically active agents, particularly those containing Na (Hart and Polan, 1984; Phy and Provenza, 1998). For example, Bergen (1972) reported 1-h after intraruminal infusion of either NaAc or NaCl, rumen osmolality increased to an average of 697 mOsm and 512 mOsm for two sheep. Phy and Provenza (1998) explained that it is the sodium in the buffers that increases the osmolality in the rumen and illustrated that despite the negative effects of Na on osmolality, preference by lambs for feed containing SB remained strong. This suggests that the lambs continued to chose to ingest feed supplemented with SB because the benefit of the bicarbonate

attenuating acidosis outweighed the detriment of increased osmolality (Phy and Provenza, 1998). Thus, SB is commonly used to prevent acidosis because the alkalinizing action of $Na^+$ is readily absorbed allowing the bicarbonate to be metabolized (Bigner et al., 1997). Sodium bicarbonate is also used as a buffer because it is more palatable compared to other buffers such as sodium chloride (Phy and Provenza, 1998 and Cumby et al., 2001). In those studies, sheep preferred feed supplemented with SB over feed supplemented with sodium chloride.

Anecdotal accounts suggest that ruminants fed high-grain diets will readily consume SB offered free choice (Phy and Provenza, 1998). Keunen et al. (2003) provided dairy cattle induced with SARA free choice access to SB. Induction of SARA did not affect DMI and did not increase SB intake compared to the intakes of SB by control cows. Also, free choice intake of SB had no affect on ruminal pH compared to control cows. The authors attributed their findings to the fact that SB was ingested at insufficient levels to significantly elevate rumen pH and may have been due to adverse organoleptic properties of SB. Coppock et al. (1986) suggested that molasses, a highly palatable ingredient, could be used to mask the flavor of buffers.

**Research Objectives**

From a welfare point of view, an economic perspective and because the current treatment of acidosis with antibiotics is considered by some to be a public health concern, it is necessary to further study the potential benefits of using buffers in the treatment and prevention of acidosis in feedlot cattle consuming high grain diets. To my knowledge, there has been no previous research on the effects of free choice SB on cattle fed feedlot diets. As well, there has been no previous research comparing the effectiveness of dietary supplemental verses free choice SB on reducing the effects of SARA. Thus, the objectives of my thesis research were: 1) to determine the effects of SARA in cattle fed a feedlot diet, 2) determine if SB, either offered free choice or mixed into the diet could minimize the effects of SARA, and 3) to determine which of these two methods of providing SB was most effective. Lastly, for this thesis research work, I attempted to develop an

objective approach of defining SARA in feedlot cattle based on ruminal pH variables. Ruminal pH thresholds of 5.8 and 5.5 were used to classify SARA, with 5.5 considered severe SARA. Furthermore, average ruminal pH across days was presented in this research for descriptive purposes to categorize, in general, the severity of acidosis in relation to the chosen pH thresholds. Time and area below pH 5.8 and 5.5 were also used to give a more accurate diagnosis of acidosis severity. As well, the duration and frequency of individual bouts of SARA that occurred within a 24-h period was defined, which has not been previously documented. Chapter II is an independent paper intended for publication and thus repeats some of the literature cited in Chapter I.

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# CHAPTER II: EFFECTS OF SODIUM BICARBONATE ON REDUCING ACIDOSIS IN CATTLE

## INTRODUCTION

Ruminal acidosis causes economic losses for the feedlot industry due to abscessed livers, lameness, morbidity, variable feed intake and reduced animal performance (Nocek, 1997; Owens et al., 1998). Acidosis results from the rapid production of ruminal VFA and lactic acid arising from ingestion of readily fermentable carbohydrates (Britton and Stock, 1986). Degree of severity distinguishes acute and subacute ruminal acidosis (SARA) (Britton and Stock, 1986). Cattle exhibit acute metabolic acidosis as an overt illness while SARA is subtle and difficult to assess, characterized by periods during which ruminal pH is below optimum for growth of cellulolytic bacteria in the rumen (Owens et al., 1998). However, brief periods of low ruminal pH (< 5.8) are unlikely to affect the health of the animal whereas extended periods (i.e. several hours) of low rumen pH are detrimental to rumen function (Keunen et al., 2002), and can also lead to acute metabolic acidosis.

Dietary buffers, such as sodium bicarbonate (SB), sometimes minimize the effects of SARA when mixed into the diet (Ghorbani et al., 1989; Solorzano et al., 1989; Zinn, 1991), but not always (Russell et al., 1980; Hart and Polan, 1984). Anecdotal accounts suggest that ruminants fed high grain diets will readily consume SB offered free choice to minimize SARA (Phy and Provenza, 1998). However, Keunen et al. (2003) provided dairy cattle experiencing SARA free choice access to SB and concluded SB intake was insufficient to significantly elevate ruminal pH. To our knowledge there has been no work investigating the effects of offering SB free choice on reducing the incidence of SARA in cattle fed high grain feedlot diets. Thus, the objectives of the present study were to: 1) determine the effects of SARA in feedlot cattle fed a high grain diet containing barley, a source of rapidly fermentable carbohydrates, and 2)

determine if SB, either mixed into the diet or offered free choice, could reduce the effects of SARA.

## MATERIALS AND METHODS

### Experiment 1

Two pens of six individually fed Hereford heifer calves, averaging 344 ± 36.9 kg BW, were used in a replicated 3 x 3 Latin square design to investigate the effects of providing supplemental SB, either incorporated directly into the control diet or offered free choice, on feed intake of feedlot cattle.

The experiment consisted of 3 periods of 3 wk each separated by 8 d during which the control diet was fed. In each period, heifers within each pen were randomly assigned to one of the following treatments: 1) control (no SB), 2) free choice access to a SB mixture (free choice SB), and 3) 0.7% SB incorporated into the TMR (DM basis) (mix SB).

### Feed and Sodium Bicarbonate Intake

Each pen was equipped with calan headgates (American Calan Inc., Northwood, U.S.A), and each heifer was fitted with a neck collar carrying a transponder that corresponded to one specific headgate within the pen. For 2 mo prior to commencing the experiment, the heifers were gradually adapted to the control diet and trained to use the headgates. The basal diet was prepared daily using a feed mixer except for the mineral-vitamin mixture, which was thoroughly mixed by hand into each feed trough (Table 2.1). Feed was offered once daily between 0700 and 0800 for ad libitum intake (at least 10% orts). Individual feed intake was measured daily during each period. Free choice SB intakes were measured weekly or more frequently when individual intakes were such that the SB mixture needed replenishing before 7 d. Intakes of SB of the cattle receiving mix SB were measured daily.

Table 2.1. Ingredient and chemical composition (mean ± SD) of the Control diet (DM basis).

| Item | Exp. 1 % | SD | Exp. 2 % | SD |
|---|---|---|---|---|
| Ingredient[a] | | | | |
| Barley silage[b] | 12.0 | | 12.0 | |
| Steam-rolled barley | 80.0 | | 81.2 | |
| Soybean meal | 7.0 | | 0 | |
| Limestone | 0.65 | | 1.4 | |
| Mineral-vitamin premix[c] | 0.35 | | 0.05 | |
| Dicalcium phosphate | 0 | | 0.3 | |
| Salt | 0 | | 0.5 | |
| Ground Barley | 0 | | 4.31 | |
| Dried molasses | 0 | | 0.17 | |
| Soy oil | 0 | | 0.07 | |
| Chemical composition, % DM | | | | |
| DM | 72.9 | 0.10 | 75.2 | 0.71 |
| OM | 92.5 | 0.26 | 95.4 | 0.97 |
| CP | 13.6 | 0.40 | 15.4 | 0.93 |
| NDF | 24.7 | 2.89 | 25.3 | 3.98 |
| ADF | 11.3 | 1.34 | 7.1 | 1.74 |
| peNDF[d] | 7.4 | | 12.1 | |

[a]All ingredients (Exp. 2) pelleted excluding barley silage and steam-rolled barley.
[b]Composition (DM basis) was 10.1% CP, 49.0% NDF, 32.0% ADF, and 67.9% pef (Exp. 1) and 13.0% CP, 41.3% NDF, 20.1% ADF, and 71.2% pef (Exp. 2) (based on three period samples per experiment).
[c]Mineral-vitamin premix supplied per kg of dietary DM: 420 mg of Ca, 420 mg of P, 350 mg of Na, 70 mg of Mg, 16.8 mg of Mn, 17.5 mg of Zn, 10.5 mg of Cu, 0.1 mg of Co, 0.56 mg of I, 10.5 mg of F, 2275 IU of vitamin A, 227.5 IU of vitamin D3, and 2.27 IU of vitamin E (Exp. 1) and 63 mg of Zn, 27 mg of Mn, 15 mg of Cu, 0.3 mg of Se, 0.19 mg of Co, 0.67 mg of I, 6000 IU of vitamin A, 600 IU of vitamin D, and 43 IU of vitamin E (Exp. 2).
[d]Physically effective fiber content (peNDF) was calculated by multiplying the physical effectiveness factor (pef, sum of the proportion of sample retained on the TMR retained on 19 and 8-mm screen of the Penn State Particle Separator) by the NDF content of the TMR.

