SmartSAMM
Powered by DairyNZ

CALVING

TECHNOTE 1

Reduce exposure to environmental mastitis bacteria

Environmental mastitis refers to intramammary infections caused by organisms that survive in the cow's surroundings – including soil, manure, bedding, calving pads, water, or on body sites of the cow other than the mammary gland. Infection of the udder with these organisms is often opportunistic, taking advantage of circumstances that favour environmental contamination and changes in the mammary gland's susceptibility to infection. There are many bacteria in the environment and some have characteristics that enable them to multiply within the udder.

Most cases of environmental mastitis occur within a few weeks of calving, when the cows' natural defence mechanisms are low and their teats have been in contact with mud and manure during calving. However, exposure of teat ends to environmental bacteria can occur at any time: before and during calving, at milking time or in paddocks during the lactation or dry periods. During lactation, factors that predispose cows to infection with environmental bacteria include milking udders that are wet or dirty, or administering an intramammary infusion if the teat orifice is not sterile. During the early and late dry period, absence of the keratin plug in the teat canal may make cows highly susceptible to infection.

Streptococcus uberis is the most common cause of environmental mastitis in New Zealand (McDougall *et al* 2007). Other environmental organisms causing mastitis include coliforms (*Escherichia coli, Klebsiella* species, *Enterobacter aerogenes), Pseudomonas aeruginosa, Bacillus cereus, Arcanobacterium* (formerly *Actinomyces* and *Corynebacterium*) *pyogenes, Serratia* species, *Enterococcus* species, *Nocardia* species, *Candida* species (yeast) and *Prototheca* species (algae). Characteristics of these bacteria are described in the following tables.

Streptococus uberis usually responds to treatment with bacteriological cure rates of the order of 80% reported (McDougall *et al* 2007). Coliforms do much of their damage through toxins released after the bacteria die. Many of the other environmental pathogens have poor cure rates so culling is usually the best option. For example, *Pseudomonas*, *Serratia, Nocardia* and *Arcanobacterium* are virtually impossible to treat and cows that survive are often culled.

Mastitis is generally divided into two types – cow-associated and environmental. The bacteria causing cow-associated mastitis usually reside in udder tissue and on teat skin and are most commonly spread at milking. The bacteria causing environmental mastitis survive in the cow's environment and, although milking may enable their entry through the teat canal, the environment is the primary source of infection. These bacteria include *Strep. uberis* and the coliforms.

Technote 14.3 and 14.4 discuss *Strep. uberis* and different approaches to the use of antibiotic Dry Cow Treatments and Internal Teat Sealants.

Characteristics of common environmental mastitis pathogens

Characteristic	*Streptococcus uberis*	*Escherichia coli**	*Enterococcus* species
Reservoir of infection	These bacteria can be isolated from the environment, faeces and many sites on cows. Some cows (5-10% of the herd) pass large numbers in their faeces. The udders of chronically infected cows are a potential reservoir and their milk may contaminate milking equipment.	These bacteria are widespread in the environment. In rare cases, udders of chronically infected cows are a reservoir of infection and their milk may contaminate milking equipment.	These bacteria can be isolated from the intestinal tract, faeces, infected udders and the environment.
Spread	Contamination of teat surfaces occurs in the environment. Passage into the udder can occur at any time, including during milking.	Contamination of teat surfaces occurs in the environment. Passage into the udder can occur at any time, including during milking.	Contamination of teat surfaces occurs in the environment.
Risk of infection	Infection most frequently occurs in the first two weeks of the dry period, and during the calving period and early lactation, especially if there is teat end damage or udder oedema. In the early dry and calving periods, changes in udder secretions, lack of flushing at milking, and absence of the keratin plug in the teat canal make cows highly susceptible to infection. Infection with *Corynebacterium bovis* may make quarters more susceptible to *Strep. uberis* mastitis during the dry period (Woolford *et al* 2001).	Clinical cases are rare in NZ, with *E. coli* isolated from fewer than 5% of cases in pasture-based herds. Downer cows with milk fever paresis may be at higher risk. Risk of infection increases for cows managed in environments that increase faecal contamination of teats i.e. feeding or calving pads, indoor housing, zero-grazing or fed diets with a high starch content (Lacy-Hulbert *et al* 2002).	Infection occurs most frequently in the first two weeks of the dry period and during the calving period particularly when teat end hygiene is poor. Infection can occur throughout lactation especially when cows are maintained in dirty environments.
Clinical signs	Severity of clinical signs may vary from slightly abnormal milk to severe swelling of the udder and an elevated temperature. Severity can increase when treatment is delayed.	The bacteria can cause a sudden and severe toxaemia where cows may develop a high temperature, or later, a low temperature, and may become recumbent and die. Coliforms do not usually invade udder tissue – toxins cause the damage. Quarters usually return to part production in the same lactation (Gröhn *et al* 2004).	It is difficult to differentiate clinical signs from those of *Strep. uberis* infection. Less research has been done on these pathogens as they are less prevalent (McDougall *et al* 2007). Between 5-15% of suspected cases of *Strep. uberis* mastitis may turn out to be *Enterococcus* spp. when fully speciated (Salmon *et al* 1998). *Cont'd…*

