SECTION 6
DAIRY CATTLE
HEALTH and BIOSECURITY
CHAPTER 54

METABOLIC DISEASES IN DAIRY COWS

Introduction

Metabolic diseases in dairy cows result in milk production and farm income losses. Metabolic diseases differ from contagious diseases as they are not spread by bacterial or viral pathogens and are usually the result of poor management related to nutritional imbalances. Some of these diseases occur mostly during the dry period and early lactation, i.e. the peri-parturient period. It is important that farmers should know how these diseases develop, as most could be prevented by sound nutritional management.

1. Milk fever

Milk fever (hypocalcaemia) usually occurs at calving or about 3 - 4 days after calving. It is not a true fever condition, as it is caused by a sudden decline in blood calcium (Ca) levels because of the production of colostrum at calving. During the dry period (late gestation, non-lactating), dairy cattle have relatively low calcium requirements, with a need to replace approximately 30 g of calcium per day due to its utilisation for fetal growth and faecal and urinary losses. At parturition, the requirement for calcium is greatly increased due to initiation of lactation, when mammary drainage of calcium may exceed 50 g per day. Colostrum contains 8 to 10 times more Ca than the levels in the blood. Due to this large increase in demand for calcium, most cows will experience some degree of hypocalcaemia for a short period following calving down as the metabolism adjusts to the increased demand. Calcium levels in the blood are maintained by the diet and Ca released from the skeleton. The failure of Ca absorption from the diet and skeleton result in a sudden decrease in blood Ca levels. When the mammary drain of plasma calcium causes hypocalcaemia severe enough to compromise neuromuscular function, the cow is considered to have clinical milk fever. It is also referred to as downer cow syndrome, as cows experience muscle failure which causes them to lie down.

While milk fever is principally caused by a sudden demand for Ca in colostrum and milk, other probable causes include excessive bone formation due to elevated levels of gonadal hormones and diet containing excessive dietary levels of cations, especially potassium (K). Other metabolic disorders that can lead to clinical and subclinical hypocalcaemia include rumen stasis, displaced abomasums, retained placenta, prolapsed uterus, metritis, and ketosis.

Milk fever is considered a herd problem when more than 10 to 15% of the cows are affected every year. Milk fever seldom develops in first parity cows, occurring mostly in older, higher producing cows. It is also more common in Jerseys than in other dairy breeds. It is a problem when a high proportion of cows in a sizable group of cows calving down are affected, such as when five out of the last eight cows calving down are diagnosed with milk fever. The main reason for the development of milk fever is the sudden increase in demand for Ca early in the lactation and the inability of the parathyroid gland to stimulate the absorption of Ca from the digestive tract while additional Ca is mobilised from the skeleton. Specific management practices have been developed to stimulate the activity of the parathyroid gland.

Mechanism

In normal Ca regulation, a decrease in plasma Ca levels causes the parathyroid gland to secrete parathyroid hormone (PTH), which regulates the activation of Vitamin D3 in the kidney. These two compounds act to increase blood Ca levels by increasing absorption of dietary Ca from the intestine, increasing renal tubular re-absorption of calcium in the kidney, and increasing resorption of Ca from bones. It has been found that tissue is less responsive to parathyroid hormone pre-partum than postpartum. It is believed that hypocalcaemia causing milk fever is due to a lower level of responsiveness of the cow’s tissues to circulating parathyroid hormone.
The resultant decreased plasma Ca causes hyper-excitability of the nervous system and weakened muscle contractions, which results in both tetany and paralysis.

The clinical signs of milk fever can be divided into three distinct stages:

1. **Stage I** milk fever often goes unobserved because of its short duration (less than 1 hour). Cows are mobile but show signs of hypersensitivity, loss of appetite, nervousness, weight shifting, and excitability, such as restlessness, tremors, ear twitching, head bobbing, and mild ataxia. Cows lie down with the sternum in contact with the ground.

2. **Stage II** milk fever can last from 1 to 12 hours. The affected cow lies down in a sternal position with its head tucked into its flank. Heart contraction and peripheral pulses are weak. Cows appear dull and listless, with dry muzzles, cold ears, and a lower-than-normal body temperature. Smooth muscle paralysis can cause bloat because of an inactive digestive system developing into constipation. Cows are not able to urinate or defecate. The heart rate is rapid, exceeding 100 beats per minute.

3. **Stage III** At this stage the affected cow lies down in a lateral position, muscles show flaccidity, and unresponsiveness to stimuli. This is followed by a loss of consciousness, progressing to coma. Heart rate can approach 120 beats per minute, with peripheral pulses becoming undetectable. If untreated, progression will continue to death.

**Treatment**

Treatment generally involves Ca injection by intravenous, intramuscular or subcutaneous routes. Before Ca injection was employed, treatment comprised inflation of the udder using a pneumatic pump. Inflation of the udder worked because the increased pressure created in the udder pushed the Ca in the udder back into the bloodstream of the cow.

Intravenous Ca, though indicated in many cases, is potentially fatal through “heart blockade”, or transient high Ca levels stopping the heart, so it should be administered with care.

In unclear cases of downer cows, intravenous calcium injection can lead to diagnosis. The typical reaction will be a generalised tremor of the skeletal muscles and sometimes
cardiac arrhythmia, defecation, urination, and eructation are frequent during the treatment, due to the pharmacological effect of calcium on the smooth muscles.

The prognosis is generally good, even in advanced cases. However, some cows can relapse the following day, and even a third time the day after.

### Prevention of milk fever

In dairy herds showing a high incidence of milk fever, some preventative measures should be put in place to reduce the risk of milk fever. These measures are mainly aimed at the dry cow feeding. In Table 54.1 conditions associated with milk fever are listed.

#### Table 54.1. Conditions associated with milk fever in dairy cows

<table>
<thead>
<tr>
<th>Factors</th>
<th>Situation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Ca-intake in dry cows (&lt; 0.40% in total diet DM)</td>
<td>Heavy maize silage feeding, inadequate supplementation of Ca, low grain intake (dry cows), low forage – high grain feeding</td>
</tr>
<tr>
<td>Low phosphorus (P) intake (&lt; 0.28% in total diet DM)</td>
<td>Inadequate P supplementation, high forage – low grain feeding (cows on pasture)</td>
</tr>
<tr>
<td>Excessive Ca (between 0.70% and 1.00% in total diet DM)</td>
<td>High legume intake by dry cows, over supplementation with Ca</td>
</tr>
<tr>
<td>Excessive P intake (&gt; 0.40% TRDM)</td>
<td>Over supplementation of P, excessive grain feeding</td>
</tr>
<tr>
<td>Excessive vitamin D intake (&gt; 100,000 units per head daily)</td>
<td>Over supplementation can lead to calcification of tissues and result in heart failure</td>
</tr>
<tr>
<td>Low magnesium intake (&lt; 0.20% in total diet DM)</td>
<td>Failure to balance low magnesium forages, i.e. corn silage, grasses, and small grains</td>
</tr>
<tr>
<td>High potassium (K) intake affecting anion-cation balance (&gt; 1.2% in total diet DM)</td>
<td>Forages high in potassium content - over 1.5% on a DM basis</td>
</tr>
<tr>
<td>Reduced mineral absorption: rumen pH over 6.8 to 7.2; higher incidence with increasing age (lack of vitamin D, alimentary tract stasis, lack of motility, constipation)</td>
<td>High legume ration, high pH water (&gt; 8.5), grain intake of less than 1.5 to 2.5 kg, underfeeding forage or effective fibre, excessive intake of protein</td>
</tr>
<tr>
<td>Selenium or vitamin E deficiency (&lt; 0.10 ppm) (&lt; 250 units per head daily)</td>
<td>White muscle disease, lack of supplementation</td>
</tr>
</tbody>
</table>

Basic principles for the feeding of cows during the dry period include the following:

i. Limit the intake of Ca to less than 80 - 100 g/day or 0.5 - 0.7% of the diet
ii. Limit the intake of P to less than 45 g/day or 0.30 - 0.35% of the diet
iii. The Ca:P ratio should be less than 2:1, preferably 1:1.

To reduce the Ca content in the diet, legume hays or silages should be replaced by grass or cereal forage crops. A nutritionist should be consulted to evaluate the present ration programme and the feeding management practices towards reducing milk fever in the herd. Provide all pertinent information, including incidence and severity of milk fever cases. Information on the mineral content of forages should be provided. Minerals to test should include Ca, phosphorus, magnesium, potassium, sodium, sulphur, and chloride.

### Dietary Cation — Anion Balance

Another method of preventing and controlling milk fever is balancing dry cow rations for anions (negatively charged molecules) and cations (positively charged molecules). Sodium and potassium are the cations and chloride and sulfur are the anions of interest in formulating anionic diets. The dietary cation-anion balance (DCAB) equation most often used to determine milli-equivalents per 100 grams of dry matter is:
mEq/100g = mEq (Na + K) - mEq (Cl + S)

Based on current research, the range that achieves the lowest incidence of milk fever is a DCAB of –10 to –15 mEq/100g dry matter (DM) or –100 to –150 mEq/kilogram. Achieving a DCAB of –10 to –15 mEq/100g requires adjustments in the major mineral levels that are quite different than what is normally programmed for regular close-up dry cow rations (no anionic salts). DCAB may be calculated from the percent element in diet dry matter.

The equation is as follows:

\[
\text{mEq/100g DM} = \left( \%\text{Na} \div 0.0230 \right) + \left( \%\text{K} \div 0.0390 \right) - \left[ \left( \%\text{Cl} \div 0.0355 \right) + \left( \%\text{S} \div 0.0160 \right) \right]
\]

For example:

\[
\text{DCAB mEq/100g DM} = \left( \left( \%\text{Na} \div 0.0230 \right) + \left( \%\text{K} \div 0.0390 \right) \right) - \left[ \left( \%\text{Cl} \div 0.0355 \right) + \left( \%\text{S} \div 0.0160 \right) \right] = 4.35 + 20.5 - 19.7 + 21.9 = 24.9 - 41.6 = -16.7.
\]

In Table 54.2 recommended mineral levels for both regular and anionic rations are presented.