The free choice SB was offered as a mixture consisting of 70% SB (Arm & Hammer feed grade, Church and Dwight Co., Inc., Princeton, NJ) and 30% dried molasses. This mixture was based on data collected during a preliminary preference study undertaken immediately prior to Exp. 1 (data not shown). The SB mixture offered free choice was provided in an 8-L bucket that was secured to either the right or left hand side of the individual's Calan gate controlled feed trough. Heifers fed free choice SB were provided an initial 500 g of the mixture on d 1 of each period. Sodium bicarbonate levels were monitored daily and replenished as needed. To

determine free choice SB intake, the orts were sieved through a 2.80-mm sieve and then a 2.36-mm sieve (standard testing sieve, Fisher Scientific, Pittsburgh, PA) to remove any bedding or TMR that was deposited in the buckets. Orts were dried in a 60°C oven for 48 h to determine the DM content (AOAC, 1990). Intakes of free choice SB were determined by dividing the orts by the number of days that the individual had access to the SB mixture and reporting this as a daily average. Intakes of free choice SB reported were corrected for the 30% dried molasses in the mixture. For the 0.7% SB supplemental diet, the amount of SB added to the heifer's diet was based on the DMI of the individual heifer. The appropriate amount of SB was weighed and hand mixed thoroughly into the individual's daily feed allotment. The amount of SB consumed was estimated by assuming that the proportion of SB in the TMR and orts was the same.

A 7 d adaptation period occurred prior to the start of the experiment, during which time all heifers in both pens were exposed to buckets containing the free choice SB mixture. This allowed the heifers to become familiar with the SB mixture contained within the buckets and to ensure that the heifers could readily access the SB. Cattle were housed outside in pens with access to shelter and fresh shavings were provided weekly. Heifers had ad libitum access to fresh water and cobalt iodized salt blocks and were cared for according to the Canadian Council on Animal Care guidelines (CCAC, 1993).

Samples of barley silage, barley grain, and TMR were collected weekly and DM content was determined by drying the samples at 60°C for 48 h (AOAC, 1990). Dry matter content was used to adjust the silage to concentrate ratio of the diet as needed. Weekly fresh samples of barley silage, barley grain, and TMR were also composited by period and frozen for analysis. Samples of TMR and barley silage were dried at 60°C for 48 h (AOAC, 1990) and sent to Cumberland Valley Analytical Services (Maugansville, MD) for chemical analysis. The physical effectiveness factor (pef) of the fresh TMR and barley silage were measured as the sum of the proportion of the sample retained on the top (19 mm) and middle (8 mm) sieves of the Penn State

Particle Separator. Physically effective fiber content (peNDF) was calculated by multiplying the pef by the NDF content of the TMR.

**Experiment 2**

A second experiment was conducted to determine the effects of supplemental SB, either incorporated into the TMR or offered free choice, on ruminal and metabolic acidosis in cattle fed high grain diets. Six ruminally fistulated (10 cm ID, Bar Diamond, Parma, ID) cattle were used in a replicated 3 × 3 Latin square design (three mature non-lactating Holstein cows and three mature Jersey steers). The average weight of cows and steers was 1013.3 and 736.6 kg, respectively. Prior to the start of the experiment, the Holstein cows had been fed a diet typical of that fed to non-lactating dairy cows, whereas the Jersey steers had previously been fed feedlot type diets. Twenty-eight days prior to commencing the experiment, the animals were adapted to the high-grain control diet.

The experiment consisted of three periods of 2 wk. In each period, animals were randomly assigned to one of the following treatments: 1) control (no SB), 2) free choice access to a SB mixture (free choice SB), and 3) 0.7% SB incorporated into the TMR (DM basis) (mix SB).

**Feed and Sodium Bicarbonate Intake**

The basal diet (Table 2.1) was prepared daily using a feed mixer and offered once daily at 1400 for ad libitum intake (at least 10% orts). Individual feed and free choice SB intakes were measured daily. Each animal in the free choice SB treatment group had access to an 8-L bucket containing 500 g/d of SB mixture that was provided once daily at 1300 h. The addition of 0.7% SB to the TMR (DM basis) was provided as described in Exp. 1.

A 7 d adaptation period occurred prior to the start of the experiment to allow the animals to adjust to their stalls and feed troughs and become familiar with the SB mixture provided in the bucket. The buckets were secured beside each animal's feed trough and remained there

throughout the trial. To minimize and account for spillage of the SB mixture, only the bottom of the buckets were filled with the SB mixture and a pan was placed under each bucket so any contents that were spilled could be collected and included as orts. During the trial, cattle on the SB free choice treatment had continuous access to the SB mixture except for 1 h/d (1300 to 1400) when all animals were let outside for exercise. Animals not on the free choice SB treatment were provided access to 500 g/d of dried molasses to maintain familiarity with the buckets in their stalls. Cattle were housed in individual stalls bedded with rubber mats and shavings and were provided ad libitum access to fresh water and were cared for according to the Canadian Council on Animal Care guidelines (CCAC, 1993).

Samples of barley silage, barley grain, and TMR were collected weekly. Dry matter was determined on a portion of each weekly sample and used to adjust the silage to concentrate ratio of the diet as needed. Weekly fresh samples of barley silage, barley grain, and TMR were composited by period, dried and ground through a 1-mm screen (Wiley Mill Standard Model No.3, Arthur H. Thomas Co., Philadelphia, PA), and retained for chemical analysis. Samples of orts were collected on d 11 to 13 (coinciding with ruminal pH measurement days) and composited by animal. Feed DM was determined by oven drying the sample at 55°C for 48 h. For each animal, DMI was calculated based on the feed DM offered and the orts DM refused.

Analytical DM content of the samples was determined by drying for 2 h at 135°C (AOAC, 1990). The samples were then combusted at 550°C for 5 h to determine the ash content (AOAC, 1990). The OM of the samples was calculated as the difference between DM and ash content (AOAC, 1990). The concentration of CP was determined by flash combustion (Carlo Erba Combustion Analyzer, model NA2100 Protein, CE Instruments, Milan, Italy). The NDF and ADF contents were determined by the methods described by Van Soest et al. (1991) with amylase and sodium sulfite used in the NDF procedure. Intakes of fiber were calculated using

the NDF and ADF of the TMR and orts. The peNDF content of barley silage and TMR was measured using the same methodology described in Exp.1.

**Ruminal pH**

Ruminal pH was continuously measured on d 11 to 13 using indwelling electrodes (model PHCN 37; Omega Engineering, Stamford, CT) inserted through the cannula into the rumen. A weight was attached to ensure that the electrode remained in the ventral sac. The electrodes were removed daily from the rumen 1 h prior to feeding when animals were exercised and calibrated with pH 4.0 and pH 7.0 standards. The pH was measured every 5 s and an average of these readings was recorded every 5 min using a data logger. Ruminal pH for each animal was summarized daily as averages for pH, maximum and minimum pH, cumulative time pH was below pH 5.8 or pH 5.5, and area the pH recording was below a straight line drawn at pH 5.8 or 5.5. The area was calculated by adding the absolute value of the negative deviations in pH from pH 5.8 or 5.5 for each 5-min interval and then expressed as pH units x hours. The degree to which ruminal pH fluctuated was calculated as pHDIFF and pHVAR, where pHDIFF is the difference between the maximum and minimum ruminal pH for an individual for that day and pHVAR is the variation in ruminal pH (measured as the standard deviation associated with the mean ruminal pH).

An individual bout of SARA was said to occur when rumen pH dropped below 5.8. The duration of each bout was calculated as the length of time that the ruminal pH continuously remained at or below this threshold. The frequency of bouts was calculated as the number of times the ruminal pH went below 5.8 each day. The incidence of severe SARA was calculated as a subset of SARA, by examining the duration and frequency that pH dropped below 5.5.

**Ruminal Fermentation**

At 0, 3 and 6 h post feeding on d 13, approximately 1 L of rumen contents from each animal was obtained from four sites within the rumen (reticulum, dorsal and ventral sacs, and the

mat) and composited. The contents were then strained through a polyester monofilament fabric (Pecap 7-1180/59, mesh opening 1,180 μm, Tetko Inc., Scarborough, ON, Canada) and VFA and lactate concentrations were measured using the methodology described in Ghorbani et al. (2002). The remaining filtrate was centrifuged (13,000 x g for 30 min at 4°C) and used to determine ruminal fluid osmolality (model 5004 Automatic Osmometer, Precision Systems Inc., Fisher Scientific, Pittsburgh, PA) within 2 h of sampling by the freezing point depression technique.