Characteristic	*Streptococcus uberis*	*Escherichia coli**	*Enterococcus* species
Duration of infection	The majority of infections are short-lived but a proportion (up to 10%) can become chronic (McDougall *et al* 2004). The median duration of subclinical infections was found to be 16 days but different strain types could cause infections within the same cow over a lactation, so many infections appear to last longer than 1 month.	Bacteria are shed in the first 6-12 hours of clinical signs, and are reduced thereafter. Most infections are of short duration – more than 50% last less than 10 days. Some (1-2%) continue for more than 100 days. Cases may be culture negative because the cow has already eliminated the bacteria.	Most infections are short-lived but a small percentage may become chronic.
Cow Somatic Cell Counts (SCC)	Most infected cows have elevated SCC. Some can be extreme (>10,000,000 cells/mL) and may result in increases in the bulk milk SCC.	Cow SCC rapidly increases following infection and persists for about two weeks. In chronic cases SCC tend to be high (about 500,000 cells/mL) although they can fluctuate.	The SCC will be elevated in infected cows.
Milk quality	*Strep. uberis* infections, if undiagnosed, can contribute significantly to bulk milk contamination.	Clinical and chronic coliform cases do not contribute significantly to bulk milk contamination. Most isolations from bulk milk are likely to be from bacteria on teat skin rather than from udder infections.	Bacteria are not usually isolated from bulk milk.
Management during outbreaks	Manage calving cows to minimise exposure to contamination. If possible, use sand rather than organic materials for calving pads. Begin milking and disinfecting teats of cows tight with milk prior to calving, especially those leaking milk. Improve pre-milking hygiene by cleaning and drying teats before attaching teat cups. Use whole herd antibiotic Dry Cow Treatment (DCT), and/or Internal Teat Sealant (ITS) at the next dry period to prevent new infections. Adjust springer mob feeding levels to decrease 'bagging up' and milk leakage pre-calving.	Manage cows to minimise exposure to contamination. The source of udder contamination should be sought and corrected e.g. feed-pads and stand-off pads, laneways and access to waterways, calving paddocks. Recent changes in feeding management should be reviewed. Improve pre-milking hygiene. Overseas, vaccination with J-5 vaccine in the dry and early lactation periods reduces the severity of many coliform infections but this vaccine is not registered for use in NZ.	Control measures are similar to *Strep. uberis*, and include good hygiene of teats and udders before calving, milking clean dry teats, good post milking teat disinfection and use of antibiotic DCT at dry off.

* *Escherichia coli* infection is often called 'coliform mastitis', which can also be caused by the less common but related bacteria *Klebsiella* spp and *Enterobacter aerogenes*.

Characteristics of less common environmental mastitis pathogens (1)

Characteristic	***Pseudomonas aeruginosa***	***Arcanobacterium pyogenes***	***Bacillus cereus***	***Nocardia* species**
Reservoir of infection	These bacteria are common in the environment – in places such as water sources and ponds. It can colonise water hoses and hot water systems and is resistant to some sanitisers.	These bacteria are common in the environment.	These bacteria are common in the environment and form heat- and chemical-resistant spores	These bacteria are common in soil. They can also survive in chlorhexidine-based teat disinfectants.
Spread	Infection may be introduced into the udder with intramammary treatment if teat ends are not dried or prepared aseptically. Spread is from contaminated water – either in the environment or water used to wash teats.	Occasional individual cases may be associated with teat or udder injury. These bacteria are one of a mix of bacteria causing 'summer mastitis' described in Europe and possibly spread by flies.	Infection may be introduced into the udder with intramammary treatment if teat ends are not dried or prepared aseptically. Mastitis has been associated with feeding heated brewer's grain containing spores.	Infection may be introduced into the udder with intramammary treatments if teat ends are not dried or prepared aseptically. Spread can be by contaminated water used to wash teats or in contaminated teat disinfectant. Spread from cow to cow at milking is possible.
Risk of infection	Cows are susceptible during milking and whenever udder infusion takes place.	Dry cows are more at risk of infection.	Risk of infection increases during udder infusion.	Infection occurs sporadically.
Clinical signs	Cases range from a mild to a severe acute form with septicaemia and death. Affected cows do not respond to treatment. If infection occurs after administering DCT and/or ITS at dry off, cows may become very sick.	Infection commonly causes a severe mastitis. Typically the quarter is hard, the teat is often very swollen, and secretions may consist of thick pus. Function may be permanently lost in affected quarters and teat obstruction following inflammation is common.	Mild to peracute forms. Haemorrhage and gangrene can occur. If infection occurs after administering DCT, clinical signs may not be seen until next calving.	Although cows are sometimes sick, infection is rarely fatal. Affected quarters are usually swollen or hard with lumps. Lumps may rupture and discharge to the surface. Infected cows do not respond to treatment. WARNING: Take care when handling infected cows as *Nocardia* species can cause respiratory infections in people. *Cont'd…*

