

100,000 cell count and 10% mastitis is an achievable target

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Bulk Milk Somatic Cell Count (BMSCC) is a reflection of a **static** and a **dynamic** situation.

- The **static** situation consists of the chronically infected cow group that makes up the majority of the high cell count group.
- The **dynamic** situation consists of the newly infected cows that enter the high cell count group, and cured cows that leave.

There is an element of dynamic in the static situation as individual high cell count cows will vary on a daily basis but their average cell count is high. Achieving low cell counts and low clinical mastitis rates involves a combination of managing both the static and dynamic situations.

Managing the static situation

Involves reducing the chronically infected group by a combination of treatment and culling. Lactational therapy is sometimes used to treat high cell count cows after herd tests, but the success of this strategy is generally disappointing. Antibiotic dry cow therapy (DCT) is the most successful method of treatment and its success is dependent on the type of bacteria (*Staph. aureus* is more difficult to treat than *Strep.* species) and the duration of the infection. Cows that don't respond to treatment should be culled, and this is often a weak point in many mastitis programmes. Look at 2 successive years of individual cow cell counts and cull cows that did not respond to DCT the previous year.

Managing the dynamic situation

Is more challenging, but is the key to success in reducing BMSCC as managing the static situation on its own is insufficient. This is because newly infected cows replace the chronically infected cows that are cured or culled. You need to understand the risks on your farm and concentrate on mitigating these risks correctly. The devil is in the detail.

For example - if you ask farmers whether they are applying the control methods they universally put up their hands. A good example of this is illustrated in a summary of 200 mastitis problems that I had involvement with in which all were teat spraying - 35% of farmers were achieving good coverage and 35% were using the correct dilution rate, but only 12% of the farmers had both the coverage and dilution rate correct. This is despite all the communication and education around the importance of teat disinfection as being probably the most important single step in a mastitis control programme.

The keys to a successful programme are:

- (a) understanding the risks on your farm, and
- (b) attention to detail.

There are basically 4 key mastitis mechanisms:

- (i) Factors that put bacteria on the teat end,
- (ii) Factors that cause teat damage,
- (iii) Factors that cause “impacts”, and
- (iv) Factors that result in poor milk-out.

1. Factors that put bacteria on the teat

The heavier the contamination the greater the risk of overcoming the cow’s natural defences.

- (a) Some bacteria could be considered “natural flora” on teat skin (*CNS*, *C. bovis* and *Strep. dysgalactiae*) –these bacteria will proliferate in the absence of teat disinfection.
- (b) Some bacteria come from the environment (*E. coli* and *Strep. uberis*) - these bacteria proliferate in faeces and the risk of infection increases with the level of contamination e.g. in housing or calving paddocks. Control is ideally achieved by reducing the faecal contamination of the environment e.g. sand bedding, reduced stock concentration on pasture. But often this is impractical, or not achievable, so control is achieved through teat disinfection prior to milking and protection of the dry cow through use of dry cow therapy and/or teat sealants.
- (c) Some bacteria come from infected cows (*Staph. aureus* and *Strep. agalactiae*) and are passed from cow to cow in the milking parlour. Control is achieved through good teat disinfection and/or cluster disinfection between cows.

2. Factors that cause teat end damage

Allows bacteria easier entry into the teat. May be environmental (e.g. harsh weather conditions), infectious (e.g. pseudo cowpox) or milking machine related (e.g. hyperkeratosis).

The most important of these are the milking machine related causes:

- a. Machines basically do damage through the effects of vacuum. Teats are damaged through the combined effects of swelling and stretching and the higher compressive load of the liner that occurs with higher vacuum. The optimal vacuum is dependent on many factors such as: the height of the milk-line, type of liner used and milking practices.
- b. Pulsation is essential for good teat health. It acts to alleviate the effects of vacuum by massaging the teat to maintain normal circulation and structure. Regular milking machine tests are essential to ensure adequate pulsation.
- c. Liner type can influence teat health and many consider liner design to be as much about art as science as evidenced by the large number of diverse shapes and sizes.

- d. Over-milking is a major cause of teat end damage as teats swell and stretch more and vacuum at the teat is higher during the over-milking phase.

3. Factors that cause “impacts”

The term “impacts” refers to the impact of bacteria containing milk droplets in a reverse direction back towards the teat with sufficient force to result in entry through the teat canal while the cups are on the cow. The two important causes of this are cup slip and poor cluster removal procedures, both of which result in air blasting into the cluster from the unattached cup back up the attached cups impacting on the teat end.

Liner type, poor cluster alignment and poor vacuum reserve are common causes of cup slip. Good cluster removal involves adequate clamping of the long milk tubes prior to cluster removal and waiting for the vacuum to drop in the cluster before removing the cluster. Poor cluster removal is caused by insufficient clamping of the long milk tube and pulling the cluster before adequate vacuum drop. This results in a sudden rushing of air into the attached cups causing impacts similar to a cup slip situation.

I also will mention here a non- milking machine cause of impacts (or its equivalent), when poor teat disinfection prior to intra-mammary treatment of clinical mastitis, or at dry cow therapy, results in the pushing of bacteria on the teat skin into the quarter.

4. Factors that result in poor milk-out

Poor milk-out is associated with an increased risk for mastitis. This effect may be direct, in that poor milk-out results in more bacteria being left in the quarter which results in more bacteria multiplying in the presence of an ideal medium of warm milk, or it may be indirect, in that poor milk-out is often caused by teat damage and cup slip. There are many other causes of poor milk-out such as: poor cluster alignment, low and high vacuum and poor liner tension in old liners.

In summary, sustainable low cell counts and low clinical mastitis rates in your herd are dependent on understanding the key risks on your farm and putting emphasis on mitigating these risks. Attention to detail is important making sure that best practices are being applied.