**Blood Variables**

At approximately 0900 on d 14 of each period, blood samples were taken from the jugular vein of each animal. Blood samples taken using 10-mL Vacutainer tubes (Na heparin) were assayed for pH, $HCO_3$, $CO_2$ and $O_2$ at the Lethbridge Regional Hospital using a blood gas analyzer (IL Synthesis System, model 15U, Instrumental Labs, Milan, Italy). Blood potassium, collected in a 10 mL Vacutainer tube, was also analyzed at the Lethbridge Regional Hospital (Dimension Clinical Chemistry System, DADE model RxL, Dade Behring, Newark, DE). Blood glucose, amylase, Ca, Mg, lactate dehydrogenase and packed cell volume were determined by methods outlined in Ghorbani et al. (2002).

**Statistical Methods**

For both experiments, treatment means were calculated by day or period for all variables. Ruminal pH, pHDIFF, duration and number of bouts of mild and severe SARA, mean DMI, mean SB intake, and differences in SB intakes among animals offered free choice SB and SB mixed into the TMR were analyzed using the mixed model procedure of SAS (Proc Mixed; SAS Inst. Inc., Cary, NC). The model included the fixed effects of day, treatment and group (cows or steers) or pen and their interactions. Animal and period were considered random effects and day was considered a repeated measure. Minimum and maximum DMI and SB intake were analyzed using the least square means procedure in GLM of SAS to account for effects of treatment and group or pen and their interactions. In Exp. 2, the correlation between daily DMI and mean

ruminal pH on d 11 to 13, daily SB intakes and mean ruminal pH on d 11 to 13, and daily free choice SB intake and daily DMI on d 1 to 14 were determined using Pearson correlation. Levenes test for homogeneity of variances in GLM was used to test pHVAR treatment differences. The mixed model procedure of SAS was also used to analyze ruminal fermentation variables to account for the effects of sampling time, treatment, group (cows or steers) and their interactions. Animal and period were random effects while sampling time was a repeated measure. Proc GLM of SAS was used to determine the effects of treatment, period and group for blood variables. For the mixed model procedures, the covariance structures used were autoregressive or compound symmetry, except for minimum and maximum ruminal pH, in which case, unstructured was the best fit. When the main effect of treatment was $P \leq 0.10$, means were separated using contrast statements. Significance was declared at $P \leq 0.05$ and trends were discussed at $P \leq 0.10$.

## RESULTS

### Experiment 1

### Feed and Sodium Bicarbonate Intake

Dry matter intake averaged (± SEM) 7.8 ± 0.43 kg/d and was unaffected by treatment (Table 2.2). Mean DMI varied daily ($P = 0.05$), but treatment did not affect this variation ($P = 0.28$; Figure 2.1A). Minimum DMI averaged (± SEM) 5.3 ± 0.61 kg/d and maximum DMI averaged (± SEM) 9.6 ± 0.46 kg/d with no effect of treatment.

Mean SB intakes for animals consuming the mix SB were higher than for animals consuming free choice SB ($P < 0.0001$; Table 2.2). Within pens, SB intakes differed greatly among heifers ($P < 0.0001$). Intake of SB tended to vary daily ($P = 0.08$), with the day to day variation in SB intake greater for mix SB than for free choice SB ($P = 0.02$).

### Experiment 2

**Feed and Sodium Bicarbonate Intake**

Dry matter intake averaged (± SEM) 8.5 ± 0.97 kg/d and was similar among treatments (Table 2.2). There were no effects of group or group by treatment interactions ($P > 0.34$). Mean DMI varied daily ($P = 0.002$), but was unaffected by treatment ($P = 0.79$; Figure 2.1B). Treatment did not affect minimum or maximum DMI which averaged (± SEM) 4.93 ± 1.10 kg/d and 10.5 ± 1.13 kg/d, respectively.

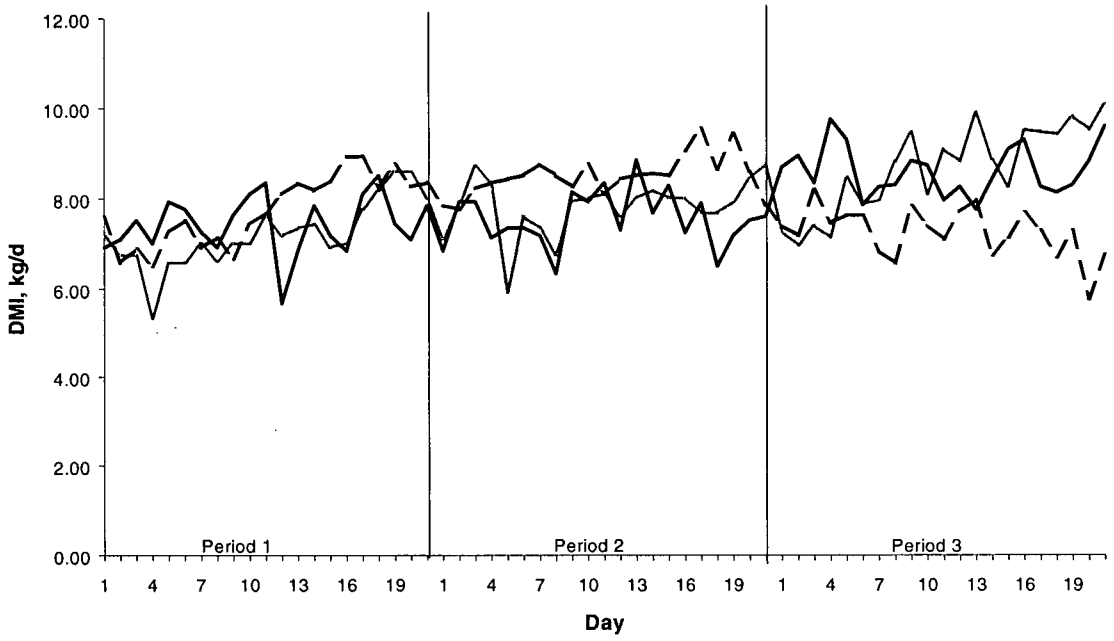
Average SB intake was greater for steers than for cows (92.6 vs. 37.6 g/d, respectively, $P = 0.04$; Table 2.2). Cows had higher SB intake when offered the mix SB treatment than when provided free choice SB, whereas the reverse occurred for steers ($P = 0.04$; Figure 2.2) and within groups, SB intakes differed amongst animals (cows, $P = 0.07$; steers, $P < 0.0001$). A trend ($P = 0.09$) was detected for a treatment by day interaction (Figure 2.2). Average maximum SB intake was greater for steers compared to cows (157.9 vs. 68.9 g/d, $P = 0.08$) and a group by treatment interaction ($P = 0.06$) was detected, with cows having a higher maximum SB intake for mix SB while the opposite occurred for steers.

**Table 2.2.** Intake of dry matter (DMI) and sodium bicarbonate for Hereford heifer calves (n = 12; Exp. 1) and non-lactating Holstein cows (n = 3) and Jersey steers (n =3) (Exp. 2) fed control with no sodium bicarbonate (Control), free choice sodium bicarbonate (Free Choice SB) or sodium bicarbonate mixed into the TMR (Mix SB).

| Item | Control | Free Choice SB | Mix SB | SEM | *P* |
|---|---|---|---|---|---|
| Experiment 1 | | | | | |
| DMI, kg/d | | | | | |
| Mean | 7.8 | 7.8 | 7.9 | 0.4 | 0.74 |
| Min[a] | 5.0 | 5.8 | 5.2 | 0.6 | 0.65 |
| Max[a] | 9.6 | 9.4 | 9.9 | 0.5 | 0.79 |
| Sodium bicarbonate, g/d | | | | | |
| Mean | | 2.1 | 55.3 | 2.9 | < 0.0001 |
| Min[a] | | 1.3 | 36.4 | 2.5 | < 0.0001 |
| Max[a] | | 2.9 | 69.3 | 2.1 | < 0.0001 |
| Experiment 2 | | | | | |
| Cows | | | | | |
| DMI, kg/d | | | | | |
| Mean | 9.2 | 9.2 | 8.3 | 1.3 | 0.69 |
| Min[a] | 5.9 | 5.8 | 4.3 | 1.9 | 0.82 |
| Max[a] | 10.9 | 11.5 | 10.9 | 2.1 | 0.98 |
| Sodium bicarbonate, g/d | | | | | |
| Mean | | 17.4 | 57.8 | 4.2 | < 0.0001 |
| Min[a] | | 1.5 | 27.4 | 5.9 | 0.04 |
| Max[a] | | 61.0 | 76.7 | 18.4 | 0.58 |
| Steers | | | | | |
| DMI, kg/d | | | | | |
| Mean | 7.7 | 7.9 | 8.0 | 1.3 | 0.69 |
| Min[a] | 4.7 | 5.2 | 3.7 | 1.1 | 0.64 |
| Max[a] | 9.8 | 9.7 | 10.1 | 0.9 | 0.95 |
| Sodium bicarbonate, g/d | | | | | |
| Mean | | 129.1 | 56.1 | 6.5 | < 0.0001 |
| Min[a] | | 18.7 | 35.9 | 9.2 | 0.26 |
| Max[a] | | 245.1 | 70.6 | 60.7 | 0.11 |

[a]Means.

