A practical guide to differential diagnosis

1 – Breeding difficulties

by Bryan G. Miller, Ruminant Technical Support Manager, Biomin.

Getting cows bred is important for lifetime milk production by increasing the number of days in early and peak production. As milk production has increased, the period between calving and successful breeding has lengthened. A longer calving interval puts additional pressure on managers to get cows bred and address underlying issues that hamper breeding success.

A healthy reproductive tract and uterus able to support a young foetus is crucial. Health conditions and production are interrelated. Reproduction efficiency can be affected by ketosis, dystocia and retained placenta, but it is not directly associated with milk fever, displaced abomasum, and mastitis. The primary ways to influence reproduction are from an energy basis and specific uterine health.

Today's cows cannot consume enough feed to meet their energy needs and can remain in negative energy balance for many weeks. Severe negative energy balance brings increased uterine inflammation, providing a poor chance for fertilisation and implantation. Increased circulating urea levels – a situation common among cows using skeletal muscles to provide carbon for glucose production – reduces reproduction efficiency. Diet energy density and total dry matter intake can lower the energy deficit duration. Reducing the drop in dry matter intake around parturition has the greatest positive effect on subsequent feed intake and energy balance. Cows in negative energy balance have also reduced immunity responses and may have more difficulty in preventing metritis.

Normally uterus involution occurs within the first 40 days postpartum. However, failure to properly expel the foetal membranes and remaining fluids sets up conditions for microbial growth and subsequent infection. Most cows will have cleaned up five weeks postpartum; however a study showed that 40% of cows have evidence of uterine disease and another 20% have subclinical endometritis.

The first steps to maximise reproductive efficiency that need to be confirmed are a good heat detection and breeding system. Then, ensure cows have the best potential for breeding heats at the appropriate time. Maximising caloric intake by increasing caloric density of the diet through manipulation of fat and grain sources is one option.

A second is maximising dry matter intake and forage fibre digestion and usage, including the use of yeast products.

Linked to, but beyond energy intake, is the need for cows to perform as efficiently as possible, including absorption of nutrients from the gut, and an effective liver that can package and distribute nutrients needed by tissues.

The liver is critical for glucose production and redistribution of fat for use by other tissues, particularly in early lactation when both are in high demand. The liver is the first organ (after the gut itself) exposed to nutrients and other compounds absorbed and transported to the blood. Toxins that decrease liver function negatively affect a cow's energy balance. Key phytogenic products have been demonstrated to improve liver health, support proper immune function and increase dry matter intake.

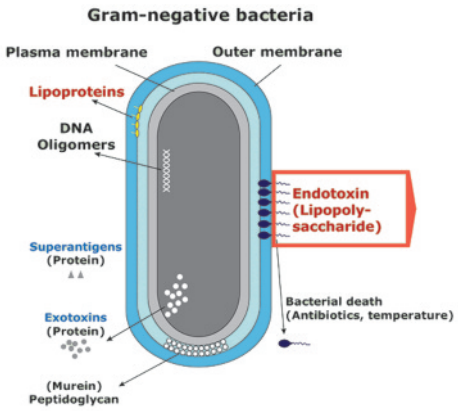
Mycotoxins in the feed can reduce feed intake, impair immune function and disrupt reproductive efficiency. Products that reduce the effects of mycotoxins are likely to assist in improving reproductive efficiency. In addition to proper management and supplying balanced rations, producers can use selected additives to prevent or reduce problems associated with breeding difficulties. ■

Check list	Potential causes
Uterine infections	
Poor immune function	Toxin issues including: • Potential nitrate poisoning, alkaloids (plant origins) • Mycotoxins including aflatoxins, deoxynivalenol, T-2 toxin, ochratoxin A • Other immune demands, general infections
Poor uterine involution	Decreased liver function: • Fatty liver syndrome, decreased nutrients provided • Mycotoxins affecting the liver (aflatoxins, deoxynivalenol, T-2 toxin) Mycotoxins' direct effect on the uterus: zearalenone causes oestrogenic effects and may result in uterine infections and vaginitis
Energy balance	
Liver function	Decreased liver efficiency due to mycotoxicosis including aflatoxins, deoxynivalenol, T-2 toxin, ochratoxin A
Decreased feed intake	Poor feed consumption just prior to and just after calving (pre-fresh feeding program) Toxins from plants, moulds and fungi (mycotoxins) Formulation including palatability, digestibility and nutrient density

References are available from the authors on request

A practical guide to differential diagnosis

2 – Preventing endotoxins



by Bryan G. Miller, Ruminant Technical Support Manager, Biomin.

Cattle are constantly in contact with endotoxins via feed, air and the environment. A healthy cow's natural defences restrict endotoxins from entering the bloodstream in sufficient numbers to cause harm. Several situations result in large quantities entering an animal's bloodstream, impairing health and production.

Definition

Endotoxins are remnants of the cell walls from Gram negative bacteria such as E. coli and salmonella. Endotoxins are comprised of fat (lipid) and starch (saccharide) components and are often referred to as lipopolysaccharides, or LPS.

