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Understanding and using somatic cell counts to improve milk quality

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The production of high quality milk is a requirement to sustain a profitable dairy industry and somatic cell count (SCC) values are routinely used to identify subclinical mastitis and define quality standards. The objective of this paper is to review the use of SCC as a diagnostic tool for subclinical mastitis in order to improve milk quality on dairy farms. Mastitis is detected based on inflammation subsequent to intramammary infection (IMI) by pathogenic organisms. Individual cow SCC values are used to detect the inflammation that results from IMI and are necessary to define the prevalence and incidence of subclinical IMI. A threshold of <200,000 cells/mL is considered to be of the most practical value used to define a mammary quarter as healthy. The development of IMI is the most significant factor that influences milk SCC and assessment of monthly values to determine newly and chronically increased SCC can be highly diagnostic for resolving problems with increased bulk tank SCC. Methods to reduce the development of new IMI are well known and adoption of best management practices for milking and herd management have consistently been shown to result in reductions in bulk tank SCC. Implementation of mastitis control programmes can be improved by focusing on three practical recommendations: 1) Farmers should work with their advisors to develop an annual udder health plan that includes clear goals for milk quality. 2) The annual udder health plan should emphasise prevention of new IMI. 3) Farmers must identify and manage chronically infected cows. Proactive management of IMI can be extremely effective in helping farmers produce milk that meets industry standards for milk quality.

Keywords: bovine; mastitis; milk quality; somatic cell count

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Introduction

In most developed countries, mastitis is the most common infectious disease of dairy cows and results in considerable economic loss for both dairy farmers and milk processors (Fetrow *et al.* 2000; Halasa *et al.* 2007; Geary *et al.* 2012). The economic impact of mastitis is greater than most other infectious diseases because the point of infection is the mammary gland; thus intramammary infection (IMI) results in reduced productive capacity of the gland and decreased processing value of milk (Barbano, Ma and Santos 2006). Inflammation subsequent to IMI can result in subclinical and/or clinical symptoms and control programmes must include methods to detect and monitor outcomes of both presentations of the disease (Ruegg 2011).

Minimising mastitis and consistently producing high quality milk is a requirement for dairy farmers who wish to be competitive in the global marketplace. Enumeration of the somatic cell count (SCC) of milk has long been used as a tool for measuring milk quality (Dohoo and Leslie 1991). Bulk tank SCC (BTSCC) values are routinely used to define the national and international regulatory standards that govern hygienic milk production. The national standards for BTSCC vary from <400,000 cells/mL (EU, Australia, New Zealand and Canada) to <1,000,000 cells/mL (Brazil) (USDA 2013). However, minimum international export requirements for milk quality are becoming more important than national regulations. The US situation is a good example of how market forces can result in rapid improvements in BTSCC, even in the absence of rigorous national regulations. While several US states have more stringent standards, the legal maximum BTSCC for most US states remains at 750,000 cells/mL (FDA 2011). However, exports of US dairy products are increasing. In 2012, 13.2% of US milk production was exported, including approximately 45–47% of whey proteins and skim milk powder/non-fat dry milk (US Dairy Export Council 2013). To ensure that US products remain eligible for export to EU nations, the USDA introduced a programme that allows processors to obtain an export certificate that verifies farm-level compliance with the 400,000 cell/mL limit adopted in the EU (USDA 2011). Most large milk processors have enrolled in this programme and enforce the more stringent requirements. The sustained industry emphasis on improved BTSCC has resulted in continued reductions in BTSCC, even though national regulations have not changed (Figure 1) (USDA 2013). These improvements demonstrate that dairy farmers can produce high quality milk in response to market demands and indicate that processors have considerable leverage in motivating farmers to adopt management practices that contribute to the production of high quality milk.

Bulk tank SCC is used to measure the quality of the milk produced by a herd, but measurement of SCC at the cow-level is necessary to estimate prevalence and incidence of subclinical mastitis. In most instances, monthly composite milk SCC values from Dairy Herd Improvement programmes (DHI) are used to monitor the dynamics of IMI (as estimated by SCC) at the herd and cow-level (Figure 2) (Laevens *et al.* 1997; Ruegg 2003). Monthly reports that are generated using SCC values can be used to monitor herd and group SCC and to identify subclinically affected cows for interventions such as culture, segregation, treatment, or removal from the herd (Cook *et al.* 2002; Rhoda and Pantoja 2012). Enrolment in a DHI testing programme is essential for managing udder health and has been associated with

