

CHANGING AND EMERGING PATHOGENS

James A Leigh

School of Veterinary Medicine and Science, University of Nottingham, Sutton Bonington Campus, Sutton Bonington, Leicestershire, LE12 5RD.

SUMMARY

Advances in molecular technology are being used to dissect the populations of bacteria that cause bovine mastitis. Although the species involved in this disease have varied little since the implementation of the Five Point Control Plan, it is becoming evident that specialised sub-groups exist within each. It is anticipated that the ability to identify clusters of bacteria with differing biological properties coupled to the greater availability and utility of bacterial genomic sequence data will lead to a massive expansion in our detailed understanding of the processes underlying this disease.

INTRODUCTION

An understanding of the causative agents of disease and their transmission is essential for sustainable disease control at local, national and international levels. Bovine mastitis typically results from infection of the mammary gland and associated tissues and is a consequence of successful colonisation, evasion of host defences and induction of marked and overt inflammatory changes. The infectious agents most associated with bovine mastitis are bacteria.

A number of studies have indicated the range of bacteria capable of causing bovine mastitis. Watts described 137 distinct infectious agents linked to mastitis in cattle (27). Given the simple route of infection (penetration of the teat canal) it is likely that the list produced by Watts 20 years ago is not exhaustive. Additionally, studies that have investigated the role of viruses, protozoa, mycoplasma and even anaerobic bacteria are relatively rare.

Despite the large number of agents capable of causing bovine mastitis, those most frequently responsible for the disease worldwide are largely restricted to species of the genera *Streptococcus*, *Staphylococcus* and a variety of coliform bacteria. The major impact on the relative distribution of these was the implementation of the 5-point control plan; formulated at the National Institute for Research in Dairying (NIRD). This resulted in a dramatic reduction in the number of cases of mastitis in the UK (6); largely due to the control of organisms that showed a predominantly contagious route of transmission (e.g. *Streptococcus agalactiae* and *Staphylococcus aureus*). Failure of the 5-point plan to control other groups of bacteria (e.g. *Streptococcus uberis* and the various coliform species) led to the hypothesis that such bacteria were transmitted to the gland from sources other than infected/diseased mammary quarters (i.e. "the environment"). Indeed, such

was the success of these procedures worldwide that the terms “contagious” and “environmental” pathogen (referring to those organisms that are and are not controlled by the five point plan, respectively) have now entered the language of researchers, veterinarians and dairy farmers. These terms have increasingly been used to describe discrete, non overlapping categories of pathogens. However, this is clearly an oversimplification of the diverse and complex interactions between host, pathogen, environment and husbandry practice that underpin the events leading to infection.

It is the intention of this presentation to briefly (but not exhaustively) review the current situation and introduce new techniques that are being employed that will enable genetic change within the mastitis causing bacteria to be assessed.

HAVE THE CAUSES OF BOVINE MASTITIS CHANGED SINCE THE IMPLEMENTATION OF THE 5-POINT PLAN?

A survey published recently describing the current situation in England and Wales (5) revealed that the rate of bovine mastitis is higher than the 35-40 cases per 100 cows per year previously reported (3, 17). It now stands at, a conservatively estimated, 47-65 cases per 100 cows per year (5). It is not clear if this represents a real increase or simply reflects previous underreporting or smaller or biased samples in earlier surveys. The increase, if indeed there has been any, did not reflect a major shift in the spectrum of pathogens, which had changed little from that described more than 25 years before (10). The absence of *Streptococcus agalactiae* from this survey would strongly suggest that the herds included in this study were largely compliant with accepted, normal procedures for the control of “contagious” pathogens and had acceptable levels of “mammary gland bio-security” in place.

It would therefore appear that in a broad sample of herds that there are no new species that have emerged as major or even significant minor causes of clinical disease in England and Wales. In such studies, a number of cases of mastitis are always reported as “no pathogen” or “no bacteria” present (3, 5, 17). Whilst this maybe true and these episodes maybe caused by events other than infection, it is also possible that the organism was not detected as it was only present in low numbers. Also, it is possible that the agent present was something other than the range of heterotrophic, aerobic bacteria that are typically sought from such samples (27).

Based on current knowledge, maintenance of the present level of disease will continue to be reliant on the use of prophylactic and therapeutic antimicrobials; a situation that will only be relieved by development of *effective*, alternate therapies, preventative products or other strategies. The rate of use of antibiotics in human medicine is again coming under scrutiny and it can be predicted that it will not be long before there is yet another review of their use in agricultural procedures.

From a biological standpoint, it is of note that despite the distinct geographical, climatic and husbandry conditions under which dairy cattle are farmed, the range of bacterial species present in mastitic samples varies relatively little. For example, data collected from Ethiopia in 2003 revealed staphylococci and streptococci, namely *S.aureus*, *S.uberis*, *S.dysgalactiae*, *S.agalactiae* and a range of coagulase negative staphylococci, as causes of clinical infection (1). A study from Tanzania (16) revealed *S.aureus* (plus other non-identified staphylococci), *S.agalactiae* (plus other non-identified streptococci) and coliforms (including *Klebsiella spp* and *E.coli*) as causes of clinical disease. Similarly, in Norway, *S.dysgalactiae*, *E.coli* and coagulase-negative staphylococci were amongst the most prevalent pathogens (28). Such consistency reveals an underlying biology that must underpin the host pathogen interactions that lead to disease.

It can be hypothesised that the host pathogen interactions that lead to mastitis present a barrier through which any new pathogen must pass before it can emerge as a significant cause of disease. We can also speculate that unless the infectious agent is allowed to pass from cow to cow unobstructed then the spread of our hypothetical new pathogen would be restricted. Furthermore, increasing our understanding of the host pathogen interactions that lead to infection and disease will leave us in a good position to develop new treatments or control strategies to cope with any new or emergent pathogen should one arrive.

GIVEN THAT NO NEW SPECIES ARE COMMONLY ASSOCIATED WITH MASTITIS, ARE THE EXISTING ONES CHANGING?

In 1988 Jeff Watts stated in the abstract of his paper;

“...Nucleic acid hybridization studies have restructured the classification of many mastitis pathogens. Availability of defined species descriptions has permitted greater insight into the distribution and pathogenicity of many previously unrecognized microorganisms associated with bovine mastitis. Precise epidemiological studies are needed to better delineate the role of some microorganisms in bovine mastitis and to aid development of improved control methods.” (27)

Twenty years on it is possible to reiterate the same sentiments in the context of the sub-species (strain/type) and even at the level of the individual gene. Nucleic acid technology has now enabled evolutionary structuring of some, if not all, mastitis causing species at the sub-species level. It is also anticipated that precise sub-species descriptions (and tools to define them) will provide the basis for gaining greater insight into pathogenic mechanisms and that precise epidemiological studies will enable delineation of the role of individual bacterial genes thus aiding development of improved control methods.

One of the most recent molecular tools to be applied to typing mastitis causing bacteria is multi locus sequence typing (MLST). In 1998 a group at the University of Oxford (20) proposed a system (MLST) that could not only separate bacterial isolates into types (sequence types or STs) but also reflected accurately an isolate's evolutionary relationship to others in the sample population. This was first applied to *Neisseria meningitidis* (a cause of meningitis in the human population), but has subsequently been applied to an ever growing list of bacterial species. MLST is a sequence based typing method in which the variation in the population is indexed through variation in the nucleotide sequence within individual isolates. Unlike other typing systems, MLST uses generic methodology to produce objective data which can be readily transferred from lab to lab electronically via the internet (26).

MLST uses the sequence variation detected in housekeeping genes (alleles) to index evolutionary change or progression. In most schemes, six or seven housekeeping genes (those encoding proteins responsible for core metabolic pathways) are analysed. Each new allelic sequence is given a new allele number and once completed each isolate has a six or seven component code termed the allelic profile. Each new allelic profile is allocated a sequence type (ST).

The sequence of other types of gene could equally be used in such a scheme, but each would index change at a different evolutionary speed. For example, 16S rRNA sequences are fairly stable and have been used to index changes at an evolutionary speed that matches the typical delineation of a bacterial species; these would be ineffective for monitoring intra-species variation. At the other extreme, genes encoding products subject to external selective pressure (e.g. those responsible for biosynthesis of antigenic capsule or a surface protein antigen) give the appearance of rapid evolutionary change at a speed dependant on the selection pressure and may vary between two individuals that are otherwise identical. Depending on the desired application, analyses of such sequences can be extremely valuable. For instance, individual species within the entire genus *Streptococcus* can be identified using 16S rRNA sequence data (2) and the appearance of individual capsule types of *S. pneumoniae* can be identified through the appearance of different biosynthetic genes (8).

Another advantage of MLST is that by analysis of several genes the speed of change is indexed around the bacterial chromosome thus offsetting the rapid evolution that may be seen in any single gene or any particular region of the genome. Also, variant sequences that have been acquired as a result of horizontal exchange of genetic material are given the same standing as one that has acquired a single point mutation, as a result of aberrant DNA replication or repair. Thus MLST negates any apparent rapid evolution due to gene acquisition. By selection of genes that are not under external selective pressure, but which are constrained by function, MLST provides a framework on which bacterial evolution and underlying population relationships can be evaluated and on to which more rapidly evolving traits can be superimposed.

MLST schemes have now been established for *S.aureus* (11), *S.agalactiae* (15) and *E.coli* (21). Two schemes exist for *S.uberis* (9, 30); one is publically accessible and follows the conventions described above (9). The other is not readily accessible and does not follow the normal conventions of MLST, as it includes genes under selective pressure (30). As a result, although it has value for typing, it is not appropriate as a tool for population biology and evolutionary analysis.

The scheme for *E.coli* is yet to be applied to isolates from bovine mastitis. However, the other schemes have been used in this context.

That for *S.aureus* was used by an international group led by a team from Warwick University (24). This revealed that isolates from bovine and human sources generally differed in their ancestral origins; implying that neither humans nor cattle were a common source of infection for the other. However, it was reported that milkers' hands did yield *S.aureus* that were found on teat skin and in the mammary gland, but the direction of movement of these bacteria cannot be certain. MLST also showed that the majority of bovine isolates (worldwide) belonged to a single group of related strains (clonal complex) based around the sequence type, ST97. This indicates that only one sub-group of the species *S.aureus* is responsible for most of the infections in the bovine udder. Surprisingly, the STs found in milk generally differed from those on teat skin; indicating that teat skin was not the principal source of *S. aureus* for the mammary gland. Also, in one herd the predominant strain found in the gland was also found in bedding material; implying that in some cases an environmental link can be established for *S.aureus*.

MLST of *S.agalactiae* (4) revealed that, not unsurprisingly, all the bovine isolates fell into a discrete group. Indicating that a restricted population was responsible for infection in cattle; an observation consistent with contagious spread of infection. In general, as with *S.aureus*, isolates from cattle and humans were shown to be of a different evolutionary origin, but with one notable exception. One type of human *S.agalactiae* (ST17) was clearly of bovine ancestry. This type and its associated clonal complex were first isolated from the human population in the 1970s and this group is now a leading cause of neonatal infection (the rate of infection with *S.agalactiae* in human neonates is approximately 1 per 1000 live births in the UK and late onset disease (occurring from 7-90 days of age) carries a mortality rate of up to 15% (7)). The genetics underlying the hypervirulent nature of clonal complex ST17 in humans is not yet clear and a subject of intense investigation.