Endotoxins are released upon the bacteria's death and increase when conditions are such that cause increased cell death of these bacteria. Common causes of endotoxin exposure in cows include antibiotic feeding and subacute rumen acidosis (SARA).

Endotoxins are known to cause strong inflammatory reactions and are classically used in research to invoke a fever situation. LPS acts by binding to key receptors in many cell types, but especially in monocytes, dendritic cells, macrophages and B cells, which promotes the secretion of pro-inflammatory cytokines, nitric oxide and eicosanoids.

Table 1. Negative effects of endotoxins in cows.

- Reduced rumen motility associated with:
  - Decreased fibre digestion.
  - Lower rumen pH.
  - Increased chance for displaced abomasum.
- Increased mastitis, metritis.
- General immune suppression.
- Liver impairment associated with:
  - Reduced nutrient availability.
  - Reduced production.
- Higher rate of respiration.

Interestingly, cattle may not exhibit a fever response to either very high or very low endotoxin administration, though a lack of fever, at least in cattle does not mean that there has not been a release of, and an effect of, endotoxins.

Harm to cattle

Endotoxin effects in cattle are closely associated with both rumen acidosis and SARA. SARA is a common condition in feedlot cattle and lactating dairy cattle that are receiving diets with increased energy from a grain source. SARA conditions can result in not only the lysing of Gram-negative bacteria but can also result in lowered rumen wall integrity. This combination can result in the passage of bacteria such as Fusobacterium necrophorum associated with liver abscesses, and increased absorption of endotoxins.

Attacking the liver

Perhaps the greatest detrimental effects of endotoxins on performance are due to the effects on the liver. The liver is the primary organ responsible for the removal of toxins from many sources including plant poisons, mycotoxins, and endotoxins. Kupffer cells are specialised macrophages that are responsible for removal of endotoxins.

They are also responsible in large part for the cascade of inflammatory events. The liver is an extremely active organ with many functions. In addition to the detoxification and immune related functions, the liver is important for the production and repackaging of nutrients such as proteins, carbohydrates and fats. An impaired liver will result in reduced nutrient availability and reduced production.

Time around parturition

Cows are in negative energy balance with requirements for both glucose, much of which is produced in the liver and fats which are repackaged for distribution to tissues via VLDL excreted from the liver. It is common to see increased fat accumulation in the liver near parturition. The condition may become severe enough, resulting in 'fatty liver' syndrome which is associated with decreased liver functions including the reduced production of glucose leading toward ketosis. It is also found that livers with increased lipid content do a poorer job of clearing endotoxins from the blood allowing for greater endotoxin concentrates to circulate to other tissues.

Prevention and mitigation

Producers can reduce their risks of endotoxicosis through management and key feed additives. The levels of endotoxin increase with increased rumen pH. Maintaining stable rumen pH through selection of feed ingredients and potential use of buffers should decrease this risk factor.

Additionally LPS challenges were worse in abrupt feed changes from fibre to grain based diets. Additionally certain mycotoxin products designed to adsorb aflatoxins and ergot alkaloids may also bind endotoxins. However not all products will have affinity for LPS and care should be taken to select products that have been tested and demonstrated to adsorb endotoxins.

Besides the toxin binding, the support of liver health and regulation of immune responses via bioprotection components is of great importance. Here again the choice of products is of great importance.

With good management and adequate feed composition your cows can overcome critical states in good conditions. ■

Table 2. Tips to counter endotoxins.

- Prevent decrease in rumen pH through:
  - Careful selection of feed ingredients.
  - Potential use of buffers.
- Avoid abrupt feed changes from fibre to grain based diets.
- Use a feed additive able to bind endotoxins and support liver and immune function.

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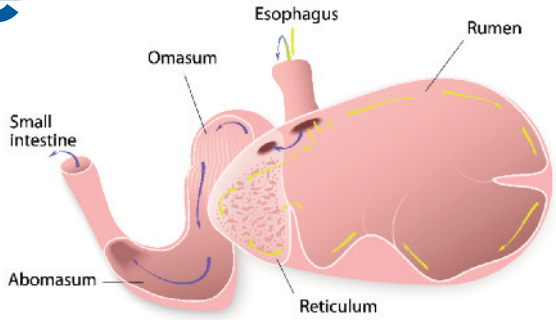
3 – Displaced abomasum



by Bryan G. Miller, Ruminant Technical Support Manager, Biomin.

Today's dairy cattle are genetically prepared to produce large amounts of milk. Along with that is the requirement for the consumption and digestion of large amounts of feedstuffs. The incidence of displaced abomasum (DA) may be increasing in our dairy herds via both genetic selection for greater milk production and the shift to more energy dense diets that may have less effective fibre content. Approximately 5% of high producing dairy cows may have a displaced abomasum.

The abomasum is the ruminant's 'true' stomach and comparable to the stomach in monogastric animals. It is not a small organ as it can contain 27 litres of fluids; however it is dwarfed by comparison to the 180+ litres in the rumen-reticulum.