Characteristic	*Pseudomonas aeruginosa*	*Arcanobacterium pyogenes*	*Bacillus cereus*	*Nocardia* species
Duration of infection	Cases are usually chronic and unresponsive to therapy. Generally low numbers of bacteria are shed, with intermittent shedding in chronic cases.	Cases are usually chronic and unresponsive to therapy. Large numbers of bacteria are passed in the secretion.	The bacteria may be a contaminant of milk samples. Clinical cases should be identified from repeat cultures.	Bacteria in fresh milk may not survive refrigeration or freezing of the sample, so culture samples should be plated promptly.
Cow Somatic Cell Counts (SCC)	Cell counts are generally >500,000 cells/mL in chronic infections.	Secretions are usually not suitable for measuring SCC.	Cell counts are usually very high.	Cell counts are usually very high.
Milk quality	Milk supply can be contaminated by water sources. These bacteria may cause milk spoilage.	Cases are usually clinical and their milk is excluded from the bulk milk.	Cases are usually clinical and their milk is excluded from the bulk milk.	Bacteria can be recovered from the bulk milk. This represents a potential milk quality hazard because the organism may not be killed by pasteurisation.
Management during outbreaks	It is important to assess the intramammary technique being used and management of cows immediately after treatment. Affected cows do not respond to available antibiotics. Culture of water sources is often unrewarding, while changing water hose rubberware is often useful. Identification of infected quarters may require repeated culture. Positive cows should be culled.	In countries where 'summer mastitis' is common, careful management during the dry period (e.g. locating cows in low risk paddocks) is essential. It can occur in areas with hot, humid summers. Whole herd antibiotic DCT and fly control are recommended if there is a herd problem. Assess intramammary infusion technique, as poor technique can be a risk factor. Teat damage is also a risk factor.	It is important to assess the intramammary technique being used and management of cows immediately after treatment.	It is important to assess the intramammary techique being used and management of cows immediately after treatment. Affected cows do not respond to available antibiotics. Culture of water sources is often unrewarding. Assess the sterility of teat disinfectant. Infected cows should be culled. WARNING: Human infections are possible.

Characteristics of less common environmental mastitis pathogens (2)

Characteristic	*Klebsiella species*	*Serratia species*	*Yeasts and Prototheca*
Reservoir of infection	Sawdust or organic bedding material used on loafing pads, calving pads or indoor housing can harbour high numbers of these bacteria. Cows grazed with access to muddy, wet, marshy areas and pools of standing water are also at risk. Research has shown that many healthy adult cows shed *Klebsiella* in their faeces so any bedding that is contaminated with manure can contain *Klebsiella* (Zadoks *et al* 2011).	Source of infection can be from soil and plants (environmental) but they can also be harboured in chlorhexidine-based teat disinfectants. Muddy, wet, marshy areas and pools of standing water pose another risk to grazed cows. Spread during milking has been reported (Isaksson and Holmberg 1984). Damaged teat ends increase risk of infection.	Both of these organisms are thought to cause infection as a result of unhygienic infusion of mastitis treatments. However *Prototheca* can be found in soil, plants, streams and stagnant ponds, faeces, sheds and yards. *Prototheca* can also be spread cow to cow (Gonzalez 1996).
Spread	Primary sources include faecal material and the environment	Infections can be spread via contaminated teat disinfectant or sourced from the environment. They can also be spread cow to cow via the milking machine.	Yeast does not spread contagiously. Contaminated intramammary tubes or materials can be a source. *Prototheca* can be sourced from the environment, and infection may be introduced into the udder with intramammary treatment if teat ends are not dried or prepared aseptically.
Risk of infection	Cows are most susceptible in early lactation. Most of the risk is for housed cattle (Zadoks and Munoz 2007).	Infection rates are highest over the dry period and usually develop into a chronic infection.	Cows are susceptible at any time during lactation or at dry off.
Clinical signs	The bacteria can cause a sudden and severe toxaemia where cows may develop a high temperature and may become recumbent and die. Similar to *E. coli*, these bacteria do not usually invade udder tissue – toxins cause the damage. Quarters usually return to part production in the same lactation (Gröhn *et al* 2004).	Many *Serratia* infections are subclinical and characterised by high somatic cell counts in infected quarters, but clinical mastitis may occur in more than half of the infected animals. Clinical signs are most often mild. Cows are rarely systemically ill. Infections tend to be chronic and clinical signs may be intermittent.	Both organisms can cause clinical mastitis. *Prototheca* and yeast infections can become chronic and subclinical. *Cont'd…*

