academicresearchJournals

Vol. 6(4), pp. 243-250, May 2018
DOI: 10.14662/ARJASR2018.019

ISSN: 2360-7874
http://www.academicresearchjournals.org/ARJASR/Index.htm

Academic Research Journal of Agricultural Science and Research

Full Length Research

Review on Sub-Acute Rumen Acidosis in Dairy Cattle and Nutritional Approaches

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Accepted 14 May 2018

Different studies revealed that, Sub-acute ruminal acidosis (SARA) is one of the most important metabolic diseases in modern dairy industry that impairs cow performance and health even well managed and high yielding dairy cows. It is characterized by repeated bouts of depressed rumen pH between 5.2 and 5.6. SARA in dairy cattle causes a disorder of ruminal fermentation caused by the ingestion of large amounts of concentrates and inadequate amounts of fibre administered in order to increase the milk production in early lactation. Furthermore, it has concern of animal welfare reasons due to lameness and laminitis impact significantly on cow comfort and general well-being. Cows in the early lactation, primiparous cows, as well as cows grazing or fed with rapidly fermentable low fibre grass are in particular exposed to high risk to develop SARA. The SARA has diverse and complex consequences, which include feed intake depression, fluctuations in feed intake, reduced diet digestibility, reduced milk yield, reduced milk fat percent, gastrointestinal damage, liver abscesses, and lameness. The suitable approach to prevent SARA is formulating adequate fibre in the diets, preparing diets with adequate particle size distribution and moisture content to reducing sorting. Feeding supplements such as yeast and exogenous buffer can be considered to stabilize rumen pH. The risk of developing SARA can be reduced by adopting a feeding regime, which balances ruminal buffering with the production of volatile fatty acids from fermentation of carbohydrates. Recommendations made for effective characterization, important management factors and good management practices of SARA in dairy cattle are further highlighted.

Key words: dairy cow, sub-acute rumen acidosis, Nutritional impact of SARA

Cite this article as: Negash D (2018). Review on Sub-Acute Rumen Acidosis in Dairy Cattle and Nutritional Approaches. Acad. Res. J. Agri. Sci. Res. 6(4): 243-250

INTRODUCTION

Cattle in the mixed crop-livestock system of Ethiopia play an important role in the provision of products and by-products, which also include services in crop cultivation processes. Crop production in Ethiopia typically uses a pair of oxen, while cows are kept for breeding and milk production. Cow milk is among the ten most important agricultural commodities in the country, and was ranked as primary animal product in terms of quantity produced (FAOSTAT, 2009). Considering the important prospective for smallholder income generation and employment opportunities from the high value dairy Products, the development of the dairy sector can contribute immensely to poverty alleviation and improved nutrition in the country. In highland areas, income earned from daily milk production is used to purchase agriculture inputs or hire labor and land, effectively increasing a household's food production potential. Although the daily income earned is marginal, especially from low milk producing local breed animals, milk sales and livestock ownership is necessary for food security (Yilma, 2010).

Evidences from different studies provided that sub-acute ruminal acidosis (SARA), also known as chronic or sub-clinical acidosis, is a well-recognized digestive disorder that is an increasing health problem in most dairy herds. Many studies indicate a high prevalence of SARA in high-producing dairy herds as producers respond to the demands for increased milk production with higher grain, lower fibre diets that maximize energy intake during early lactation. If SARA is experiencing in dairy herds these is a decreased efficiency of milk production, impaired cow health and high rates of involuntary culling. The economic cost associated with SARA can be staggering. The challenge for dairy farmers and dairy nutritionists is to implement feeding management and husbandry practices that prevent or reduce the incidence of SARA, even in high-producing dairy herds where higher levels of concentrate are fed to maximize energy intake. Russell and Rychlik, 2001 revealed that ruminal acidosis can be a direct human health concern as well. Low ruminal and intestinal pH due to high grain feeding increases the risk for shedding entero hemorrhagic E. coli such as 0157:H7. Switching cattle to a high forage diet just prior to slaughter decreases this shedding.