Located on the right side and along the bottom of the rumen, the abomasum can undergo movement or position changes due to changes in rumen fill or the size and place of internal organs.

Key to abomasal displacements are changes due to calving and feed intake.

The more time the rumen remains filled, the less opportunity there is for the abomasum to shift. However, at calving there is a great void in the cow with the loss of the calf, placenta, and associated fluid. Organs shift in location and the abomasum can slip from its normal position.

Additionally, cows often have reduced feed intake associated with calving which can also make it easier for the rumen to slip from its normal position. Ketosis that occurs during the first months of lactation can also contribute to decreased feed intake and an increase in the incidence of displaced abomasum. About 80% of the DA cases occur within the first month of lactation.

A 'left' displacement where the abomasum 'slides' up the left side of the rumen is the most common and accounts for 80% of displacements.

Symptoms

The first signs are decreased feed intake, listless behaviour and a drop in milk production. The amount of faeces produced may be reduced and have more fluids than normal. The heart and respiration rates may remain fairly normal.

The most diagnostic test is usually the 'ping test'. Using a stethoscope one can hear a ping as a result of thumping the area. As the abomasum is displaced, gases tend to build as regular flow is decreased due to twisting of the duodenum.

Occasionally, DA can be corrected by 'rolling' the cow or jogging the cow to encourage the organs to resume their normal position. On a practical basis, by the time a cow is observed and a DA is suspected, surgery is the likely outcome.

The surgery can be done with minimally invasive techniques, but all surgery has a risk and the cow will still have a recovery period of poor production, or if treated with antibiotics, disposed milk.

Prevention

The best prevention is consistent feed intake. Rumen fill and continuous flow of material helps maintain both rumen and abomasum in their proper position.

Unfortunately, there are times when consistent intake is not possible. In addition to the issues surrounding calving, cattle can go off feed because of disease, mycotoxin challenges, and diet changes. Even weather can affect intake. Certain management decisions can reduce the interruptions to feed intake.

Tips for maintaining feed intake

- Have a transition program that encourages feed intake and rumen fill. Numerous studies have demonstrated that the less a cow's feed intake drops at calving, the more quickly they will increase dry matter intake after calving.

- Encourage feed intake through the use of better quality forages.

- Consider adding yeast or yeast culture products into the ration. Yeast products can support fibre digestion and feed intake. Yeast culture products have also been used as palatability enhancers.

- Avoid ketosis while additionally maintaining this feed intake is critical to reducing both clinical and subclinical ketosis. Ketosis is known to result in reduced feed intake and explains the increases in DA associated with it. A reduction in rumen motility can also be related to DA.

- Fibre length and proportion of the diet can influence DA. A lack of fibre can result in lowered rumination and the fill to keep the rumen in its normal position. At the same time, high producing dairy cows need energy. Fibre sources that have good digestibility or the addition of yeast based products which may assist fibre digestion can help in both meeting the need for fibre and the greater energy need of these cattle.

Summary

Displaced abomasum are becoming more common in dairy operations as a combination of changing genetics and feeding programs. Management and feeding programs that help ensure rumen fill through good dry matter intake, yet reduce energy deficits should help reduce the number of observed displaced abomasum. ■

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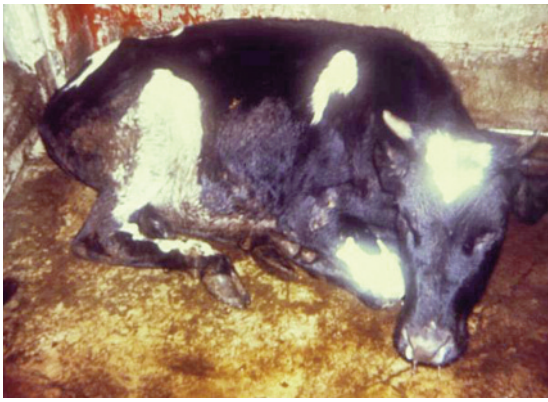
4 – Milk fever



by Bryan G. Miller, Ruminant Technical Support Manager, Biomin.

Milk fever, also known as hypocalcaemia or parturient paresis, is by no means a new condition for modern dairy cattle. Calcium, in addition to being a major component of bone, is also needed metabolically to transfer the message for both skeletal and smooth muscles to contract. A shortage of calcium can result in tremors, cows found in a 'sitting' stance, eventual collapse, and potentially death.

Timing and susceptibility

Milk fever can be caused by the large need for calcium for the production of colostrum. As a result, about 80% of milk fever occurs within one day of calving.

Older cows (two or more lactations) seem to be more likely than first calf heifers to have milk fever, but cows of any age are susceptible. Additionally, Jersey cattle are more predisposed to milk fever than other breeds. Milk fever is most common in high producing dairy cows. An incidence of 5% is not unusual, but incidences over 10% certainly indicate a larger problem needing specific management changes.

The incidence of milk fever can increase due to the presence of other common metabolic disorders. Milk fever incidence is greater in over-conditioned cows. This is most likely related to clinical and subclinical ketosis which reduces feed intake post-partum and puts additional pressure on an already limited calcium supply.