Characteristic	*Klebsiella species*	*Serratia species*	*Yeasts and Prototheca*
Duration of infection	Cows that survive clinical *Klebsiella* mastitis often develop chronic mastitis, more so than for *E. coli*. Milk may appear normal, but somatic cell counts are high, and repeated clinical cases may occur. Cows with chronic *Klebsiella* mastitis are often culled for high cell count, recurrent mastitis or production loss.	Shedding in milk has been documented from 55 days to 3 years in duration (QMPS 2005).	*Prototheca* can be shed in milk and passed from cow to cow. Neither organism responds to antibiotics, and yeast infections may even be exacerbated by antibiotic use. Infections by these bacteria can become chronic.
Cow Somatic Cell Counts (SCC)	Cell counts are generally high, consistent with chronic mastitis.	Many *Serratia* infections are subclinical and characterised by elevated somatic cell counts in the infected quarter. Clinical mastitis may occur in more than half of the infected animals.	Both yeast and *Prototheca* can cause high cow SCC due to their poor response to antibiotic therapy.
Milk quality	Cases are often clinical and their milk is excluded from the bulk milk.	*Serratia* may be intermittently shed by infected cows and therefore may not be detected in bulk milk consistently.	Yeasts do not usually affect milk quality but may be detected in bulk milk cultures. *Prototheca*, being an alga, will not be detected by a standard plate count but may be detected by Bactoscan.
Management during outbreaks	Remove cows from sources of mud, manure, dirt, soiled organic bedding. Reduce stocking density if required. Improve pre-milking teat hygiene. Consider segregating and/or culling infected cows because response to therapy is poor (Zadoks and Munoz, 2007).	Remove risks due to poor hygiene, use of loafing or calving pads with organic bedding, muddy, wet, swampy areas, over stocking, poorly or infrequently scraped feed pads. Improve pre-milking teat hygiene. Check for contamination of the teat spray and change to an iodine-based product if using chlorhexidine.	Culling or drying off infected quarters of infected cows is the only way to manage these infections. Once infected, cows should be identified and milked last to reduce risk of spread. Minimise access to muddy and wet areas (*Prototheca*) and review intramammary treatment technique.

Cont'd from page 1…

Streptococcus dysgalactiae has characteristics of environmental and cow-associated causes of mastitis, so is not easy to categorise. The bacteria can be isolated from the environment and from sites on the animal such as the mouth, udder and vagina. Teat end damage is a risk factor for mastitis caused by *Strep. dysgalactiae* (Ericsson-Unnerstad *et al* 2009).

Technote 5 describes characteristics of mastitis caused by common cow-associated bacteria, and by bacteria that share characteristics of both types.

1.1

Calve on clean, dry pasture or a clean, dry calving pad.

The udder is very susceptible to new infection at calving, and many infections detected in early lactation are established around calving (Hogan and Smith 1998; Parker *et al* 2007). The ideal place for cows to calve is a clean, sheltered, dry area. The ideal situation is a paddock with a good cover of grass, not irrigated or contaminated with farm dairy or feed pad effluent, on an elevated site that is not wet, boggy or poorly-drained.

Unfortunately, this is a challenge on most NZ dairy farms and most farm managers choose to use calving paddocks where the cows can be supervised easily or calving pads, especially in areas of high rainfall during the calving period.

Confidence – High

Local observations of the importance of hygiene in calving areas are consistent with overseas research and experience.

Research priority – Moderate

Further information on what constitutes a successful calving pad surface, including measurement of pathogen counts, would be useful.

Grazing management

Grazing is the main process by which pasture becomes contaminated with environmental bacteria such as *Strep. uberis*. For this reason, it is generally considered important to avoid 'back-grazing' (where cows have access to recently contaminated areas in addition to their new area). In times of inclement weather, it may be appropriate to increase the grazing area, to reduce stocking density.

If possible heifers should be calved separately from the adult herd. Heifers are more likely to be bullied and be forced to calve in the less suitable areas of the calving paddock or calving pad.

When allocating pasture, sufficient area should be offered to the springer cows so as to minimize pugging and subsequent udder contamination. If electric fences are shifted across a paddock at regular intervals, clean areas can be provided for new batches of calving cows. It is advisable to shift those cows which have recently calved (<12 hours), or those which are about to calve, through into a clean break.