### Table 54.2. Guide to mineral composition (dry matter basis) for close-up dry cows

<table>
<thead>
<tr>
<th>Mineral</th>
<th>Regular</th>
<th>Anionic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium</td>
<td>0.45 to 0.55</td>
<td>1.40 to 1.60</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>0.30 to 0.35</td>
<td>0.35 to 0.40</td>
</tr>
<tr>
<td>Magnesium</td>
<td>0.22 to 0.24</td>
<td>0.28 to 0.32</td>
</tr>
<tr>
<td>Potassium</td>
<td>0.80 to 1.00</td>
<td>0.80 to 1.00</td>
</tr>
<tr>
<td>Sulphur</td>
<td>0.17 to 0.19</td>
<td>0.35 to 0.40</td>
</tr>
<tr>
<td>Chlorine</td>
<td>0.20 to 0.24</td>
<td>0.70 to 0.80</td>
</tr>
<tr>
<td>Sodium</td>
<td>0.10 to 0.12</td>
<td>0.10 to 0.12</td>
</tr>
</tbody>
</table>

Feeding a combination of different anionic salts is necessary for achieving the desired DCAB. The most commonly fed salts are ammonium sulfate, calcium sulfate, magnesium sulfate, ammonium chloride, calcium chloride, and magnesium chloride. Special attention should be paid to the degree of hydration of specific salts in formulating rations, as well as their costs and availability. In Table 54.3 the chemical composition of commonly available anionic salts are presented.

### Table 54.3. Chemical composition of commonly available anionic macro-mineral salts

<table>
<thead>
<tr>
<th>Mineral salt</th>
<th>Chemical formula</th>
<th>N (%)</th>
<th>Ca (%)</th>
<th>Mg (%)</th>
<th>S (%)</th>
<th>Cl (%)</th>
<th>DM (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ammonium sulphate</td>
<td>(NH₄)₂SO₄</td>
<td>21.2</td>
<td>-</td>
<td>-</td>
<td>24.3</td>
<td>-</td>
<td>100.0</td>
</tr>
<tr>
<td>Calcium sulphate</td>
<td>CaSO₄*2H₂O</td>
<td>23.3</td>
<td>-</td>
<td>-</td>
<td>18.6</td>
<td>-</td>
<td>79.1</td>
</tr>
<tr>
<td>Magnesium sulphate</td>
<td>MgSO₄*7H₂O</td>
<td>-</td>
<td>9.9</td>
<td>-</td>
<td>13.0</td>
<td>-</td>
<td>48.8</td>
</tr>
<tr>
<td>Ammonium chloride</td>
<td>NH₄Cl</td>
<td>26.2</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>63.3</td>
<td>100.0</td>
</tr>
<tr>
<td>Calcium chloride</td>
<td>CaCl₂*2H₂O</td>
<td>27.3</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>48.2</td>
<td>75.5</td>
</tr>
<tr>
<td>Magnesium chloride</td>
<td>MgCl₂*6H₂O</td>
<td>-</td>
<td>12.0</td>
<td>-</td>
<td>-</td>
<td>34.9</td>
<td>46.8</td>
</tr>
</tbody>
</table>
Before incorporating DCAB into a dry cow programme, there are several factors to consider. For instance, some of the anionic salts are very unpalatable which can depress intakes significantly in conventional feeding programmes. In particular, ammonium salts may result in more intake and palatability problems, especially when a silage based ration is not being fed. Reduced dry matter intakes as a result of feeding anionic salts can lead to the development of other metabolic disorders.

Much of the success with anionic salts has been in herds feeding a total mixed ration. The use of an anionic diet is appropriate when high calcium forages are fed at relatively high levels during the close-up dry period. Animals should receive the anionic diet at least three to four weeks prior to the expected calving date.

Forages presumed to be good dry cow forages might actually contain high potassium levels that interfere with DCAB. When the potassium level in the total ration dry matter exceeds 150 grams (or > 1.2%), it is difficult to add the proper amounts of anionic salts to meet the ideal DCAB range. Re-evaluating the ration and forages may be necessary if more than 295 g to 340 g of anionic salts are needed.

If DCAB is to be implemented in a herd, sodium, potassium, chloride, and sulphur must be included in the forage analyses. Buffers must not be used in anionic salt rations, because they will counter the effect of DCAB.

### 2. Bloat

Bloat causes severe losses because of cow deaths and lower milk production. Bloat is the result of an excessive accumulation of gas in the rumen of cows, which is usually belched away by the cow. Bloat is generally associated with cows grazing high legume pastures such as clover or lucerne in spring and autumn. A clover content of over 50% is considered dangerous. However, problems have occurred at lower clover content levels when abundant new succulent growth is available. Occasionally, pastures containing young grass growth can cause bloat because of a high content of soluble protein. Hungry cows, which gorge themselves when introduced to spring or young pasture, are particularly at risk. Mornings with dew on the grass or overcast, windy days are frequently associated with outbreaks. It has been found that heifers are three times more likely to die of bloat than mature cows. Jerseys are three times more susceptible than Friesians and crossbreds are twice as susceptible to bloat.

Treatment and prevention are costly and it is important to remember that, apart from restricting access to dangerous pastures, there is no single method which will guarantee complete protection from bloat. Many farmers use two or more of the following techniques to reduce the incidence of bloat on their farms:

#### Signs of bloat

Cattle with bloat may display the following signs:
- no longer grazing,
- a reluctance to move,
- distended left abdomen,
- appear distressed — vocalise, eyes bulging,
- strain to urinate and defecate,
- rapid breathing — mouth may be open with tongue protruding, and
- staggering.

#### Prevention:
**Pasture management**

Legume pastures should be introduced into the diet gradually over several days. Avoid cows gorging new pastures by feeding them before letting them out to graze. Silage, hay or more mature pasture can be used to reduce the cow’s appetite. Initially, cows should only be allowed access to legume pastures for short periods (one hour or so). They should be monitored closely during grazing and immediately after removal from the pasture. Cows become accustomed to suspect pastures over several days by modifying their intake to prevent bloating. It has been suggested that mowing the pasture and allowing it to wilt for 2 - 3 hours should reduce the risk of bloat.

#### Preventative medication

Three types of medication can be used to control bloat in cows, namely:
- Fermentation modifiers (Anti-bloat capsules),
- Detergents (alcohol ethoxylate), and
- Anti-foaming agents such as Paraffinic oil and tallow.

The systems used to administer these chemicals aim to provide a continuous supply of medication over the whole grazing period.
Movements within the gut ensure that the chemical is thoroughly mixed with the contents of the rumen, preventing the formation of stable gas foam.

**Direct medication**
Directly drenching cows with a detergent is perhaps the most successful way of controlling bloat and is widely practiced in pasture-based countries. It is usually applied after milking. Cows have to be trained to accept the medication. Molasses can be mixed with the detergent to improve its palatability.

Bloat oils may also be directly drenched; however, larger volumes are required and the duration of action is significantly shorter than for the detergents. Anti-bloat capsules are a longer lasting alternative to aid in the control of bloat in cattle. Administered as a large plastic pellet down the throat (into the rumen), they provide a continuous supply of chemicals for 80 - 100 days. The capsules have been found to reduce bloat deaths by about 80%. Trials have shown that cows with capsules have an increase in milk and protein production. Butterfat test (%) may be depressed, but the total fat yield for the season is unaffected.

**Spraying the pasture with oil**
Spraying the whole day’s grazing with anti-foaming agents, such as paraffinic oil, is a very reliable method of bloat control if carried out properly. It is best suited to ‘strip grazing’ systems that provide small, fresh areas for intensive grazing at least once per day. Oils only give 2 - 4 hours’ protection in the rumen and consequently need to be consumed over the entire grazing period. Spraying the total area of each day’s grazing (24 hours) at a rate of 85 ml of oil per cow is recommended. It is important that the grazing area is not over estimated or the oil may be spread too thinly and some cows may not eat enough. Make sure the oil will emulsify with water prior to purchase. Do not spray more than 2 - 3 days’ grazing at a time. Respraying may be required after heavy rain. Boom spraying equipment and fencing must be in good condition to avoid failures.

**Feeding in the milking parlour**
Anti-bloat medications can be mixed with concentrates and fed during the bloat season. Detergents and oils have been added to supplementary feeds (pellets or grain) with good results, providing that the cows eat sufficient amounts twice daily. Feed companies may include anti-bloat detergents. Rumensin (monensin) mixed with supplementary feed may be used for bloat control in dairy cattle.

**Water trough application**
Detergent can be added to troughs, if it is the only source of water available to the herd. Detergents are bitter tasting and need to be gradually introduced into the water supply over a week or two, until the herd becomes accustomed to the taste. The fresh water supply must be disconnected, unless a metering device can supply a measured amount of detergent as the water flows in. Troughs must be replenished with the concentrate at regular intervals to maintain protection. Despite this, the daily water consumption of individual cows can fluctuate greatly, leading to variable control within the herd.

**Flank applications**
Between 30 and 70 ml of thick bloat oil can be applied to the flank of each cow with a brush or automated spraying system. Most cows, but not all, will lick this off during grazing. Adding molasses or tallow increases palatability, but encourages some cows to lick it off others. Wet weather and variable consumption reduce the effectiveness of this control method.

**Treatment**

**If the cow is still standing**
There are a number of products specifically registered for the treatment of bloat in cattle. If these are not available, bloated cows should be drenched immediately with 60 - 120 ml of bloat (paraffinic) or vegetable oil. Cows should be removed from the pasture as soon as possible to prevent losses.

**If the cow is down and in extreme distress**
If treatment with oil has not been successful and the cow is likely to die before a veterinarian can attend, the pressure in the rumen should be relieved surgically. Often a wide bore (14G) milk fever needle, or trocar, and cannula are sufficient for this purpose. The site for stabbing is on the left flank, an open hand’s width behind the last rib and a similar distance below the ends of the short ribs (spinal vertebrae). If a needle or cannula is not available, or if they become blocked with foam (and the cow is in severe distress), a sharp narrow bladed knife can be used as a last resort. A 2.5 cm stab wound is ample and the knife should be left in the incision and twisted until gas and foam have ceased escaping.
A veterinarian will be required to suture the wound and administer antibiotic therapy if the rumen wall has been punctured during treatment.

3. Ketosis

Ketosis is a common disease in adult cattle. It typically occurs in dairy cows in early lactation and is most consistently characterised by partial anorexia and depression. It rarely occurs in cattle in late gestation, at which time it resembles pregnancy toxemia of ewes. In addition to a poor appetite, signs of nervous dysfunction, including pica, abnormal licking, uncoordinated movement, and abnormal gait, bellowing, and aggression are occasionally seen. The condition is in distribution worldwide, but is most common where dairy cows are bred and managed for high production.