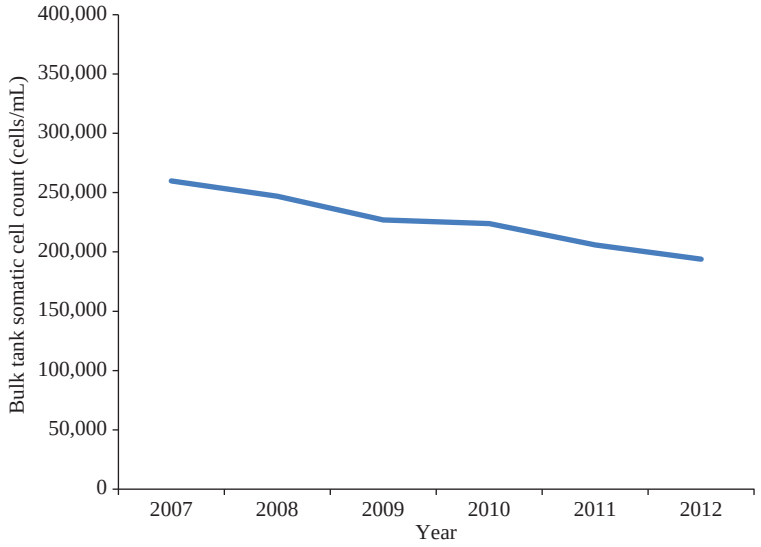

Figure 1. Trends in bulk tank somatic cell count in US states monitored by the Federal Milk Mark Orders (representing 47% of milk produced in the US) (adapted from USDA 2013).

reduced prevalence of subclinical mastitis (Wilson, Gonzalez and Sears 1997). The objective of this paper is to review the use of SCC as a diagnostic tool for improving milk quality on dairy farms.

Detecting Intramammary Infection and Inflammation

The technical definition of mastitis is "inflammation of the mammary gland" but on a practical basis, almost all mastitis occurring in dairy cows is caused by bacteria, although some cases are caused by yeasts, fungi or algae (Hogan *et al.* 1999). Mastitis is initiated after an infective dose of a pathogenic organism passes through the streak canal, followed by bacterial growth during an incubation period and then progression to either subclinical or clinical states or resolution of the infection as a result of the cows immune response (Ovideo-Boyso *et al.* 2007). Mastitis is virtually never detected at the precise moment of infection but is recognised based on observation of the resulting inflammation. Thus, the presentation of mastitis (as a clinical or subclinical case) depends on pathogen and cow characteristics that influence the extent of the immune response, whereas recognition by farm personnel depends on the intensity and accuracy of the detection methods (Ruegg and Erskine, in press).

Consistency in collection of samples used for analysis of SCC is important because the milk fraction from which samples are collected can influence SCC. In general, in both healthy and infected quarters, concentrations of SCC are greatest in foremilk and post-milking strippings and least in milk collected during peak flow (Urech, Puhan and Schällibaum 1999; Bansal *et al.* 2005; Nielsen *et al.* 2005; Sarikaya and Bruckmaier 2006). The time interval between milking periods influences the magnitude of this diurnal variation (Reneau 1986). Somatic cell counts have been reported to be greater in milk samples collected in the evening