Studies on *S.uberis* showed that the population isolated from the bovine mammary gland was more diverse than those of *S.aureus* and *S.agalactiae* (9). This is consistent with the assumed transfer of this organism from widespread environmental sources rather than other infected mammary quarters. However, the population of *S.uberis* is not without structure and

possible specialisation. Approximately 50% of the typed population shows little if any relation to others, however, the remainder can be grouped into three distinct clonal complexes (centred on ST5, ST143 and ST86). Isolates from the UK and Europe appear to belong largely with the clonal complex of ST5; those from Australasia are more commonly associated with ST143 (19, 23). Researchers in New Zealand have been able to make a collection of environmental *S.uberis* that is contemporary with their mammary gland isolates. This revealed that the population structure was similar in each niche (23). However, there were some common environmental isolates that were rarely seen in cattle and common mammary gland isolates that were not detected in the environment (19, 23). It is tempting to speculate that the former were of low infectivity and/or virulence and that the latter were also capable of cow to cow transmission. Without further experimental evidence this is, however, just conjecture. A subsequent study conducted in Australia has confirmed that the clonal complexes based on ST5 and ST143 appear to be correlated with overt mammary gland infection (clinical signs and/or high cell count), but very interestingly they also noted that isolates belonging to clonal complex ST86 were associated with low cell counts (covert infection) (25). A limited survey conducted with isolates from dairy herds in the UK undergoing organic conversion showed that infections of long duration (50-260 days) and those that spontaneously cured (in the absence of antibiotic) in less than one month did not correlate with particular STs or related groups; although in this small study none of the isolates corresponded to clonal complex ST86 (22).

The level of detailed information that can now be gleaned from the use of molecular tools in an epidemiological context is immense. The use of MLST as a base-line typing and population biology tool has only just begun within the mastitis research arena, but has already allowed objective information to be stored, transferred and interrogated on a global scale. Such information will allow us to make rational, objective and scientifically valid assertions as to whether the bacterial species that cause mastitis are changing and how these changes are manifest within the population as a whole.

It is interesting to note from the analyses of these populations that the appearance of new genes in *S.aureus* is equally likely to have resulted from point mutation as genetic exchange (acquisition of DNA from another individual) (24), whereas in *S.uberis* the overwhelming route of diversity is due to genetic exchange (25).

Although at present we cannot conclude that mastitis pathogens are changing in any meaningful way, we can now determine that not all members of a species are equal. In some cases specific traits or tropisms can be aligned with particular genetic sub-sets within the species. In the development of the tools used to determine this, we also have seen that both *S.aureus* and *S.uberis* have the ability to exchange genetic material with other members of their own species and possibly other bacterial species present in the same niche. Such a property will permit rapid acquisition and spread of new traits of benefit to these organisms.

ENVIRONMENTAL & CONTAGIOUS PATHOGENS; JUST HOW DEFINITE ARE THESE DEFINITIONS?

As mentioned previously the 5-point control plan had a major impact on both the rate of new infection and the causative agents of mastitis (6). The same, or similar, procedures have also been shown to be similarly effective on a global scale. The underlying basis of this scheme was to reduce the probability of an infection spreading from one quarter to another quarter by reducing both the duration and likelihood of transfer of existing infections. To this end, it recommended;

1. The use of antibiotic treatment on all clinical cases (to reduce the duration of infection).
2. The use of blanket dry cow therapy (to eliminate any residual unapparent infections present at the end of lactation and to protect the gland from infection during the early dry-period).
3. Culling of persistently infected cows (in an attempt to remove chronically infected and highly susceptible animals from the herd).
4. Dipping of milked (susceptible) teats in disinfectant (to prevent "invasion" by bacteria deposited on the teat during (or immediately after) milking).
5. Correct maintenance and use of the milking machine (to reduce the possibility of transferring any milk harbouring infectious agent between quarters).

The exclusive "environmental" and "contagious" labels that have been allocated to different mastitis pathogens are somewhat misleading. Whilst these descriptions describe the bacterium's most probable or typical route of infection, the outcome of the host pathogen interaction also significantly influence the possible routes of transmission.

At either extreme of the spectrum are *S.agalactiae* and *E.coli*. The former seems not to occupy environmental reservoirs likely to result in teat end contamination, whereas *E.coli* quite clearly does. Infection with *S.agalactiae* is relatively low grade and usually sub-clinical in nature (12), whereas infection with *E.coli* is typically of rapid onset and acute (14). Therefore once in the gland *E.coli* is quickly recognised and persists for only short period of time whilst *S.agalactiae* may remain undetected for considerably longer. Thus the opportunity for *S.agalactiae* to transmit is greater, but once removed from the gland the ability to acquire a new infection is limited. In the case of *E.coli* the opportunity to transmit is low but, under the correct circumstances, the ability of the gland to acquire a new infection from other sources is high.

The situations with *S.aureus* and *S.uberis* are less clear cut. The former would be considered “contagious”, but in some cases environmental reservoirs containing the specific sub-set involved in mammary gland infection have been detected (24). The latter would be considered “environmental”, but instances where apparent contagious spread can be detected (i.e. the same organism (type) detected in different glands at one time) are not uncommon. Certainly, *S.uberis* is more readily isolated from the environment than *S.aureus*. Also, the diverse population of *S.uberis* found in milk samples (9, 22) would indicate a large and diverse reservoir such as the environment. The restricted population of *S.aureus* that infect the gland (24) is indicative of infection from a narrow restricted reservoir such as other infected mammary glands.

However, the diverse nature of *S.uberis* could well hide sub-populations that can not only gain access to the gland from environmental contamination but once there could also transmit from gland to gland. Field strains of high and low virulence have already been shown in *S.uberis* (13). Factors that alter duration of infection (time from infection to clinical signs), number of bacteria in milk and infectious dose will all impact on the contagious potential of *S.uberis*. Given the high numbers that *S.uberis* can attain in milk (18) and the long duration of infection prior to the onset of clinical signs (23), it is possible that this organism (or specific sub-sets of this organism) may have an even greater propensity for contagious transmission than some of those currently labelled as “contagious”.

A Report describing the apparent contagious transfer of *S.uberis* within a herd has been published (29). Although such transmission may in many cases be attributed to a breakdown in “biosecurity”, it is also possible that due to high bacterial numbers and long duration of infection that under certain circumstances this bacterium can overcome the barriers that are suitable to prevent the spread of *S.aureus* and *S.agalactiae*. We do not yet know the level of *S.uberis* infection in the UK that results from contagious transmission of an environmentally acquired organism.

WHAT CAN BEEN GLEANED FROM FURTHER EXPERIMENTAL RESEARCH

Being able to cluster isolates in discrete genetically related groups using MLST is a solid foundation on which particular biological properties (e.g. site of isolation/colonisation, contagious nature, and cause of acute/chronic infection) can be superimposed. Such epidemiological analysis can be used to direct further analysis to determine the specific genetic differences between the sub-sets within the population.

These different attributes of bacterial strain/types are embedded within the bacterial genome and genomic sequences are becoming more accessible on a routine basis. For example, in January 2001 sequencing of the first *S.uberis* genome was initiated using the latest technology available at the Sanger

Centre. The genome was completed in November 2004 (sanger.ac.uk/Projects/S_uberis/) at a cost of £100,000s. Due to current advances in technology (biochemistry and computing power) it is now possible to commission a commercial sequencing company to generate a new sequence for £1,000s and the data is available in weeks. It is predicted that as the cost of the newer sequencing technologies continues to fall it will not be long before there is the opportunity to obtain whole bacterial genome sequences in days for a matter for just £10s. Such advances in technology underpinned by accurate analysis of bacterial populations, solid epidemiological models, sound bioinformatics and a good understanding of the biology of the disease pathogenesis will permit investigations of mastitis at a level of detail never before considered possible.

These data will continue to test and challenge the existing dogma and where appropriate will be used to develop new interventions to reduce the burden of bovine mastitis.

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THE GENETIC INFLUENCE OF MASTITIS

Marco Winters

DairyCo *breeding+*, Trent Lodge, Stroud Road, Cirencester, Gloucestershire, GL7 6JN.
Marco.winters@dairyco.org.uk

SUMMARY

Direct selection against (sub) clinical mastitis is difficult, because mastitis events are not widely recorded and because its heritability is very low. However, since 1998 the UK has been calculating and publishing genetic indices for Somatic Cell Count (SCC), which can be used to indirectly select against mastitis with about 70 percent efficiency.

Analysis of UK data shows that farmers have been making effective use of these Predicted Transmitting Abilities (PTA's) for SCC and have now managed to successfully reverse the genetic increase of SCC.

On-farm data has shown that the impact of good SCC PTAs is very effective in reducing phenotypic lactation average SCC of cows, and therefore a long term reduction of phenotypic SCC can be achieved.

However, for long term economic sustainability a balanced approach of selection for mastitis resistance alongside other fitness and milk production traits is needed. The current national Profitable Lifetime Index (PLI) with over 50 percent emphasis on 'fitness' traits is the most effective way of achieving this.

INTRODUCTION

The economic impact of mastitis on a dairy herd is considerable. The various factors responsible for this are mainly loss of production, increased labour, veterinary treatments, therapeutics and increased culling.

Although no accurate national statistics exist on the exact reasons for culling, the results of numerous surveys suggest that mastitis is likely to be the second biggest reason for animals and is in the order of 20% of all culls in the dairy herd (e.g. 4). Fertility is the biggest reason.

Traditionally mastitis control programs have mainly focussed on management. Managing environmental factors has been shown to be effective in controlling infections in the short term, but have been limited in controlling the disease long term.

To the animal breeder, the aim has always been to take care of long term needs. Historically this has been geared towards higher outputs (e.g. milk yield), but over recent years there has been a significant shift in emphasis

away from maximising outputs, to maximising long term economic sustainability. Numerous studies have shown that selection on production alone, has led to negative genetic trends in several 'fitness' traits, including mastitis. In order to improve this situation a balanced selection approach is required, which addresses the need to maximise output, without sacrificing these 'fitness' traits. Over the years total merit indices used for genetic selection have incorporated many of these non production traits (1).

Use of SCC to select for mastitis resistance

Like many of the disease traits, recording of mastitis is subject to a number of difficulties. The two main ones are; recording fails to detect all cases of infection (e.g. sub-clinical) and secondly, registration can be incorrect, depending on the method and timing of recording.

A way of solving these problems is by using an easy to measure, objective indirect correlated trait. Various studies have shown that Somatic Cell Count (SCC) can be used as such, and literature estimates of the genetic correlation between mastitis and SCC are moderately high and average around 0.7 (2). The additional benefit of SCC over clinical mastitis is its higher heritability of around 0.11, compared to around 0.03 for mastitis, making genetic improvement easier to achieve using the currently available data.

Because SCC is heritable, we are able to estimate PTA's for bulls, which can be used in selection. This provides a measure of an animal's ability to pass its genes for SCC on to the next generation. Although we are most familiar with genetic indexes for milk, fat and protein, it's a common misconception that indexes only relate to production. Genetic indexes are available for many more than these alone and their application for non-production traits is becoming increasingly important.

Even though no direct distinction can be made for the reason for elevated SCC levels, which may be due to contagious or environmental infections, past studies have shown that, although not ideal, selection for a general reduction in SCC is an effective tool to reduce mastitis in general and will tackle both clinical and sub-clinical mastitis.

UK milk recording organisations started to collect SCC data routinely through the monthly milk recording from 1990 onwards. Since 1998, the UK has been using this national data to calculate SCC PTAs and the selection of service sires has been influenced by this for the last 10 years (3).

Somatic cell count PTAs in the UK are expressed as a percentage and generally fall within the range +30 to -30. For every 1% in a bull's SCC PTA, a change of 1% in his daughters' SCC is predicted. For example, daughters of a bull with a -10% SCC are expected to have cell counts 10% lower than daughters of a bull with a SCC PTA of zero. So, negative figures for SCC PTAs are desirable as these indicate a reduction in cell counts. The advice to

farmers is to avoid excessively high SCC bulls (>15), and when possible favour the good SCC bulls (<0).