Ethiology and pathogenesis
The pathogenesis of bovine ketosis is incompletely understood, but it requires the combination of intense adipose mobilisation and a high glucose demand. Both of these conditions are present in early lactation, at which time negative energy balance leads to adipose mobilisation, and milk synthesis creates a high glucose demand. Adipose mobilisation is accompanied by high blood serum concentrations of non-esterified fatty acids (NEFAs). During periods of intense gluconeogenesis, a large portion of serum NEFAs is directed to ketone body synthesis in the liver. Thus, the clinicopathologic characterisation of ketosis includes high serum concentrations of NEFAs and ketone bodies and low concentrations of glucose. In contrast to many other species, cattle with hyperketonemia do not have concurrent acidemia. The serum ketone bodies are acetone, acetoacetate, and β-hydroxybutyrate (BHB).

It seems that the pathogenesis of ketosis cases occurring in the immediate postpartum period is slightly different from that of cases occurring closer to the time of peak milk production. Ketosis in the immediate postpartum period is sometimes described as type II ketosis. Such cases of ketosis in very early lactation are usually associated with the fatty liver syndrome. Both fatty liver and ketosis are probably part of a spectrum of conditions associated with intense fat mobilisation in cattle. Ketosis cases occurring closer to peak milk production, which usually occurs at 4 – 6 weeks postpartum, may be more closely associated with underfed cattle experiencing a metabolic shortage of gluco-neogenic precursors than with excessive fat mobilisation. Ketosis at this time is described as type I ketosis.

Epidemiology
All dairy cows in early lactation (first 6 weeks) are at risk of ketosis. The overall prevalence in cattle in the first 60 days of lactation is estimated at 7% – 14%, but prevalence in individual herds varies substantially and may exceed 14%. The peak prevalence of ketosis occurs in the first 2 weeks of lactation. The incidence of ketosis rates vary between herds with some herds showing high levels and others low levels. Ketosis is seen in all parities (although it appears to be less common in primiparous animals) and does not appear to have a genetic predisposition, other than being associated with dairy breeds. Cows with excessive adipose stores (body condition score ≥ 3.75 out of 5) at calving are at a greater risk of ketosis than those with lower body condition scores. Lactating cows with subclinical ketosis are also at a greater risk of developing clinical ketosis and displaced abomasum than cows with lower serum BHB concentrations.

Clinical Findings
In cows maintained in confinement stalls, reduced feed intake is usually the first sign of ketosis. If rations are offered in components, cows with ketosis often refuse grain before forage. In group-fed herds, reduced milk production, lethargy, and an “empty” appearing abdomen are usually the signs of ketosis noticed first. On physical examination, cows may be slightly dehydrated. Rumen motility is variable, being hyperactive in some cases and hypoactive in others. In many cases, there are no other physical abnormalities. Some cows show abnormal licking and chewing, e.g. chewing on pipes and other objects in their surroundings. Muscle incoordination and gait abnormalities are seen occasionally, as well as aggression and bellowing. These signs occur in a clear minority of cases, but because the disease is so common, finding animals with these signs is not unusual.

Diagnosis
The clinical diagnosis of ketosis is based on presence of risk factors (early lactation), clinical signs, and ketone bodies in urine or milk. When a diagnosis of ketosis is made, a thorough physical examination should be performed, because ketosis frequently occurs concurrently with other peripartum diseases.
Especially common concurrent diseases include displaced abomasum, retained fetal membranes, and metritis.

Cow-side tests for the presence of ketone bodies in urine or milk are critical for diagnosis. Most commercially available test kits are based on the presence of acetoacetate or acetone in milk or urine. Dipstick tests are convenient, but tests aimed to detect acetoacetate or acetone in urine samples, are not suitable for milk testing. All of these tests are read by observation for a particular colour change. Care should be taken to allow the appropriate time for colour development as specified by the test manufacturer. Also, handheld instruments designed to monitor ketone bodies in the blood of human diabetic patients, are available. These instruments quantitatively measure the concentration of BHB in blood, urine, or milk and may be used for the clinical diagnosis of ketosis.

In a given animal, urine ketone body concentrations are always higher than milk ketone body concentrations. Trace to mildly positive results for the presence of ketone bodies in urine does not signify clinical ketosis. Without clinical signs, such as partial anorexia, these results indicate subclinical ketosis. Milk tests for acetone and acetoacetate are more specific than urine tests. Positive milk tests for acetoacetate and/or acetone usually indicate clinical ketosis. BHB concentrations in milk may be measured by a dipstick method that is available in some countries, or by the electronic device mentioned above. The BHB concentration in milk is always higher than the acetoacetate or acetone concentration, making the tests based on BHB more sensitive than those based on acetoacetate or acetone.

Treatment
Treatment of ketosis is aimed at re-establishing normal glycogen levels and reducing serum ketone body concentrations. Bolus intravenous (IV) administration of 500 mL of 50% dextrose solution is a common therapy. This solution is very hyper-osmotic and, if administered perivascular, may result in severe tissue swelling and irritation, so care should be taken to ensure that it is administered correctly. Bolus glucose therapy generally results in rapid recovery, especially in cases occurring near peak lactation (type I ketosis). However, the effect is frequently transient and relapses are common. Administration of glucocorticoids, including dexamethasone or isoflupredone acetate at 5 – 20 mg/dose, intramuscular (IM) may result in a more sustained response, relative to glucose alone. Glucose and glucocorticoid therapy may be repeated daily as necessary. Propylene glycol administered orally (250 – 400 g/dose) once per day acts as a glucose precursor and is effective as ketosis therapy. Indeed, propylene glycol appears to be the most well documented of the various therapies for ketosis. Overdosing propylene glycol leads to central nervous system (CNS) depression.

Ketosis cases occurring within the first two weeks after calving (type II ketosis) are frequently more refractory to therapy than cases occurring nearer to peak lactation (type I). In these cases, a long-acting insulin preparation given IM at 150 – 200 IU/day may be beneficial. Insulin suppresses both adipose mobilisation and ketogenesis, but should be given in combination with glucose or a glucocorticoid to prevent hypoglycemia. Use of insulin in this manner is an extra-label, unapproved use. Other therapies that may be of benefit in refractory ketosis cases are continuous IV glucose infusion and tube feeding.

Prevention and control
Prevention of ketosis is through nutritional management. Body condition should be managed in late lactation, when cows frequently become too fat. Modifying diets of late lactation cows to increase the energy supply from digestible fibre and reduce the energy supply from starch may aid in partitioning dietary energy toward milk and away from body fattening. The dry period is generally too late to reduce body condition score. Reducing body condition in the dry period, particularly in the late dry period, may even be counterproductive, resulting in excessive adipose mobilisation pre-partum. A critical area in ketosis prevention is maintaining and promoting feed intake. Cows tend to reduce feed consumption in the last 3 weeks of gestation. Nutritional management should be aimed at minimising this reduction. Controversy exists regarding the optimal dietary characteristics during this period. It is likely that optimal energy and fibre concentrations in rations for cows in the last 3 weeks of gestation vary from farm to farm. Feed intake should be monitored and rations adjusted to meet, but not greatly exceed, energy requirements throughout the entire dry period. For Holstein cows of typical adult body size, the average daily energy requirement throughout the dry
period is between 12 and 15 Mcal, expressed as net energy for lactation (NEL). After calving, diets should promote rapid and sustained increases in feed and energy consumption. Early lactation rations should be relatively high in nonfibre carbohydrate concentration, but contain enough fibre to maintain rumen health and feed intake. Neutral-detergent fibre concentrations should usually be in the range of 28% – 30%, with nonfibre carbohydrate concentrations in the range of 38% – 41%. Dietary particle size will influence the optimal proportions of carbohydrate fractions. Some feed additives, including niacin, calcium propionate, sodium propionate, propylene glycol, and rumen-protected choline, may help prevent and manage ketosis. To be effective, these supplements should be fed in the last two to three weeks of gestation, as well as during the period of ketosis susceptibility. In some countries, monensin sodium is approved for use in preventing subclinical ketosis and its associated diseases. Where approved, it is recommended at the rate of 200 – 300 mg/head/day.

Ruminant animals are adapted to digest and metabolise predominantly forage diets; however, growth rates and milk production are increased substantially when ruminants consume high-grain diets. One consequence of feeding excessive amounts of rapidly fermentable carbohydrates in conjunction with inadequate fibre to ruminants is subacute ruminal acidosis, which is characterised by periods of low ruminal pH that resolve without treatment and is rarely diagnosed. Dairy cows, feedlot cattle, and feedlot sheep are at risk of developing this condition.

4. Acidosis

Ruminal pH fluctuates considerably during a twenty-four-hour-period (typically between 0.5 – 1 pH units) and is determined by the dynamic balance between the intake of fermentable carbohydrates, buffering capacity of the rumen, and rate of acid absorption from the rumen. In general, subacute ruminal acidosis is caused by ingestion of diets high in rapidly fermentable carbohydrates and/or deficient in physically active fibre. Subacute ruminal acidosis is most commonly defined as repeatedly occurring, prolonged periods of depression of the ruminal pH to values between 5.6 and 5.2. The low ruminal pH is caused by excessive accumulation of volatile fatty acids (VFAs) without persistent lactic acid accumulation and is restored to normal by the animal's own physiologic responses.

The ability of the rumen to rapidly absorb organic acids contributes greatly to the stability of ruminal pH. It is rarely difficult for peripheral tissues to utilise VFAs already absorbed from the rumen; however, absorption of these VFAs from the rumen can be an important bottleneck.

Ruminal VFAs are absorbed passively across the rumen wall. This passive absorption is enhanced by finger-like papillae, which project away from the rumen wall and provide massive surface area for absorption. Ruminal papillae increase in length when cattle are fed high-grain diets; this presumably increases ruminal surface area and absorptive capacity, which protects the animal from acid accumulation in the rumen. Dairy cows are especially at risk in the transition period, because the ruminal mucosa needs several weeks to adjust to high-grain diets, and in peak lactation, when high levels of easily fermentable carbohydrates are fed to avoid excessive negative energy balance.

One mechanism by which affected animals resolve ruminal acidosis and return ruminal pH to normal is by selecting long forage particles, either by choosing to preferentially consume long dry hay or by sorting a mixed ration in favour of longer forage particles. Another mechanism is by reducing overall feed intake. Depressed dry-matter intake becomes especially evident if ruminal pH falls below ~5.5. Intake depression may be mediated by pH receptors and/or osmolality receptors in the rumen. Inflammation of the ruminal epithelium (rumenitis) could cause pain and also contribute to intake depression during subacute ruminal acidosis.