A)




B)

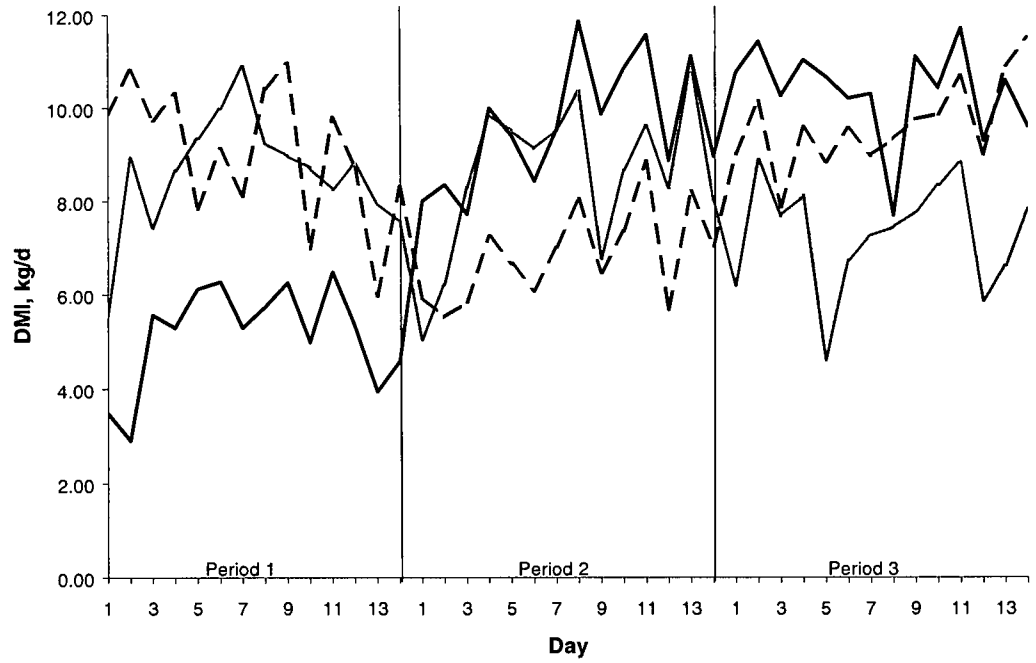
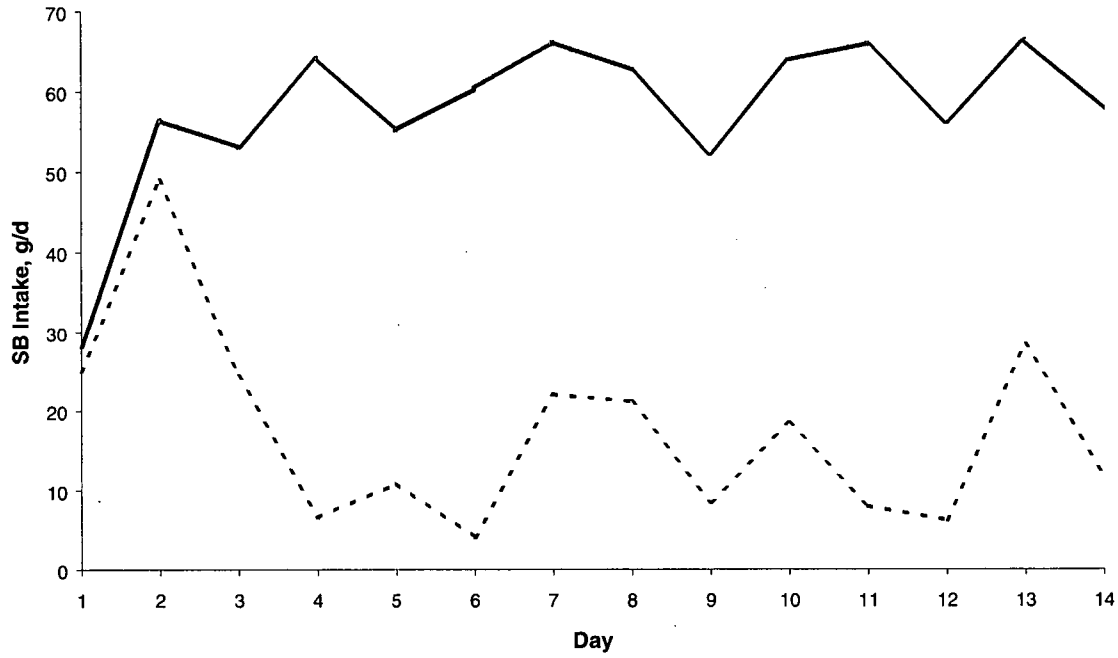



**Figure 2.1**. Mean dry matter intake (DMI) for A) Hereford heifer calves (n = 12; Exp. 1) and B) non–lactating Holstein cows and Jersey steers (n = 6; Exp. 2) fed Control (solid line), Free Choice SB (dashed line) or Mix SB (gray line). Average DMI was unaffected by treatment for A and B ($P = 0.74$, 0.69, respectively). For A and B, feed intake varied daily ($P = 0.05$, 0.002, respectively), however, the daily variation in DMI did not differ by treatment ($P = 0.28$, 0.79, respectively).

A)




B)

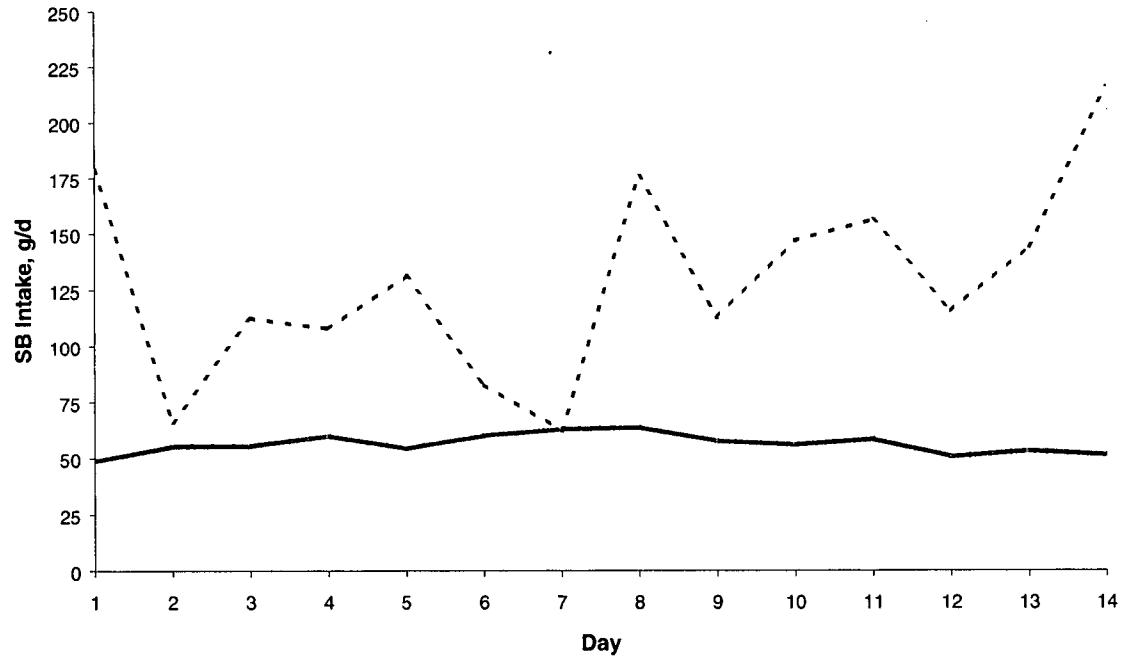



**Figure 2.2.** Mean sodium bicarbonate (SB) intakes for A) non–lactating Holstein cows (n = 3) and B) Jersey steers (n = 3) fed free choice SB (dotted line) and mix SB (gray line) (Exp. 2). A group by treatment interaction occurred ($P = 0.04$); cows average SB intake was greater for mix SB than free choice ($P < 0.0001$), whereas steers average intake of SB was greater for free choice SB than mix SB ($P < 0.0001$).

**Ruminal pH**

Ruminal pH for cows and steers averaged (± SEM) 5.86 ± 0.08 and was similar among treatments (Table 2.3). Mean ruminal pH tended ($P = 0.08$) to be higher for steers than cows, however, no treatment by group interaction occurred ($P = 0.34$). Ruminal pH did not vary among days ($P = 0.61$; Figure 2.3).