Related issues

Milk fever can also predispose the cow to other metabolic disorders and infectious diseases. Cows with milk fever may have greater plasma cortisol levels which can cause immunosuppression.

Additionally, the cells' internal calcium is used as a secondary signal once outside of the cell to stimulate an immune response. Lowered plasma calcium concentrations, as in milk fever, can result in reduced cellular calcium concentration and a blunted immune response. It is not uncommon to have increased cases of mastitis and metritis in cows that have suffered from milk fever.

Milk fever can also contribute to dystocia, retained foetal membranes, and uterine prolapse. Additionally, cows will have reduced feed intake which can lead to more cases of ketosis and displaced abomasum.

Feed recommendations

Low calcium diets (less than 20g per head per day) have been implemented by some producers successfully, though it can be difficult to formulate rations with such low levels of calcium. Proper attention to dietary anion strength is a better way to manage diets to reduce milk fever.

Dietary Cation-Anion Difference (DCAD) diets balance four macrominerals: the anions chloride and sulphur; and the cations sodium and potassium. This balance can help determine blood (and urine) pH.

Slightly acidic conditions are needed for the proper mobilisation of bone so that calcium can be released for colostrum and milk production.

Diets designed for dry cows in the weeks prior to calving should have a negative DCAD. The goal is to lower blood pH. Fortunately, producers can easily monitor pH via the urine. A pH of 7.0 or greater would suggest that a producer should consider balancing for cations-anions.

Proper balance does require regular monitoring as producers should not let urine pH drop below 5.5. A urine pH of 6.0 to 6.5 indicates an effective DCAD diet.

Potassium content of forages can greatly affect DCAD: increased potassium content is a contributor to milk fever. Also, as temperatures rise, cows pant more, expelling more CO_2 resulting in lower pH.

Products used to increase the negative balance include magnesium sulphate, calcium sulphate, ammonium sulphate, calcium chloride, ammonium chloride, and magnesium chloride.

Many of these products can be unpalatable. Producers are cautioned to ensure dry matter intake is not negatively affected. Protein sources treated with hydrochloric acid provide an additional way to increase negative charges and avoid some of the palatability problems associated with anionic salts.

Further steps

In addition to managing calcium in the diet and blood pH, producers need to consider the overall herd management in regards to feed intake, energy balance and other challenges.

Maintaining feed intake through diet balance and potential intake enhancers, such as yeast products and phytogenic products, can have the additional benefit of reducing the effects of milk fever.

Reducing other challenges to the cow, including pathogens and mycotoxins, should help reduce the secondary effects of milk fever that can impact disease status and reduce milk production. ■

Dietary Cation-Anion Difference

The pH can be manipulated by controlling the concentration of sodium (Na), potassium (K), chloride (Cl), and sulphur (S) that the cows consume.

What actually is determined is based on the charges of each anion (Cl and S) and cation (Na and K) mineral. The equation below takes into account the molecular weight of the respective minerals.

DCAD Equation:

$$\frac{\text{Sodium (Na)} \times 435 + \text{potassium (K)} \times 256 - \text{chloride (Cl)} \times 282 + \text{sulphur (S)} \times 624}{= \text{milliequivalents (mEq)/kg dietary dry matter}}$$

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5 – Ketosis

by Bryan G. Miller, Ruminant Technical Support Manager, Biomin.

Even if you only own one dairy cow, if she has had a calf it is likely that you have had a case of at least mild subclinical ketosis. Yes, it is that prevalent. Most cows go through at least a short time period where they lack the glucose to meet all of their needs. In many ways, the herd's milk production and health status are linked to the duration and severity of subclinical and clinical ketosis within the herd.

The negative energy balance that causes ketosis can decrease milk production and often accompanies other metabolic diseases such as milk fever (hypocalcaemia), metritis, mastitis, and displaced abomasums (DA).

Cows with ketosis conditions take longer to be bred successfully and often will not reach their full potential in peak milk production.

Symptoms of ketosis

One of the primary effects of ketosis is a decrease in dry matter intake, which is much easier to diagnose in tie stall barns vs. free stall barns. A classic detection is for the odour of acetone on the breath of cows, which can be useful for clinical ketosis but will probably not identify the subclinical ketosis cows.

If a cow herd has a high level of DA (over 3.5%), consider the link to ketosis. As both afflictions affect dry matter intake, it is not necessarily a cause and effect relationship, but the two are highly associated. When viewing cows early in their lactation that have dull coats and appear unthrifty or listless, consider ketosis as a possible source.

Additionally, cow herds with more ketosis also tend to have increased levels of metritis. If one is looking at increased disease conditions, consider potential links to subclinical ketosis.

What is ketosis?

By definition it is the result of finding ketone bodies – primarily made up of acetone, and alpha keto butyrate – in the blood.These byproducts come from poor utilisation of fats. There is a saying that 'fats burn in the flame of carbohydrates', which is to say you need sufficient carbohydrates (or carbohydrate derived compounds) to properly utilise fat, much like a pilot light is needed for the furnace to run properly.