It has been suggested that it may not be desirable to reduce SCC too much, as these are a defence mechanism against infections. However, past studies suggest that SCC levels should be seen as a measure of the level of infection, rather than a measure of ability to resist an infection. Therefore, unless SCC levels in the population substantially reduce, this worry has not been shown to be a valid one.

For this paper, historic data has been analysed to show the impact that genetic selection for SCC is having on the national dairy herd, and to project where we are heading.

MATERIALS & METHODS

For this study a subset of the nationally recorded SCC data, used for the purpose of routine genetic evaluation, was extracted. This data was based on validated records for cows calving in 2005 and Q1 2006 and was collected by the Cattle Information Services (CIS), National Milk Records (NMR) and United Dairy Farmers (UDF).

Table 1 gives a description of the 551,262 data records used in this analysis. The summary shows how SCC increases by about 25% with each increase in parity and also shows how the geometric means are lower than the simple arithmetic means. The geometric means here are based on the mean of the log transformed test day records, transformed back to the original SCC scale. This transformation results in a less severe impact of the outlying high SCC tests, resulting in a slightly lower overall mean. For the remainder of this paper geometric SCC is used.

Table 1 Geometric and Arithmetic means plus number of records by lactation number of the data used

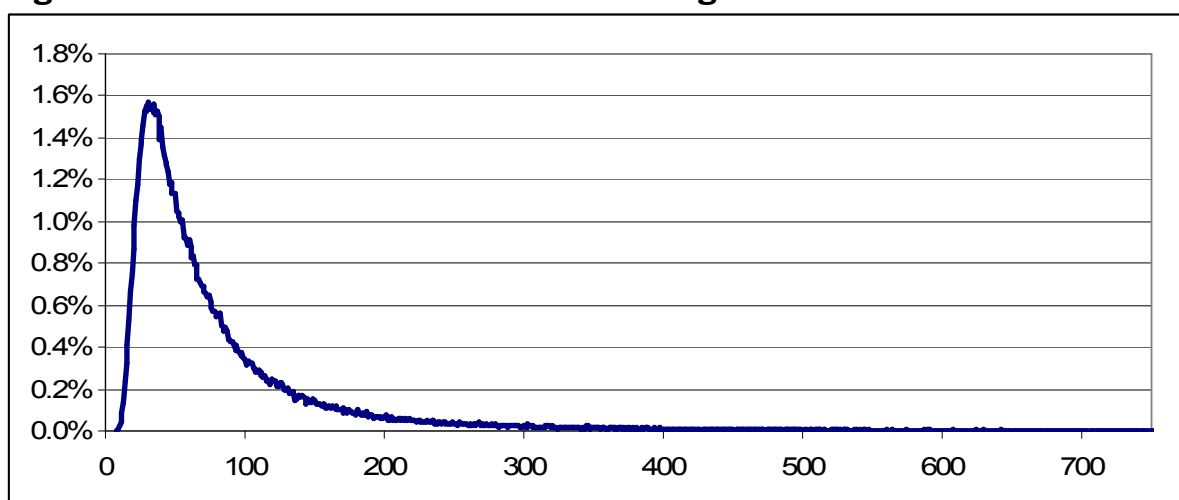
Lactation	Geometric Mean (x1000)	Arithmetic Mean (x1000)	Records
1	88	139	188052
2	108	171	152430
3	139	220	117842
4	167	264	92938

RESULTS

Distribution of Somatic Cell Counts

SCC data shows a highly skewed distribution, with the mean roughly 2.5 times the median of the data. Figure 1 shows the distribution of first lactation SCC average which clearly illustrates the long extended tail for high SCC values. Although the median of the population is at a very acceptable level already, improvements are still desired to reduce the number of clinical and sub-clinical mastitis cases, represented in this graph as animals that feature in the right hand tail. So will breeding for reduced SCC by means of sire PTAs for SCC help us achieve this goal?

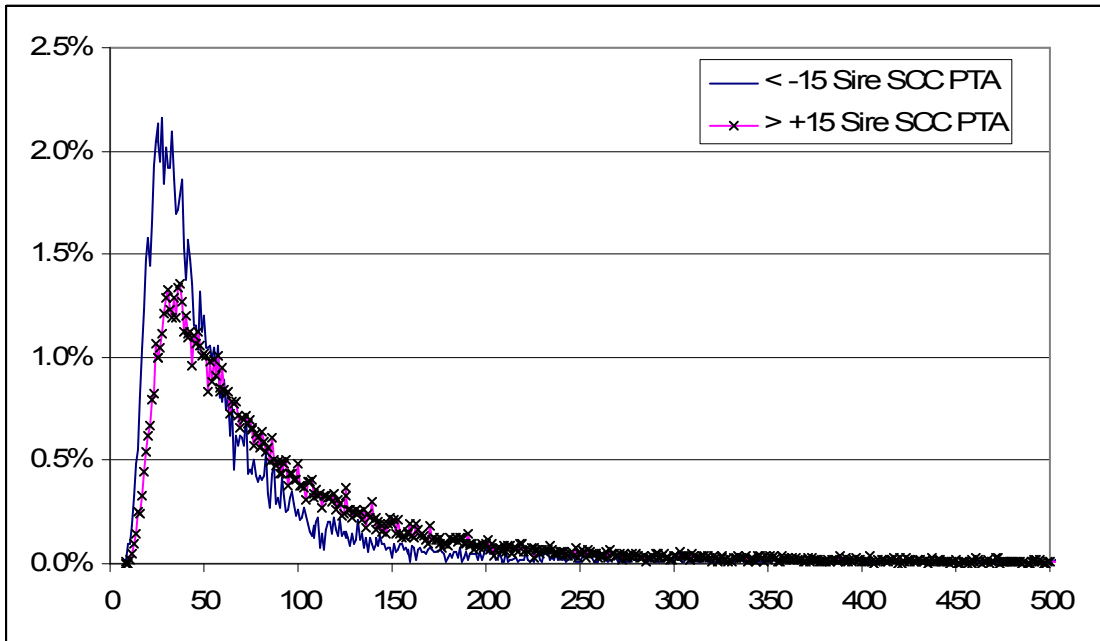
Figure 1 Distribution of lactation average SCC for first lactation



Impact of genetics on animal performance

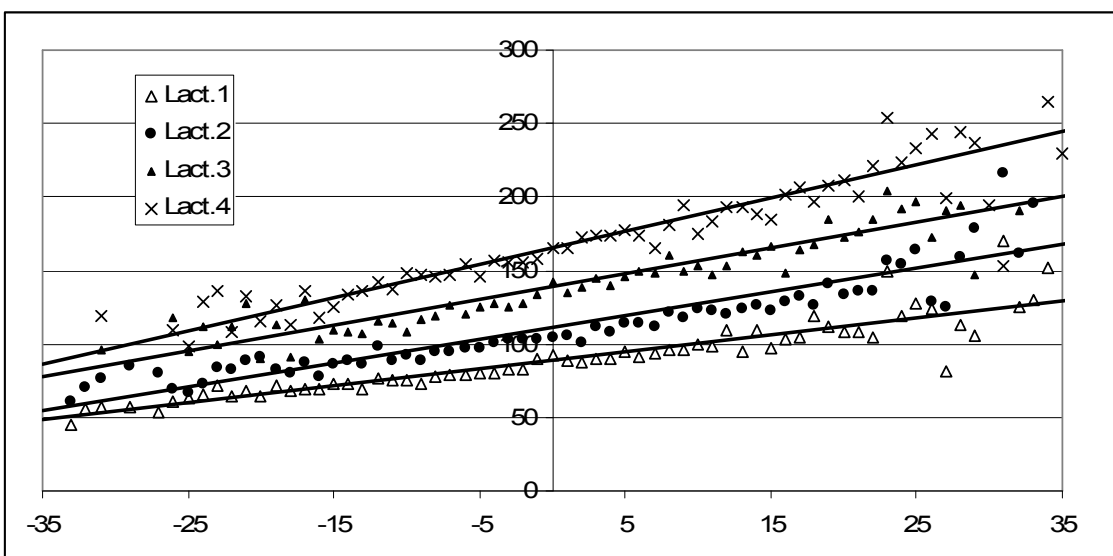
Whereas figure 1 presented the distribution of all first lactation cows, in figure 2 the data is further split, and a separate distribution of lactation average SCC is given for cows who are sired by very good SCC bulls (less than -15 SCC) and for cows that are sired by poor SCC bulls (greater than +15 SCC). This graph illustrates that not only does selection for good sire SCC PTAs results in a slightly reduced median, but more importantly has a significant impact on animals with elevated lactation SCC's. The difference in proportion of animals that are 100,000 SCC or below is 86 percent for the daughters of good SCC PTA bulls, versus only 69 percent of daughters of the high SCC PTA bulls.

Figure 2 Distribution of first lactation average SCC for good and bad SCC PTA sires



The data was further analysed to determine the impact of sire PTAs for SCC on the daughter average lactation performance of SCC. Figure 3 shows the SCC PTA of bulls (x axis) plotted against the average lactation SCC's of their daughters (y axis). This plot clearly shows that the link between genetics and phenotypic performance is strong over a large group of animals, and the fact that the lines are not quite parallel but diverge as the figures get higher, indicates that the biggest cell count improving bulls generally have the greatest impact on the worst cell count cows. Once again this figure shows that as animals get older, lactation SCC increase.

Figure 3. Average daughter lactation SCC against their sire SCC PTAs for lactations 1 to 4

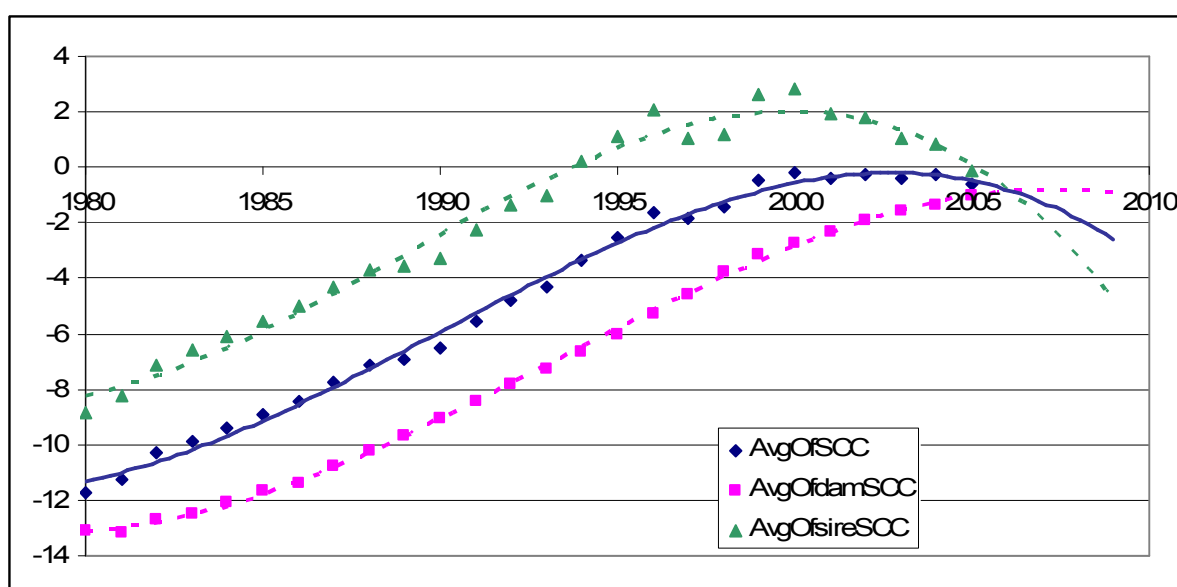


Past genetic trend and future projection

As mentioned previously, UK dairy farmers have had access to SCC PTAs since 1998. In order to measure the success rate of the use of these figures, we can look at the genetic trend for SCC in the national herd.