**Table 2.3.** Ruminal pH for non-lactating Holstein cows (n = 3) and Jersey steers (n = 3) fed the control diet with no sodium bicarbonate (Control), free choice sodium bicarbonate (Free Choice SB) or sodium bicarbonate mixed into the TMR (Mix SB) (Exp. 2).

| Item | Control | Free Choice SB | Mix SB | SEM | *P* |
|---|---|---|---|---|---|
| Cows | | | | | |
| Mean | 5.65 | 5.75 | 5.91 | 0.09 | 0.23 |
| Min[a] | 5.17 | 5.15 | 5.29 | 0.12 | 0.65 |
| Max[a] | 6.25 | 6.58 | 6.44 | 0.11 | 0.17 |
| < 5.8, h[a] | 15.6 | 12.7 | 11.4 | 1.9 | 0.35 |
| Area < pH 5.8, pH × h[a] | 28.4 | 28.4 | 19.6 | 6.1 | 0.41 |
| < 5.5, h[a] | 8.5 | 8.9 | 7.1 | 2.1 | 0.84 |
| Area < pH 5.5, pH × h[a] | 10.6 | 11.8 | 5.6 | 3.4 | 0.33 |
| pHDIFF[b] | 1.08 | 1.43 | 1.15 | 0.19 | 0.43 |
| pHVAR[c] | 0.04 | 0.22 | 0.15 | | 0.19 |
| Steers | | | | | |
| Mean | 5.97 | 6.01 | 5.89 | 0.13 | 0.82 |
| Min[a] | 5.38 | 5.17 | 5.38 | 0.18 | 0.64 |
| Max[a] | 6.54 | 6.59 | 6.45 | 0.16 | 0.84 |
| < 5.8, h[a] | 7.4 | 6.9 | 10.5 | 3.6 | 0.76 |
| Area < pH 5.8, pH × h[a] | 10.9 | 7.9 | 17.1 | 5.8 | 0.14 |
| < 5.5, h[a] | 3.4 | 1.1 | 5.5 | 2.1 | 0.38 |
| Area < pH 5.5, pH × h[a] | 3.3 | 2.2 | 4.6 | 1.9 | 0.38 |
| pHDIFF[b] | 1.16 | 1.42 | 1.06 | 0.13 | 0.26 |
| pHVAR[c] | 0.22 | 0.06 | 0.34 | | 0.17 |

[a]Means.
[b]pHDIFF Difference between the maximum and minimum ruminal pH for each animal for each day averaged over treatment.
[c]pHVAR Variation in ruminal pH measured as the standard deviation associated with the mean pH for each treatment.

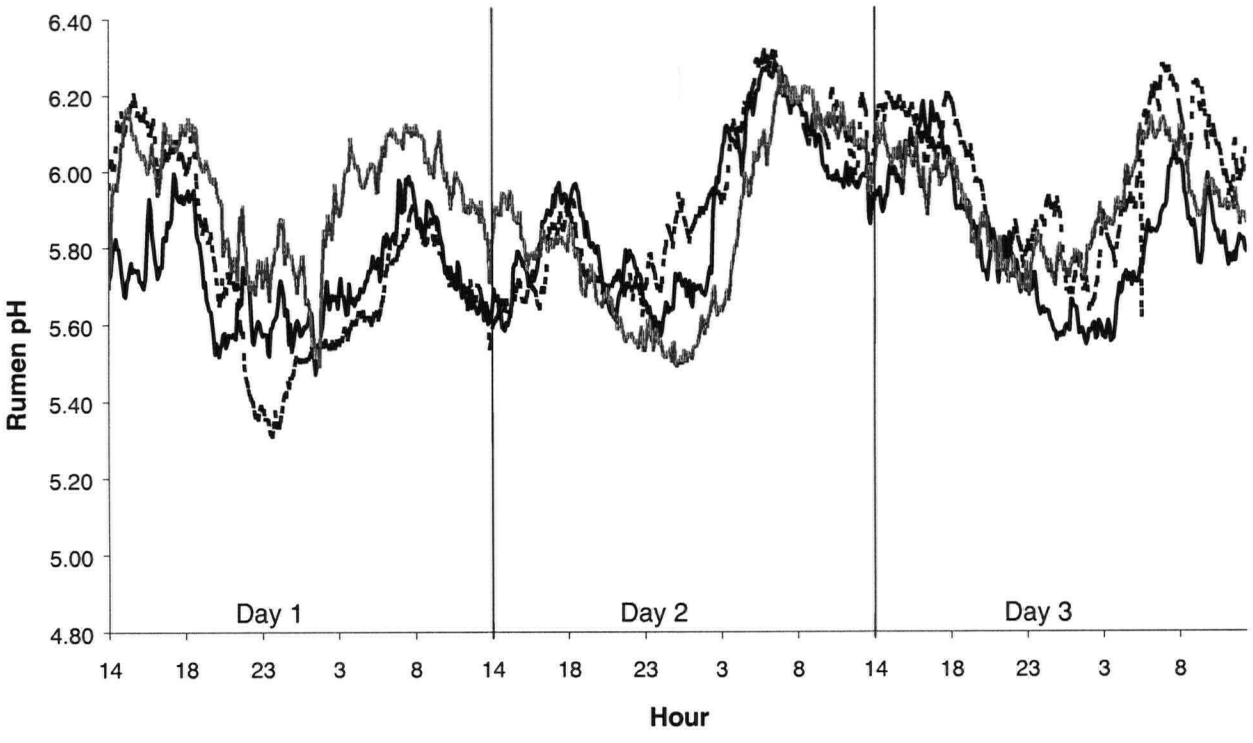



**Figure 2.3.** Mean ruminal pH for non-lactating Holstein cows and Jersey steers (n = 6; Exp. 2) fed Control (solid line), Free Choice SB (dotted line) or Mix SB (gray line). Mean ruminal pH was similar among treatments ($P = 0.75$) and there was no daily variation ($P = 0.61$).

No treatment differences were detected for the other ruminal pH variables measured. Compared to cows, steers had a higher minimum ruminal pH ($P = 0.01$), less time below ruminal pH 5.5 and 5.8 ($P = 0.07$, 0.09, respectively) and less area below ruminal pH 5.5 and 5.8 ($P = 0.05$, 0.02, respectively). There were no group by treatment interactions for area and time below the pH thresholds ($P > 0.24$).

On average, the cattle experienced 4 to 5 bouts of SARA each day (Table 2.4). A bout of SARA lasted approximately 5.1 h, however, the duration of these bouts was highly variable, ranging in length from 0.08 to 22.6 h/d. Animals fed mix SB had longer ($P = 0.05$) SARA bouts than those fed free choice SB, but neither method of supplementation was different ($P > 0.05$) from the control. Bouts of severe SARA (< pH 5.5) lasted approximately 1.7 h, but ranged from 0.08 to 5.4 h/d. Feeding free choice or mix SB tended ($P < 0.10$) to reduce the duration of severe SARA bouts compared to the control. The number of severe SARA bouts each day was similar

for control and free choice SB fed animals, but tended ($P < 0.10$) to increase when animals were provided the mix SB diet.

Compared to cows, steers had more daily bouts of SARA (3.1 and 5.8, respectively, $P = 0.02$) and tended to have shorter bouts of severe SARA (2.4 and 0.9 h, respectively, $P = 0.003$), however, no group by treatment interactions occurred ($P > 0.50$).

**Table 2.4.** Duration and number of SARA bouts for non-lactating Holstein cows and Jersey steers (n = 6) fed the control diet with no sodium bicarbonate (Control), free choice sodium bicarbonate (Free Choice SB) or sodium bicarbonate mixed into the TMR (Mix SB) (Exp. 2).

| Item | Control | Free Choice SB | Mix SB | SEM | *P* |
|---|---|---|---|---|---|
| pH 5.8 | | | | | |
| Duration of SARA bouts, h[c] | 4.7[ab] | 2.5[a] | 8.1[b] | 1.45 | 0.05 |
| No. SARA bouts[c] | 4.8 | 4.5 | 4.1 | 0.76 | 0.83 |
| pH 5.5 | | | | | |
| Duration of SARA bouts, h[c] | 2.3 | 1.4 | 1.3 | 0.34 | 0.11 |
| No. SARA bouts[c] | 3.3[a] | 3.8[a] | 8.7[b] | 1.68 | 0.09 |

[ab]Within row, means without a common superscript differ ($P < 0.05$).
[c]Means.