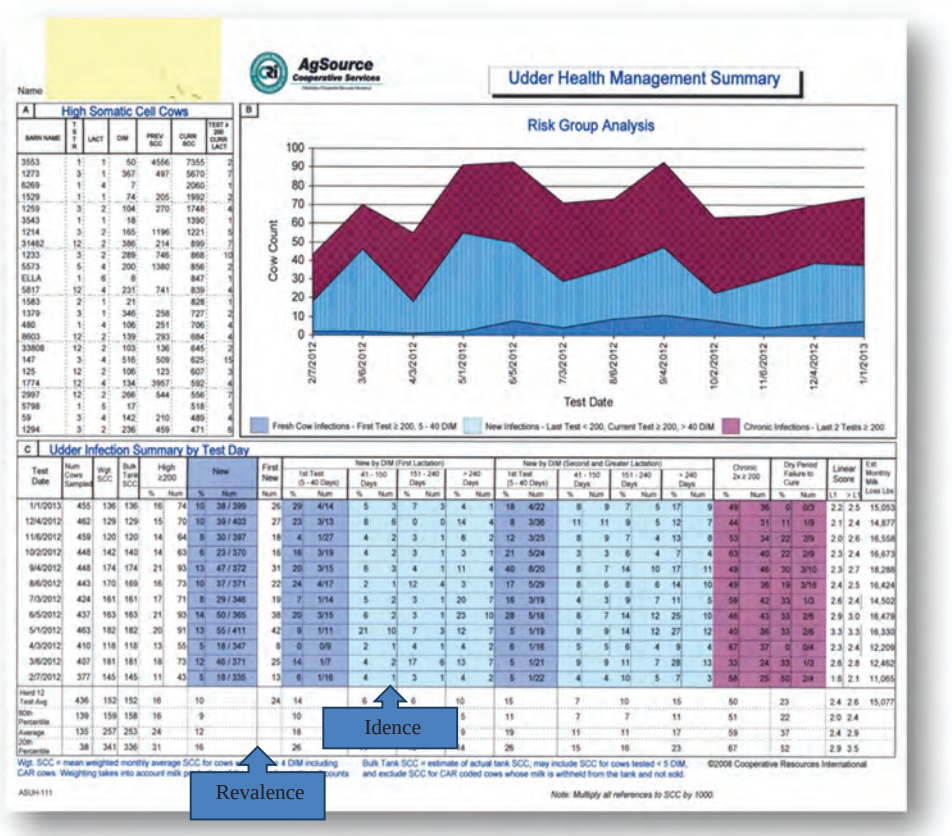

Figure 2. Example of summarised somatic cell count report for one herd.

as compared to samples collected in the morning (Reneau 1986). Although failure to consider diurnal variation of SCC will probably not affect diagnosis of major pathogens, it may lead to misclassification errors.

By definition, milk obtained from mammary gland quarters of cows experiencing *subclinical mastitis* appears visually normal (even when millions of somatic cells are present) but contain an excessive number of somatic cells, (with or without the detectable presence of pathogenic organisms) (Dohoo and Leslie 1991). Somatic cells in milk consist of neutrophils, macrophages, lymphocytes, and a smaller percentage of epithelial cells (Sordillo, Shafer-Weaver and DeRosa 1997). The SCC of healthy quarters is usually well below 100,000 cells/mL and is composed primarily of macrophages (Hamann 2005). After establishment of IMI, inflammatory mediators attract large numbers of phagocytes to migrate from the bloodstream to the udder, dramatically increasing the SCC in milk and shifting the distribution of cells from macrophages to primarily neutrophils.

Detection of subclinical mastitis is based on measurement of SCC in milk collected from individual mammary gland quarters or composite milk samples that are a

mixture of milk from all functional glands of an individual cow. Composite milk samples collected through milk meters are routinely used in DHI programmes for monitoring SCC of individual cows. It is important to recognise that each mammary gland quarter becomes infected independently, so most infections occur in single quarters. When composite milk samples are used, some subclinical infections will not be detected because the SCC in the sample will be reduced by dilution with milk from healthy quarters that contain few somatic cells (Figure 3) (Ruegg and Reinemann 2002; Ruegg 2011).

The most accurate relationship between IMI and SCC exists at quarter level (Schukken *et al.* 2003). Researchers have reported that uninfected quarters have a mean SCC of approximately 70,000 cells/mL and reduced milk yield is observed once cell counts exceed 100,000 cells/mL (Schukken *et al.* 2003). While the SCC of healthy quarters is consistently quite low and usually remains below 100,000 cells/mL (Hamann 2005), a threshold of <200,000 cells/mL is usually considered to be the most practical value to use to define a mammary quarter as healthy (Dohoo and Leslie 1991; Schepers *et al.* 1997; Djabari *et al*. 2002; Pantoja, Hulland and Ruegg 2009). The probability of isolating a major pathogen increases as SCC exceeds 200,000 cells/mL. When SCC are used for detection of IMI, a threshold of approximately 200,000 to 250,000 cells/mL has been considered optimal to reduce diagnostic error under field conditions (Dohoo and Leslie 1991; Schepers *et al.* 1997; Djabri *et al.* 2002).

The selection of the appropriate threshold for defining subclinical mastitis is dependent on the goal of the control programme. When the goal is to detect microbiologically positive quarters, the use of lower thresholds will identify more animals with IMI (increased sensitivity and fewer false negatives) whereas the use of higher thresholds (increased specificity) will result in fewer false positives (Pantoja *et al.* 2009). Regardless of the threshold selected, review of the SCC history of a cow is much more informative as compared to observation of a single monthly value.