Figure 4 shows the genetic trend for SCC in UK Holstein cows, based on over 6 million records.

Figure 4 Historic actual genetic trends and future projections for SCC PTAs of UK cows, together with the average PTAs of their sires and dams.



The data in graph 4 shows an almost linear increase (i.e. undesirable) genetic trend for SCC since early 1980 until 1999. After 1999, we see a distinct levelling of the SCC values, and most recent data of 2005 born cows indicate that a small reduction of SCC is now beginning to happen. The data points for cows born after 2006 are based on forward projections.

Alongside the cow average SCC PTAs, we can also look at the average genetic merit of their sires and dams. This picture reveals an interesting trend. From 1980, with the increase in genetic selection on milk production and without the aid of SCC PTAs available, we can observe a strong lift in SCC due to the introduction of high production Holstein genes into the population. However, when SCC PTAs were made available for use in bull selection in 1998, we can almost immediately see that the sires used to breed the next generation of cows are showing an improvement. Due to the generation interval time lag and the inability to perform strong selection in dams to breed the next generation of cows on the average farm, we see that the dam genetic trend was still in the upward (undesirable) direction for quite a while, and is only starting to show a levelling off some 6 years later.

This is in line with expectation, knowing that the average age of dams is about six years.

Based on the historic trend, we are also able to forward project the SCC trends. This projection is very encouraging, and based on this we expect to see a genetic reduction (desirable) of SCC for cows born in 2006 for the first time. Analysis of insemination data (not published), indeed shows that the genetic merit of service sires has been following the projected trend as given in figure 4. So, bearing in mind that 2006 born cows will calve for the first time in 2008, we would normally expect to see a phenotypic reduction of SCC's and associated mastitis incidence for heifers from 2008 onwards. Obviously this prediction is relying on external environmental influences not deteriorating. If we take this one step further and use these genetic trends to establish a trend for the live cow population, we are similarly expecting to see a gradual improvement in the national herd average SCC values from 2008 onwards.

DISCUSSION

The fact that actual (phenotypic) link between mastitis and production is generally far lower than the genetic link, indicates that farmers are doing a good job of managing the underlying genetic issue. However, if the genetics behind the problems continues to be ignored, the farmer's job in time will become harder and herd health will eventually decline beyond acceptable levels.

That's why genetic information for health and welfare traits is becoming increasingly more important, and although these traits don't tend to be inherited to the same degree as milk production, it is certainly possible to breed for them since many show sufficient genetic variation to enable selection between animals. In addition, it is important to remember that genetic improvements are very cost effective, are permanent and accumulate over the generations.

The availability of SCC alongside other 'fitness' traits have enabled us to broaden our national breeding goal for an improved long term sustainability of the dairy industry. The most recent revamp of our national Profitable Lifetime Index (£PLI) in the UK now places over 50% of its weight on non production traits (5).

Future improvement in the quality and quantity of animal performance data, enhancement of genetic evaluations and the structure of genetic selection indexes are all needed to help us provide for a sustainable dairy industry.

A new research project is about to start in the UK which will aid in this, and is planning to re-look at the national available data and new more powerful

genetic evaluation models to determine whether we can further improve 'udder health'. (Defra LINK 'expanding indices').

So far, historic assessment of the genetic trends has shown us that farmers have been adopting the new genetic indices for fitness traits very effectively. The latest indications are that lactation SCC's are finally starting to show an improvement and guided by forward projections, we anticipate a gradual improvement of on-farm phenotypic SCC and (sub)clinical mastitis as a result.

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IS THERE A PLACE FOR ANALGESICS IN MASTITIS THERAPY?

Jon Huxley

School of Veterinary Medicine and Science, University of Nottingham, Sutton Bonington Campus, Loughborough, LE12 5RD. Email: Jon.Huxley@Nottingham.ac.uk

SUMMARY

This paper reviews the literature on the pain associated with mastitis and summarises a number of recent questionnaire surveys which have asked cattle farmers and veterinary surgeons, from around the world, to score the pain associated with mastitis on a ten point pain scale. In all the data sets the median score attributed to a case of mastitis with clots in milk only was 3; the median score attributed to a case of acute toxic mastitis was 7 in the UK veterinary surgeon and European veterinary surgeon data sets and 8 in the UK cattle farmer and New Zealand veterinary surgeon data sets.

Data currently available indicates that mastitis is a painful condition; those of us working in the dairy industry should firstly be striving to reduce the number of cases of mastitis that occur and secondly to control the pain caused when cases do arise. Currently, NSAIDs are the only suitable products licensed within the UK; their usage for this indication should be encouraged.

INTRODUCTION

Mastitis remains one of the most important endemic production diseases of dairy cattle world wide. Most evidence would suggest that its incidence has actually increased in recent years as average yields have improved (1). Whilst the production losses and the financial implications of mastitis are well documented, little work has concentrated on the pain associated with this very common condition.

The International Association for the Study of Pain has described pain as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage”. It is reasonable to suppose that animals experience pain in a similar way to humans because experimental work has demonstrated that the neural pathways of pain sensation are similar in human and other mammals. Molony *et al* concluded that “If a lesion poses the same biological threat, then the pain suffered by cattle should occupy an equivalent place on its pain scale, to that of similar lesion on the human pain scale” (2). However, assessing pain in animals is extremely difficult because pain is a “personal” experience and they are unable to communicate the levels of discomfort they experience. Therefore we must rely on other indicators in order to identify animals that are suffering, such

as behavioural changes or markers of a stress response (e.g. blood cortisol levels).

Indicators of pain caused by mastitis

Indicators of discomfort suggest that mastitis causes pain. Mastitis can be graded on a three point scale (Grade 1: Changes to milk; Grade 2: Changes to the affected quarter e.g. heat, swelling; Grade 3: Systemic signs). Unsurprisingly, many papers have demonstrated that indicators indicative of discomfort such as elevated heart rate and respiratory rate, decreased ruminal activity and food intake and alterations to behaviour and general condition occur in grade 3 mastitis (e.g. 3, 4, 5, 6).

More recently, research work has demonstrated that the heart rate and respiration rate of cows with grade 2 mastitis were significantly higher than normal animals and those with grade 1 mastitis (7, 8, 9) and the hock-hock distance was significantly higher in cows with both grade 1 and grade 2 mastitis compared to their unaffected herd mates (7, 9). This last observation is particularly interesting because it indicates that even when mastitis is mild, animals alter their stance, presumably to reduce pressure on the udder, when they walk and stand.

Markers of pain caused by mastitis

Mediators of inflammation and pain such as prostaglandins, thromboxanes and bradykinins are released in response to tissue injury and infection. Elevated levels of bradykinins and other pain mediators have been demonstrated in quarters suffering from even mild cases of mastitis (e.g. 10, 11)

Modulation of pain processing caused by mastitis

If a disease or injury causes pain, the presence of high levels of inflammatory mediators around the site of injury and persistent activation of pain fibre pathways in the spinal cord leads to a decrease in pain threshold, so that stimuli are perceived as more painful than would normal be the case. This phenomenon is known as hyperalgesia. Another finding associated with chronic pain is allodynia, whereby similar mechanisms lead to perception of normally non-painful stimuli as painful. Recent findings have demonstrated that the response to a mechanical stimulus (a blunt ended pin applied to the hind legs) is altered in cows suffering mastitis i.e. a lower pressure was required to elicit a response on the leg ipsilateral to the case of mastitis compared to the contralateral leg (7, 8, 9).

Use of NSAIDs in the treatment of bovine mastitis

Non-steroidal anti-inflammatory drugs (NSAIDs) are currently the only practically useful drug class with analgesic properties licensed for use in cattle in the UK. Many papers have investigated their usage in the treatment

of bovine mastitis (e.g. 3, 4, 5, 12, 13, 14, 15, 16, 17, 18, 19, 20), however in virtually all cases they have been assessed for their anti-inflammatory rather than their analgesic properties. If analgesic properties have been investigated it has been indirectly via their impact on aspects of behaviour or physiological responses.

However, one study has specifically investigated the impact of treatment with meloxicam on the pain associated with mastitis. Cows that received antibiotics alone showed higher threshold differences for a longer period compared to cows that received either one or three doses of meloxicam i.e. cows with mastitis demonstrated hyperalgesia (7, 8). There were no differences between the two treatment groups which received either one or three doses of meloxicam. This work suggests that treatment with a NSAID at the time of diagnosis reduced the pain associated with grade 1 and grade 2 mastitis.

Subjective assessment of discomfort

As an alternative to measuring behavioural and physiological changes, experienced animal workers can make a value judgement in order to gauge the level of pain an animal is suffering. In these circumstances it is likely that observers draw on their experience of assessing a range of indicators including behaviour, posture, demeanour and other cues. Additionally it is likely that they, consciously or subconsciously, draw on their own personal experience of similar conditions.

MATERIALS & METHODS

Between 2004 and 2006 a questionnaire designed to assess the attitudes of respondents towards pain and the use of analgesics in cattle was distributed to 2,391 practicing veterinary surgeons in the UK, 10,373 practicing veterinary surgeons in a further eight European countries (France, Germany, Holland, Belgium, Spain, Norway, Sweden and Denmark), 455 practicing veterinary surgeons in New Zealand and 7500 cattle farmers in the UK. The detailed methodology has been described previously (21, 22, 23). Briefly the questionnaires collected background data about the respondent and their business (practice or farm) and attitudes towards pain and the use of analgesics in cattle. Specifically the questionnaire asked respondents to grade the level of discomfort suffered by cattle with two grades of mastitis ("Clots in milk only" and "Acute, toxic mastitis") on a ten point pain scale (1 - "No pain at all" through to 10 - "The worst pain imaginable").

RESULTS

Pain Associated with Mastitis

The median pain score attributed to mastitis by respondents are outlined in Table 1. The scores are remarkably similar; in all the data sets the median score attributed to a case of mastitis with clots in milk only was 3, the median score attributed to a case of acute toxic mastitis was 7 in the UK veterinary surgeon and European veterinary surgeon data sets and 8 in the UK cattle farmer and New Zealand veterinary surgeon data sets.

Table 1 Median Pain Score Attributed to Two Grades of Mastitis by Veterinary Surgeons and Cattle Farmers

Country and type of respondent	Mastitis (Clots in milk only)	Acute Toxic Mastitis
UK Veterinary Surgeons	3	7
UK Cattle Farmers	3	8
European Veterinary Surgeons	3	7
New Zealand Veterinary Surgeons	3	8

DISCUSSION

There is now a weight of evidence in the literature, based on physiological and behavioural indicators, which suggests that mastitis is a painful condition. Recently, the subjective assessments of thousands of veterinary surgeons and cattle farmers from around the world suggests that the discomfort associated with the condition ranges from 3 to 7/8 on a ten point pain scale.

Currently, NSAIDs are the only analgesic products licensed in the UK which can be used clinically to control the pain associated with this condition. The actions of NSAIDs (anti-inflammatory, analgesic and anti-pyretic) are due to their ability to inhibit arachidonate cyclo-oxygenase (COX) enzymes which leads to a reduction in the synthesis of prostaglandins and thromboxanes. Prostaglandins and thromboxanes cause inflammation and pain. The NSAIDs, carprofen, flunixin, ketoprofen, meloxicam and tolfenamic acid are all licensed for use in cattle in the UK. Flunixin, ketoprofen, meloxicam and tolfenamic acid have a licensed indication for use in mastitis and therefore under the cascade are the first line choices.