Absorption of VFA inherently increases as ruminal pH drops. These acids are absorbed only in the protonated state. Because they have a $pK_a$ of ~4.8, the proportion of these acids that is protonated increases dramatically as ruminal pH decreases below 5.5. Lactate levels in the ruminal fluid of cattle with subacute ruminal acidosis, if measured, are usually not increased; however, the pathogenesis of excessive lactate production in the rumen is well-described. Ruminal carbohydrate fermentation shifts to lactate production at lower ruminal pH (mostly due to *Streptococcus bovis* proliferating and shifting to lactate instead...
of VFA production); this can offset gains from VFA absorption. Ruminal lactate production is undesirable, because lactate has a much lower pKa than VFAs (3.9 vs. 4.8). For example, lactate is 5.2 times less protonated than VFAs at pH 5. As a result, lactate stays in the rumen longer and contributes to the downward spiral in ruminal pH.

Additional adaptive responses are invoked if lactate production begins. Lactate-utilising bacteria, such as *Megasphaera elsdenii* and *Selenomonas ruminantium*, begin to proliferate. These beneficial bacteria convert lactate to other VFAs, which are then easily protonated and absorbed. However, the turnover time of lactate utilisers is much slower than that of lactate synthesisers. Thus, this mechanism may not be invoked quickly enough to fully stabilise ruminal pH. Periods of very high ruminal pH, such as during feed deprivation, may inhibit populations of lactate utilisers (which are sensitive to higher ruminal pH) and leave them more susceptible to severe ruminal acidosis.

Besides disrupting microbial balance, feed deprivation causes cattle to overeat when feed is reintroduced. This creates a double effect in lowering ruminal pH. Cycles of feed deprivation followed by overconsumption greatly increase the risk of subacute ruminal acidosis.

Low ruminal pH during subacute ruminal acidosis also reduces the number of species of bacteria in the rumen, although the metabolic activity of the bacteria that remain is very high. Protozoal populations are particularly limited at lower ruminal pH; the absence of ciliated protozoa in ruminal fluid is often observed during bouts of subacute ruminal acidosis. When fewer species of bacteria and protozoa are present, the ruminal microflora are less stable and less able to maintain normal ruminal pH during periods of sudden dietary change. Thus, periods of subacute ruminal acidosis leave animals more susceptible to future episodes of ruminal acidosis.

The pathophysiologic consequences of ruminal acidosis have mainly been described in feedlot cattle and in cattle surviving acute ruminal acidosis. Low ruminal pH may lead to rumenitis, erosion, and ulceration of the ruminal epithelium. Once the ruminal epithelium is inflamed, bacteria may colonise the papillae and leak into the portal circulation. These bacteria may cause liver abscesses, which may eventually lead to peritonitis around the site of the abscess.

Subacute ruminal acidosis has traditionally been associated with claw horn lesions, assumed to be caused by subacute laminitis. However, this pathophysiologic mechanism has not been experimentally characterised or reproduced.

**Clinical Findings**

The main clinical signs attributed to subacute ruminal acidosis are reduced or cyclic feed intake, decreased milk production, reduced fat, poor body condition score despite adequate feed intake, and unexplained diarrhea. High rates of culling or unexplained deaths may be noted in the herd. Sporadic cases of caudal vena cava syndrome may also be seen. The clinical signs are delayed and insidious. Actual episodes of low ruminal pH are not identified; in fact, by the time an animal is observed to be off-feed, its ruminal pH has probably been restored to normal. Diarrhea may follow periods of low ruminal pH; however, this finding is inconsistent and may be related to other dietary factors as well.

**Diagnosis**

Subacute ruminal acidosis is diagnosed on a group rather than individual basis. Measurement of pH in the ruminal fluid of a representative portion of apparently healthy animals in a group has been used to help make the diagnosis of subacute ruminal acidosis in dairy herds. Animal selection should be from highest-risk groups: cows between ~15 - 30 days in milk in component-fed herds and cows between ~50 - 150 days in milk in herds fed total mixed rations. Ruminal fluid is collected, and its pH is determined using a pH meter. Twelve or more animals are typically sampled about 2 - 4 hr after being fed grain (in component-fed herds) or 6 - 10 hr after the first daily feeding of a total mixed ration. When more than 25% of animals tested have a ruminal pH below 5.5, then the group is considered to be at high risk of subacute ruminal acidosis. This type of diagnostic tool should be used in conjunction with other factors, such as ration evaluation, evaluation of management practices, and identification of health problems on a herd basis.

Milk fat depression is a poor and insensitive indicator of subacute ruminal acidosis in dairy herds.
Treatment and prevention

The key to prevention of subacute ruminal acidosis is allowing for ruminal adaption to high-grain diets, as well as limiting the intake of readily fermentable carbohydrates. This requires both good diet formulation (proper balance of fibre and non-fibre carbohydrates) and excellent feed bunk management. Animals consuming well-formulated diets remain at high risk of this condition if they tend to eat large meals because of excessive competition for bunk space or after periods of feed deprivation.

Field recommendations to feed component-fed concentrates to dairy cattle during the first 3 weeks of the lactation period are usually excessive. Subacute ruminal acidosis is commonly caused by feeding a diet containing high levels of concentrates and low levels of forages. The same situation may be seen during the last few days before parturition if the ration is fed in separate components; as dry-matter intake drops before calving, dry cows preferentially consume concentrates instead of forage and develop acidosis.

Including long-fibre particles in the diet reduces the risk of subacute ruminal acidosis by encouraging saliva production during chewing and by increasing rumination after feeding. The provision of adequate long-fibre particles reduces the risk of ruminal acidosis, but cannot eliminate it. If a total mixed ration is fed, it is important that the long-fibre particles not be easily sorted away from the rest of the diet; this could delay their consumption until later in the day or cause them to be refused completely. Sorting can be prevented by providing long-fibre particles less than ~5 cm in length, by having adequate (~50% - 55%) moisture in the mixed ration, and by including ingredients such as liquid molasses that help ration ingredients stick together.

Ruminant diets should also be formulated to provide adequate buffering. This can be accomplished by feedstuff selection and/or by addition of dietary buffers, such as sodium bicarbonate or potassium carbonate.

Supplementing the diet with direct-fed microbials that enhance lactate utilisation in the rumen may reduce the risk of subacute ruminal acidosis. Yeasts, propionobacteria, lactobacilli, and enterococci have been used for this purpose. Ionophore (e.g. monensin sodium) supplementation may also reduce the risk by selectively inhibiting ruminal lactate producers and by reducing meal size.

In closing

Metabolic diseases in dairy cows may be prevented through correct feed formulation and feeding management. Milk fever is triggered by the demand for Ca in colostrums and milk at calving. Cows should be prepared for this by feeding them correctly during the steam-up period. Bloat is caused by cows consuming lush legume-rich pastures when they are hungry. Bloat can be prevented by feeding management, such as feeding hay or silage before going onto suspect pastures. Ketosis is usually caused by a combination of intense adipose mobilisation and a high glucose demand, such as in early lactation. Subacute ruminal acidosis is caused by cows consuming diets high in rapidly fermentable carbohydrates and/or deficient in physically active fibre.
CHAPTER 55
VACCINES AND IMMUNISATION OF DAIRY CATTLE

Introduction

Maintaining an effective animal health programme is an essential part of a successful dairy enterprise. Good animal health is vital for maximum production since dairy cows must be healthy to reach their production potential. Since each dairy operation is unique, owners (or managers) must establish a herd health plan in cooperation with the herd veterinarian. It is important that appropriate records are correctly kept of animals that are being culled. It is also important to record the reason for animals dying, to keep record of ongoing treatments, as well as all aspects of herd health. This information should be used to plan a herd health programme involving vaccination, deworming, dipping, calf rearing, prevention of metabolic diseases, metritis and reproduction management, and/or mastitis control, etc. Therefore, a sound vaccination programme is only part of a herd health plan and requires planning and consultation with the herd veterinarian who should be aware of the diseases of importance in a specific area.

Vaccination

Vaccination stimulates the immune system of the animal producing protective antibodies that will help to combat invading disease organisms in the body of an animal.

The most common vaccines available on the market are either killed or live vaccines. So when vaccines are prepared, it is essential that one of the following alternatives must be followed:

1. The organism should first be killed, or the toxin should be inactivated. These vaccines are known as killed or inactive vaccines.
2. Vaccines where the organisms are alive, but weakened, are referred to as live or attenuated vaccines.
3. In some cases, the organism used in the preparation of the vaccine is not altered, or certain strains are used that are not as virulent – these are also live vaccines.

In practice, for the dairy manager, the differences between killed and live vaccines are the following:

1. Killed vaccines are distributed as in suspension, and need only be shaken before they can be used. Live vaccines are distributed in small bottles and the live micro-organisms are freeze-dried into a pill or powder form. The pill or powder must be reconstituted to a fluid by adding sterile water just before use.
2. Killed vaccines can be stored at 20°C inside a cool and dark place while live vaccines must be kept at 4°C inside a refrigerator. Some live vaccines must even be stored at much lower temperatures. Killed vaccines can be stored in refrigerators, but they must not be frozen.
3. The immunity against killed vaccines is usually of short duration. For that reason, it is recommended to repeat vaccination after 3 - 4 weeks and thereafter annually. The immunity against some live vaccines is of a life-long duration and it is not necessary to repeat vaccinations annually.
4. Killed vaccines normally don’t cause a fever reaction or symptoms of the disease, and they are safe to be given to pregnant and high producing milk cows. On the other hand, live vaccines may cause a mild form of the disease and even a fever reaction. Some live vaccines should therefore never be given to pregnant or high producing dairy cows.

It is critical to always read the vaccine instructions carefully and to follow them. Due to the fact that vaccines are highly perishable products, they should be handled with great caution. In order to ensure good results, the following precautionary measures should be carried out very strictly:

1. When buying vaccines one must make sure that they are stored in the correct way. All vaccines have an expiry date printed on the container. Vaccines should be used before the expiry date has elapsed.
2. Vaccines should be kept in a cool bag/
container when being transported. They should never be left inside the cabin of a vehicle, because it can become extremely hot inside the cabin.

3. Exposure to direct sunlight must be avoided at all times, especially for live vaccines.

4. At home, vaccines should be stored under the prescribed conditions, i.e. inside a refrigerator at all times, but not frozen at any stage.

5. Live vaccines should be reconstituted using sterile water just before vaccination is to start. Killed or inactivated vaccines need only to be shaken before being used.

6. Only sterilised syringes and needles should be used when vaccinating animals. Due to the fact that disinfectants and alcohol substances especially can harm the organisms in live vaccines, they should never be used before and during the vaccinations. The boiling of syringes and needles in water for 30 minutes remains the best method of sterilisation. Only clean, unused needles should be used to penetrate the rubber stopper of the vaccine bottles.