Planning is needed to create access lanes and allow for continuous access to drinking water, for example by using mobile troughs. When cows are calving on grass it is also important to ensure that preventative measures for metabolic diseases have been taken.

Alternative arrangements

In extremely wet areas, farmers may use loafing, calving or stand-off pads or indoor housing in which to calve cows. These facilities may increase the risk of environmental mastitis (Washburn *et al* 1992; Lacy-Hulbert *et al* 2002). Bedding selection and effective drainage are the most important factors in reducing udder contamination, and the risk of mastitis.

Usually some bedding material is provided to make the cows more comfortable. Organic bedding materials (straw, post peelings, shavings or sawdust) support higher bacterial populations than non-organic materials (washed sand, or ground limestone). The particle size is also important. The surface area for bacterial growth and the chance of bacterial attachment and colonisation is increased in finely chopped or ground organic material. For this reason, long straw is generally better than finely chopped straw, and shavings are better than sawdust (Hogan and Smith 1998).

Download the "Minimising muck, maximizing money" resource from the DairyNZ website for tips on design of stand-off and feeding pads.

Sawdust and other wood products tend to harbour coliform bacteria and straw may contain large numbers of environmental streptococci, such as *Strep. uberis* (Bramley 1982, Smith and Hogan 1997). Sawdust may have high pathogen (*Klebsiella* species) counts even when fresh (Hogan *et al* 1989). Kiln-dried sawdust is less of a problem and pathogen counts are much lower when contaminated sawdust is removed and replaced with fresh sawdust daily rather than weekly (Bramley 1992). Sawdust should not be used as bedding if it cannot be kept dry (Blowey and Edmonson 1995).

All bedding materials (organic and inorganic) will support high pathogen counts after becoming contaminated with manure (Blowey and Edmonson 1995). The area must be kept well drained and contaminated material removed and replaced on a regular basis. Pathogen counts will be high before the bedding looks soiled, and chemical disinfection (agricultural or hydrated lime, formalin, etc) of contaminated bedding is not effective (Hogan and Smith 1998).

Rubber mats are an alternative that are easier to keep clean but can harbour bacterial films. Concrete or concrete slats are easier to keep clean but are not particularly suitable for calving. Cows maintained on concrete for 12 hours or more per day for more than three days should be given at least one full day on an alternative surface where they are free to lie down and rest (NAWAC, 2010).

A copy of the Animal Welfare code can be downloaded from: www.biosecurity.govt.nz.

In summary, the recommendations that no more than two pats of manure are present per square metre and no water is visible in foot prints are crude estimates designed to focus attention at least on gross contamination, given the lack of other more sophisticated monitoring techniques. Checking for fresh liquid manure, rather than dried pats, gives an indication of recent faecal contamination. Practical and economic factors influence the surfaces available for calving cows and must be individually assessed for each farm.

Options for surface types, in preferred order (highest to lowest) are:

- clean, grassed paddock with no surface water;
- well-drained inorganic material, such as sand;
- well-drained organic material; or
- poorly drained calving paddock (a last resort).

1.2

Monitor the number of cases of mastitis occurring, especially in recently calved heifers.

This is an indicator of pre-calving management. Mastitis in recently calved heifers (first calvers) may result from infection that has occurred during their development since puberty, in the few weeks immediately before calving, or in the hours associated with calving (Compton *et al* 2007a).

Clinical mastitis was observed at calving in 8% of first-calf heifers in a study of 11 herds in NZ (Pankey *et al* 1996). Environmental streptococci were isolated from 68% of these clinical cases. More recent studies have observed that the incidence of clinical mastitis at calving varied from 13% to 23% of heifers and 13% of cows (Compton *et al* 2007a; McDougall *et al* 2007).

Heifers may be particularly susceptible to infection during the calving period, because they tend to spend longer calving, especially on the ground, and may leak milk prior to calving (Waage *et al* 2001). Also they often suffer from some degree of udder oedema that may reduce the ability of the teat and udder tissues to resist bacterial challenge (Slettbakk *et al* 1995). Research indicates a greater tendency of animals with udder oedema to develop *Strep. uberis* infections (Compton *et al* 2007b).

Various studies (Slettbakk *et al* 1995, Waage *et al* 1998, Compton *et al* 2007a, b, Parker *et al* 2008, McDougall *et al* 2009) report other key risk factors for *Strep. uberis* infections, including:

- breed (Friesians are more at risk than other breeds),
- difficult calving and/or retained placenta,
- milk leakage prepartum,
- short distances from the teat end to the ground,
- dirty udder,
- high milk flow rate,
- large herd size and higher levels of herd production,
- high stocking density,
- peripartum nutritional management.