According to De Brabander *et al.,* 2002 the average milk production per cow has increased dramatically over the last 20 years. Not only genetic improvement, but also changed feeding management as basis for optimal production has contributed to this development. Use of total mixed rations and increased energy supply are today's standard in successful dairy farming. Here, the proportion of energetic dense feed components is often maintained at the highest level possible to deliver proper energy supply for the high producing dairy cow. The complexity of managing a large dairy herd and the tendency of cattle to prefer concentrate rather than structured components, however, does not always deliver the necessary intake of feedstuffs high in structure. This may lead to an imbalance of energy with the proportion of structure, a component of crucial importance for the ruminant digestive system. The rise of acid in the rumen originating from carbohydrates and the decreasing buffering capacity of structured feeding components lead to an acidic ruminal environment. Already in the early 1960's, the tendency of increasing the carbohydrates providing necessary energy supply at the cost of roughage-components has been characterized as putting the animals at the edge between maximal productivity and illness due to acidosis (Dunlop, 1961). Although today modern dairy farming is based on research and feeding technology, it has to be expected, that non-acute forms of ruminal acidosis are indeed present in high-producing dairy herds. These forms are believed to contribute to milk fat depression (MFD), laminitis and other impairment of health status, finally leading to increased culling rates on herd-level (Oetzal, 2000). In the last years, this form of non-acute ruminal acidosis in dairy farms has been described as sub- acute ruminal acidosis (SARA).

Although it has been characterized in a number of publications, the real prevalence could only be estimated. A field study in the U.S. in the early 1990's revealed a SARA-prevalence of up to 26% in the whole study population of 15 dairy farms reaching as much as 40% on single farms (Garrett *et al.,* 1997). This would furthermore enable researchers to establish the possible influence of SARA on milk production and evaluate the proposed diagnostic schemes made for the evaluation of SARA-prevalence. Thus, SARA is the most important nutritional disease and represents a significant concern as it can negatively impacts the dairy industry by decreasing dry matter intake, milk production, profitability, and increasing culling rate and death loss (McCann *et al.,* 2016). However, there is paucity of well documented information on its general aspect and consequence in dairy cattle. Therefore, the main objectives of this manuscript were to review the general aspects of Sub-acute ruminal acidosis including it clinical sign, diagnostic methods and its consequence in dairy cattle by focusing on both past and recent researches at global prospective.

WHAT is SARA?

The data from different studies explain that SARA is a disorder of ruminal fermentation that is characterized by extended periods of depressed ruminal pH below 5.5-5.6. Ruminal fluid pH is a measure of the acidity or alkalinity of ruminal contents. A lower pH means higher acidity. For optimum ruminal fermentation and fibre digestion, ruminal pH should lie between 6.0 and 6.4, although, even in healthy cows, ruminal pH will fluctuate below this level for short periods during the day. This drop in ruminal pH is a result of the breakdown of dietary carbohydrates (e.g. starch), particularly from cereal grains such as corn and barley. Grains are high in readily fermentable carbohydrates that are rapidly broken down by ruminal bacteria, leading to the production of volatile fatty acids (VFA) and lactic acid. Under normal feeding conditions, VFA are readily absorbed by papillae (small finger-like projections) on the rumen wall. Once absorbed, VFA enter the cow's bloodstream and can be used for milk production. A graph of rumen pH is shown in Table 1, from an experiment that fed limited amounts of grain to simulate SARA.

Different studies stated that during periods of SARA in dairy cows, lactic acid in the rumen is rarely the cause of depressed pH, unlike the acidosis condition seen in feedlot cattle. SARA results from excessive VFA production that exceeds the ability of the ruminal papillae to absorb them. Volatile fatty acids accumulate in the rumen and, as a result, reduce ruminal pH. A common cause of SARA occurs at calving when dry cows are abruptly switched from high fibre diets to higher concentrate milking cow diets. An abrupt dietary change does not allow ruminal bacteria and ruminal papillae adequate time to adjust, thereby leading to the rapid production and accumulation of VFA. Another common cause of SARA is improperly balanced or mixed rations, such that effective fibre content falls below recommended levels or particle size is too small. This suppresses rumination (cud-chewing) and the production of saliva, which can buffer changes in ruminal pH.