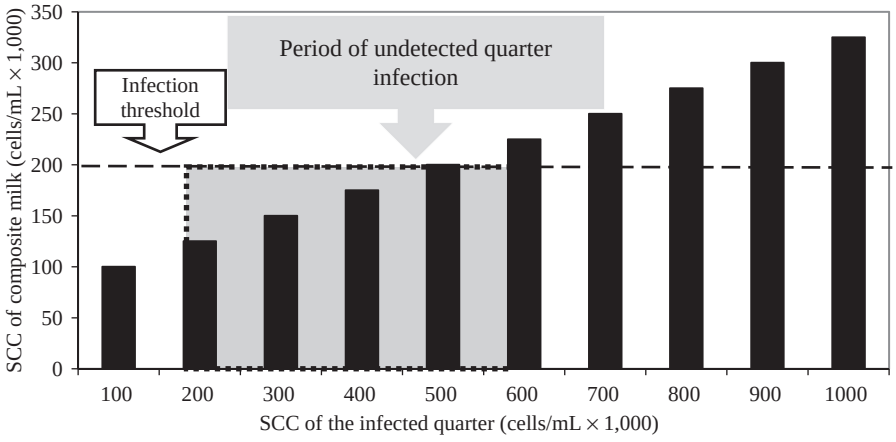

Figure 3. Example of estimated composite milk somatic cell count (from all four quarters) when a single mammary gland quarter is infected and the baseline SCC in uninfected quarters is 100,000 cells/mL.

The occurrence of SCC >200,000 cells/mL is an extremely specific indicator of IMI, but the failure to recover bacteria from a high SCC gland does not indicate that the gland is healthy. An increased SCC in a microbiologically negative milk sample is a common occurrence that can occur because the immune response has reduced the number of bacteria to below normal laboratory detection limits (usually 100 cfu/mL). The increased SCC is part of an immune response that has the purpose of elimination of pathogens. This response is often effective and at least 10–25% of quarters that have SCC >200,000 cells/mL will be apparently bacteriologically negative (Dohoo and Leslie 1991; Schepers *et al.* 1997; Pantoja *et al.* 2009). When using monthly SCC data to identify potentially infected cows, the positive predictive value (PPV) for recovery of bacteria (IMI) is relatively poor for dairy herds that have moderate prevalence of subclinical infections. In recent research, the PPV (defined as the probability of recovering mastitis pathogens from milk samples when the first test SCC was >200,000 cells/mL) was only 41% (Pantoja *et al.* 2009). In contrast, the negative predictive value (NPV; defined as the probability of NOT recovering mastitis pathogens from milk samples when the first test SCC was <200,000 cells/mL) was 85%. This indicates that farmers should be educated to expect that many milk samples used to determine aetiology of IMI are likely to be negative and results of single milk samples should not be over-interpreted relative to infection status nor treatment efficacy. After effective treatment or spontaneous cure, the SCC will gradually return to <200,000 cells/mL, but the time required to return to normal is often dependent on the aetiology (de Haas *et al.* 2004; Ruegg 2013).

The SCC values of individual cows are not usually normally distributed so arithmetic mean values of groups or herds may not be representative of the prevalence of IMI. To account for the skewed distribution, most North American DHI centres report SCC using a linear score transformation (usually termed Somatic Cell Score; SCS), which is calculated using a simple formula ($LS = \log_2 (SCC/100) + 3$). The use of SCS removes the influence of very high values and is associated with a linear reduction in milk yield. When linear scores are used, each 1 unit increase in SCS has been demonstrated to result in 91 kg and 182 kg reductions in milk yield for primiparous and multiparous cows, respectively (Ali and Shook 1980).

Clinical Mastitis

Clinical mastitis is defined as inflammation that results in visible abnormalities of milk, regardless of SCC level. Hygienic regulations usually require that abnormal milk is discarded (FDA 2011), thus if the cases are accurately detected, the occurrence of clinical mastitis should not contribute to increased BTSCC. However, most symptoms of clinical mastitis are quite mild and cannot be detected unless foremilk is observed before attaching the milking cluster. In a study that enrolled almost 800 cases of clinical mastitis occurring on 51 Wisconsin dairy farms, only 15% of clinical cases presented with systemic symptoms, while 50% and 35% of cases presented with solely abnormal milk or abnormal milk and swelling of the affected quarter, respectfully (Oliveira, Hulland and Ruegg, 2013).

The relationship between the subclinical and clinical phases of IMI is dependent on aetiology (Sheldrake *et al.* 1983; Smith, Todhunter and Schoenberger 1985; Schepers *et al.* 1997). Many