Warning indicators (otherwise known as SmartSAMM “triggers for action”) have been identified using the median performance for NZ herds. The indicators are based on the incidence of clinical cases observed during the calving period (two weeks before and two weeks after calving). Current triggers are:

- For all animals, **10 clinical cases** per 100 calvings
- For first calving heifers, **15 clinical cases** per 100 calvings
- For older aged cows, **7-8 clinical cases** per 100 calvings.

If the clinical case rates at calving are above trigger, a reassessment of the calving environment and management should be made. It is often useful to have an independent adviser help with this to obtain the benefit of a ‘fresh pair of eyes’. It is also worth noting that sometimes a high rate of mastitis occurs at calving, even though the environment appears clean and dry. These may be infections that occurred at an earlier time (for example at drying-off) and then became clinical at calving.

Confidence – Moderate

This recommendation assumes that most heifers have a low prevalence of infection when they enter springer mobs.

Research priority – Low

Analysis of the timing of clinical cases of mastitis, as part of the herd improvement statistics, would provide better benchmark information on new infection rates.

SmartSAMM Mastitis Focus report can calculate these indicators if clinical case treatment records and cow SCC records are available in herd improvement systems.

Technote 2 describes strategies for reducing mastitis in first calving heifers.

1.3

Bring cows into the dairy as soon as possible to be checked for mastitis, and milked.

In 1996 Pankey *et al* noted that many 'over-conditioned' heifers leaked milk prior to calving and had a higher prevalence of mastitis. They believed preferential treatment of heifers during the calving period might reduce the incidence of new infections and suggested several factors to aid mastitis control in heifers including:

- minimising exposure to muddy conditions;
- milking them out as soon as possible after calving; and
- applying an effective teat disinfectant after every milking.

Since then, research studies have examined some of these suggestions. Milking newly calved heifers at an average of 9 hours after calving was found to reduce the risk of clinical mastitis by 45%, compared to milking them on average 19 hours after calving (Compton and McDougall 2008). This was achieved by ensuring that the farm team brought newly calved animals to the farm dairy twice daily, for milking rather than once daily.

Applying teat spray three times weekly precalving was found to reduce the number of *Strep. uberis* bacteria isolated, by swabbing heifer teats, and tended to reduce the incidence of clinical mastitis associated with *Strep. uberis*, but did not reduce the overall clinical mastitis incidence (Lopez-Benavides *et al* 2009).

Although these studies were conducted on heifers, early removal of calves and teat disinfection prior to calving, are management strategies that are likely to reduce mastitis in older animals.

For older cows, machine milking prior to calving may also be appropriate, on the basis that "If she's dripping milk, she should be milking." There is no research data to support this approach, however.

In the past, preventive management of milk fever (a sudden reduction in the calcium level in the blood) sometimes involved leaving milk in the udder of fresh cows. This practice is now discredited because it predisposes to mastitis (O'Shea 1987). Rather than treating milk fever by incomplete milking, it should be controlled by managing the diet before and at calving to manipulate calcium availability in this period.

Technote 4.1 and 4.2 describe how to check udders and milk from quarters of freshly calved cows.

1.4

Take special care with high risk cows.

First lactation heifers and cows induced to calve prematurely have a higher risk of mastitis due to environmental bacteria than other cows in the herd. Management strategies for first calving heifers are described in detail in Technote 2.

NB. Using inductions as a tool to tighten calving is becoming less common in NZ, but its use is still allowed, within certain limits.

Long-acting corticosteroids (such as dexamethasone) are used to induce parturition in dairy cattle. They can impair secretion of proteins that are critical to normal cellular and humoral immune responses (Nonnecke *et al* 1997), an effect that is strongly linked with changes in the proportion of different types of the white blood cells. The ability of cows to respond to stressors may be reduced by the use of long-acting corticosteroids to induce premature calving.

Browning *et al* (1990) described a collapse syndrome associated with the use of dexamethasone to induce calving. It appeared to result from Gram-negative endotoxaemia associated with subclinical infections as three of the seven cows in this study had peracute *Escherichia coli* mastitis confirmed at post-mortem examination.

Immune suppression resulting from the use of long-acting corticosteroids to induce parturition is still profound at the time of parturition. Steps must be taken to minimise exposure of induced cows to mastitis-causing pathogens at this time.

To minimise the risk of environmental mastitis in induced cows, these procedures should be followed:

- Maintain cows in clean, well-drained paddocks (the best calving area available on the farm) from the time they receive their first injection to induce parturition until after they have calved.
- Milk cows once the udder is tight with milk. Induced cows often bag-up tightly before calving and may drip milk. Machine milking is recommended once the udder gets tight with milk, even though the cow may not yet have calved. “If she’s dripping milk, she should be milking.”
- Watch udders carefully for signs of mastitis. Some cases can be rapid and severe with few initial changes or abnormalities in the milk (e.g. water milk, clots or flecks).
- Monitor induced cows very closely for signs of systemic illness. Cows may become acutely ill with an *Escherichia coli* mastitis endotoxaemia, even though visible changes in the udder may be limited and the secretion from the affected quarter is difficult to differentiate from colostrum.