OCCURRENCE of SARA

Subacute acidosis is caused by the accumulation of volatile fatty acids (VFA) in the rumen. As feed is digested, VFA (acetate, propionate, butyrate) are produced. The pH in the rumen drops if VFA production is rapid and exceeds the capacity of the rumen to maintain equilibrium. With time, the VFA are absorbed, buffered, or passed from the rumen, causing the pH to rise. As a consequence, a cyclical pattern of pH occurs causing periods of SARA. During SARA, lactic acid concentrations remain very low (< 1 mM).(Karen A. 2016).

There are probably 4 types of cattle at high risk of developing SARA: transition animals, high DMI animals, and those subject to either a high degree of vari ability in their ration and meal patterns, or to poorly formulated diets. Transition animals have been considered to be more prone to developing SARA if their rumen bacterial populations and papillae have not been gradually acclimated to a higher starch ration prior to freshening. Rumen papillae significantly increased in size and ability to absorb VFA when animals were switched from a diet mainly of hay and straw (70% NDF, converted from crude fiber according to (Mertens, 1992) to a higher energy diet containing a mixture of grass hay and grain 2 wk prior to freshening .

WHAT are the SYMPTOMS of SARA?

Dairy cows experiencing SARA often do not exhibit any clear, overt clinical symptoms. Often, the most common clinical sign associated with SARA is reduced or erratic feed intake. Dairy cows experiencing an episode of SARA will reduce their feed intake in order to reduce the acid load in their rumen. Cows will start eating again when ruminal pH is above 5.6. This results in erratic feed intake that often goes unnoticed in individual cows experiencing SARA, particularly in large dairy herds where cows are housed and fed in groups. Other clinical signs often observed during SARA may include:

- reduced rumination (cud-chewing)
- mild diarrhea
- foamy feces containing gas bubbles
- appearance of undigested grain (> 1/4 in. or 6 mm) in feces.

In the long term, dairy herds experiencing SARA usually exhibit secondary signs of the disease, usually 3-6 months after an episode of SARA. These secondary signs include episodes of laminitis, weight loss and poor body condition despite adequate energy intake, and unexplained abscesses. Undiagnosed, the secondary health effects of SARA can lead to high herd culling rates. SARA should be investigated as a cause, if the secondary signs are occurring for no apparent reason.

LAMINITIS

Laminitis is the most common clinical sign which is repeatedly mentioned with subacute ruminal acidosis (SARA) in cattle (Mathew and Ajithkumar, 2014). It imparts an important role in dairy farming by considerable economic loss, impaired animal welfare and pre-disposition to other diseases (Nelson and Cattell, 2000). Cattle fed with a diet high in concentrates are more prone to laminitis (Kelly and Leaver, 1990). The feeding regime in which frequency as well as quantity of concentrate feed has a certain influence and more concentrate in a shorter period of time will cause locomotive pathology.

SARA can cause release of certain vasoactive substances like histamine, lipopolysaccheride endotoxins in to the circulation by the disintegration of Gram negative bacteria. It will cause injury to microvasculature of corium followed by hypoxia to extremities leading to laminitis (Plaizier *et al.,* 2009). Body condition of dairy cows is having a certain relation in the development of non-infectious laminitis. Over conditioned cows at drying off period are at greater risk for foot problems while cows experiencing moderate net energy balance are at slightly lower risk of having non-infectious lameness. Hoof discoloration, ulcers, abscesses, sole haemorrhages and misshapen hooves have been observed as a complication of SARA in cattle (Oetzel, 2000). The existence of SARA should be examined in herds having reports of clinical lameness.

HOW is SARA DIAGNOSED?

On a herd basis, SARA can be a difficult condition to diagnose because its appearance is subtle. Depressed milk-fat content is commonly used as a diagnostic tool for

SARA. The basis for this is that low pH suppresses fibre digestion in the rumen and the end products of fibre digestion are necessary for milk-fat synthesis. Normal milk-fat test in Holstein dairy cows is around 3.5%, so a milk-fat test of < 3% can indicate SARA. However, bulk tank testing of milk fat is inappropriate to diagnose SARA at the herd level. Individual cows with SARA may have a low milk-fat test, but pooling their milk with that of the rest of the herd will mask detection of SARA. Individual milk-fat tests provide a better indicator of SARA.