The use of NSAIDs in cases of grade 3 mastitis e.g. sick cows and cases of acute toxic *E. coli* mastitis, are well established both experimentally and clinically. In these cases they are principally used for their anti-

inflammatory and anti-pyretic properties. Their use should be routine in all severe cases, not only for these properties but also to control the pain associated with this unpleasant and painful condition.

The use of NSAIDs in cases of grade 1 and grade 2 mastitis is much less clearly defined. However, recent work has suggested that meloxicam can influence the pain modulation caused by this level of mastitis and that a single dose is just as affective as three doses administered daily. Other experimental evidence is currently lacking; however, the existing data on pain now makes the use of NSAIDs in mild and moderate cases a logical treatment option. Whilst undoubtedly the cost associated with NSAID treatment for analgesia alone may well be a barrier in commercial situations, the additional benefits they offers in these cases should also be considered because they will reduce inflammation and promote rapid recovery of the gland. Consequently, the use of NSAID should be considered and encouraged in all cases of mastitis, regardless of their severity.

CONCLUSIONS

Those of us working in the dairy industry should be doing more firstly to reduce the number of cases of mastitis that occur and secondly to control the pain caused when cases do arise. Currently, NSAIDs are the only suitable products licensed within the UK; their usage for this indication should be encouraged.

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IS THERE AN OPTIMUM CUBICLE SYSTEM FOR MASTITIS CONTROL?

Martin Yeates

Kingshay, Bridge Farm, West Bradley, Glastonbury, Somerset, BA6 8LU.
01458 851555. martin.yeates@kingshay.co.uk.

SUMMARY

Cubicle systems can be excellent for mastitis control, but in practice very often result in dirty cows, dirty beds and high mastitis challenges. This can be the case for old sheds and new alike.

Better matching of cubicles to cow size, improving ventilation and attention to detail with management of cubicles will all reduce mastitis challenge quickly. Assessing cubicles using a holistic approach, looking at ventilation, lameness, cubicles, slurry management etc. is essential. Very often it is the basics that are wrong and a fresh pair of eyes is needed to spot these problems and suggest practical and cheap solutions.

INTRODUCTION

A good dairy cow housing system needs to provide excellent cow comfort, a low mastitis challenge, minimal interruption to natural cow behaviour and movement and do all this without it costing a fortune to install or manage day to day. Whilst cubicles have their drawbacks, if properly set up they can achieve all of the above requirements. In practice many cubicle systems are far from satisfactory.

If the cows cannot fit into the cubicles, then as comfortable as they are, they will not be well used. In practice this is a major problem on farm. Some of this problem is a result of modern bigger cows in older sheds, but ironically much of it is due to poorly set up modern sheds. Very often many of these basic problems can be resolved with little more than a spanner or at the most some angle grinding and welding.

This paper will help identify good practice, summarise the findings from Kingshay's Cubicle Demonstration Unit on setting up the cubicles to fit a specific herd, and show how outdated buildings can be improved to reduce mastitis risk without this being prohibitively expensive.

ASSESSING THE CURRENT SYSTEM

Let the cows tell you their problems! The first thing to do is to watch the cows closely during a quiet time, ideally when they move back to the cubicles after milking and then feeding. What are the cows doing? Do they

show signs of injury from the cubicle system? Do you see healthy cows being able to stand squarely with all four feet in the cubicle before the lying down within a short time? If not, it is worth asking yourself some pointed questions. Do you see the following?

1. **Resting cows are standing up.** If more than 10% of your resting cows are standing up then it's a good indicator that the beds are uncomfortable or the cubicles are too small. The more time cows spend standing the less rest they are getting and the higher the risk of lameness. Increasing lying time has been associated with an extra 1 litre of milk for each hour of lying time (2).

2. **Damaged hocks and knees.** If more than 10% of your herd has damaged knees or hocks then an abrasive or uncomfortable lying surface is damaging your cows. This can be exacerbated by a poorly ventilated shed, resulting in damp beds and soft skin.

3. **Injured cows.** Are the cows hitting into the cubicles as they stand up or lie down. Where cubicles are incorrectly positioned and cows find it difficult to stand up or lie down then they will be reluctant to do so, resulting in increased standing times.

4. **Dirty cubicles.** All your cubicles need not be spotless – if they are, then even your heifers find them too small. Ideally, about 5% of cubicles should have muck on the back of them.

Of those cows that are in the cubicles it is important to understand what their behaviour tells you about how well the cubicles are set up for them. To do this you need to understand natural cow standing up and lying down behaviour. Cows stand up by lunging their head forward and using this as a counter weight to lift their hindquarters. They then step forward with one front foot and rise to a standing position. Compare the way your cows stand up and lie down out in the field with the movement of cubicle housed cows.

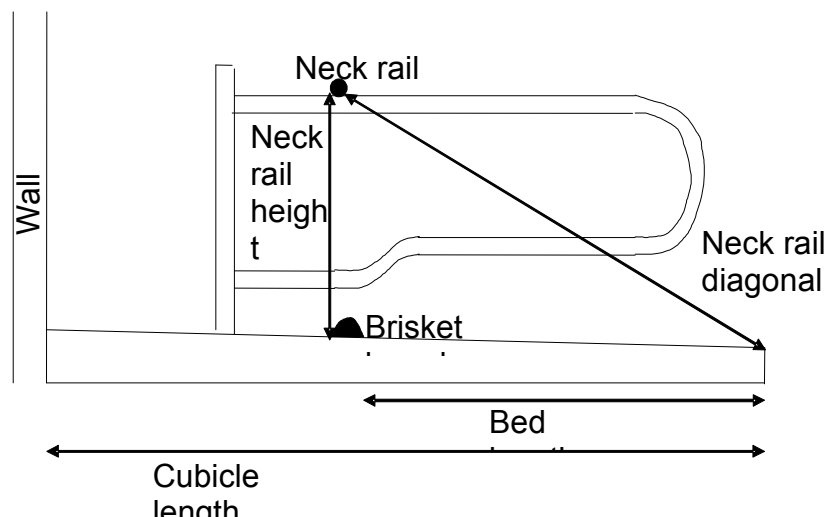
Table 1 gives details of behaviours often seen when cubicles are suboptimal. It also describes the likely causes of these problems and lists the negative effects caused by them.

CALCULATING THE IDEAL SIZE OF CUBICLES FOR A PARTICULAR HERD

If your assessment of cow behaviour points to problems with your cubicles, it's likely that cubicle size is an issue. It's therefore helpful to calculate the ideal size of cubicles to suit your herd. In order to do this Kingshay has used its Cow Housing Demonstration Unit to develop the work from Neil Anderson (1), who produced a matrix so that the ideal cubicle size could be calculated, based on the rump height and hook bone width of the larger cows in a herd. Table 2 gives this matrix and includes guide cubicle dimensions for a typical Holstein Friesian. Figure 1 shows how these should

be applied to a cubicle set up. The same sizes should also be applied to other designs of cubicles.

Figure 1: Key dimensions to check on any cubicle system



ADJUSTING CUBICLES TO FIT YOUR COWS

Having established your target cubicle dimensions, you need to adjust them to suit your cows. Luckily it isn't always necessary to spend a lot of money on these adjustments. Very often you are limited more by lack of imagination than by lack of finance! Whether your cubicles were installed last year or 25 years ago, most cubicle problems fall into one of five categories:

1. **Neck Rail:** This positions the cow in the cubicle whilst she is standing, ensuring she can stand with all four feet in the cubicle and mucks into the passage way. Often these are missing or improperly located.

The main problems seen on farm are modern cubicles installed with only a 2m (6'6") diagonal between the neck rail and the kerb. This needs to be 2.15m (7') for most Holstein Friesian herds. This seems a relatively small difference but makes a huge difference to the cow. Usually you can loosen the bolts on modern cubicles to easily make this adjustment.

With older cubicles, you can often raise the neck rail height by the construction of brackets. With more modern cubicles the rail can be moved forwards or backward to the ideal location.

2. **Brisket board:** This helps to locate cows correctly in the cubicles when lying down and limits mucking on the beds. Often these are incorrectly located, or are missing. If the brisket board is taller than 10cm (4") it can limit the ability of cows to extend a front foot, which forms a natural part of standing and lying.

Brisket boards should be 10 - 12.5cm (4-5") high with a smooth round top. Specifically designed products can be purchased or you can improvise using pipes or timber fixed to the cubicle bed

3. Insufficient or blocked lunging space: Ensure your cows have enough forward lunging space. Don't forget that a Holstein Friesian at grass will use 3m of space including its lunging space, yet many cubicles are little over 2.15m in length.

Extra lunging space can often be made by knocking out walls or removing bars that run through the lunging area (10-75 cm from the ground). If increasing lunging space is impossible, then provide side lunging instead. Make sure this is not blocked by the side rails of the cubicles. These need to be less than 30cm from the ground, and open between this and 100cm from the ground.

Older cubicles with back legs and bottom rails can be improved by moving the bottom rail so that it runs diagonally from the ground at the front and gets higher towards the back of the cubicle. This will allow the cow to lunge sideways into the next cubicle.

4. Excessively wide cubicles: This is generally less of a problem than it seems, especially when neck rails are in the right place and extra lunging space can be made available. Remember a cow that can stand square in the cubicle and can lunge forwards will generally lie down straight.

If necessary, the width of rail mounted modern cubicles can be easily adjusted. Older cubicles can be trickier, but low cost solutions are available e.g. try hanging pairs of tyres wired together over the cubicles. This works well where you need to make them much narrower so they can be used by heifers.

5. Uncomfortable beds: Use the tried & tested method to see how comfortable your cows' beds are: drop to your knees on the beds! If the beds are hard and/or abrasive then consider your options. More bedding may reduce the abrasiveness (see below for pros and cons of each type) and, at least in the short-term, is cheaper than investing in either mattresses or sand, though these will be a better bet in the long-term.

OTHER SETUP ISSUES TO CONSIDER

Cubicle slope

Cows naturally lie slightly up hill. Creating cubicles with a slope of 7.5cm (3") achieves this, helps drainage and reduces cows shuffling forward.

Kerb height

A kerb height of approximately 17.5cm (7"), including the height of any mat or mattress, is ideal, although this can be between 20cm (8") and 15cm (6") depending on the expected slurry depth and scraping type.

Number of cubicles

Farm assurance schemes will generally require 5–10% more cubicles than the number of cows. Check that you are not overstocking sheds by providing some cubicles that cows never use as they are broken, flooded by leaking gutters or have very uncomfortable surfaces.

Passageways

Keeping cubicles clean when passageways are dirty is near impossible. When a cow lies down, one foot generally touches the udder and so teats can only be as clean as the feet. Dirty feet lead to dirty cubicles and dirty udders. Tightly stocked sheds, higher yielding cows and ineffective scraping all lead to a build up of slurry.

Targets:

- Minimum of 7.5m² (80 sq feet) per cow, ideally 8m² (86 sq feet) per cow, including: loafing area, cubicles, lunging space. Excluding feed troughs.
- 3m (10') passageway widths between banks of cubicles is ideal.
- 4.5m (15') passageways to a feed face allows cows to move freely behind feeding cows.

After conventional scraping check if a good enough job is being done by using a hand scraper to run over a 1m square area. If you find there is more than the equivalent to a dung pat then your scraping needs improving

Ventilation

Good ventilation is key to keeping cows healthy during the winter. Poor ventilation leads to damp cubicle beds, stress to the cow and in turn an elevated risk of mastitis.

Targets:

- Aim for 7cm of ridge opening for every 3m of shed width, with double this for inlet area.
- Open or elevated ridges are far better than ones that are enclosed.
- Test the ventilation by setting off a smoke bomb, lighting straw in a metal wheelbarrow or a bucket. Make sure you do this with the cows in the shed. Watch to see where the smoke goes. Does it clear quickly or circle back onto the cows?