The rumen, which allows cows to digest fibre, is of great benefit. However, it does not allow for the passage of sugars and most of the starch. As a result, cows rely on the liver to produce needed glucose through gluconeogenesis.

This process can take propionate, a volatile fatty acid produced in the rumen, amino acids, and other compounds that can be broken down into a unit containing three directly attached carbon atoms and convert them to glucose. Because the liver is critical to the manufacturing of glucose, any factors that decreases its ability to produce glucose increases the risk of developing ketosis.

This is particularly true in early lactation where the energy demands for high milk production cannot be met by the dry matter that cows typically consume. Fatty infiltration of the liver can decrease liver function and is common in transition cows, particularly in cows with excess body fat (body scores of 3.75 and above). This is a major contributor to increased ketosis observed in over conditioned cattle.

Treatment for ketosis

The first course of action is to increase the circulating glucose concentrations in the blood. This is done through infusion of

500ml of 50% dextrose solution IV. This is more effective in cows that develop ketosis soon after calving and may need to be repeated. Sometimes, glucocorticoids, such as dexamethasone or isoflupredone acetate (5-20mg) are administered intramuscularly to stimulate glucose production.

Rumen precursors for glucose production can be fed, usually propylene glycol (400g/day) to allow the cow to produce her own glucose.

Treatments should be discontinued after cattle both appear more normal and increase their dry matter intake to appropriate levels.

Preventing ketosis

Ketosis is prevented primarily through improving liver health and maintaining dry matter intake, in particular in the first five days after calving. The most important thing a producer can do is to maintain proper body condition and avoid fat cows. Research indicates that cows of normal condition to slightly thin have less fatty liver infiltration and less ketosis.

Increasing the rumen bypass of methionine and choline has been demonstrated to reduce ketosis and foster greater feed intake post calving. Niacin supplementation can also be helpful, but tends to work better with fat cattle than those in better body condition. Where legal to do so, feeding monensin can increase the amount of propionate (glucose precursor) produced in the rumen.

Herds that experience high levels of ketosis should also consider the possibility of mycotoxin contamination. Many mycotoxins have a negative effect on liver function which can reduce the ability to produce glucose. Producers that use mycotoxin control products in the lactating herd should also use them in the dry cow herd as well.

Although it is important to avoid fat cows, it is also important to minimise the drop in dry matter intake commonly associated with calving.

Products that can encourage dry matter intake, including the use of yeast products and phytogenic compounds should also help reduce the incidence of ketosis. ■

A practical guide to differential diagnosis

6 – Calf growth

by Bryan G. Miller, Ruminant Technical Support Manager, Biomin.

Economically important in both the short and long term, calf health and growth rate can have a large impact on a dairy operation's profitability. The first parameter to consider is simply mortality. A reduction in the number of calves in the system decreases the future herd replacement opportunities. Hopefully, a well-planned breeding program has provided replacement heifers with greater genetic potential for milk production than previous generations. Increased number of replacements allows for greater culling options or may provide an additional revenue stream to the farm.

There are multiple feeding and rearing programs. Different plans will fit better or worse within each unique dairy operation. This article will not try and cover each scheme, but rather factors which are important for all operations.

Consumption of quality colostrum

Calves are born without the necessary antibodies needed to protect them. Colostrum contains these needed antibodies (IgGs). During the first day of a calf's life, these antibodies (proteins) can be absorbed and subsequently utilised.

Colostrum should be consumed as quickly as possible as the gut will 'close' and absorption will no longer be available.

Calves should be fed 10% of body weight within the first six hours of life: the sooner, the better. It is important that the quality of colostrum is maintained. Generally speaking, colostrum quality is better from older cows than younger cows.

Cows should also be vaccinated so that the IgG to those vaccines will also be in the colostrum. For good quality colostrum, the cow must have a strong immune system. Poor protein and energy content or the presence of mycotoxins in the feed can reduce colostrum quality.

Maternity or calving pens

General recommendations are for pens 3.5-4.0m x 3.5-4.0m in size. They should have a deep bedding of straw or sawdust. Hygiene and comfort are important. Cows prefer an isolated location if possible. Getting the calf dry and ready to receive colostrum are important parts of the calving process.

Many producers prefer to feed calves colostrum rather than letting them naturally nurse. If one does allow nursing, make sure that the teat is clean.

Calf milk replacers

There are a multitude of calf milk replacers available with a wide variety of ingredient qualities and compositions. Regardless of formulation it is important that they provide the protein, energy, along with vitamins and minerals, to support rapid growth of the young calf. Depending upon jurisdiction, antibiotics and coccidiostats may be included in the milk replacer.

Alternatively, producers can use acidifying products and phytogenic compounds (essential oils) that have also been demonstrated to reduce threats from dietary pathogens and support health and growth.

These types of products can be particularly important when the calf reaches 7-10 days of age. At this time the immunity received from the colostrum may not have been fully supplanted by the calves' own antibody production. In addition, at this age calves begin to test eating dry feeds (calf starters) and scours are common. Organic acids and phytogenic compounds can reduce the severity and time of these scours (typically E. coli related).