The only reliable and accurate diagnostic test for SARA is measuring ruminal fluid pH. While stomach tubing has been used on-farm to collect ruminal fluid samples for pH measurement, the results are often inaccurate due to contamination with saliva. A practical method to obtain ruminal fluid samples under field conditions is a technique called rumenocentesis. Rumenocentesis, sometimes referred to as percutaneous needle aspiration, involves inserting a needle (16-gauge, 5 in. long) into the ventral rumen, and withdrawing a sample of ruminal fluid using a 10-mL syringe. Because of its invasive nature, it is recommended that only a qualified veterinarian do this procedure. The time of ruminal fluid sampling relative to feeding is important to allow proper interpretation of results (See Table 1).

The research done on different cows show that only sample cows within 60 days of calving, as these are most at risk of SARA. To account for cow-to-cow variation, which is usually considerable, sample at least 10 cows per feeding group. the result revealed that >30% of the cows sampled have ruminal pH less than or equal to 5.5, consider the whole feeding group as experiencing SARA. Review and make appropriate changes to the feeding management and husbandry practices. some authors say that pH values between 5.6 and 5.8 are considered marginal. Ruminal pH values greater than or equal to 5.8 are considered normal.

IMPACT of RUMINAL ACIDOSIS

It can be difficult to identify animals suffering from subacute acidosis because the clinical signs are not unique to acidosis. According to Donovan,1997 cows with acidosis can experience diarrhea, weight loss, reduced milk production, and increased susceptibility to other metabolic disorders. Ruminal acidosis is a major problem for the North American dairy industry (Krause and Oetzel, 2006) costing between $500 million to $1 billion a year Financial losses occur due to treatment of sick animals, reduced productivity, and increased feed costs due to poor fiber digestion and lower feed efficiency (Donovan, 1997)..

Poor Health and Increased Lameness

Lameness is a multi factorial condition and the most important welfare problem in dairy cows. Lameness is also regarded as a cause of economic loss owing to a reduction in milk yields, lowered reproductive performance and an increased risk of culling. Farmers are often reported to underestimate the prevalence of lameness, thereby prompting a low perception of its impact on cow welfare, health and production (Horseman, 2014).

According to Krause and Oetzel, 2006 repeated bouts of subacute acidosis can damage the surface of the rumen wall. As expressed by Gohzo *et al.,* 2005 once the rumen wall is damaged, bacteria and the toxins produced by bacteria can enter the portal circulation, causing liver abscesses and an inflammatory response. In addition, there is increasing evidence that these toxins are implicated in laminitis. They also find that, the hoof, the horn (or exterior surface) is joined to the major bone in the hoof (pedal bone) by highly vascularized connective tissue (corium), which acts as a shock absorber when the hoof comes into contact with the ground.

Mungall *et al.,* 2001 say that, during laminitis, the mechanical strength of the connective tissue within the hoof is disrupted which allows the bone to rotate laterally or sink into the corium of the hoof. Blowey,1993 say that the corium can then shift laterally, expanding the white line, or upwards, causing swelling around the coronary band. Solar compression can lead to sole ulceration. The impact of acidosis on laminitis is thought to be mediated by proteinases within the connective tissue that are activated by the bacterial toxins. Once activated, these proteinases degrade the connective tissue within the hoof.

Poor Feed Conversion Efficiency

Different authors agreed with, ruminal acidosis decreases the digestibility of fiber in the rumen, which decreases feed conversion efficiency and increases feed costs. In a study with ruminally and duodenally cannulated cows, we observed that NDF digestion in the rumen declined from 52% for cows with a mean ruminal pH of 6.4 to 44% for cows experiencing repeated episodes of acidosis with a mean ruminal pH of 5.8. This reduction in potential fiber digestion is equivalent to a loss of 2.5 kg/d of milk.

Low Feed Intake

Ruminal acidosis can cause erratic fluctuations in feed intake. Low ruminal pH can causes the cow to go “off-feed,” which reduces the production of VFA, allowing the pH to recover. The cow then resumes a high feed intake that causes excessive production of acids, and the cycle is repeated.