Confidence – High

Field experience in Australia and NZ shows that induced cows are more susceptible to *Escherichia coli* infection.

Research priority – Low

Technote 2 describes strategies for reducing mastitis in first calving heifers.

Consult your veterinarian to find out about more about managing the risk of mastitis for induced cows.

1.5

Take care with pre-milking preparation of udders.

Keeping teats and udders clean helps reduce the number of bacteria around the teat end, which is an important step in preventing mastitis (Schreiner and Ruegg, 2003). Keeping udders clean is easier if the tail switch is kept well-trimmed, if the hair on the udder is kept short through clipping or flaming, and if the legs are kept free of dirt and manure.

Technote 5 describes good milking technique, including pre-milking teat preparation, and the importance of consistent routines.

The first few hours or days after calving is an ideal time to trim up tails and clip or flame the udder. Areas on the farm that cause legs and udders to become dirty should also be checked to see if they can be cleaned up.

Technote 5.3 describes udder flaming.

Acknowledgements

DairyNZ and NMAC (NZ National Mastitis Advisory Committee) acknowledge the huge contribution of Dairy Australia's Countdown Downunder as the original source material from which SmartSAMM Technotes are derived, being updated and adapted for NZ dairy farming in 2011.

These SmartSAMM adapted resources are made available to NZ dairy farmers and advisors through a Memorandum of Understanding between Dairy Australia and DairyNZ.

The SmartSAMM programme is funded by DairyNZ, and supported by the MPI Sustainable Farming Fund.

Key papers

Blowey R, Edmondson P. The environment and mastitis. In: *Mastitis control in dairy herds*, Chapter 8, Farming Press Books, Ipswich, United Kingdom, 1995:103-118.

Bramley AJ. Bovine Medicine. Blackwell Science Publications, Oxford, United Kingdom, 1992.

Bramley AJ. Sources of *Streptococcus uberis* in the dairy herd. I. Isolation from bovine faeces and from straw bedding of cattle. *J Dairy Res,* 1982; 49:369-373.

Browning JW, Slee KJ, Malmo J, Brightling P. A collapse syndrome associated with gram-negative infection in cows treated with dexamethasone to induce parturition. *Aust Vet J,* 1990; 67:28-29.

Compton CW, Heuer C, Parker K, McDougall S. Epidemiology of mastitis in pasture-grazed peripartum dairy heifers and its effects on productivity. *J Dairy Sci,* 2007a; 90:4157-70.

Compton CW, Heuer C, Parker K, McDougall S. Risk factors for peripartum mastitis in pasture-grazed dairy heifers. *J Dairy Sci*, 2007b; 90:4171-80.

Compton CWR, McDougall S. Effect of early milking of calved heifers and selenium supplementation on incidence of clinical mastitis in dairy heifers. *Hungarian Veterinary Journal,* 2008; 130: Supplement 2, 48.

Ericsson-Unnerstad H, Lindberg A, Persson-Waller K, Ekman T, Artursson K, Nilsson-Ost M, Bengtsson B. Microbial aetiology of acute clinical mastitis and agent-specific risk factors. *Vet Microbiol,* 2009; 137:90-97.

Gonzalez R. Prototheca, yeast, and Bacillus as a cause of mastitis. In: *Proceedings of the 35th National Mastitis Council Annual Meeting,* Nashville, Tennessee, 1996:82-92.

Gröhn YT, Wilson DJ, González RN, Hertl JA, Schulte H, Bennett G, Schukken YH. Effect of pathogen-specific clinical mastitis on milk yield in dairy cows. *J Dairy Sci,* 2004; 87:3358-74.

Hogan JS, Smith KL, Hoblet KH *et al*. Bacterial counts in bedding materials used on nine commercial dairies. *J Dairy Sci,* 1989; 72:250-258.

Hogan JS, Smith KL. Occurrence of clinical and subclinical environmental streptococcal mastitis. In: *Proceedings of the symposium on udder health management for environmental streptococci*, Ontario Veterinary College, Canada, 1997; 36-41.

Hogan JS, Smith KL. Risk factors associated with environmental mastitis. In*: Proceedings of* *the 37th National Mastitis Council Annual Meeting*, St Louis, Missouri, 1998:93–97.

Isaksson A, Holmberg O.[Serratia-mastitis in cows as a herd problem]. *Nord Vet Med,* 1984; 36:354-60

Lacy-Hulbert SJ, Kolver ES, Williamson JH, Napper AR. Incidence of mastitis among cows of different genotypes in differing nutritional environments. *Proceedings of the New Zealand Society of Animal Production,* 2002; 62:24-29.