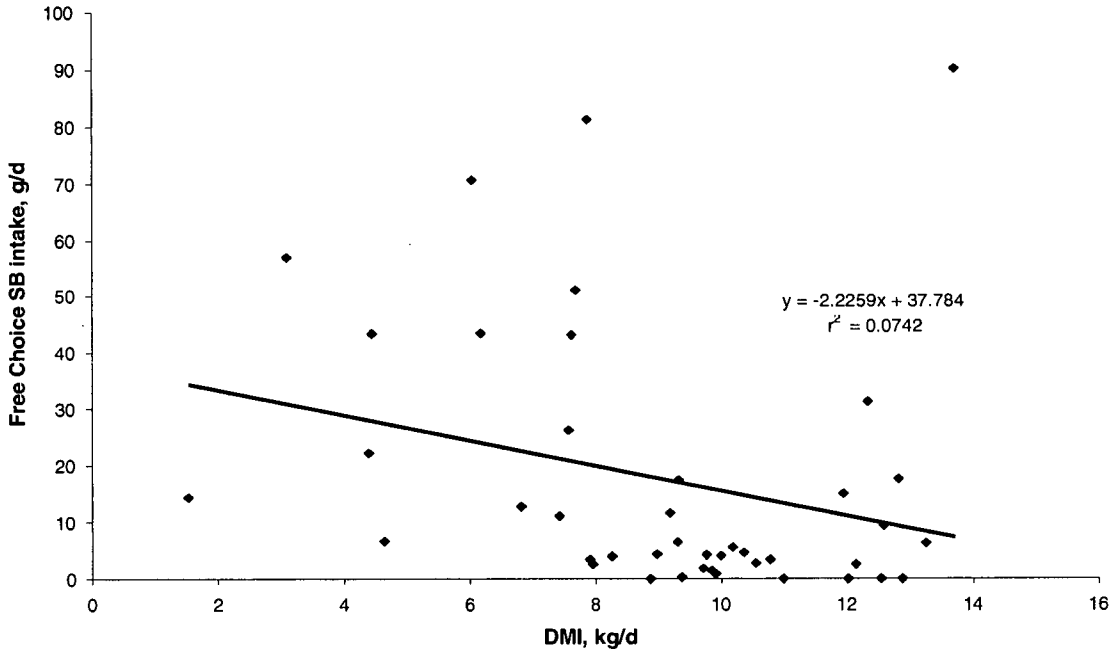
Daily DMI and mean ruminal pH on d 11 to 13 were not correlated for cattle fed the control diet or mix SB ($P > 0.05$). However, a negative correlation between DMI and ruminal pH was observed for cattle fed free choice SB ($P = 0.02$, $r^2 = 0.28$). Further analysis revealed that this correlation was predominately due to cows ($P = 0.04$, $r^2 = 0.47$) and not steers ($P > 0.05$).

The correlation between SB intake and mean ruminal pH on d 11 to 13 was examined. However, for the SB free choice treatment, only data for steers was used for the correlation, because SB intakes of cows were so low. Daily intakes of SB and ruminal pH were not correlated for free choice SB or mix SB ($P > 0.05$).

A moderate correlation between daily DMI and daily free choice SB intake on d 1 to 14 occurred ($P = 0.002$, $r^2 = 0.16$), with higher intake of free choice SB occurring in animals with low or reduced DMI. Cows showed a trend towards an inverse relationship between DMI and

free choice SB intake ($P = 0.08$, $r^2 = 0.07$; Figure 2.4A), with a stronger relationship observed for steers ($P = 0.0004$, $r^2 = 0.27$; Figure 2.4B).

A)




B)

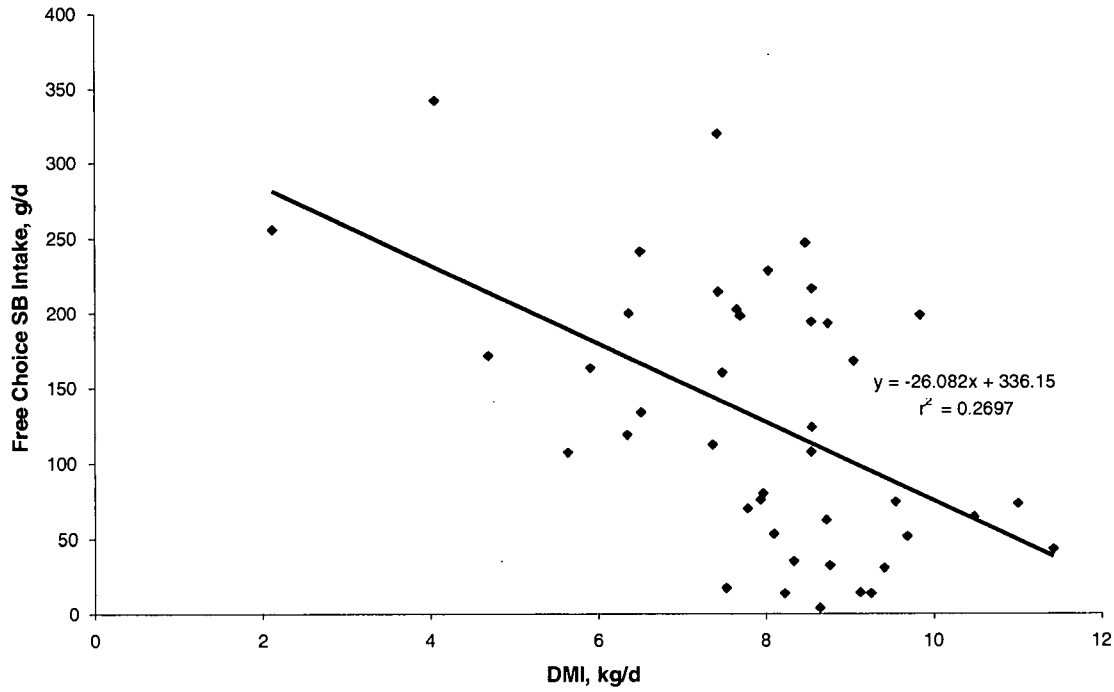



Figure 2.4. The correlation between daily DMI and SB offered free choice on d 1 to 14 for A) non-lactating Holstein cows (n = 3) ($P = 0.08$) and B) Jersey steers (n = 3) ($P = 0.0004$) (Exp. 2). Higher intake of free choice SB occurred when DMI was reduced. Cows had numerically greater average DMI for free choice SB compared to steers (9.2 vs. 7.9 kg/d), however, steers had a greater mean intake of free choice SB than cows (129.1 vs. 17.4 g/d).

The variation in ruminal pH (pHVAR) averaged 0.22 for cows and steers and did not differ by treatment (Table 2.3). Treatment had no affect on the range in ruminal pH (pHDIFF), which averaged (± SEM) 1.21 ± 0.12 for cows and steers.

**Ruminal Fermentation**

Treatment had no effect on total VFA ($P = 0.82$; Table 2.5). For the individual VFA proportions and the acetate to propionate ratio, there were no treatment differences detected. Lactate concentrations for all treatments were below the threshold of detection (< 2 mM). Rumen fluid osmolality was similar among treatments ($P = 0.41$) and steers had higher osmolality than cows (338 vs. 326 mmol/L; $P = 0.04$).

**Table 2.5.** Mean ruminal fermentation for non-lactating Holstein cows and Jersey steers (n = 6) fed the control diet with no sodium bicarbonate (Control), free choice sodium bicarbonate (Free Choice SB) or sodium bicarbonate mixed into the TMR (Mix SB) (Exp. 2).

| Item | Control | Free Choice SB | Mix SB | SEM | *P* |
|---|---|---|---|---|---|
| VFA | | | | | |
| Total, mM | 116.8 | 114.1 | 119.3 | 11.4 | 0.82 |
| Acetate (A), % | 53.1 | 53.2 | 51.8 | 2.8 | 0.92 |
| Propionate (P), % | 28.5 | 30.5 | 28.2 | 2.4 | 0.76 |
| Isobutyrate, % | 1.1 | 1.1 | 0.9 | 0.1 | 0.37 |
| Butyrate, % | 12.3 | 10.7 | 13.3 | 2.9 | 0.63 |
| Isovalerate, % | 2.3 | 2.1 | 2.5 | 0.3 | 0.46 |
| Valerate, % | 2.3 | 2.1 | 2.8 | 0.7 | 0.77 |
| Caproic, % | 0.4 | 0.3 | 0.4 | 0.1 | 0.70 |
| A:P | 1.9 | 1.8 | 1.9 | 0.2 | 0.86 |
| Lactate, mM | ND[a] | ND | ND | | |
| Osmolality, mmol/L | 327 | 333 | 336 | 4.5 | 0.41 |

ND[a] = not detectable.

**Blood**

There were no treatment effects for any of the blood variables measured (Table 2.6). Packed cell volume percentages were lower for cows than steers (42.3 vs. 52.0%; $P = 0.05$). A treatment by group interaction ($P = 0.06$) was detected for blood osmolality. Cows blood osmolality was highest for control (292 mmol/L) compared to free choice SB (288 mmol/L) or

mix SB (289 mmol/L), whereas for steers, blood osmolality was highest for free choice SB (291 mmol/L) compared to control (288 mmol/L) or mix SB (288 mmol/L).