Table 1. Recommended Timing of Ruminal pH Measurements.

Feeding Program	Rumen Sampling Time to Measure pH
TMR fed once daily	5-8 hours after feeding
Forage and concentrate fed separately	2-5 hours after concentrate feeding

Reduced Microbial Protein Synthesis

Ruminal acidosis can lowers the efficiency of microbial protein production in the rumen (i.e., the amount of microbial protein produced per unit of carbohydrate digested in the rumen). A decrease in microbial efficiency will decrease the yield of microbial protein (g/d), unless more fermentable carbohydrate is supplied, which further increases the risk of acidosis. When microbial protein synthesis is decreased it can increases the need for supplemental feed protein in the diet, which in most cases increases feed costs.

How to Prevent SARA

Once diagnostic testing has identified a dairy herd to be experiencing SARA, it is important to make the appropriate adjustments in feeding and management practices to reduce the incidence of SARA. The rapid or abrupt introduction of fresh cows to high concentrate diets is taken as the most common cause of SARA. Changes in ruminal bacterial populations when exposed to higher concentrate rations require about 3 weeks, and it is recommended that concentrate levels be increased at 5 to 7-day intervals during this period to avoid SARA. When higher concentrate diets are fed takes longer the adaptation of ruminal papillae, becomes approximately 4-6 weeks. Close-up dry cows should consume enough carbohydrates in their ration so that changes in the rumen are minimized when the milking cow ration is offered after calving.

Pay attention to ration formulation, particularly dietary fibre levels. The National Research Council (2001) developed recommendations for the fibre content of lactating cow rations, with a minimum neutral detergent fibre (NDF) requirement of 27%-30% of ration dry matter (DM), with 70%-80% of the NDF being supplied from forage. Supplying 70%-80% of dietary NDF requirement from forage ensures there is adequate "effective" NDF (eNDF) in the ration. Diets with adequate amounts of NDF stimulate rumination or cud chewing, which increases saliva production that buffers ruminal pH. A risk factor for SARA in the dietary content of readily fermentable carbohydrates like starch, sugar and pectin. Balance the dietary level and type of these non-fibre carbohydrates (NFC) to prevent SARA. NRC (2001) recommends that dairy rations contain 35%-45% of ration DM as NFC. NFC sources can be digested at different rates in the rumen, and knowing the relative rates of NFC fermentation of grains can assist in ration formulation. There is a greater risk of SARA when more rapidly fermentable NFC sources such as wheat and barley are fed, and grain processing such as steam flaking or high moisture ensiling increase the rate of NFC fermentation in the rumen. To minimize the risk of SARA while maximizing energy intake, balance your NFC types and sources.

Dietary buffers (e.g., sodium bicarbonate and sodium sesquicarbonate) are commonly added to dairy rations to help manage SARA. Research indicates that the above buffers increase ruminal pH. A feeding rate of 0.75% of ration DM can be recommended. Other preventative measures to avoid SARA include:

- Avoiding of over-mixing or over-processing of the TMR that reduces particle size and eNDF content
- Minimize separation of feed ingredients during TMR mixing and delivery
- when feeding a TMR, monitor and minimize sorting in the feed bunk
- avoiding of "slug feeding" of higher concentrate rations by allowing adequate bunk space or feed provision so as to reduce meal size
- Ensure adequate length of cut for forages and silage.

SARA is a subtle condition in most high-producing dairy herds, leading to unnecessary economic losses. Dairy cattle nutrition can advanced sufficiently over the years to avoid SARA. If you suspect a SARA problem in your dairy herd, work closely with your veterinarian and nutritionist to reduce the incidence of this problem.