Calf starter (grain)

It is important for calves to transition from a milk diet to a solid diet. Consumption of calf starters also stimulates rumen development. Generally, it is better to limit the amount of forage presented to calves during the first two months.

Calf starter will actually stimulate rumen development better and contain more energy to stimulate growth. As such, we want to encourage starter intake.

Molasses is often used to encourage feed intake. Studies have shown that it should be limited to 5% of the diet. Molasses can potentially lead to greater cleaning requirements or encourage greater fly problems. Using a flavouring system (as with the essential oils) can encourage calves to make the move from a milk-based to a grain-based diet.

Summary

It is sometimes difficult to know if calves are healthy because they eat more and have a better diet, or whether they eat more because they are healthier. In the end it may not matter, as we strive for both from the beginning.

Faster growing calves not only allow for transition to a less expensive diet and potentially reduce days to first breeding, but are also better producing cows once they enter the lactation trial.

An analysis of multiple calf trials has suggested that for every 100g increase in average daily gain prior to calving, they will produce an additional 155kg during their first lactation. As such, better growing calves not only provide dividends though lower medication and feed costs, but also add to the bottom line as adults. ■

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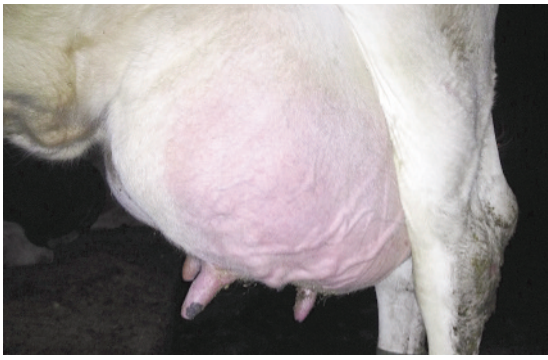

7 – Mycotoxins and mastitis

by Paige Gott, PhD, Ruminant Technical Manager, Biomin.

Mastitis, a costly disease affecting the dairy industry worldwide, is a complex disease with many factors influencing its occurrence. Mycotoxins can increase the risk of mastitis and negatively impact milk production and milk quality.

Mastitis is an inflammation of the mammary gland which is typically caused by an intramammary infection. Bacteria are the most common causative agent of mastitis in dairy cows, but other micro-organisms have been isolated from the milk of quarters experiencing mastitis including yeasts, fungi, mycoplasmas, algae, and viruses. Physical trauma or chemical irritation can also cause mastitis.

There are multiple ways to classify cases of mastitis. The first major classification has to do with the source of the pathogen (Table 1). Major contagious pathogens include Staphylococcus aureus, Streptococcus agalactiae, and Mycoplasma spp. Common environmental pathogens include Escherichia coli and Klebsiella spp. as well as environmental streptococci including S. uberis and S. dysgalactiae. A third group exists, 'skin flora opportunists', which consists of the coagulase negative staphylococci (CNS) species that colonise healthy teat skin.

The second classification of clinical vs. subclinical mastitis deals with the presentation of the disease. Clinical cases result in visible abnormalities of the milk and/or quarter, and range from mild to severe. Subclinical infections do not cause overt changes in the milk or quarter. Both mastitis types cause increases in somatic cell count (SCC). An elevated SCC often signals subclinical mastitis.

A third classification is acute vs. chronic mastitis. This has to do with the timing and duration of the disease. Acute cases are characterised by sudden onset, but are often quickly resolved. Chronic cases continue over a longer period of time.

Costs

Economic losses stem from reduced milk production and decreased milk quality. Farmers must discard milk from cows with clinical cases of mastitis and from cows undergoing antibiotic treatment (according to withdrawal periods). Treatment and veterinary costs rise, as do labour costs. Mastitis also alters the composition and properties of milk which reduces cheese yield, and can reduce shelf life of manufactured products.

In addition, animal welfare is a concern. Studies have shown that mastitis can be painful and cause discomfort to cows. Furthermore, udder health issues are frequently cited as one of the top three reasons for culling of dairy cows. Low milk production, potentially associated with mastitis, is another leading cause of culling in dairy herds. Toxic mastitis, an acute form of the disease, can even lead to cow death.

Mycotoxins

Some of the main consequences of mycotoxins in dairy cows in relation to udder health and milk production are:

- Reduced milk yield and quality.
- Toxic contaminants in milk, especially Aflatoxin M1.
- Increased risk of intramammary infections and mastitis.
- Altered milk composition.

Reduced milk yield results from several factors, including a decrease in intake or feed refusal that is associated with certain mycotoxins. Additionally, mycotoxins can alter rumen function, reducing nutrient absorption and impairing metabolism, which ultimately leads to reduced availability of the precursors needed for milk synthesis.

Addressing predisposing factors

Proper milking parlour management and milking routine are essential to limiting mastitis risk in a herd. The milking system must be well maintained to ensure properly functioning, clean equipment is used to harvest milk.