7. It is essential that each animal be vaccinated with a clean needle, as some of the animals being vaccinated may be sick. In practice it often happens that more than one animal is vaccinated with the same needle, but this must be kept to a minimum. Needles need to be changed as often as possible. When vaccinating a sick animal, or when an abscess has been struck, the needle must be replaced at once.

8. The correct dose must be given, using the correct route of vaccination. Most vaccines are administered under the skin (subcutaneous) or in the muscle (intramuscular). Subcutaneous vaccination should be done in front of the shoulder in the neck area, and intra-muscular injections are done in the rump area.

9. At the end of the day, when only a portion of the vaccine is left in the container, the container and its contents must be destroyed, because pathogenic bacteria may easily enter the container where they will multiply and cause problems later on.

Animal vaccines are not to be used in humans and special care should be taken not to inject oneself by mistake. Accidental contact with the Brucellosis vaccine, for instance, either by a needle prick, or contact with drops of vaccine in the eyes, can infect the vaccinator with brucellosis. Brucellosis is a serious disease for humans. Symptoms may show up anytime from a few days to a few months after being infected. Signs and symptoms are similar to those of flu and include: fever, chills, sweats, general weakness, fatigue, joint and muscle pain and headaches. In some people, brucellosis becomes chronic with symptoms persisting for years.

The basic principle in compiling a vaccination programme for a dairy herd is to divide the type of vaccines to be used into three major categories.

A. Vaccination of cows during the dry period

The aim of vaccinating cows in the pre-partum period (before calving down) is to stimulate colostrum immunity to protect the new-born calf up to three to four months of age. The following diseases of calves can be protected through colostrum immunity:

- **Escherichia coli**,  
- Rota/Carona virus infection causing scours,  
- *Salmonella* infection causing scours,  
- Pasteurella lung infection, and  
- Bovine viral diarrhoea (BVD).

These vaccines are administered to the cows at least one month to a fortnight before calving to ensure enough antibodies are excreted through the colostrum.

The absorption of maternal antibodies through the digestive tract of the new-born calf takes place within the first four to eight hours after birth. It is therefore important for the survival of the newborn calf to receive at least one to two litres of colostrum within the first four to eight hours of their lives.

B. Vaccination of calves and heifers from one month of age to 15 months

The vaccination programme for calves and heifers from one month of age should protect them against diseases like:

- Pasteurella,  
- BVD,  
- Bovine herpes virus 1 (Infectious bovine rhinotracheitis or IBR),  
- Para-influenza virus type 3,  
- *Salmonella*,  
- All the *Clostridium* diseases, like Botulism,
Black Quarter,
- Anthrax, Brucellosis, Leptospirosis,
  Lumpy Skin Disease.

Tick-borne disease vaccines can also be administered in this age group. Deep frozen blood vaccines that are available include:

- Babesiosis (Redwater),
- African and Asiatic Anaplasmosis (tick-borne gall sickness), and
- Heartwater (Ehrlichiosis).

The blood vaccines cause the respective disease in animals, which may require appropriate differential treatment to prevent clinical symptoms, but at specific times in order to allow sufficient immunity to develop. The vaccination of pregnant animals is not advised.

When young animals are vaccinated using blood vaccines, the treatment of vaccine reactions should be unnecessary.

- Redwater – vaccinate calves when they are 3 to 9 months of age.
- Anaplasmosis – vaccinate calves when they are 3 to 9 months of age.
- Heartwater – vaccinate calves when they are 4 to 6 weeks of age.

C. Annual vaccination programme for the adult cow herd

This annual vaccination programme should protect cows against diseases like:

- Lumpy skin disease,
- Anthrax,
- *Clostridium* diseases,
- Rift Valley fever,
- Ephemeral fever, and
- *E. coli* mastitis.

In closing

For the best results with regards to vaccination and dosing programmes in dairy herds, the manufacturer’s recommendations with regards to dosage amounts, methods of administration, number of times treated, and storage of products, should always be followed. To set up a vaccination programme, the local or herd veterinarian should be consulted as he would be familiar with the animals in the herd.
INTRODUCTION

E
evry dairy farm is unique in its layout, number of animals, the way it is being managed, and challenges to be faced. A practical bio-security programme should be developed individually for each farm in close collaboration with the herd veterinarian. This should be based on risk-assessment. A bio-security programme basically involves the management of a dairy herd in such a way that the introduction and spreading of infectious diseases is prevented. It is the responsibility of the farmer or herd owner to develop and apply a bio-security programme at all times.

KNOW THE RISK

Dairy farmers have to be aware of all diseases of concern to cattle in the local area, as well as endemic and foreign diseases that may occur in cattle. A risk assessment of the farm should be done by carefully scrutinising the layout and design of the farm, all on-farm activities, as well as activities of service providers and visitors to identify all hazards. The risk associated with each different hazard should be carefully analysed. Appropriate ways to eliminate or control hazards and include those measures in a bio-security programme for the farm should be determined. As a general guideline biosecurity measures should include the following principles:

1. **Keep a closed herd**
   Keeping a closed herd is probably the best way to protect cattle being exposed to infectious diseases. A herd is not closed when cattle are purchased or rented or boarded from other herds or owners, when cattle have attended and returned from agricultural shows, or when cows use commonages or have attended and returned from performance testing centres. The herd is also not closed when bulls are purchased, borrowed or loaned, when cattle utilise pasture that shares a fence line with cattle on pastures on a neighbouring farm, or when cattle from the herd are transported by someone else or in someone else’s vehicle. When any of the above actions take place on the farm, measures should be added to the bio-security plan towards minimising the risk of introducing diseases to the farm.

2. **Isolate “new” animals**
   Cattle, embryos or semen should only be bought from reputable sources. The introduction of “new” dairy cattle usually presents the greatest risk, since these animals’ disease status is unknown. All “new” cattle to the farm should be isolated for a minimum period of 30 days (the incubation period of the majority of diseases) in an area at least 10 metres away from the milking parlour and herd. These animals should be observed daily for signs of diarrhoea, reduced feed intake, weight loss, breathing difficulty, lameness, strange behaviour, etc. In addition, these animals’ temperature should be recorded every day or at least every other day for signs of fever. Any problems should be reported to the herd veterinarian. The isolation period should be used to de-worm and treat animals against external parasites. A comprehensive vaccination programme should also be applied during this period. The sooner this process is started, the sooner “new” animals can be introduced into the herd.

3. **Prevent contact with the neighbour’s cattle**
   Fences should be kept in a good condition; this will keep animals in and the neighbour’s animals outside the farm. In addition, pastures should preferably not be used that share a fence line with cattle on pastures on a neighbouring farm. Any animal that has been off the farm or that has come into contact with non-resident cattle should be considered as a “new” animal and treat them as described above.

4. **Recognise susceptibility and maintain separation**
   There are a number of different systems and groups of animals on a dairy farm. The distinctive production areas and their risk levels should be identified, and contact between these areas should be minimised. Keeping the younger, more
susceptible animals away from the older ones, separating the sick animals from the healthy ones and the “new” from the resident animals, can go far in preventing the transmission of diseases.

**Monitor regularly for any sign of disease**

Cattle should be observed regularly for early detection of signs of possible diseases. Animals must be tested regularly for diseases. The following principles should apply:

- **Vaccinate strategically**
  Strategic vaccination is an essential component of disease prevention. An Animal Health Management Plan should be developed in consultation with the herd veterinarian. Such a plan must determine what diseases occur in the area, which animals are the most susceptible and when to vaccinate to get the best protection. Cattle should be vaccinated according to the recommendations of the manufacturer and herd veterinarian.

- **Isolate sick animals**
  Pathogens can spread by direct animal-to-animal contact through secretions and faecal material. Sick animals should be isolated immediately from the rest of the herd and kept in a “hospital” camp or stall. Any unusual or unknown cases of diseases should be reported immediately; sick animals should be subjected to clinical examinations and get an accurate and prompt diagnosis by the herd veterinarian. Animals should be treated as soon as possible after diagnosis and sick animals should not be allowed back into the herd. All cases of diseases should be recorded, as well as treatments administered. This data should be summarised and analysed at regular intervals so as to revise the effectiveness of the on-farm biosecurity programme.

- **Be aware of possible “carriers” of disease**
  Resident cattle that have seemingly recovered from a disease can still be “carriers” and could therefore pose a threat to other cattle. The herd should therefore be observed for any symptoms of diseases for at least 4 weeks after “healthy” animals have been returned to the herd.

- **Minimise contact with dead cattle**
  Most infectious agents can survive in a carcass for a considerable period of time, so as to prevent spreading of pathogens, carcasses should be removed carefully as quickly as possible from places where they have been sick and died. No material related to the diseased and dead animals should be left behind. In order to rule out exotic disease, an accurate and prompt diagnosis of the cause of death by the herd veterinarian through a post mortem should be determined. The number of animals that have died should be recorded, as well as the cause of their death.

- **Limit contact with other farm animals and pets**
  Animals, other than cattle of the appropriate age, should not be permitted access to cattle rearing or holding facilities. No animals (including home pets) should have access to buildings that is used to store feed, equipment, or the milking parlour. Potential risks posed by other farm animal species should be considered. Salmonellosis has been reported to be transmitted from feral cats, dogs, pigs, goats and poultry. Cats can spread toxoplasmosis, a frequently incurable protozoan disease.

- **Control insects**
  Flies, blowflies, and other Arthropods are reservoirs and vectors of a wide variety of pathogenic organisms. Moist manure in cattle rearing facilities presents an ideal habitat for the development of large populations of the house fly and related species of flies normally found in manure. Breeding of flies should be controlled by regularly cleaning out rearing facilities and by minimising the accumulation of manure (e.g. spreading it out on pastures or by having it removed).

- **Control rodents—rats/mice**
  Rodents should be discouraged as far as possible from entering the milking parlour, feed stores, other stores, etc., by keeping areas around the buildings free from unwanted vegetation and debris that could attract or harbour pests. Rodents should be controlled by maintaining baited poison stations. If possible, feed should be stored in rodent-proof containers as rodent droppings can harbour disease-causing organisms such as Salmonella.
**Safe disposal of carcasses**

Carcasses should be carefully transported and disposed at a secure disposal site. Rodents, flies, wildlife and other scavengers (including pets e.g. dogs and cats) should be prevented from coming into contact with carcasses as they can serve as mechanical vectors, spreading disease all over your farm and to neighbouring farms. Potential disease-agents should be contained by burying carcasses at a depth of at least 4 - 5 m, but not lower than the water-table. The carcass should be covered with 400 mm of soil, and on top of that, a layer of chalk should be added. This will ensure that earthworms do not carry pathogens to the surface. In addition, at least 2 m of soil should be added to cover the carcass to ensure containment of the disease-agent. Please note that in case of a notifiable disease, methods of disposal as prescribed by the Provincial State Veterinarian, or as prescribed by other legislation applicable to the disposal of dead animals, will apply.