Lactic Acid Production Versus Accumulation

Money researchers say that lactic acidosis is the scourge of concentrate feeding for beef and dairy cattle. Excess amount or rate of concentrate consumption favors lactate-producing *bovis*, the “weed of the rumen”. High rate of concentrate although it can metabolize glucose to lactate at one-half the ATP yield per molecule of glucose, it can still metabolize glucose to lactate at least 5 times faster and yield more ATP per unit of time than to volatile fatty acids (**VFA**). Lactic acid is 10-fold more acidic than the VFA per molecule, so the low pH tolerance of *Streptococcus bovis* allows it to outcompete the resident lactate consumers such that while increasing lactate concentration is decreasing the pH, the decreasing pH is further increasing lactate concentration. After further cycling, eventually *S. bovis* is replaced by lactobacilli, further exacerbating the cycle. Lactate can be produced in L and D forms, but the conversion of D to L is very slow, so the buildup of D-lactate in the blood causes acute systemic acidosis. Although Nagaraja and Titgemeyer (2007) document these findings for cattle with acute acidosis, they also explain why subacute rumen acidosis (**SARA**) leads to many problems in feedlot cattle even though lactate concentration in the rumen (and blood) remains only briefly increased and then only peaks at about 5 mM (< 5% of total organic acids). Moreover, induction of acute acidosis only consistently raised lactate concentration when wheat was used, whereas induction of acute acidosis with corn or beet pulp elevated butyrate and propionate, respectively (Lettat *et al.,* 2010).

Grain-induced SARA can associated with reduced bacterial diversity and increased occurrence of *E. coli* phylotypes (). As discussed below, a resilient bacterial community should have many redundant fluid-associated bacterial groups, including those that utilize lactic acid. Khafipour *et al.,* 2009 also steeped barley grain with an equal quantity of water or water plus 0.5% lactic acid. Although Iqbal *et al.,* (2009) described how lactic acid decreased starch digestibility statistically, the difference in effective degradability was trivial (< 1%). As a result the increased in milk fat% resulting from the lactic acid steeping was arguably most physiologically related to a significantly decreased immune response from Gram negative bacteria such as *E. coli* (Iqbal et al., 2010). The same researchers have linked bursts of serum virulence factors from *E. coli* in dairy cattle with grain-induced SARA (Khafipour et al., 2011). Although *E. coli* can considered as a minor player based on its low abundance, fluctuating bursts could augment important systemic responses. They could increase and suddenly die as pH lowers; their lysis leaves behind lipopolysaccharide cell wall fragments that can pass through the rumen epithelial membrane (Emmanuel *et al.,* 2007) but Gozho *et al.,* 2007, says it is not necessarily into blood to trigger a host immune response. To prepare the cow's system for an expected bacterial infection, the system would prioritize energy use for the immune response and thereby decrease fatty acid synthesis in the mammary gland (Zebeli and Ametaj, 2009). Thus, it is not the production or even the long-term accumulation of lactate that is the problem; the cow's rumen microbial population helps to prevent a disruption in her normal rumen function unless the normal "good" bacteria are intermittently challenged by undesirable bacteria such as certain phylotypes of *E. coli*. The rumen contains of a variety of lactilytic bacteria and entodiniomorphid protozoa most of which do not use lactate as their main energy source (Nagaraja and Titgemeyer, 2007). These authors also explained how entodiniomorphid protozoa can store cache dietary starch and isotrichid protozoa can convert dietary sugars to stored glycogen to help "buffer" the rumen unless a lowered pH inhibits them. Martin, 1998, say that organic acids direct-fed microbials (Martin and Nisbet, 1992), and even residual fermentation extract in distillers byproducts. (Fron *et al.,* 1996) can enhance the lactilytic populations of the rumen to help prevent a rapid pH decline in the rumen. According to Nagaraja and Titgemeyer, 2007 as lactate production increases, though, strains of *Megasphaera elsdenii* are probably the most well known to shift from glucose to lactate as substrate such that *S. bovis* rarely increases in abundance.