Gram-positive IMI have long subclinical phases before the occurrence of a clinical case while most Gram-negative IMI have relatively short subclinical phases as compared to Gram-positive pathogens (Smith *et al.* 1985, de Haas, Barkema and Veerkamp 2002). Pathogen specific SCC patterns before and after the occurrence of clinical cases have been evaluated in the past (de Haas *et al.* 2002, 2004). A period of subclinical infection with increased SCC was observed before cases of clinical mastitis associated with typical Gram-positive mastitis pathogens (*Staphylococcus aureus*, *Streptococcus dysgalactiae*, *Streptococcus uberis*, and other *Streptococci*), whereas SCC values were generally <200,000 cells/mL before the occurrence of clinical *E. coli* mastitis (de Haas *et al.* 2002). Several distinct patterns of SCC have been developed based on analysis of consecutive test-day records (de Haas *et al.* 2004). These patterns differentiated between a short or longer period of increased SCC with or without recovery and were used with the aim of identifying profiles of pathogens causing IMI and clinical mastitis in dairy herds. After resolution of clinical signs, the SCC of clinical cases associated with isolation of *E. coli* or no bacterial growth resulted in a relatively rapid return to normal levels of SCC, whereas clinical cases caused by *Staphylococcus aureus* or coagulase-negative *Staphylococci* were associated with slow recovery and increased SCC during lactation. In contrast, *Streptococci* were not consistently associated with any of the defined patterns of peaks in SCC (de Haas *et al.* 2004). While occurrence of clinical mastitis cannot be measured using SCC values, a review of the SCC history preceding a clinical case can give useful clues regarding the potential aetiology and need for antimicrobial therapy (Ruegg 2013). For example, a clinical case that is preceded by a long history of increased SCC is more likely to be a case caused by bacteria that may require antibiotic therapy as compared to a case that has no prior history of inflammation.

Factors that Influence Somatic Cell Counts

Immediately after calving, SCC are usually >1,000,000 cells/mL and decrease to approximately 100,000/mL by 7–10 days after parturition. Barkema *et al.* (1999) reported that the geometric mean SCC of cows with culture-negative quarters decreased from 588,000 cells/mL on the first day post-calving to 166,000 cells/mL on the third day after calving. After initiation of lactation, in the absence of IMI, few leucocytes migrate into milk and SCC are normally <100,000 cells/mL. As lactation progresses, both the SCC and the proportion of neutrophils in milk gradually increase. In the absence of apparent IMI, the SCC at the end of lactation has been reported to increase in very low producing cows, but the increase was not significant for cows producing more than 4 kg/day on the last DHI test (Bodoh, Batista and Schultz 1976). Thus, on most modern dairy farms, increased SCC at the end of lactation is indicative of IMI. Characteristic increases in SCC at different stages of lactation will reflect exposures to pathogens found on individual farms. The timing and duration of IMI caused by contagious and environmental pathogens are usually distinctive. For example, in herds with many cows infected with contagious mastitis pathogens, the prevalence of IMI increases with parity and stage of lactation because of the increased likelihood of exposure to these pathogens from milk of subclincally infected herdmates. In contrast, the incidence of IMI (as evidenced by increased SCC)

caused by environmental pathogens is usually greatest in early lactation because post-partum immune suppression reduces the ability of animal to respond to exposures to these opportunistic pathogens.

Age, number of quarters with IMI (in composite samples), season and diurnal variation are other significant factors that have been associated with SCC (Dohoo and Meek 1982; Wiggans and Shook 1987; Schepers *et al.* 1997). As parity advances, cows have greater probability of developing IMI and increased SCC with parity may be attributed to increased prevalence of IMI and greater cellular response to certain pathogens. As expected, when composite milk samples are used, there is an observed increase in SCC as the number of infected quarters increased. Seasonal variation in SCC for dairy herds in North America and Europe are consistently reported (Ruegg and Tabone 2000; Berry *et al.* 2006; Summer *et al.* 2007; Cicconi-Hogan *et al.* 2013). In general, due to increased exposure to pathogens (as a result of favourable climatic conditions for microbial growth) more IMI occur in warmer, wetter seasons (such as summer) and reduced IMI occur in cooler periods (such as winter or spring) (Ruegg and Tabone 2000; Berry *et al.* 2006; Summer *et al.* 2007; Cicconi-Hogan *et al.* 2013). There is also some evidence that heat stress can reduce the phagocytic ability of neutrophils, resulting in reduced capability of the cow to respond to IMI (do Amaral *et al.* 2011). Thus, during warm and wet seasons, cows experience increased exposure to mastitis pathogens while simultaneously having decreased ability to spontaneously clear pathogens, resulting in increased probability of persistent IMI and increased SCC.

The development of an IMI is the most important factor that will affect milk SCC for an individual quarter, a cow and at the herd-level (Dohoo and Meek 1982). A difference in SCC response can be observed between quarters infected by major (e.g. *Staphylococcus aureus, streptococci* and coliforms) and minor pathogens (e.g. *Corynebacterium* spp. and coagulase-negative *Staphylococci*). Several studies that used various epidemiological approaches (retrospective or prospective) and various units of analysis (quarter or cow level) have shown that minor pathogens normally induce less intense SCC responses than major pathogens. Cows infected with minor pathogens have been reported to have composite SCC levels that ranged from 190,000 to 519,000 cells/mL (average of 227,000 cells/mL), as opposed to cows infected with major pathogens, which had SCC greater than 600,000 cells/mL; (Sheldrake *et al.* 1983; Barkema *et al.* 1999; and Schukken *et al.* 2003). At the cow level, experimental studies have demonstrated that different pathogens may cause specific SCC patterns of response after infection. Quarters with experimental infection induced using *E. coli* had short peaks of SCC (at about 2 days after challenge) and a period of three to four weeks until normalisation (Erskine 1992; Pyorala *et al.* 1994). However, within 24 hours after inoculation with *Staphylococcus aureus*, SCC increased and remained increased for at least 48 days (Shoshani *et al.* 2000).