**Sanitise facilities**

To disinfect buildings used for cattle rearing, the following steps should be taken:

1. Clean the building by removing all old feed, manure, loose dirt, cobwebs, etc.; scrub all surfaces with a detergent and then rinse detergent and organic matter from surfaces (a steam or high-pressure water hose may be helpful).
2. Sanitise the building by applying disinfectant and allow it to dry completely, before reappplying the disinfectant and allowing it to dry a second time.
3. Rinse all water and feeding equipment before refilling them. Use only detergents and disinfectants registered for use in dairy production operations.

**Limit access to the farm**

Access to the farm should be controlled to ensure that only authorised persons and vehicles enter the production areas. A dedicated parking area or “Visitors parking”, which is isolated from the production areas, should be provided and clearly identified. Commercial vehicles that move between farms transporting and delivering bulk feed should be prevented from entering the production area by storing bulk feed supplies as far away as possible from the production area. Warning signs should be posted at the main entrance to the production area to indicate the concern for the spreading of diseases. The number of visitors from other farms should be restricted.

**Prevent introduction and spreading of infectious agents by visitors and personnel**

Infectious agents can be spread on shoes, clothing and the hands of people who move from farm to farm, between production areas or from one herd to the next herd. Visitors from urban areas or visitors who have no contact with livestock, present very little risk of introducing disease or pathogens to the farm. However, neighbouring farmers, veterinarians, and some sales representative coming in direct contact with dairy cattle or other farm animals are considered high risk visitors. Such visitors should preferably shower and change into clean clothes and footwear before entering production areas on the farm. As a general preventative measure, clean coats or overalls and footwear should be supplied to all visitors to the production areas. As infectious agents remain viable in soil for a long period, footwear should be disinfected before entering a unit and all demarcated areas in a production unit. Although strategically placed foot baths for visitors may help to decrease the transfer of organisms by footwear, a poorly managed foot bath may become a source of infection. The disinfectant solution in the foot bath should be changed on a regular basis to ensure its efficacy. The disinfectant’s dosage, whether in a hand spray or in the foot bath, should be according to the manufacturer’s recommendations. Production area personnel and visitors can either use a properly maintained disinfectant foot bath or a boot spray.

Production area personnel must wear freshly laundered overalls every day. Overalls and footwear should not be shared between one production area and the next. All visitors and production area personnel should, on entering a production unit, as well as on leaving the unit, wash their hands with soap and water or sanitise them using a disinfectant while also disinfecting shoes.

**Manage on-farm activities: Personnel**

Although not always feasible, it would be best to appoint dedicated personnel for each production area or for a specific...
group of animals, like young calves, heifers, dry cows, and sick animals normally kept in isolation. Therefore, as standard practice, production area personnel must, on a daily basis, start with the most susceptible animals (usually young calves) and then only move on to the ones that are not as susceptible. Personnel responsible for taking care of sick animals should only work with them after they have finished their tasks concerning healthy animals and preferably as the last task of the day. Movement between these groups should not be done without a change in overalls and shoes, and before hands and shoes have been properly washed and/or disinfected.

- **Manage on farm activities: Usage and movement of vehicles**
  In a farming environment, it is well accepted that some vehicles have a multi-purpose use. Small trucks or trailers are often used to transport feed bales, bales of bedding material, and newly-born calves from the maternity area to the calf pen area, as well as young calves to auction yards. Such vehicles may also be used to transport dead animals to the veterinarian’s office for post mortem examinations. The risk of spreading infectious agents should be considered when using such multi-purpose vehicles. The route should be planned to reduce this possibility by visiting the most susceptible animals first and then only move on to the rest of the animals. The potential spreading of diseases by using the same piece of road repeatedly should also be considered. The sick animals or animals in isolation should be checked up on after being to the “healthy” animals. Any vehicle entering the “isolation area” or “hospital camp” must be cleaned and disinfected before moving onto the rest of the farm. Cattle should only be transported in vehicles specifically adapted for this purpose. Vehicles should be cleaned and disinfected prior to loading a consignment of animals. Vehicles used for the disposal of dead cattle must be properly cleaned and disinfected before using it for anything else.

- **Minimise contact with waste coming from cattle**
  Potentially infectious material and material that is known to provide a pathway for pathogens (e.g. manure and body fluids, discarded milk, soiled bedding, and spilled or leftover/excess feed) should be removed. Alleyways should regularly be scraped and manure and waste should be removed from housing and milking areas. Manure and waste should be handled with equipment that is not used for other functions, or that is cleaned and disinfected between uses. Waste should be moved to a secluded area to minimise contact with cattle and to limit contamination of pathways and production areas. The storage area should be constructed so as to ensure that runoff will not reach and contaminate active production, feed storage or feed transfer areas. Other waste should be removed to municipal landfills or to a hazardous waste site as appropriate.

  **Water**
  Water supplied to cattle must be according to the standard for drinking water for livestock. Water troughs should be cleaned and disinfected on a weekly basis.

  **Feed**
  Feed could be contaminated or become contaminated by raw materials being used, during transportation, post-production, or by exposure to rodents and birds. Bacteria and mould in poor quality or damaged feed are also a concern. Commercial feeds should be acquired from officially registered feed mills. Feed should be stored in clean and sealed containers (silos or bags). New batches of feed should be stored separately from previously bought feeds. All documents should be kept for traceability purposes. It should be ensured that feed bins and containers are free of vermin and that wild birds do not have access to stored feed.
**Recordkeeping**

Up-to-date records of all movements of cattle (on and off farm), all on-farm actions (e.g., cleaning and disinfection, rodent control, etc.), and all transactions (feed, semen, etc.) should be maintained for traceability purposes in case of a disease outbreak. Records should also be maintained on an individual animal basis and include data on identification, animal health, production, medications, withholding-period, vaccination, mortality, and surveillance.

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**In closing**

Dairy farms are unique in their layout, number of animals, management style, and challenges. A practical bio-security programme, based on risk-assessment, should be developed for each farm by the farmer in collaboration with the herd veterinarian. Bio-security is managing the herd in such a way to prevent the introduction and spreading of most infectious diseases. For successful on-farm bio-security, everyone on the farm should be appropriately trained in bio-security measures while committing to apply it totally.
CHAPTER 57
LAMENESS IN DAIRY CATTLE

Introduction

Due to ever-increasing production costs and erratic farm-gate milk prices, dairy farmers today must always find alternative ways to keep farming profitable and sustainable. Due to economic pressures, modern dairy farms have become highly intensive. Increasing numbers of cows are kept on the same farm for the benefit of economy of scale for a higher dairy farm income towards financial sustainability. However, this inevitably leads to increased health and welfare problems among dairy cows. This is because dairy cows are increasingly kept under more intensive production systems, being exposed for longer periods to concrete surfaces and with their hoofs in daily contact with manure. This has resulted in lameness becoming the third largest problem in dairy cows following reproductive failure and mastitis.

Lameness reduces herd income

Lameness not only causes major economic losses in dairy herds, it also causes considerable pain to cows leading to serious welfare issues in dairy herds. Farm output is affected when cows are lame. Lame cows are in pain and stressed, and therefore eat less, start to lose live weight, and show a reduction in milk yield. Cows may lose up to 1.5 kg of milk/day when suffering from lameness problems. Lame cows need veterinary attention (incurring veterinarian cost) and treatments like antibiotics that cost extra money. Because of the antibiotic treatment, milk has to be discarded during the treatment period and up to the end of the withdrawal period. The farmer needs to spend extra time caring for the sick cows that he could have used more productively. Cows, to be culled for lameness, often have a lower price at the abattoir due to the reduction in live weight. The withdrawal period of antibiotic treatments for lameness could also affect the time of slaughter. Therefore, lameness not only affects milk yield, but also the value of the culled cows at sale.

Lameness also affects the reproductive performance of dairy cows. Cows that are lame would be hesitant to allow other cows to mount them when they are on heat, mainly because they are unstable as moving around would be painful. This would make heat detection very difficult. Conception from artificial insemination may be low because the body is fighting the infection caused by the lameness. Such cows require more services per conception or may not conceive at all, having to be replaced by expensive heifers. These factors can lead to increased calving intervals. The extra heifers that need to be raised use up valuable land space that could have been used to produce own feeds like forages. Lame cows are culled at a greater rate than healthy cows, increasing the overall herd culling rate. Therefore, more cows must be put in calf by using semen from dairy bulls instead of semen from beef bulls, reducing the total bull calf income considerably. If too much extra culling is enforced on the farm, a reduction in the genetic level of the herd can be the result.

Although studies determining the incidence of lameness occurring in South African dairy herds are limited, anecdotal observation indicates that approximately 20% of the average dairy herd could be affected by lameness at any time. In an earlier study conducted in England during the 1992/1993 calving season, it was found that 27% of the costs of production diseases were due to lameness problems. Due to the economic and reproductive impact that lameness has on dairy herds today, it is imperative that more attention and research needs to be focused on lameness in dairy herds.

Lameness and its associated claw lesions

Lameness is caused by foot lesions on the claws of cows. To prevent lameness from occurring in a dairy herd, it is necessary to understand the different types of lesions on the claws causing lameness. For this purpose, a dairy cow claw lesion identification information sheet was
developed by The International Lameness Committee to help dairy farmers identify the type of lesion occurring on the claw of cows. This claw lesion guide includes photographs and descriptions of the 14 most commonly found claw lesions in dairy cows, which all lead to lameness being observed in the cows.

Lesions on the claws of dairy cows are divided into infectious and non-infectious lesions. To manage lameness in a herd, managers should know which category of lesions is most prevalent in the herd. Records must be kept of which lesion of the two categories occurs in which claw zone of the cow. This information will enable veterinarians and managers to determine the correct treatment, as well as shedding some light on the potential factor(s) causing lameness. Each lesion within each category has its own potential cause associated with it. Preliminary data from an evaluation conducted among South African dairy herds suggests that infectious lesions, such as digital dermatitis, heel erosion, interdigital dermatitis and foot rot, occur more in confinement dairies, while pasture dairies show more non-infectious lesions. These include white line lesions, sole ulcers, sole haemorrhages, toe ulcers, corkscrew claws, horizontal hardship grooves, vertical and axial grooves, inter-digital growth, and thin soles.