Henning *et al.* (2010) in his research, selected *M. elsdenii* strains as potential probiotics for dairy cattle to reduce SARA during the shift from high forage to higher grain diets and showed that the treatment improved pH control and concurrently increased molar proportion of butyrate. In a previous study (Klieve *et al.,* 2003), a *M. elsdenii* probiotic established at functional abundance in dairy cattle, but when given enough time to adapt to a shift from a forage- to a grain-based diet, the resident *M. elsdenii* populations in the control group lagged but increased to the same abundance. Additives therefore probably do not necessarily increase the abundance in the longer term, but reduce the transition time needed to increase the abundance of the lactilytic populations. Targeting on the obvious transition phase from a dry cow diet to a lactation ration for such a role, but any feeding situation that promotes variation among cows (e.g., overcrowding) and variation among diets (e.g., forage sorting) promotes intermittent patterns of microbial transition that could lead to unexpected SARA or at least milk fat depression. Although studied with additives, addition of sugars probably also increase the lactilytic populations and thereby helps to buffer against transient bursts of opportunistic sugar fermenters.

CARBOHYDRATES METABOLISM IN THE RUMEN

Carbohydrates contribute 70 to 80 percent of the diet dry matter while protein, fat, and minerals make up the remaining portion. Carbohydrates can be taken as the primary energy source for the cow and supports rumen function and microbial growth. Two carbohydrate categories occur in feeds: cell solubles (sugar and starch) and cell wall (cellulose, hemicellulose, lignin, and pectin). Sugar, starch, and fiber can be digested by rumen microbes converting carbohydrates to VFA. These VFA are the main source of energy. When the VFA ratios and levels shift, milk yield and components change. According to some researches on the issue says, rumen availability and digestibility of cell wall and cell solubles vary depending on growth stage and maturity (forages), source of carbohydrate (starch or cellulose) and processing (grinding of grain or chopping of forages). Dairy farmers and nutritionist must decide the correct source and rate of starch fermentation in the rumen based on rumen pH, forage sources, level of non-fiber carbohydrate, dry matter intake, and price of starch containing grains. If more rumen fermentable carbohydrate are needed, finely processed corn, high moisture corn, corn starch, or corn solubles can be added. If high levels of corn silage or subacute acidosis is occurring, shifting to corn gluten feed or less corn could be the correct decision.

Non-structural carbohydrates (NSC) and sub-acute ruminal acidosis (SARA)

According to Nocek *et a*l., 1988 70% to 80% of dairy cows' diet are Carbohydrates; from which NSC (compounds of plants containing simple sugar or those more complex like starch) are the most used in Livestock. The main source of supplement in dairy cows is grains, as they are an effective-cost option of digestive energy to fulfill the high requirements of dairy cows that forages cannot meet (Peyraud JL *et al*.,2013). According to Kleen JI *et al*.,2003 feedstuffs with nutritionally dense diets increases the amount of volatile fatty acids and lactic acid in the rumen, which exceeds its absorption capacity and therefore the buffer power of the animal organism. The combination of these factors creates a decrease in the ruminal pH for long periods during the day (pH <5,6 for more than 3 hours) creating SARA. This sub-clinic acidosis affects dry matter intake, milk yield, ruminal microflora, and digestion; it may cause diarrhea, damage in the mucosa of the gastrointestinal tract, laminitis and liver abscesses in dairy cows.

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VOLATILE FATTY ACID PRODUCTION

End products of microbial digestion are VFA which are absorbed from the rumen and serve as a source of energy for the dairy cow. The primary VFA is acetate which is a two carbon VFA, represents 55 to 70 percent of the total VFA production, and produced from the digestion of fiber. Propionate or propionic acid is a three carbon VFA produced by starch and sugar digestion bacteria. Propionate is converted to glucose by the liver. Glucose is used to synthesize milk lactose sparing amino acids from gluconeogenesis. The level of propionate varies from 15 to 30 percent of the total VFA production. The third main VFA is butyrate and contributes 5 to 15 percent of the VFA produced. Butyrate is used as an energy source and for milk fat synthesis. When evaluating VFA patterns, the ratio of acetate to propionate or A:P ratio (60 percent acetate:25 percent propionate or 2.4:1) reflects the rumen fermentation pattern. Under optimal rumen fermentation conditions, the A:P ratio should be greater than 2.2 to 1. High levels of acetate can indicate a high fiber-low fermentable carbohydrate ration. High levels of propionic acid can indicate reduced fiber digestion and acidosis. VFA analysis in the field in not available, but would be a useful tool to evaluate rumen fermentation and digestion.

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