Lopez-Benavides MG, Williamson JH, Lacy-Hulbert SJ, Cursons RT. Heifer teats sprayed in the dry period with an iodine teat sanitizer have reduced *Streptococcus uberis* teat-end contamination and less *Streptococcus uberis* intra-mammary infections at calving. *Veterinary Microbiology,* 2009; 134:186-91.

McDougall S. Arthur DG, Bryan MA, Vermunt JJ, Weir AM. Clinical and bacteriological response to treatment of clinical mastitis with one of three intramammary antibiotics. *NZ Vet. J,* 2007; 55:161-170.

McDougall S, Parkinson TJ, Leyland M, Anniss FM, Fenwick SG Duration of infection and strain variation in *Streptococcus uberis* isolated from cows' milk. *J Dairy Sci,* 2004; 87:2062-72.

NAWAC. Animal Welfare (Dairy Cattle) Code of Welfare. National Animal Welfare Advisory Committee, MAF Biosecurity New Zealand, Wellington, New Zealand. 2010:1-47.

Nonnecke BJ, Burton JL, Kehrli ME. Associations between function and composition of blood mononuclear leukocyte populations from Holstein bulls treated with dexamethasone. *J Dairy Sci* 1997;80:2403-2410.

O'Shea J. Machine milking factors affecting mastitis. In: *Machine milking and mastitis, Bulletin of the International Dairy Federation, No. 215*, Brussels, Belgium, 1987:25-27.

Pankey JW, Pankey PB, Barker RM, Williamson JH, Woolford MW. The prevalence of mastitis in primiparous heifers in eleven Waikato dairy herds. *NZ Vet J,* 1996; 44:41-44.

Parker KI, Compton C, Anniss FM, Weir A, Heuer C, McDougall S. Subclinical and clinical mastitis in heifers following the use of a teat sealant precalving. *J Dairy Sci,* 2007; 90:207-18.

Parker KI, Compton CW, Anniss FM, Heuer C, McDougall S. Quarter-level analysis of subclinical and clinical mastitis in primiparous heifers following the use of a teat sealant or an injectable antibiotic, or both, precalving. *J Dairy Sci,* 2008; 91:169-81.

QMPS *Serratia* species and Mastitis - QMPS fact sheet. Quality Milk Production Services, Cornell University College of Veterinary Medicine. Accessed September 2011 at: *http://ahdc.vet.cornell.edu/sects/QMPS/Articles.*

Salmon SA, Watts JL, Aarestrup FM, Pankey JW, Yancey RJ Jr. Minimum inhibitory concentrations for selected antimicrobial agents against organisms isolated from the mammary glands of dairy heifers in New Zealand and Denmark. *J Dairy Sci,* 1998; 81:570-8.

Schreiner DA, Ruegg PL. Relationship between udder and leg hygiene scores and subclinical mastitis. *J Dairy Sci*, 2003; 86:3460-5.

Slettbakk T, Jorstad A, Farver TB, Holmes JC. Impact of milking characteristics and morphology of udder and teats on clinical mastitis in first- and second-lactation Norwegian cattle. *Prev Vet Med,* 1995; 24:235-244.

Smith KL, Hogan JS. Risk factors for environmental streptococcal intramammary infections. In: *Proceedings of the symposium on udder health management for environmental streptococci*, Ontario Veterinary College, Canada, 1997; 42-50.

Waage S, Odegaard SA, Lund A, Brattgjerd S, Rothe T. Case-control study of risk factors for clinical mastitis in postpartum dairy heifers. *J. Dairy Sci,* 2001; 84:392-399.

Waage S, Sviland S, Odegaard SA. Identification of risk factors for clinical mastitis in dairy heifers. *J Dairy Sci,* 1998; 81:1275-84.

Washburn SP, White SL, Green JT Jr, Benson GA Reproduction, mastitis, and body condition of seasonally calved Holstein and Jersey cows in confinement or pasture systems. *J Dairy Sci*. 2002; 85:105-11.

Woolford MW, Williamson JH, Day TM, Lacy-Hulbert SJ, Henderson HV. Effect of localised antibiotic infusions applied to the teat-canal and teat sinus at drying-off on mastitis in the dry-period and at calving. *J. Dairy Res,* 2001; 68:551-558.

Zadoks RN, Griffiths HM, Munoz MA, Ahlstrom C, Bennett GJ, Thomas E, Schukken YH. Sources of *Klebsiella* and *Raoultella* species on dairy farms: be careful where you walk. *J. Dairy Sci*, 2011; 94:1045-51.

Zadoks RN Munoz MA. The emergence of *Klebsiella* as a major mastitis organism. In: *Proceedings of the 46th NMC Annual Meeting,* San Antonio, Texas, 2007; 100-111.