Factors affecting lameness in dairy cattle

Lameness is a disease that is multi-factorial in origin as there are a range of factors affecting it which are inherent to dairy farms. The management of lameness in dairy cows is therefore very challenging. The basic net result of all the factors causing lameness is that claws become increasingly soft and therefore more prone to injuries and damage that may occur in the day-to-day life of the dairy cow. The causes and sequence of events during lameness is shown in Figure 57.1.

The flow chart shows that there are a number of factors that can, on their own, cause lameness, but some of these factors can also play a combined role in causing lameness. For example, non-infectious lesions can be triggered by trauma to the claw, increased standing times, and/or shearing forces on the white line of the claw (associated with cow flow), nutritional problems like sub-clinical acidosis, or by changes occurring at calving. These triggers will result in very similar lesions at the sole surface (e.g. sole, toe and heel ulcers, white line disease, and sole haemorrhaging). Therefore, every lesion may have a number of factors that can trigger its development usually associated with specific factors on the farm which can be managed to reduce the incidence of lesions. Different factors are responsible for the development of non-infectious and infectious lesions.
Factors affecting non-infectious lesions

1. Nutrition
   The ingestion of large amounts of ruminal fermentable carbohydrates (high levels of concentrates) often leads to the overproduction of volatile fatty acids in the rumen. This overproduction of fatty acids eventually exceeds the buffering capacity of the rumen. The increase in acidity in the rumen leads to the fermentation process in the rumen slowing down, which results in acidosis when high levels of carbohydrate consumption continue. Acidosis causes the death of certain gram negative bacteria in the rumen. Furthermore, in situations where the rumen comes to standstill because of acidosis, certain endotoxins are produced and released which triggers the release of histamine. Histamine causes vaso-
constriction and dilation of blood vessels, destruction of the laminar layers in the claw and eventual claw deterioration. Acidosis simultaneously leads to depressed fibre digestion, reduced feed intake, reduced butterfat in the milk, and increased metabolic diseases like laminitis.

Since effective fibre stimulates rumination, which stimulates the secretion of saliva, a small amount of effective fibre in the diet leads to less chewing, less saliva production, and therefore a decrease in ruminal pH. The problem is similarly aggravated by too short effective fibre pieces in the diet.

Other nutritional factors associated with faster digestibility in the rumen and therefore more acid being produced include the grain source in the concentrate mixture, i.e. wheat is digested quicker than maize grain, smaller particle size of grain, treatment of the grain (grinding, steam flaking, and cooking), and moisture content of the grain (especially if high moisture grains are ensiled). Poorly ensiled and mouldy feed should also be avoided due to histamine and mycotoxin release.

The amount of protein in the ration may also affect the incidence of lameness, but little information is available to identify the role protein might play in the development of lameness.

Deficiencies in certain trace minerals, like copper, zinc, and manganese, can lead to lameness because of their role in horn formation. Certain vitamins, like Vitamin A, beta-carotene, Vitamin E, and biotin, also play a role in cell replication and formation of hoofs. If rations are high in concentrates, biotin synthesis in the rumen is reduced, leading to softer claws.

According to some studies, the incidence of acidosis is highest during the first month after calving, while being almost non-existent during the last trimester of pregnancy. This is probably due to higher concentrate diets containing high levels of highly fermentable carbohydrates being fed during early-lactation, while less concentrates are fed during the dry period. Due to the tremendous hormonal and nutritional changes that occur before and during parturition, it is advisable that feeding management be changed during these times to limit the incidence of lameness during early lactation.

Whenever switching from one diet to another, like from the dry period to early lactation, the transition in diets should be gradual. Usually, during the dry period, cows are fed no or small amounts of concentrates per day. For this reason, concentrate feeding of cows should gradually be increased from about three weeks before the expected calving date for the rumen bacteria to adapt to the higher concentrate feeding level. At and around parturition, it is also essential that cows consume sufficient amounts of fibre while maintaining a normal dry matter intake. During the milk production phase, dairy cows should be fed concentrates at least twice a day. When large amounts of concentrates have to be fed to high-producing dairy cows, this should be done three to four times a day. More incidences of lameness and lesions were observed when concentrate feeding was less frequent during the day, less time was spent at the feed bunk and when feeding concentrates before feeding roughages. Roughages, like hay or silage, should be fed before grain is offered or, preferably, it should be part of a total mixed ration (TMR). Total mixed rations must be mixed in such a way to minimise the sorting of feeds as cows tend to eat the more palatable feeds, such as the concentrate mixture, first, while eating roughages last. This is especially a problem when feeding low quality roughages, such as wheat straw or mature lucerne hay, as part of the total daily diet. When large amounts of concentrates are fed to dairy cows, dietary buffers should be included in the diet to aid maintaining rumen pH at acceptable levels and to keep the incidence of acidosis at a minimum. Cows in first lactation should be kept in a separate group receiving a specific diet which takes into account their smaller live weight and specific nutrient requirements as in many cases they are still growing towards their mature live weight. When cows have limited access to the feed trough because of overstocking or insufficient bunk space, they will most likely overfeed when they do get access to food.

2. Environment and herd management
Dairy cows should get the opportunity to lie down for 12 - 14 hours per day to ruminate.
Housing systems that are overcrowded and poorly designed with regards to ventilation with stalls that are uncomfortable (being too small) will result in cows not lying down, thereby increasing standing time. As cows are standing for longer periods, the possibility of their feet being injured increases, making them more susceptible to lameness. Stall space should be sufficient to allow cows to lie down and to ruminate. Proper clean bedding should be supplied; it must be soft without being abrasive and without small stones that can penetrate the horn of the sole as cows lie down and get up. Studies have shown that bleeding of the soles decreases when rubber flooring, rather than concrete floors, is used. The floor surface used in housing systems and the milking parlour must not be conducive to excessive horn wear. This is especially the case in concrete floors.

When cows are standing for long periods in the holding areas before and after milking in addition to being milked, it can lead to the development of lesions. This is because the mechanical overload and tissue compression (associated with long standing times) inside the claw can interfere with the movement of fluids inside the claw or the supply of nutrients to the horn-producing tissues within the claw. This is often the case that when large groups of cows are being milked that the time standing before, during, and after milking could be more than three hours per day for each milking session.

When cows are rushed on their way to and from the milking parlour, they do not have time to look where to put their front feet. Cows have to put their front feet on an even and stone-free walking surface, because this enables them to put their hind feet in the same place, minimising the chances of punctures to the claws. When cows are rushed into the milking parlour and/or around stalls, they often have to twist and turn on unyielding surfaces, like concrete, putting extreme shearing forces on their claws or across the white line region. Putting rubber mats on concrete floors in turning areas can reduce the shearing forces on their claws. A slow and non-rushing cow flow is required in preventing claw injuries. Cows bullying each other in the housing system can also lead to sharp twists and turns when the bullied cow has to move away from the dominant cow. The condition of the walkways should be good with no loose stones that may cause hoof punctures.

Lesions can develop when the claws are not trimmed at all or when they are not trimmed regularly. However, trimming of claws must also be done properly by a trained hoof trimmer. Experts suggest that claw trimming should be done every six months in dairy herds that are prone to the development of lameness. Caution against over-trimming is also necessary as claws that have been over-trimmed can lead to unbalanced weight bearing, as well as thin soles, which in turn increases the cow’s risk in developing lesions.

To prevent laminitis in first lactation cows, it is essential that they are managed separately from older cows. The sudden introduction of first lactation cows to a group of older cows often upsets the social structure (or pecking order) of the group of cows. This may also result in overcrowding, while first lactation cows may be introduced for the first time to long-term exposure to concrete floors, especially if they have been kept outside on pasture as heifers. The higher concentrate feeding level during early lactation combined with these housing factors may result in a higher incidence of laminitis in first lactation cows. Usually because of overcrowding and being of a lower social order, first lactation cows may not find space at the feed bunk. They may therefore only eat 3 - 4 times a day instead of the normal 13 - 14 meals per day. This may predispose them to rumen acidosis. Any stress on the cows through management, like vaccination, transportation, and/or reduced or increased exercise imposes stress on their bodies. Stress depletes the body’s nutrient reserves and reduces the animal’s resistance to diseases, leading to lameness. This is why first lactation cows are more susceptible to lameness and laminitic challenges than multiparous cows.

Cows close to parturition experience an increase in the elasticity of the connective tissue that suspends the pedal bone in the claw capsule. The hypothesis around this is that this increase in elasticity is due to the hormone, relaxin, which is being released around parturition. This helps in the expulsion of the calf during the birth
process. However, the increased elasticity of the connective tissue allows for greater movement of the pedal bone in the claw capsule. This puts increased pressure on the corium between the sole of the claw and the pedal bone itself, increasing the risk of claw lesions in cows.

While too little exercise can decrease the blood flow to the claws, leading to swelling or oedema, too much exercise (especially on unyielding floors like concrete) can lead to contusions in the claws.

At high ambient temperatures (above the thermal neutral zone), cows will start panting and drooling saliva. The increased respiration rate may lead to alkalosis. The body's response to this is an increasing urinary output of bicarbonate. Drooling, because of heat stress, results in cows losing saliva which should have been used to buffer the rumen. Cows also stand for longer periods to alleviate heat stress. This increases time on their feet. Since there is less saliva and bicarbonate to buffer the rumen against a low pH, acidosis can develop, which in turn, can lead to a risk for laminitis.

3. Infectious diseases & metabolic disorders
Metabolic disorders, like milk fever and ketosis, and infectious diseases, like mastitis and metritis, which develop after calving lead to toxins being circulated in the blood. This causes thrombosis in the capillaries and narrowing of the blood vessels in the corium of the claw. This increases the development of haemorrhaging in the claws.

4. Genetics
The physical characteristics of feet and legs in dairy cattle are fairly heritable. Good feet and leg conformation has huge value in the long-term prevention of lameness occurrences. Therefore, the breeding programme followed by the dairy herd should include choosing bulls and cows with good feet and leg conformation to prevent lameness as far as possible. It is advisable to not breed herd replacements from chronically-lame cows or cows that have a history of mobility problems.