**Table 2.6.** Mean blood variables for non-lactating Holstein cows and Jersey steers (n = 6) fed the control diet with no sodium bicarbonate (Control), free choice sodium bicarbonate (Free Choice SB) or sodium bicarbonate mixed into the TMR (Mix SB) (Exp. 2).

| Item | Control | Free Choice SB | Mix SB | SEM | *P* |
|---|---|---|---|---|---|
| pH | 7.38 | 7.39 | 7.39 | 0.01 | 0.57 |
| $HCO_3$, mmol/L | 26.5 | 27.8 | 28.0 | 0.9 | 0.46 |
| $pCO_2$, mmHg | 44.9 | 45.5 | 45.9 | 1.2 | 0.80 |
| $pO_2$, mmHg | 36.7 | 45.3 | 40.0 | 3.5 | 0.75 |
| Osmolality, mmol/L | 291 | 289 | 289 | 0.8 | 0.39 |
| PCV, % | 46.5 | 43.2 | 51.8 | 0.3 | 0.25 |
| Magnesium, mg/L | 17.4 | 18.4 | 20.4 | 2.1 | 0.59 |
| Amylase, 16 units/L | 12.0 | 12.7 | 13.5 | 3.6 | 0.96 |
| Glucose, g/L | 0.8 | 0.8 | 0.8 | 0.1 | 0.90 |
| Calcium, mg/L | 77.4 | 79.5 | 83.7 | 6.9 | 0.81 |
| Potassium, mmol/L | 4.3 | 4.2 | 4.3 | 0.1 | 0.55 |
| LDH, units/L | 1368 | 1278 | 1196 | 157 | 0.74 |

## DISCUSSION

The cattle in this study exhibited signs of SARA including occasional diarrhea (personal observation), fluctuating feed intake, and low ruminal pH (Braun et al., 1992; Phy and Provenza, 1998, Ghorbani et al., 2002). The incidence and severity of SARA observed in this experiment was typical of feedlot cattle adapted to barley based high grain diets (Ghorbani et al. 2002).

Sodium bicarbonate was provided either free choice or mixed into the TMR in an attempt to minimize SARA. For cattle receiving SB as part of the TMR, the supplementation rate was within the recommended range of 0.6 to 0.8% of DMI (NRC, 2001). When SB was offered free choice, the Jersey steers (Exp. 2) consumed SB free choice at levels (129.1 g/d) that exceeded NRC (2001) recommendations, but interestingly the animals in Exp 1 and cows (Exp. 2) consumed far less. Similarly, Keunen et al. (2003) reported that only on one day of their entire study was a single Holstein cow able to consume sufficient free choice SB to meet the

recommended NRC (2001) levels. Ensuring high intake of SB may be a limitation to providing free choice access to SB, even when the SB is mixed with dried molasses to improve its palatability (Coppock et al., 1986).

Regardless of method of delivery, SB did not elevate mean ruminal pH. The effects of feeding SB on ruminal pH have been inconsistent in previously published literature. For example, Hart and Polan (1984) reported for calves fed a corn based diet with added SB (1.5, 3.0, and 4.5% of DM), ruminal pH was not affected, however, pH values remained relatively high in that study. Similarly, Russell et al. (1980) reported ruminal pH of steers fed a finishing diet based on corn and supplemented with 0.9% SB averaged 6.5 and did not differ from control. In contrast, other studies have reported that the addition of buffers to high concentrate diets increased ruminal pH. Ghorbani et al. (1989) reported that adding 1% SB to a lactating dairy cow diet (60% corn grain) significantly increased mean ruminal pH (from 6.06 to 6.24). Zinn (1991) reported feedlot cattle fed a finishing diet of steam-flaked corn supplemented with 0.75% SB had a higher mean ruminal pH (5.87) compared to cattle fed a non-buffered diet (5.83). The variable responses of cattle to SB added to high concentrate diets are likely due to the variation in quantities of SB consumed and the extent that pH is depressed by feeding the control diet, with greater effects expected when pH is low.

Time and area below ruminal pH thresholds were used to characterize SARA (Mackie and Gilchrist, 1979; Cooper and Klopfenstein, 1996; Keunen et al., 2002). Schwartzkopf – Genswein et al. (2003) considered SARA to exist when ruminal pH fell below 5.8 for a total of 12 h/d, and by this definition, only the cows in our study exhibited SARA. Further, SB provided either free choice or mixed into the TMR had no effect on the total time and area that ruminal pH was below the pH thresholds of acidosis. Similarly, Keunen et al. (2003) provided SB free choice and observed no affect on time and area below ruminal pH 6.0 and 5.6.

The definition of SARA used by Schwartzkopf – Genswein et al. (2003) was refined in this study to include the duration and number of individual bouts of SARA occurring within a day. It is well documented that pH below 5.8 is detrimental to growth of fibrolytic bacteria in the rumen, with long periods of low pH having more severe effects than frequent, short periods of low pH (Calsamiglia et al., 2002). In our study, a bout of SARA was defined as the continuous duration that ruminal pH was equal to or less than pH 5.8. Bouts of SARA were further categorized as severe (< pH 5.5) to indicate their potential to be detrimental to rumen function and health of the animal (Keunen et al., 2002; de Veth and Kolver, 2001). The main benefit of providing SB free choice or mixed into the TMR was a tendency for the duration of severe SARA (pH 5.5) bouts to be shorter compared to those of control. When comparing the effectiveness of method of providing SB, free choice SB was more effective in reducing SARA because the reduction in the duration of severe acidosis bouts was not accompanied by a concomitant increase in the number of bouts, as was the case for animals fed the mix SB diet.

Ruminal pH patterns remained relatively consistent from day to day, however, wide ranges in ruminal pH within a day were observed for all animals irrespective of treatment. Daily fluctuations in ruminal pH typically increase when cattle are fed high grain diets (Cooper and Klopfenstein, 1996; Hristov et al., 2001). The variation and magnitude of the daily ruminal pH fluctuations observed in the present study (pHVAR and pHDIFF) were comparable to values for cattle fed a 92.5% grain diet (Cooper and Klopfenstein, 1996), but providing SB had no effect on minimizing these fluctuations.

After feeding, the pH tended to remain constant or increase, which is in contrast to the pattern typically reported for cattle fed once daily. Ruminal pH profiles usually decrease following feeding reaching nadir 8 to 16 h post feeding, then subsequently increase overnight, peaking prior to feeding (Beauchemin et al., 2001; Keunen et al., 2002; Nocek et al., 2002). The atypical ruminal pH profiles observed in this study may be related to heat stress. Cattle were fed

in the afternoon when temperatures were as high as 32°C, but they tended to consume little at this time. Lowest pH values occurred between 2300 and 0400, approximately 9 to 13 h post feeding. Ruminal pH gradually increased and peaked at around 0800 and then slowly declined again until the afternoon feeding. Sodium bicarbonate, offered either free choice or mixed into the diet, had no affect on the diurnal pattern of ruminal pH.

Keunen et al. (2003) subjected dairy cows to an acidosis challenge by replacing 25% of the TMR with pellets containing 50% ground barley and 50% ground wheat. The cows were then offered free choice SB. Despite cows exhibiting SARA (average ruminal pH was 5.86), free choice SB was not effective in raising ruminal pH because intakes of SB were low (26.8 g/d), attributed to the adverse organoleptic properties of SB. In the present study, even when intake of SB exceeded the recommended level, there was no correlation between SB intake and ruminal pH, indicating that factors other than SB intake, such as feed intake, were primarily influencing ruminal pH. The inverse relationship between DMI and ruminal pH indicated that animals with high feed intakes were at greater risk for acidosis. Thus, the differences among cows and steers for time and area below ruminal pH 5.8 and 5.5 likely resulted from cows having a higher feed intake. The higher DMI of cows also led to the higher SB intakes when it was mixed into the diet.

Reductions in feed intake are common when cattle are fed high grain diets and experience SARA. In this study, offering SB free choice or mixed into the diet did not attenuate the negative effects of SARA on DMI. The effects of SB on DMI have been inconsistent in previous studies. While some work has reported no effect of SB on DMI (Hart and Polan, 1984; Coppock et al., 1986; Ghorbani et al., 1989), other researchers have reported that dietary buffers improved feed intake and increased animal performance (Fulton et al., 1979b; Solorzano et al., 1989; Zinn, 1991). Keunen et al. (2003) reported that offering free choice SB to dairy cows with SARA did

not increase DMI, similarly observed in our study despite the high intake of free choice SB consumed by steers.

The cyclical or erratic feeding pattern often displayed by cattle exhibiting SARA was apparent in this study as indicated by the significant effect of day on DMI. It has been suggested that cattle adjust feed consumption and intake patterns in attempts to attenuate SARA and regulate high levels of VFA in the rumen (Slyter, 1976; Owens et al., 1998). Fluctuating feed intake patterns have been documented in other studies with ruminants fed high grain diets (Fulton et al., 1979a; Cooper et al., 1999). Ghorbani et al. (2002) reported that the reductions in feed intake of steers fed barley grain diets experiencing SARA lasted for a single day before recovering, as was similarly observed in this study. Providing SB free choice or mixed into the diet did not reduce the daily variation in DMI for Exp. 1 or 2.