IMPROVING COW COMFORT

Cubicle housing must provide accommodation that is comfortable to lie on and will not cause injury to the cows. Cows given access to good cubicles spend less time standing in slurry and have fewer feet problems. This can be provided by mats, mattresses or deep bed systems.

Mats

Better quality mats can provide good cushioning, but many are little more than hard rubber surfaces with little give in them. Depending on the type, they may stretch after installation and curl up at the edges, making cleaning more problematic.

Mattresses

The top cover is as important as the mattresses material inside. The surface needs to be non-slip, yet not abrasive to the skin. Smooth surfaces may be easier to clean but retention of bedding can be poorer. Select a top cover which has an internal woven mesh to stop it stretching and some surface texture to help hold bedding.

Deep bed systems

Deep bed sand, straw and paper systems can all be made to work well, although deep bed sand is the main type being installed.

Deep bed sand cubicles can be installed from scratch or made by digging out the beds within existing cubicles. Cubicles can either be filled purely with sand to a depth of 15cm (6") or tyres can be placed in the base.

Tyres can stop cows digging out the beds. They must be placed 7.5cm (3") below the curb, thoroughly hand packed with sand and tied together, otherwise they will work their way up through the bed. Make sure the sand is kept well topped up otherwise the bed will be very uncomfortable.

Kerb stone shape is critical with deep bed systems. Movement of the sand bedding inevitably exposes the inside of the kerb, so it must be comfortable for cows to lie on or they will lie within the bed and muck onto the kerb. Ensuring that the inside of the kerb is rounded and smooth will limit hock damage.

Shallow bed systems

These can be made by increasing the kerb stone height of existing cubicles and filling with 5cm (2") of sand. Keeping a good covering of sand is essential if abrasive damage is to be avoided; therefore, top up frequently.

- Any liquid entering the beds cannot drain away so be very sure to remove any damp sand.

BEDDING TYPES IN PRACTICE

All traditional bedding materials (straw, shavings, sawdust and paper) are reservoirs for environmental mastitis pathogens (*E coli* and *S Uberis*) and exposure can only be reduced not eliminated by good management. Sand can be better in this regard, if it is kept clean. Each bedding type has other pros and cons (see Table 3), beyond its affect on mastitis. All can be made to work well if the shed is properly set up (well ventilated) and is managed with a high degree of attention to detail.

Straw

For maximum absorbency and minimum straw usage it is important to chop straw for cubicles as short as possible. Remove soiled material from the beds at least once a day and bed up regularly to limit mastitis challenge. Use only good quality dry straw (15% moisture or less) and protect staff from the dust if chopped and blown into the beds.

Sawdust and shavings

Sawdust and shavings, like straw, are carbon based so bacteria and bugs can multiply on them, especially when moist.

- Wood products must be dry and clean. Kiln dried products are better but more expensive.
- Shavings achieve lower pathogen levels than powder but are more expensive and don't stick on the beds as well.
- Remove wet and soiled bedding before adding fresh, otherwise it is just adding fuel to the fire.

Paper

Recycled paper offers another option for cubicle bedding which can be used as a direct alternative to sawdust, shavings or chopped straw.

- Store in the dry or sheet over well.
- Remove all damp material from the cubicles regularly.
- Paper can clump together and stick to the cubicle bed, forming lumps and hollows.
- Highly absorbent, keeps the cows clean if well managed.

Sand

Sand is an inorganic material and, if kept clean, does not support bacterial growth. This means that it can present a lower mastitis challenge compared to other bedding options. The main downside of sand is handling weighty bedding and sand laden slurry.

Sand needs to be non abrasive. Sea or estuary sand is excellent, as is washed quarry sand. Ensure that the sand is free from contamination.

- Stones are abrasive and uncomfortable.
- Sand containing clay will tend to pack down and form a hard abrasive surface.
- Organic material will provide a breeding ground for bacteria.
- Sea sand will have some liming effect on your land.

Cows will have sand on their teats as they enter the parlour. Thorough teat cleaning is essential before clusters are attached, as any sand getting into the milking systems can rapidly wear out milk pumps.

- Check your filters and regularly flush out any sand settling in the milk lines.
- Barrier teat dips used in combination with sand bedding can be problematic because sand sticks to the teats.
- Sand blocks up parlour drains; remember to install (and clean out!) sand traps.

Slurry and sand can make a difficult combination.

- Sand will often settle out of the slurry and form a layer in the base of your slurry pit
- If you have slats, slurry channels or a pit that cannot easily be dug out then sand is very unlikely to be suitable
- Pumping sand filled slurry will cause wear to equipment and contractors may be unhappy about pumping slurry containing sand
- Umbilical systems cannot pump slurry with a high proportion of sand
- Sand may settle out in slurry tankers, requiring flushing to remove it

CONCLUSIONS

No one cubicle system will solve your mastitis problems, but many systems provide such a high challenge to the cows that it is very difficult to out-manage the problems and keep mastitis low and cow health high. Observing your cows' behaviour and modifying the cubicles to suit them can often be achieved at a very low cost and have dramatic consequences. Improving ventilation is often not difficult and will dry out sheds and reduce mastitis risk. Changing bedding type is an option, but will not solve mastitis problems if they are predominantly caused by shed set up or lack of attention to detail.

Often these problem are best identified by use of an independent expert in reading cows, rather than by the farmer themselves who is too used to looking at his or her herd.

Small changes to your cubicle and shed setup will result in major changes to cubicle usage, cow health and mastitis.

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Table 1: Interpreting cow behaviour in cubicles

	Cow Behaviour	Likely Cause	Negative Effect
Incorrect Standing	Perching: Cow stands with her front feet in the cubicle and her back feet in the passageway	Neck rail incorrectly located – too near the kerb and/or too low Cows reluctant to lie down on very uncomfortable bed	Increased standing time puts excessive pressure on the back feet, leading to lameness and stress on the cow
	Diagonal Standing: Cow stands diagonally across cubicle with all four feet in it	Lack of lunging space Neck rail incorrectly located – too near the kerb and/or too low Excessively wide cubicles	Muck is seen in the back corners of the bed Cows that stand diagonally tend to lie diagonally
	Standing too far forward in the cubicle	Neck rail positioned too far forward	Muck seen in the middle at the back of the bed
Incorrect Lying	Cows lying diagonally in cubicles	Lack of lunging space Lunging blocked by rails across the front of the cubicles Excessively wide cubicles	Muck is deposited in the back corners of the beds
	Cows lying too far forward in the cubicle	Brisket board incorrectly located or missing	Muck seen in the middle at the back of the bed
	Lying backwards in the cubicle	Wide or broken cubicles	Muck seen in the cubicles and risk of cows getting stuck

Table 2: the relationship between cow size and cubicle dimensions

Cubicle dimension	Dimension calculation	Rump height	Hook width	Ideal cubicle dimensions
Cubicle length (solid front)	1.9 x rump height	1.45m		1.9 x 1.45 = 2.75m (9')
Cubicle length (head to head)	1.65 x rump height	1.45m		1.65 x 1.45 = 2.40m (8')
Bed length	1.16 x rump height	1.45m		1.16 x 1.45 = 1.68m (5'6")
Height of neck rail	0.87 x rump height	1.45m		0.87 x 1.45 = 1.26m (50")
Neck rail diagonal	1.48 x rump height	1.45m		1.48 x 1.45 = 2.15m (7')
Cubicle width	1.9 x hook width		0.6m	1.9 x 0.6 = 1.14m (45")

Adapted from Anderson, 2007

Table 3: Pros and cons of different bedding materials

	Typical usage and cost per cow during 200 day Winter	Pros	Cons
Straw	0.5 – 0.8t £23 - £36	Provides some long fibre for the cows to pick at Good quality straw typically contains fewer mastitis bugs than sawdust and shavings	An ideal medium for mastitis pathogens when over 20% moisture Large DRY storage area required Price varies hugely across the country Dust levels can be very high if you are chopping the straw
Sawdust/ Shavings	0.15 – 0.2t £28 - £37	Easy to handle using a trolley and shovel or mechanised dispenser	Can be variable in quality and availability Can stick to the cows’ teats and takes time to remove at milking Provides a good growth medium for mastitis bugs if not kept dry The finer the sawdust the higher the pathogen loading Can be dusty to spread
Paper	0.2 – 0.4t £14 – £28	Very absorbent Good for mastitis control	Can develop hard lumps in the beds if not well managed Thickens the slurry Can be dusty to spread
Sand	1.5 - 2.0t £27 - 36	Comfortable surface Excellent for mastitis control Bedding frequency can be reduced Sand can add grip to polished concrete – good for bulling, but will add to the polishing effect	Slurry pumps and channels don’t like it Heavy to handle Cows can get caught under cubicle bars if the beds are not topped up Hock damage can be a problem due to contact with kerbs in poorly topped up beds

Based on Straw @ £45/t, Shavings @ 185/t, Envirobed @ £65/t, Sand @ £18/t

MILKING MACHINE INFLUENCES ON MASTITIS

Norm Schuring

GEA WestfaliaSurge Inc, Illinois, USA

SUMMARY

This paper will primarily focus on the how the milking machine can influence or impact mastitis that is found on the modern dairy farm. It is important however, to note it is well established through proven research and extensive field experience that udder infections are normally caused by multi faceted events and rarely can mastitis be viewed as occurring by one single event. As a result Mastitis Management includes the industry's recommended "Six point mastitis control program" as defined below:

- Proper milking hygiene
- Use of functionally adequate milking machines in the correct manner
- Dipping teats
- Treat all cows at the beginning of the dry period
- Treat all clinical cases promptly
- Cull chronically infected cows from the herd

All of these points are important to Mastitis Management and have proven to be an essential element in mastitis control. This paper will focus on the second point of Mastitis Management; "The use of functionally adequate milking machines in the correct manner".

INTRODUCTION

Research and field experience have proven the milking machine has little effect on the new infection rate if properly operated and functioning in accordance to manufacturer's and industry's specifications. Graeme Mein *et al.* in the paper "Milking Machines and Mastitis Risk: A storm in a teat cup", states the influence of the milking machine to new intramammary infections is less than 20 to 25%. Other papers suggest the milking equipment will only influence the rate of new infections at between 6 to 15%.

The milk harvest process should have goals of being a pleasurable experience for the cow, maintaining teat integrity and protecting the production of the highest milk quality possible. The milking machine plays a major role in accomplishing these goals when they are used in the correct manner, but if improper operator techniques are allowed during milking time, the expected accomplishments of these goals will normally fall short of expectations.

Research and field experience reveal that herds with high somatic cell counts and higher than normal incidences of clinical mastitis do not

practice recommended milking procedures. The most common problem in these herds is a failure to properly clean, sanitize and dry teats (which positively influences milk letdown) prior to attaching the milking units. Failure to do this exposes the milking equipment to high colonies of microorganisms, reduces peak milk flow, increases milking time and reduces milk quality. It has been proven, proper milking procedures will reduce the risks of new infections and increases the potential of milk harvested.

The paper written by Kiro Petrovski and Mel T Eden, March 2006, "The role of the milking machine in the aetiology and epidemiology of bovine mastitis" states: Although not a direct prerequisite for mastitis, the milking machine has significant effects upon the aetiology and epidemiology of mastitis. These effects may operate directly by increasing the new intramammary infection rate, or indirectly by increasing exposure to mastitis causing organisms and/or by reducing disease resistance in the animal. There are four major identifiable ways in which milking machines can influence the development and severity of mastitis:

- Physical transport of mastitis-causing organisms between quarters and cows
- Causing damage to the teat end
- Increasing the risk of mastitis-causing organisms penetrating the teat canal
- Increasing colonization of the teat canal with mastitis-causing organisms

There are approximately 140 known organisms that cause mastitis infections. These organisms either live in the cow, on the cow or in her environment. The four categories of these microorganisms can be classified into:

- Contagious
- Environmental
- Opportunistic
- Other

The following facts and theories will be discussed on how the milking machine influences mastitis and how the operating parameters may also influence the risks of new infections.