5. Age and stage of lactation
The susceptibility of dairy cows to develop laminitis seems to increase with age. Some studies showed that 10-year-old cows were four times more likely to develop laminitis than 3-year-old cows. Other studies have also shown that lameness is more common during the first 120 days post-partum. Therefore, non-infectious lesions can be prevented by implementing a sound nutritional plan, implementing a regular hoof trimming programme, ensuring cow comfort, and ensuring less trauma to the claws by ensuring that walkways and holding areas are comfortable; namely, level without loose gravel on the floors. Special attention needs to be given to the claws of cows that are in milk, and especially first lactation cows due to the hormonal changes occurring during this time.

Factors affecting infectious lesions
Whereas the previous non-infectious lesions are mostly lesions of the claw horn or sole of the claws, the infectious lesions mostly affects the skin close to the claws. The following are potential factors on the farm that may cause infectious lesions:

1. Wet and poor hygienic conditions
When cows have to stand for long periods in wet, dirty- or manure-filled holding areas (holding pens outside in the rain), the skin of the claws above the bulbs and in between the claws are in contact with this wet, unhygienic environment. This usually leads to the softening of the skin, allowing bacteria to penetrate into the superficial layers of the skin, causing digital dermatitis and interdigital dermatitis in the interdigital space between the claws. Rough and uneven walking surfaces may also traumatised the interdigital skin and leave it more prone to infection risk.

2. Presence of infected animals in the herd
These infectious lesions could be transmitted from cow to cow in the same herd. Therefore, it is very important to make sure that these lesions are contained by keeping cows separate so as to not infect the whole herd. New animals brought into the herd may also infect other cows.

3. Poor foot bath management
Implementing a proper intensive foot bath programme should contain and control infectious lesions. The anti-bacterial treatment, which is put into the foot bath,
should specifically help in controlling digital dermatitis and interdigital dermatitis. The design of the foot bath should promote cow flow. It must be long enough to increase the number of foot immersions as cows walk through the bath. The passage way with the foot bath should have a removable or drop-down barrier to enable reaching and helping cows that have fallen down while walking through the foot bath.

The control of infectious lesions is based on keeping feet (claws) clean and dry. An effective foot bath programme will help towards this goal. Treating infected animals and keeping them separate from the rest of the herd should prevent the spread of bacteria.

**Where does laminitis fit into the lameness picture?**

In the past, sole haemorrhages, sole, heel and toe ulcers, and white line disease have been grouped together and referred to as “laminitis”. However, laminitis is only one cause of lameness and as stated previously, these specific lesions can also be caused by trauma to the claw, increased standing times, and/or shearing forces on the white line of the claw or by changes occurring at calving. Although they are most likely connected to nutritional challenges, they could also be caused by metabolic and digestive disorders, stress associated with parturition, mastitis, metritis, hard or poorly bedded stalls, too little exercise, and excessive bodyweight. Non-infectious lesions are clinical signs seen on the surface of the sole, which reflects a disease process within the animal which can alter the structure of the claw. This is why these lesions can be caused by more than one trigger factors. Of all the lesions identified in dairy cow lameness, approximately 62% could be associated with laminitis in some form. Cows may display these lesions on their claws, while not displaying any signs of lameness, i.e. sub-clinical laminitis. This can develop into more serious lesions such as sole ulcers and sole abscesses. The lesions associated with laminitis all fall within the non-infectious category in the dairy claw identification poster and therefore all the factors affecting the development of non-infectious lesions will also affect the development of laminitis. The lesions most connected to laminitis include white line disease, sole and toe ulcers, sole haemorrhage, corkscrew claws, horizontal, vertical or axial grooves, interdigital growth, and thin soles.

**What happens in the cow during laminitis?**

To understand what happens when laminitis occurs in the claw, it is necessary to understand what the claw looks like. The anatomy of cow’s claw is shown in Figure 57.2.
Looking at the cow’s claw from the outside going inwards, the hard outer covering of the claw, known as the hoof wall or horn, is seen. This is a hard surface and the cells that form the horn are produced by the tissue directly underneath the hoof wall. This is known as the corium, which is a nutrient-rich tissue that contains all the blood vessels and nerves of the claw. The corium continuously produces new cells, which are pushed away from the corium. These cells eventually die, producing the hard outer growth known as the hoof. The cells have therefore been keratinised and the new growth appears from the coronary band. Very often rings can be seen on the claws of animals which indicate that the horn was produced at different rates. These rates differ due to nutritional factors, health status, and living conditions. Generally, the claw grows at around 0.6 cm per month.

Underneath the claw is a slightly softer region, called the sole, which is also formed out of the corium layer. Where the sole is bound to the claw wall is a flexible junction, called the white line. This allows the cow’s claw to be more flexible as she moves. There are two soft bulbs at the back of the claw, which acts as a shock absorber when the cow moves. Between the corium and the sole of the claw is the digital cushion, which is a pad of fatty tissue which protects the corium and aids in blood transport in the leg. Within the claw of the cow, the four bones (phalanx 1, 2, 3 and navicular bone) rest within the claw capsule. These bones play a key role as a support structure for the leg and the rest of the body. The pedal bone, also known as phalanx 3, is situated directly above the digital cushion. The pedal bone is attached to the corium of the claw by sensitive connective tissue known as the laminae. Therefore, the laminae hold the cow suspended within her claw. The pedal bone is the only bone that is completely inside the actual claw and provides the framework for the general shape of each claw.

Factors affecting laminitis

Laminitis is a multi-factorial metabolic disorder that occurs when the laminae within the corium layers in the claw wall becomes inflamed. As mentioned earlier, these layers produce the horny tissue of the claw wall and sole. Any disruption in the blood flow to these folds will damage the tissues, impairing their ability to produce high-quality horn. This disruption can be caused by any of the previously mentioned factors.

The primary, but not only, cause of laminitis in cows is rumen acidosis. Acidosis occurs when cows are fed too much carbohydrates and too little effective fibre (as described earlier). This is a systemic metabolic challenge that causes a decrease in ruminal and systemic pH. The reduction in pH activates the circulatory system into increased blood flow and pulse. Due to the increased blood pressure, the corium and laminae swell, as well as damaging the vessel walls within the corium. Fluid seepage occurs through the vessel walls, resulting in oedema, internal bleeding of the solar corium from thrombosis, and ultimately expansion of the corium layer, leading to severe pain. All of this puts pressure on the claw wall and cuts off the circulation in the blood vessels of the corium. The laminae become starved for blood, oxygen, and nutrients, leading to abnormal claw formation, reducing elasticity, and inhibiting blood supply to the claw. As these tissues die, the bond that holds the pedal bone to the claw wall becomes weak and the pedal bone begins to separate from the claw wall. Because the pedal bone now has less support and a weaker bond to the wall, it begins to sink and rotate within the claw capsule. This causes compression of the soft tissue between the sole and pedal bone. It now becomes very susceptible to mechanical damage like penetration by sharp stones or long standing times on unyielding surfaces. Claws that are afflicted with laminitis also begin to grow at abnormal rates due to the increased circulation in the blood vessels. This alters the shape of the claw, causing cows to have an uncomfortable gait.

Laminitis, therefore, occurs as a result of blood vessel dysfunction, as well as softening of the ligaments of the suspensory apparatus, leading to rotation of the pedal bone, and compression of the digital cushion. This leads to haemorrhaging in the soles, as well as the formation of lower quality horn in the claw. As soon as the pedal bone separates from the claw wall, it can cause the sole to separate from the wall at the white line; this is known as white line disease. When the white line pulls too far away from the sole, it can expose the claw to infections. The reduction in ruminal pH also causes the death of rumen bacteria, which releases endotoxins and histamines into the blood stream. This further increases vascular constriction and dilation. Therefore, laminitis
puts the claw of the cow in a weakened state and will pre-dispose it to the other risk factors as depicted in Figure 57.1 (e.g. compromised cow comfort, stress, trauma to the claws and improper trimming of claws).

The most common form of laminitis occurring in dairy herds is sub-clinical laminitis, which often goes undetected because cows may not show any obvious signs of lameness. During sub-clinical laminitis, serum seepage and bleeding in the corium are evident, as well as inadequate blood and oxygen supply to the claws. It is this damage that leads to the production of poor-quality horn, and the wall and sole of the claw becoming softer and prone to wear and physical damage. The signs of sub-clinical laminitis include sole haemorrhages and yellow discoloration, white line separation, double soles, and sole ulcers.

Sub-clinical laminitis is mostly a problem in high-producing, intensively managed dairy herds. This is mostly due to the feeding of too much concentrates in the diets of lactating cows, which leads to acidosis in the rumen and therefore predisposes cows to develop laminitis. As the production level of the cow increases, she becomes more sensitive to all the risk factors depicted in Figure 57.1, especially trauma to the claws and, therefore, development of laminitis.

Western Cape dairy feeding programmes

Feeding large amounts of concentrates to dairy cows is a standard practice for dairy farmers in the Cape Winelands and Swartland regions of the Western Cape Province. These regions have a winter rainfall pattern with cold, wet winters and hot, dry summers. This creates the problem that mainly cereal crops, such as oats, barley, and triticale, can be grown here which, in comparison to lucerne hay and maize silage, are low to medium quality roughages having lower protein and energy levels while forage production is also less. Furthermore, because of seasonal droughts, there is a lack of water for irrigation purposes in the summer months. This makes the production of higher quality summer growing crops, like maize and lucerne, as well as ryegrass or legume pastures, very difficult.

Harvesting cereal crops for hay is also problematic, as late winter rains may interrupt harvesting or may result in extended harvesting dates. Roughages harvested at a late maturity stage have a poor quality, having low protein and energy levels while containing high levels of fibre which reduces roughage digestibility. Such roughages require large amounts of concentrates to support high milk yield levels. Using cereal roughages increase the cost of total diets since a more expensive concentrate containing a higher crude protein level must be used. The higher feeding cost of total mixed ration (TMR) production systems increases the vulnerability of these systems. To combat higher feeding costs, TMR systems require higher milk yields per cow and increased cow numbers. Both these factors may increase the probability of cows developing laminitis.

In closing

Lameness is a disease that causes great economic losses in dairy herds today. Nutrition plays a major role in the development of acidosis while there is a distinct link between acidosis and the development of laminitis. Sub-clinical laminitis without cows showing signs of lameness complicates the management of the disease. Laminitis can be controlled through nutrition by feeding higher fibre diets, although this reduces milk yield. Increasing the concentrate intake leads to higher milk yields which may improve financial sustainability. Because dairy farmers maximise energy intake to increase milk production, it remains a challenge to manage acidosis and laminitis profitably. Management in the following areas is critical in preventing laminitis: the diet being fed and feeding management, comfortable cow environment, and routine trimming and care of claws.