Good hygiene is critical, both in the milking parlour as well as in the barn. Clean sand is considered the gold standard bedding, as inorganic material does not support the growth of pathogens. The environment influences mammary health as increased temperatures and humidity better support pathogen growth in the cow's surroundings. Additionally, heat stress reduces the cow's resistance to infection.

Nutrition and feeding management can also play a role in udder health. Cows in negative energy balance, especially transition cows, are more susceptible to infection. Diets must also meet vitamin and mineral requirements to support proper immune function. Coordinating the delivery of fresh feed while cows are in the parlour will entice cows to eat and remain standing upon return to the pen. This provides time for the teat ends to close and limits exposure to pathogens following milking. Finally, feed should be monitored for the presence of mycotoxins and an effective mycotoxin counteracting product should be incorporated into the feed.

Many factors influence the development of mastitis, making mastitis control and prevention a constant challenge for dairy producers striving to produce high quality milk for consumers. ■

Table 1. Contagious and environmental mastitis (Biomin).

	Contagious mastitis	Environmental mastitis
Reservoir	Infected mammary glands	The cow's environment including bedding/stalls/soil, manure, water and feedstuffs
Exposure	Spread from cow-to-cow via milking equipment, milkers' hands or towels and flies and other vectors	Constant exposure exacerbated by heat and humidity

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8 – Subacute ruminal acidosis (SARA)

by Luis Cardo, PhD, Technical Ruminant Manager, Biomin.

Though not easily detected, subacute ruminal acidosis (SARA) can have a serious impact on milk production, general health and longevity. It is caused by an imbalance between production of volatile fatty acids (VFA) and their absorption by the rumen walls and the buffering mechanisms of the rumen.

Technically, a bout of SARA occurs when rumen pH drops below pH 5.8 for at least three hours, or pH 5.6 according to other authors. Fibre digestion is reduced and noticeably affects production. It can also result in lower feed intake, lower feed efficiency, and hoof problems (laminitis).

Rumen effects

SARA will affect feed efficiency, therefore increasing feeding costs, due mainly to the decrease of fibre digestibility.

When pH drops below 6.0, the populations and growth of cellulolytic bacteria and the ruminal fungi decline, impairing fibre digestibility. According to several sources, every 0.1 decrease in pH reduces fibre digestibility by 3.6%. Poor fibre digestibility and lower feed efficiency resulting from SARA translate into increased feeding costs for producers.

One study showed that short bouts of SARA (less than 30 minutes) did not reduce neutral detergent fibre (NDF) digestibility, while repeated bouts of four hours did so. These findings support the use of TMR and free 24-hour access to the feed bunker as key management tools to control SARA.

Main causes of SARA

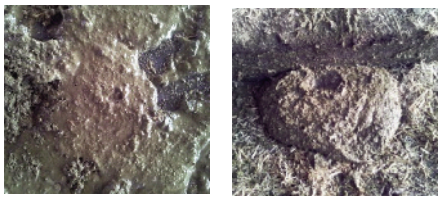
- Poor adaptation of rumen microflora to diet changes. Common at calving, pairing with other metabolic diseases such as ketosis and related conditions.
- Improper feeding patterns and cows selectively choosing their feed.
- Inappropriate forage size.
- Formulation mistakes.

Feed intake effects

SARA commonly causes erratic eating patterns and reduces feed intake. When pH drops, the cow reduces its feed intake, decreasing the production of acids and driving the pH back to normal levels. Then the cow will resume eating, resulting in another bout of SARA and repeating the cycle. This variation will not only decrease production due to the lower feed intake, but will also reduce the efficiency of the rumen fermentations due to the variation of the nutrients supply, causing further economic losses.

Faeces assessment

A heterogeneity of faeces in a cow's group in the same lactation stage can be caused by SARA. In this situation some faeces will be normal and some too loose. You can use the 1 to 5 scoring system to assess them.



Above left, liquid faeces score 1; above right, score 3.

Lameness

Lameness is a major concern in modern dairy and beef production, due to implications for welfare and profitability.

There is a clear link between acidosis and the inflammation of the lamellar tissue of the hoof, a condition known as laminitis that not only causes problems by itself, but is also a predisposing factor for other conditions such as sole ulcers and white line haemorrhages.

Although the mechanism of laminitis is not yet totally clear, it is thought that the condition is due to lower systemic pH during acidosis and substances such as histamine (involved in immune response) and endotoxins entering the bloodstream.

Lameness, in its turn, can exacerbate SARA as cows suffering this condition will change their feeding patterns due to the lower number of meals caused by the pain suffered when moving to the feeding bunker.

Two detection tips

- Check feeding patterns on TMR. If cows are selectively choosing their feed – evidenced by lots of holes in the TMR – then the ingested fibre and concentrates can differ considerably from the theoretical ration.
- Routinely assess and document indicators of possible SARA: butterfat content, manure assessment, laminitis, and individual feed intake patterns.