Use of Somatic Cell Counts as a Diagnostic Tool for Mastitis

It is impossible to manage any disease without knowledge of the infection status of individual animals. The first step in monitoring subclinical mastitis is to ensure that SCC values are obtained from all cows on a regular basis. Generally all cows with composite SCC values >200,000 cells/mL (SCS of approximately 4.0) are considered to have subclinical mastitis caused by an IMI.

At the herd-level, evaluation of the pattern of newly and chronically increased SCC can be highly diagnostic for troubleshooting high BTSCC. For example, when many cows have increased SCC in early lactation, exposure to environmental mastitis pathogens during the dry and transition periods should be evaluated. In these herds, transition and dry cow management should be evaluated with special emphasis on the condition of pastures, lanes and animal housing (during periods of confinement). In contrast, when contagious mastitis is a problem, the proportion of cows with increased SCC usually increases as lactation progresses and as cows age (because of the longer a cow milks, the greater the opportunities for exposure to infected milk). In these herds, emphasis should be placed on detecting inadequate teat dipping or the presence of fomites that can transfer infected milk among cows (such as towels used to clean or dry teats on more than one cow). When a large proportion of cows have chronically increased SCC (more than 2 consecutive monthly tests with increased SCC) it indicates that cows are infected with host adapted pathogens that are usually transmitted in a contagious manner. In these instances it is useful to review a list of individual cows sorted by SCC to identify cows that may require specific interventions. The use of a rapid cow-side quarter-level SCC test, can help farmers make important management decisions such as whether or not to segregate, treat, culture, withhold high SCC quarters or cull the cow.

Most DHI centres produce a summarised SCC report (Figure 2) that should be routinely reviewed to manage udder health. Monthly individual cow SCC values should be summarised to provide estimates of *prevalence* (usually defined as the proportion of cows with SCC >200,000 cells/mL and to monitor *incidence* (usually defined as the proportion of cows with SCC >200,000 cells/mL for the first time (Figure 2). Assessments of subclinical mastitis should begin with the following questions (from Ruegg 2011): 1) What is the prevalence of subclinical mastitis? 2) What is the incidence of subclinical mastitis? 3) What are the most common bacteria recovered from cows with SCC values >200,000 cells/mL? 4) What proportion of subclinical cases are chronic (persist more than two months)? 5) What is the prevalence of subclinical mastitis by days in milk and parity? 6) What proportion of cows have subclinical mastitis at the first test and the last test? Common key performance indicators for evaluation of subclinical mastitis are: 85% cows with somatic cell counts <200,000 (prevalence) and <5% of cows developing new subclinical mastitis infections per month (incidence) (Table 1 – Ruegg 2011). When herds do not meet these goals, a plan to improve milk quality should be initiated.

Practical Methods to Reduce Somatic Cell counts and Improve Milk Quality

Mastitis is a bacterial disease that results from insufficient management of people, cows, technology, and/or the environment. Mastitis is a bacterial disease that occurs in individual animals but mastitis control programmes must be implemented at the herd level. Fortunately, methods to control mastitis are well known. Numerous studies have indicated that effective implementation of best management practices results in reduced prevalence of IMI and reduced BTSCC (Rodrigues, Caraviello and Ruegg 2005; Olde Riekerink *et al.* 2010; Dufour *et al.* 2011). However, control of mastitis requires a multidisciplinary approach that is focused on prevention of new infections and appropriate interventions

Table 1. Calculation of suggested key performance indicators for subclinical mastitis (from Ruegg 2012).