The decline in ruminal pH post feeding was predominantly due to VFA accumulation and not lactate, as ruminal lactate concentrations were below levels of detection (< 2mM). This has been reported in other studies with cattle fed diets containing barley grain (Hristov et al., 2001; Ghorbani et al., 2002). Because high levels of lactate were not detected, we concluded that the adaptation phase provided sufficient time for microbial adaptation to the high concentrate diet to occur. Sodium bicarbonate, either offered free choice or mixed into the TMR had no affect on total VFA concentrations, similar to Hart and Polan (1984), but in contrast to others (Ha et al., 1983; Hart and Doyle, 1985). In addition, SB did not affect the molar proportions of individual VFA, similarly reported in other studies (Horn et al., 1979; Ha et al., 1983; Solorzano et al., 1989). In contrast, others have observed buffers increased acetate, valerate, isovalerate and butyrate concentrations (Fulton et al., 1979ab; Russell et al., 1980; Hart and Doyle, 1985), reduced propionic acid (Russell et al., 1980; Coppock et al., 1986; Zinn, 1991) and widened the acetate to propionate ratio (Coppock et al., 1986). Lack of treatments effects on VFA suggests

that supplementation with SB likely had negligible effects on the ruminal microbial population and feed digestion.

Clinical diagnosis of metabolic acidosis relies on blood pH levels equal to or less than 7.35 (Owens et al., 1998). Changes in systemic acid-base status have been reported to be minimal during SARA because the ruminal pH eventually recovers during the day and homeostasis of blood is maintained (Burrin and Britton, 1986; Goad et al., 1998; Brown et al., 2000). In the present study, blood pH and gases were similar to values reported for steers with SARA (Brown et al., 2000). Even though the cattle in our study exhibited periods of severe SARA, they did not experience metabolic acidosis. However, it must be recognized that blood samples were only collected once per period at a time that did not coincide with nadir ruminal pH.

Blood metabolic profiles of cattle in this study (Exp. 2) were similar to those reported for steers diagnosed with SARA (Bigner et al., 1997; Brown et al., 2000). Packed cell volume readings were higher in our study compared to those reported for steers with SARA (Brown et al., 2000; Ghorbani et al., 2002), likely because cattle in this study had higher ruminal osmolalities. Providing SB had no affect on blood variables, consistent with some studies (Ha et al., 1983; Ghorbani et al., 1989), but not others (Aslam et al., 1991; Bigner et al., 1997).

## IMPLICATIONS

Subacute ruminal acidosis is prevalent in cattle fed barley based finishing diets. Cattle with high feed intakes were at greater risk for acidosis. Providing sodium bicarbonate did not eliminate ruminal acidosis, but it tended to reduce the duration of individual bouts of severe SARA (below pH 5.5). Sodium bicarbonate provided free choice was more effective in reducing ruminal acidosis than mixing it into the diet. However, intake of free choice sodium bicarbonate was highly variable amongst animals, but not related to acidosis.

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# CHAPTER III: CONCLUSION

The feedlot industry is managed such that consumer demand for grain fed beef is met in an economical manner, whereby production costs are minimized while the product, in this case, beef, is maximized through nutritional programs that are designed to increase the weight gain of the animal in short periods of time. In addition, feedlot cattle are often monitored on a per pen basis rather than on an individual basis, particularly in large feedlots that often have 100 cattle or more per pen. Thus, it is not uncommon for individual animals experiencing SARA to go undetected as their symptoms are masked by the feeding patterns of the large group (Huntington, 1988; Cooper and Klopfenstein, 1996; Owens et al., 1998). Scientific research in the area of feedlot cattle nutrition has tended to focus on limiting metabolic disorders, such as acidosis, so that animal performance is not hindered and producer profitability is maintained. Reducing acidosis so that the welfare of the beef animal is improved is another advantage of this research.

This thesis illustrated that the behavioral responses of cattle in both experiments were similar to responses reported in other experiments involving animals afflicted with ruminal acidosis, signifying that they were likely experiencing SARA. For example, kicking at the belly, a sign indicative of abdominal pain according to Radostits (1994), was observed by the heifer calves used in Exp. 1. Lethargy, dull appearances, and eating dirt were also observed during visual observations of the heifer calves in Exp.1. Teeth grinding was reported to occur in acidotic sheep (Braun et al., 1992), a behavior also observed for several individuals in Exps. 1 and 2, perhaps adopted as a pain coping mechanism or as an aversive response to the high grain diet. All animals used in experiments described within this thesis were observed to have occasional bouts of diarrhea, consistent with other studies (Stock and Britton, 1993; Phy and Provenza, 1998; Stock and Britton, 2002). Although not previously documented in the literature as being associated with acidosis, I frequently observed the heifer calves in Exp. 1 chewing the wood

fence of their pens, possibly in attempt to satisfy fiber cravings or in response to being fed a diet that was to some degree unpalatable to them.

In this study, the diet was supplemented with SB, either by mixing it into the TMR or offering it free choice, with the objective of reducing the incidence of SARA in cattle fed barley grain based diets, and ultimately to improve feedlot cattle welfare. Although supplementing with SB did not elevate mean ruminal pH or improve feed intake, some benefits of consuming SB were realized. In this study, the definition of SARA previously documented in Schwartzkopf – Genswein et al. (2003) was refined to include the duration and frequency of bouts within a day, and in doing so, we established that providing SB free choice reduced the severity of acidosis by limiting bout duration and frequency, thus demonstrating that SB may provide some benefit as a nutritional management strategy. However, it is unlikely that this corrective measure would eliminate acidosis and improve cattle welfare.

Additionally, results from metabolic feedlot trials on the effectiveness of using buffers as a method of reducing acidosis in ruminants fed high grain diets have been inconsistent, as described in Chapter I. The buffering capacity of SB is dependant on various aspects, such as quantity consumed, time of SB ingestion in relation to feed intake, type and level of grain in the diet (Slyter, 1976), as well as $pCO_2$ in the rumen (Kohn and Dunlap, 1998). Individual animal traits will also influence an animal's response to buffers (Slyter, 1976; Radostits, 1994). Thus, further research is required to more accurately determine the effectiveness of supplemental SB, either mixed into the diet or offered free choice, on minimizing acidosis in relation to the source of rapidly fermentable carbohydrate in the TMR. There is a need to further test NRC (2001) recommended guidelines for buffer supplementation to beef feedlot cattle and how this corresponds to different grain sources. Research is essential to determine the relationship between feeding behavior and the intake of free choice SB (times within meals, duration, frequency and quantities of SB consumed) to determine when cattle will actively seek out and

consume SB relative to meals. It has been suggested that buffer additions may only be useful during the early adaptation phase of high concentrate feeding and lack of a sustained response is indicative of adjustment to the bicarbonate load by the animal (Russell et al., 1980; Wheeler, 1980; Ha et al., 1983). Therefore, a study examining the effectiveness of supplemental SB in minimizing acidosis while beef calves are being adapted to feedlot diets is required. The work undertaken in this thesis illustrated that intake of SB free choice is highly variable even when the palatability was improved. Thus, cattle may not consume SB when offered it free choice despite the fact they are experiencing SARA. Further palatability trials are necessary to pinpoint alternative flavor preferences.

Reflections on improving this study include allowing a longer adaptation period for Exp. 2 cattle to the final 80% barley grain level prior to exposing animals to treatments. As described in Chapter II, the Jersey steers had previously been exposed to a high grain diet while the Holstein cows had not, which may have been reflected in the various group differences that were detected in Exp. 2. Increasing animal numbers may have resulted in a greater number of animals that would consistently consume SB free choice in quantities similar to that observed for the Jersey steers (Exp. 2). It would have been ideal to use newly weaned beef calves in Exp. 2 to simulate a typical feedlot scenario, however, this was not an option at the time that Exp. 2 occurred. Implementing the use of feed scales to measure feed intake hourly instead of daily would have been useful to try and substantiate that periods of elevated ruminal pH occurred following periods of reduced feed intake. Lastly, the impact of reduced bout duration and frequency needs to be determined in terms of the long term affects on animal performance and health, thus, longer period duration and greater than 3 d of ruminal pH monitoring would have allowed us to gain further insights.

To conclude, improvements to the welfare of beef feedlot cattle are more likely to be achieved by altering current feedlot nutritional programs so that beef cattle are fed higher

percentages of forage in finishing diets and cattle are finished over a longer time period and have slower growth rates. However, changes to the current feeding strategy are unlikely as economically, grain prices are lower and easier to handle and mix in the diet compared to forage sources, which tend to be bulky, require more effort to preserve, and provide less energy (Stock and Britton, 1993).

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