Steps to address SARA

SARA control aims at improving adaptation of rumen papillae and microflora and optimise effective fibre intake. Here is a list of management practices to mitigate the risk of SARA:

- Ensure proper rumen adaptation especially at calving when shifting cows from the dry group to the lactation group.
- Control ingredients' palatability.
- Ensure homogeneity of TMR and proper forage length cut. Keep records of maintenance of mixer (balances, knives).
- Ensure proper access to the feed bunks and an adequate supply of water.
- Avoid stressful situations such as moving animals too much between production groups.
- Keep first calving heifers separated from older cows when possible.
- Resting. Ensure good layout, maintenance and bedding. Insufficient lying time will cause cows to change their feeding pattern.
- When formulas or forages are changed a smooth transition is highly advisable. ■

A practical guide to differential diagnosis

9 – Rumen fermentation

by Bryan Miller, Technical Sales Manager, Biomin.

Dairy cattle provide their true benefit by allowing the utilisation of forages, which in turn allows us to utilise land that may not be appropriate for traditional grain crops. Through a symbiotic relationship with a microbial population in the rumen, fibre that would have been indigestible can be converted to microbial protein and volatile fatty acids (VFA), which are converted by cows into a final, highly nutritious end product within the mammary glands: milk.

However, genetic improvements in cows and the pressure for greater production have resulted in the feeding of more energy-dense products, including grains and specialty products. This additional opportunity to increase protein production also comes with potential problems, such as subacute rumen acidosis.

Protein production

Animals do not really have a dietary protein requirement, but rather a dietary amino acid requirement. The concept of an 'ideal protein' is providing proteins to the cow's digestive system that will best meet her needs for the essential amino acids (building blocks of proteins).

The protein reaching the intestines will be a combination of amino acids from the growth of micro-organisms in the rumen and protein that remained undigested and comes from the original feed ration.

Microbial protein has the correct ratio of essential amino acids and, as such, maximising microbial protein provides a very efficient source of proteins for cows. The two most limiting (in short supply) amino acids are methionine and lysine.

A market has developed for providing rumen protected sources of both of these amino acids. Corn grain can also act as a good source of by-pass methionine and certain 'treated' soybean meals can be a source of lysine.

Rumen microbial population can also take advantage of non-protein-nitrogen sources like urea and convert it to microbial protein. However, to fully utilise NPN there must be a readily available source of energy that both encourages microbial growth, but also provides the carbon that is additionally needed to manufacture amino acids.

Multiple computer modelling systems are available to assist the nutritionist in maximising microbial protein production.

Energy from forages

Fibre portions are not digestible by mammalian enzymes. Rumen microbes can digest the fibres and, as a byproduct of this digestion, the bacteria produce volatile fatty acids which the cow can use for energy. Acetic acid (acetate) and propionic acid (propionate) are the two most prevalent and important VFA.

Acetic acid is a two carbon sugar closely associated with milk fat production. Decreases in acetate production occur in diets that have a limited amount of fibre, or fibre that is poorly utilised. As part of the rumination process the cow eructates and re-chews her feed. In doing so, additional buffering capacity from saliva is introduced.

This pH balance is essential for healthy rumen fermentation and subsequent VFA production. This is one of the reasons that poor milkfat is associated with diets that lack either a total fibre content or effective fibre, that is of sufficient size to stimulate re-chewing of feed.

Because rumen bacteria utilise starch, the cow does not receive enough digestible carbohydrate to meet her needs. The liver is responsible for the production of glucose from non-carbohydrate sources.

One source is the deamination of amino acids and using the carbon from certain amino acids. Fortunately, propionate can also be used for the production of glucose. In jurisdictions where it is legal, feed additives may be applied.

Monensin

An ionophore, monensin, is capable of shifting the ratio of VFA in the rumen, resulting in increased propionate production to support glucose production which is used for energy and lactose synthesis. Milk lactose content varies less than milk fat or protein concentration.

A shortage of glucose can be manifested in reduced fluid milk production as lactose helps drive milk volume by regulating the amount of water drawn into the secretory cells of the mammary glands.

Buffering compounds

Grain feeding can result in an over production of VFAs and also lactic acid production, which can lead to subclinical/clinical acidosis and potential liver abscess development. Acidosis can lead to decreased integrity of the rumen wall and the potential for liver abscess development.

Some of the effects of grain feeding can be compensated for through feeding various buffering compounds which help to increase pH.

Yeast products

Live yeast, autolysed yeast and yeast cultures can be used to improve rumen fermentation resulting in greater fibre and organic matter digestibility of the diet.

These yeast products may act through different mechanisms, whether improving fermentation through a more anaerobic environment or supplying needed nutrients to stimulate bacterial growth.

Toxin mitigation products

While rumen bacteria have the potential to breakdown toxins of plant and fungal origin, certain toxins can decrease microbial production and subsequent animal production. Producers should avoid feeding contaminated feedstuffs to higher producing cows, which are subject to more disturbances; and consider mitigation programs to minimise effects. An effective rumen allows cattle to utilise forages and produce food for human consumption.

Feeding programs that maximise rumen fermentation through a healthy environment for microbial growth, will help maximise subsequent animal production. ■