Indicator	Calculation	Suggested goal
Prevalence	Number of cows with SCC > linear score 4[a] divided by the number of cows with somatic cell counts	<15% of the herd
Incidence	Number of cows with SCC > linear score 4[a] for the first time in the time period of interest[b] divided by the number of cows with SCC below the threshold in the previous time period	<5% if incidence is determined based on the first SCC above threshold in the lactation; up to 8% if calculated based on month to month changes in SCC[b]
Prevalence at 1st DHIA test	Number of cows with SCC > linear score 4[a] at the 1st DHIA test divided by the number of cows with first test DHIA somatic cell counts	<5% of 1st lactation <10% of lactation 2+
Prevalence at last DHIA test before dry off	Number of cows with SCC ≥ linear score 4[a] at the last DHIA test before dry off of the lactation divided by the number of cows with last test DHIA somatic cell counts	<30% of cows with last test days before dry off

[a]For the purpose of herd monitoring, a linear somatic cell score of 4 is used interchangeably with somatic cell count of >200,000 cells/mL.
[b]The appropriate time period will vary depending on the intended use of this index. Many DHIA centres and computer management programmes will calculate this index based on changes between two months. Others may calculate it based on the SCC values available in the current lactation.

for infected cattle. In general, exposure to contagious pathogens is based on reducing the possibility that teats of healthy cows come into contact with milk that came from udder of cows with subclinical IMI. More than 40 years ago, Neave *et al.* (1969) developed the 5-point plan that is the basis for control of contagious pathogens. This plan remains effective and in regions that have adopted these recommendations, successful control of contagious pathogens has occurred (Makovec and Ruegg 2003). However, on many modern dairy farms, the BTSCC is low but environmental pathogens continue to cause excessive cases of clinical mastitis (Oliveira *et al.* 2013). To address the increased incidence of mastitis caused by environmental pathogens, the NMC expanded the 5-point plan to 10-points that focus on comprehensive mastitis control (NMC 2013). Based on these plans, implementation of successful mastitis control can be summarized in three practical recommendations:

1. Each farm should routinely work with their advisors to develop an annual udder health plan that includes clear goals for milk quality. Barriers to improvement in milk quality are often related to motivation and implementation rather than lack of technical knowledge or skills (Rodrigues and Ruegg 2004). Without having clear goals, deadlines to accomplish tasks and routine access to advisors, few farmers actually set aside sufficient time to develop and implement an udder health plan. The development, implementation and evaluation of an annual udder health plan can be increased by involvement of veterinarians and other industry professionals (Rodrigues *et al*. 2005; Ruegg 2009). The plan should include outcome measurement and key performance indicators for managing mastitis (Ruegg 2011; Anon. 2013a). Successful mastitis control is dependent on effective detection, accurate diagnosis, evaluation of appropriate treatment options and implementation of preventive practices that address herd specific risk factors associated with exposure to mastitis pathogens. Evaluation of cow factors, environmental factors and milking machine factors that can contribute to exposure to mastitis pathogens should be a considered as the udder health plan is developed. An effective surveillance system for mastitis includes clear case definitions and effective mechanisms to detect both clinical and subclinical mastitis, the use of recording systems that allow for timely evaluation of risk factors and feedback mechanisms that allow management personnel and veterinarians to regularly assess progress toward meeting the agreed upon goals. Implementation of udder health plans is often improved when processors provide incentives for producing higher quality milk.

2. The annual udder health plan should emphasise prevention of new infections. Emphasis should be placed on proactive planning to prevent new IMI rather than reactive strategies to limit losses after occurrence of IMI. Management of the environment to reduce exposure to pathogens is especially important as numerous studies have demonstrated that exposure to environmental pathogens is strongly associated with animal hygiene (Barkema *et al.* 1998; Peeler *et al.* 2000; Schreiner and Ruegg 2003). Maintaining clean and dry udder is especially important. Using a four point udder hygiene scoring (UHS) system, Schreiner and Ruegg (2003) demonstrated that cows that were scored as having dirty udders (scores 3 and 4) were at increased risk of IMI and had increased SCC. Forms to perform udder hygiene scoring are readily available (Anon. 2013b).

The milking process is a critical control point for limiting new IMI and should include well recognised practices that are known to contribute to production of high quality milk. Several studies have indicated that the risk of new IMI can be reduced by ensuring that milking technicians wear disposable nitrile or latex gloves (Rodrigues *et al.* 2005; Dufour *et al.* 2011). Essential aspects of an effective pre-milking routine include effective teat disinfection, examination of foremilk, sufficient stimulation to allow for effective milk let-down, complete drying of the teats and timely attachment of the milking unit. Research has definitively established that the gold standard for premilking teat disinfection is the application of an effective disinfectant for a sufficient contact time, followed by drying the teat using a single cloth or paper towel for each cow (Galton *et al.* 1984; Galton, Petersson and Merrill 1986). To ensure that clinical mastitis is detected and abnormal milk does not enter the human food chain, two or three streams of foremilk should be removed and examined before attaching milking units. Drying of teats has been demonstrated to reduce bacterial counts of teat ends (Galton *et al.* 1986) reduce the number of colonies of spore forming bacteria in bulk milk (Rasmussen, Galton and Petersson 1991), and to effectively reduce the risk of contaminating milk with iodine residues from pre-dips (Borucki-Castro *et al.* 2010). The use of automatic take-off units is encouraged as they increase consistency of milking and allow milking technicians to spend more time on pre-milking preparation. Effective post-milking teat disinfection is fundamental to control of contagious pathogens (Keefe 2012). The principle is based on killing pathogens deposited on the teat skin during the milking process before they colonise the teat orifice and invade the gland and effective application of post-milking teat dips can result in >50% reduction in new IMI (Harmon 1996). Post-milking teat dipping is a highly adopted practice, but it is not always properly implemented. Milking technicians should be trained to use an application method that ensures that at least 75% of the teat skin is covered with an approved, commercially formulated teat disinfectant. Efficacy of the post-milking teat dip should have been demonstrated through properly performed scientific trials. A list of peer-reviewed publications about efficacy trials of teat dips is updated annually by the NMC (Anon. 2009).