- Milking management
- Vacuum levels and fluctuation
- Pulsation
- Milking clusters and liners
- Detachers and milk flow rates

MILKING MANAGEMENT

Environmental pathogens are found on the teat skin prior to milking. These organisms may also contaminate the milking machine and increase the risk of infecting the udder during milking and lower milk quality. It is important to reduce these environmental pathogens on the teat prior to milking. It is a known fact that milking equipment can aid in the transfer of pathogens at milking time. Care should be taken to make sure the teat is clean and sanitized to reduce the potential spread of pathogens from cow to cow or teat to teat through the milking claw and/or liner at milking time. This situation is amplified when the hours per milking session increases to greater than three hours of milking.

Post milking procedures should include sanitizing the teats after milking to kill the bacteria that may have been placed on the skin surface at milking time. Operator hands, liners and milk from the udder are all contributing factors that contaminate the teat skin. Post milking teat sanitizing (teat dipping) should kill these bacteria to prevent colonization at the teat end. The power of the teat sanitizer should also help minimize the growth of the bacteria at the end of milking especially during the first two hours when the teat canal is open and more susceptible to bacterial invasion.

Milking cluster alignment to the udder is an important step with the milking machine influence on mastitis. Later in this presentation we will make reference to this important step and describe why it is so important.

VACUUM LEVELS AND FLUCTUATIONS

The basic principle of the milking machine is to use vacuum to extract milk from the cow. Industry recommends a milking vacuum level at the teat end of 32 to 42 kPa for safe and efficient removal of milk from the teat. One must recognize higher vacuum levels will normally produce higher milk flow rates but may also compromise teat health by allowing more tissue trauma and may also be offset by producing higher strip yields. Lower vacuum levels are normally associated with less tissue trauma but the speed of milking is normally decreased which makes this a less attractive option for many dairy producers. It is safe to say many of the farmers in North America have chosen to milk at higher vacuums while many of the European farmers have elected to use a lower vacuum.

It would be easy to suggest one milking vacuum level across the globe if all things remained equal but the fact that we have different clusters, liners; a variety of cows and various operator routines and procedures makes one recommended vacuum level impossible even within one community or country. We may want to divide our approach into two categories of (1) Aggressive Milking and (2) Non-Aggressive Milking.

Aggressive Milking (higher vacuum) starts with the knowledge that we incur higher risks of increased tissue trauma and other less desirable conditions

that may be associated with a greater incidence of mastitis. Considerations include:

- Weight of the milking cluster
 - Higher vacuums should use heavier milking clusters and lower vacuum levels should use lighter milking clusters
- Udder Preparation
 - Higher vacuum levels required excellent pre milk stimulation to assure a constant milk flow at the time of attachment. Tissue damage is associated with low milk flow especially at higher vacuum levels. During low flow, the pressure inside the teat decreases to a level that vacuum is created inside the teat and therefore increases the risk of bacterial invasion during this time.
 - Low milk flow combined with higher vacuum levels challenge alignment of the cluster to the udder and normally allows the teat to penetrate deeper into the liner resulting in higher strip yields. *One may consider this happens only at the end of milking but many times is associated more at the beginning of milking rather than the end of milking.*
- Liners
 - Liner design should be considered when considering vacuum level. Liners are the component of the milking machine providing teat massage during the rest phase. The vacuum level influences the closing position of the liner. The goal is to assure the liner massages the blood and fluids out of the teat to relieve congestion while still maintaining the integrity of the teat and teat tissue. Liners with a low collapse specification should not be used in high vacuum systems.
 - Liners having high collapse characteristics should not be used with low vacuum systems. The liner must have the ability to close around the teat end to massage the congestion away from the teat end. Liners not massaging the teat end lead to slower closure of the teat canal increasing the potential of new infections.

The relationship of vacuum fluctuations to new mastitis infections remains difficult to prove. This theory of milk droplets to new mastitis infections remains difficult to prove since bacterial penetration through the teat orifice will most likely be influenced during the time it occurs. The risk is noted to be much higher at the end of milking as compared to peak flow conditions. Common belief remains that the action of the liner causes a cyclic fluctuation in the milking claw and cows may become infected when contaminated milk droplets strikes the end of the teat with sufficient force to carry this droplet into or through the teat canal. However, it has been learned that these cyclic fluctuations alone do not have a large potential risk of intramammary infections unless they are combined with large transient vacuum fluctuations.

Common belief as stated in Graeme Mein's *et al.* paper "Milking Machines and Mastitis Risk: A storm in a teat cup" states machine induced infections are more likely to result with large transient vacuum fluctuations occurring in the liner during low milk flow periods. Liner slips, vigorous machine stripping and rough or abrupt cluster removal are normally considered as

potential causes of large transient fluctuations. Other factors such as poor cluster alignment, improperly positioned long milk tubes and over milking are risks causing these large transient vacuum fluctuations.

Research collectively agrees pathogen concentration in or near the environment of the teat orifice has a dominant influence on the rate of infections. However, the effects of the milking machine functions appear to be low as compared to milking procedures and herd management. This concentration of pathogens will influence the rate of new intramammary infections especially if the condition is compounded with poor teat health such as cracking or dry skin, hyperkeratosis or lesions.

PULSATION

Pulsation acts to massage the teats, promote blood circulation and relieve the oedema and teat end congestion caused by exposure to vacuum. Faulty pulsation is a major risk to teat end damage especially if the pulsators are not cleaned and maintained on a regular basis.

Effective pulsation is the combination of vacuum levels, liner and shell characteristics, together with teat size, length and shape. Vacuum and/or atmospheric air in the pulsation chamber combined with the vacuum level inside the liner causes the liner to be open (milking) and closed (massage) during the milking process. The industry guidelines suggest a liner open time of greater than 500 milliseconds and a full liner closed time of approximately 200 milliseconds. Effective pulsation is achieved when the action of the components provide an adequate milk/massage time on the teat with proper milk flows and minimal tissue changes normally evident at the end of milking. Research and field experience suggests improper massage times are regarded as harmful or undesirable and failure of pulsation has direct effects on the health of the teat end and the skin of the teat.

Typical pulsation ratios can be used with a safety range from 50% to 65%. The higher number may milk quicker but require monitoring to avoid poor massage time and associated increased teat congestion. These wider ratios (milk compared to massage time) have higher risks of causing irritation of the teat tissue especially when service or cleaning of the pulsation ports are not completed on a regular basis. This condition may cause skin irritation and increase the potential of bacterial growth on the skin and encourage the penetration of microorganisms into or through the teat orifice.

Milking machines perform well with pulsation rates operated between 55 to 65 pulses per minute if the remainder of the milking system is operating properly and serviced on a regular basis. Excessive rates may not provide adequate massage times especially when liner selection is considered as part of the equation.

Early signs of pulsation failure can be observed on many occasions at the end of milking by observing the teats for swelling or discoloration of the skin and cows either kicking or stepping. Since many components of the milking system are mechanical with parts that wear over time it is important to establish benchmarks for teat skin condition especially at the end of milking.

The science of matching the liner, vacuum and pulsation continues to improve and should be noted when planning pulsation and its desired performance. Simply guessing and changing of components without considering the interaction of all components with teat size and shape normally causes goals and objectives to be missed and unfortunately the rate of intramammary infections to increase.

MILKING CLUSTERS AND LINERS

Milking clusters are the components attached to the cow's udder and are responsible for collecting the milk from all four teats and allowing it to flow through the long milk tube to the milk transfer lines. This process sounds simple but several factors may influence the performance of this task and should be considered with every milking operation.

The milking claw should be designed to apply equal weight to all four teats when properly attached and aligned to the cow's udder. This balance improves the liner wall and teat wall seal which helps prevent liners from slipping up or down on the teat causing improper milk-out or irregular vacuum fluctuations. Factors such as poor cluster adjustment, milking vacuum, liner selection, improperly positioned long milk tubes or uneven shaped udders must be considered when evaluating milking cluster performance.

When the milking cluster is attached to the cow; the liner wall should seal against the teat wall. This seal maintains the position of the liner on the teat and applies a vacuum at the teat end to extract the milk from the teat. When these proper conditions exist the milk is rapidly removed by the vacuum or reduced pressure below the teat and cow comfort is accomplished. Additional weight or tension on this seal will normally result in high vacuum in the mouthpiece area of the liner, deeper teat penetration in the liner, and liner slips that may influence milk droplet impacts.

Vacuum in the mouthpiece causes congestion and discomfort to the cow. This swelling will reduce milk flow and may close the annular fold preventing milk flow from the udder cistern to the teat cistern. This improper liner position is normally noted by a noticeable ring at the teat base immediately after removal of the milking cluster. These rings noticed on one or two teats can indicate poor unit alignment and result in improper milking.

The milking liner provides the accumulative action of the entire system. If the liner does not perform properly on the teat the entire system may be at risk of an incomplete milk harvest. The opposite is also true. If the liner is designed and matched to the system's components the successful operation of the milk harvest process is highly likely to occur. The design of the liner should consider the following points.

- Liner closure and the massaging action applied to the teat
- Liner wall seal against the teat
- Reduced liner slip
- Maintain contact to teat wall to prevent high mouthpiece vacuum
- Provide adequate tension for compression against the teat end
- Maintain clear path for milk flow for fast and efficient milking

Research studies suggest new infections rates do increase with liner slips but the effects of liner slips on large scale milking herds has never been established. Field experiments on devices placed in the liner to prevent milk droplets impacting on the teat end did indicate an overall reduction in new infections of approximately 10%.

In one research project liner slips were recorded whenever a vacuum drop of 10kPa or more occurred within a time of 250 milliseconds or less. The high slip averaged 7.6 major slips per cow milking, compared with 3.1 for the low slip average. New infection rates were 0.49 per 100 cow days for high slip compared with 0.27 for low slip liner. Interestingly, this works out to about one new infection per 2500 liner slips for both high and low liner slips. Not surprisingly, the new infection rate was higher in cows with one or more quarters already infected (1500 – 1850 slips per new infections) compared with previously uninfected cows (over 6000 slips per new infection).

An additional driving force required to force milk droplets into a teat canal can be generated by sudden air leakage past one or more teats as a result of liner slips. The common irregular vacuum fluctuations normally associated with poor vacuum pump performance, poor vacuum regulation and flooded milk lines do not produce changes at the teat end vacuum to provide speeds great enough to impact the teat and penetrate the teat canal. However, when these irregular vacuum fluctuations occur, the alignment of the milking cluster may be influenced and altered, causing liner slips.

A study at milking time is valuable to find liners suited to a particular herd by examining the teat ends at the end of milking. It should also provide an opportunity to monitor operator procedures and equipment performance for other operational factors that may contribute to new intramammary infections.

Unsuitable liners or those not matched to the tea cup may also cause damage to the teat. In the same way liners which are allowed to lose their resilience and shape through over-use are a serious risk to herd health.

Selection of the liners used on dairy farms requires careful thought and consideration for the operating parameters of the entire milking system.

DETACHERS AND MILK FLOW RATES

The aim of good milking routines is to maximize the amount of quality milk removed from the udder at each milking while maintaining the integrity of the teat. Improper milk harvest has been demonstrated to increase the risk of new intramammary infections and the severity of this costly disease in affected quarters. The large number of bacteria and toxins left in the udder after milking impair defence mechanisms leading to the exacerbation of mammary infections. This is one of the reasons increased frequency of milkings per day has shown benefits as it relates to udder health. The most common causes of poor milking out are poor selection or care of liners, improper alignment of the milking cluster, poor location of the long milk tube, very low or very high vacuum level, improper detacher settings and/or poor handling of cows especially at milking time.