3. *Farmers must identify and manage chronically infected cows.* Cows that maintain more than 2 months of individual SCC >200,000 cells/mL and cows that experience repeated (>2 episodes) of clinical mastitis can be considered to be chronically infected. There are 6 options that can be considered for managing chronic cows to reduce BTSCC. 1) Economic models have demonstrated that treatment of subclinical infections during lactation is almost never cost effective (Swinkels *et al.* 2005; Steeneveld, Swinkels and Hogeveen 2007). Due to the value of milk and uncertainty of treatment responses, treatment of cows with subclinical mastitis is rarely cost effectiveness and is generally only recommended when the IMI is caused by *Streptococcus agalactiae* (Keefe 2012). 2) Physical segregation of chronically infected cows has been shown to be an effective method of reducing the new infection rate (Wilson, Gonzalez and Sears 1995; Zecconi, Piccinini and Fox 2003), but is labour intensive and dependent on use of consistent use of diagnostic methods that can accurately detect infected cows. Many farmers arrange the milking order so that lower risk animals (first lactation cows or cows with

lower SCC) are milked first to reduce the risk of transmission of mastitis pathogens during the milking process. The use of automated disinfectants to back-flush the teat cups between cows has been demonstrated to reduce the risk of developing new IMI caused by *Staphylococcus aureus* (Hogan *et al.* 1984), but the cost effectiveness of these systems is dependent on the prevalence of infection within the herd. 3) Early dry off of the affected cow. While the use of intramammary dry cow therapy (DCT) remains an effective practice to prevent new IMI during the dry period, cure rates for chronic mastitis caused by *Staphylococcus aureus* remain relatively low even when multiple treatments or systemic therapies are added (Cummins and McCaskey 1987; Erskine *et al.* 1994; Nickerson *et al.* 1999). In general, most studies do not indicate that use of DCT and/or additional therapies at dry off result in cure of most chronically affected cows. Thus, when this strategy is used it should be accompanied by an effected surveillance programme that will limit the risk of transmission if the cow remains infected in the next lactation. 4) Dry off of the affected quarter. When a single mammary gland quarter of a cow is chronically infected, farmers have the option of permanently drying off that quarter. Therapeutic cessation of lactation should be performed under veterinary supervision and is often accomplished by infusion of an irritating substance such as iodine or chlorhexidine into the mammary gland (Middleton and Fox 2001). This strategy reduces the shedding of SCC into milk and decreases the potential for transmission among cows. However, this intervention is not therapeutic and must be accompanied by an intensive strategy to prevent new infections. 5) Segregation and discard of milk from affected quarters. In some instances, when there is a need to rapidly reduce the BTSCC without excessive culling, individual "quarter-milking" containers can be used to reduce cross contamination of milking clusters and to reduce the BTSCC. These small containers are inserted into the milking cluster to divert from chronically affected quarters away from the bulk tank. If these devices are used, they should be considered as potential fomites for transmission to other cows, and they should be carefully washed and dried after each use. 6) Culling of the cow. The prevalence of chronically infected cows is a strong predictor of risk of new IMI for pathogens that can be transmitted in a contagious manner. When possible, culling is a preferred strategy to manage many chronically affected cows. One study demonstrated that the odds of a cow becoming infected with *S. aureus* doubled with each 5% increase in the herd prevalence of existing infection (Dufour *et al.* 2012). Chronically infected cows must be identified through routine testing of individual cows SCC and microbiological analysis should be performed to identify the causative pathogens. Cows that develop chronic infections with pathogens that are refractory to treatment should be culled to reduce the risk of transmission to healthy animals.

Summary

The production of high quality milk is vital for dairy producers to remain competitive in the global dairy industry. Reducing bulk tank SCC is based on adoption of well-known best management practices that minimise development of new IMI. Regular monitoring of individual cow SCC is an important tool that can be used by farmers and their advisors to develop and implement annual udder health plans.

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