Correcting factors that cause under milking will usually result in a reduction in the bulk milk somatic cell count and in clinical mastitis in problematic herds.

Another critical area of concern is overmilking. Field experience and years of history clearly does not support the theory of over milking resulting in higher somatic cell counts or increased intramammary infections. This is supported by the fact that front quarters are normally over milked more than rear quarters but rear quarters normally have higher incidences of new infections.

Overmilking can influence the teat condition which in turn may lead to an increased risk on new intramammary infections. Factors included in this theory are:

- Overmilking causes the teat to collapse due to the fact that internal pressure is decreased, resulting in a vacuum inside the teat cistern. This vacuum allows the teat wall to respond to the opening and closing of the liner by rubbing the tissue together each time the liner closes for massage. This action can irritate the inner teat walls, rupture the blood vessels and/or cause roughness which increases the risks of harbouring microorganisms leading to bacterial growth. This may result in new infections.
- During over milking the teat end is normally stressed when the liner is closing. The radius of the liner wall closing on the teat end is normally altered at this time. This change can apply additional stress showing a higher level of damage to the epithelial lining and removal of more keratin from the teat canal. Over time this continuous action on the team will cause a condition known as hyperkeratosis. Keratin lines the teat canal and provides protection against bacterial penetration into the teat sinus. During

normal milking approximately 40% of the keratin is removed. This process is healthy for the cow because it cleanses the teat orifice and removes some of the bacteria that are trapped within the orifice between milking. If less keratin is removed surviving bacteria may have better opportunities to grow and multiply causing these bacteria to result in new infections. If conditions allow more keratin removal between milking, the orifice may not close and environmental pathogens may enter the teat canal between milking. Excessive keratin removal occurs during low milk flow periods and will be observed by a build up around the orifice that is many times referred to as a donut.

- Modern milking machines have increased milk flow capacities over the years and with today's genetically improved cows one should expect higher milk flow rates. Excellent pre-milk procedures and proper prep-lag time allows peak milk flows to reach 5 to 6 litres per minute with average milk flows averaging 3 to 3.5 litres. Lower flow rates may be an indication of the milking conditions.
- Automatic detachers and end of milking sensing points must consider the overall herd milking conditions and cannot be averaged from farm to farm.

CONCLUSIONS

Milking Machine Influences on Mastitis is a complex subject and rarely can one designate an operating parameter as being the major cause of new intramammary infections. Mastitis normally results from an interaction of cow, machine and operator and the environment. To assure milk quality and herd health goals are met one must monitor the entire milking performance as well as the system's cleaning routine and determine if the operation is within the set and agreed upon operating parameters.

TEAT MANAGEMENT: LIFE OUTSIDE THE PARLOUR

Nigel B. Cook

University of Wisconsin-Madison, School of Veterinary Medicine, Madison, Wisconsin, USA

INTRODUCTION

Infection of the mammary gland with environmental bacterial pathogens is the most significant udder health problem facing the dairy industry in North America and many other parts of the world. Forty years ago, Neave et al. (9) stated that the rate of new intra-mammary infection is related to the number of bacteria that the teat end is exposed to, and several studies have made associations between clean housing, clean cows and lower bulk tank somatic cell counts (2, 3 and 5). In addition, Bartlett *et al.* (4) found that an index of environmental sanitation based on the amount of manure on the cow and in her environment was a predictor of the occurrence of coliform mastitis, and in a study of four herds, the lowest incidence of mastitis occurred in the herd with the cleanest cows and the most satisfactory beds (12).

Despite the improvements made in so many other areas of the dairy industry, our ability to keep cows clean and to reduce the bacterial load at the teat end has improved little. Increases in herd size, poor stall design, infrequent alley scraping and manure removal, pressure for milkers to increase parlour throughput, and changes in the availability and use of different bedding materials have all worked against significant progress in this area.

The predominant sources of coliforms and environmental streptococci (*S.uberis*, *S.dysgalactiae*, *Enterococcus spp.*) are manure and bedding materials. The cleaner we can keep the cows and the lower the bacterial count of the bedding, the fewer problems we will see.

TIME BUDGETS

Once the cow leaves the parlour – what does she do? From an analysis of 250 total 24-hour time budgets, we have collected from 208 cows housed in 17 freestall barns in Wisconsin, the average time spent performing each of five key behaviours is shown in Table 1. On average, cows spend 2.6 h/d milking – reflecting the three times a day milking schedule most large freestall dairies operate at. Other components of the cow's day are also fixed and non-negotiable. The cow has to spend a large proportion of the day eating. The TMR fed, free stall housed dairy cow eats for an average of 4.4 h/d (range 1.4-8.1). Note that this is about half the time that a grazing cow spends eating per day – pasture cows average around 8-9 h/d eating. She also needs to drink around 95 litres of water per day (more in hot climates) and she will spend an average of 0.4 h/d at or around a waterer. With these

fixed non-negotiable time slots, we have already taken $4.4 + 0.4 + 2.6 = 7.4$ hours out of the time budget, leaving under 17 hours remaining in the pen.

Table 1. The mean (range) 24-h time budgets for 208 cows filmed over 250 filming periods on 17 freestall barns in Wisconsin

Activity N=250	Mean (h/d)	Range (h/d)
Time lying down in the stall	11.3	2.8-17.6
Time standing in the stall	2.9	0.3-13.0
Time standing in the alley	2.4	0.2-9.4
Time drinking	0.4	0-2.0
Time feeding	4.4	1.4-8.1
Time milking	2.6	0.9-5.7

Time left in the pen will be spent performing three activities – lying down, standing in an alley and standing in a stall. The average freestall cow spends 2.4 h/d standing in an alley socializing, moving between the feed bunk and stalls and returning from the parlour. Once in the stall, the average cow spends 2.9 h/d standing in the stall (range 0.3-13.0) and 11.3 h/d lying in the stall (range 2.8-17.6) on average – but note the wide ranges in these behaviours. Lying behaviour is typically divided into an average of 7.2 visits to a stall each day (called a lying session), and each session is categorized by periods standing and lying – called bouts. The average cow has 13.6 lying bouts per day and the average duration of each bout is 1.2 h (range 0.3-2.9). Most cows will stand after a lying bout, defecate or urinate, and lie back down again on the contra-lateral side.

From this analysis of cow behaviour, it is clear that in order to limit the bacterial challenge at the teat end we must:

1. Limit the splashing of manure on the udder as the cows walk around alleyways.
2. Limit contamination of the stall bed when the cow rises in the stall and defecates.
3. Provide a comfortable stall so that we maximize lying time and time out of the alleyways.

MANURE TRANSFER TO THE TEAT END

Several different methods of hygiene scoring have been documented (6, 10 and 11) and some have been used to prove that poor hygiene results in udder health problems. Schreiner and Ruegg (11) used a 4-point udder hygiene scoring system to document the degree of contamination of 1250 cows in 8 herds. Udder hygiene scores averaged 22% score 3 and 4 and a significant association between poor udder hygiene and increasing individual cow linear score and the prevalence of intramammary infection with an environmental pathogen was reported. In fact, cows with udder

scores of 3 and 4 were 1.5 times more likely to be infected with a major pathogen than cows with scores of 1 or 2. The study reported only a weak association between leg hygiene score and the prevalence of pathogen isolation from the udder.

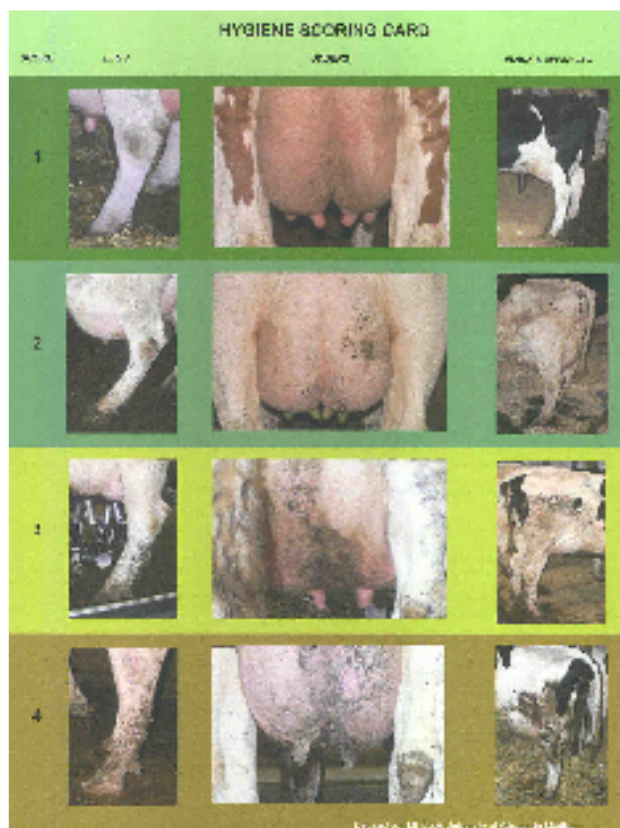
A more complex scoring system (10) was used to document hygiene in 1,093 cows in 8 herds and showed a significant association between udder and lower leg hygiene and individual cow linear score measured within 2 days of recording.

We have used the simplified multi-zone hygiene scoring system in Figure 1, scoring the udder, lower legs and upper leg and flank zones of cows on a 1-4 scale to communicate the reasons for manure contamination of the udder. There are four basic manure transfer mechanisms to the udder, and the relative importance of each differs with the type of housing under consideration:

1. **Direct Transfer.** Cows may lie down in a manure contaminated stall or bedded area (or sometimes in a traffic alley!) and transfer bacteria directly to the udder.
2. **Leg Transfer.** Cows may walk through manure, coating their feet and legs, which transfers bacteria to the teat ends and bedding when the cow lies down and the udder comes to rest (1).
3. **Splash Transfer.** Cows walking through deep liquid slurry will splash manure up toward the udder.
4. **Tail Transfer.** In some situations, the tail may become heavily contaminated with manure and transfer bacteria to the rear udder and flank areas (1).

The pattern of manure contamination and the mechanism of transfer therefore becomes a very important concept to communicate to the herd owner and we calculate the proportion of scores 3 and 4 in each zone, rather than a mean hygiene score. During investigations, we typically score 20% of the cows in each pen, or all of the cows in small herds.

Figure 1. A hygiene scoring card which documents the degree of manure contamination on a 1-4 scale for each of three zones, the udder, the lower leg and the upper leg and flank. The score sheet is available at <http://www.vetmed.wisc.edu/dms/fapm/fapmtools/4hygiene/hygiene.pdf>



The hygiene scoring data from 60 farms collected by the SVM food animal production medicine group suggests that on average, 16-23% of udders are score 3 and 4 and have an elevated risk of infection (Table 2). While tie stall cows generally have cleaner lower limbs and less leg transfer, direct transfer is the predominant means of manure contamination of the udder – from manure deposited on the stall surface. Upper leg and flank scores are usually much poorer than in free stalls, reflecting the risk associated with spending around 22 hours per day in a tie stall.

In contrast, the lower legs of free stall cows are far more contaminated than tie stall cows and leg transfer is a significant risk for udder contamination. Splash transfer in poorly draining alleys is also significant.

Table 2. Median and upper quartile proportion of hygiene scores 3 and 4 for each zone for cows in 60 Wisconsin dairy herds by housing type (46 Freestall and 14 Tiestall).

Housing Type	Proportion Hygiene Scores 3 and 4 (%)					
	Udder		Lower Leg		Upper Leg and Flank	
	Median	Top 25%	Median	Top 25%	Median	Top 25%
Freestall n=46	16	10	64	52	15	11
Tiestall n=14	23	13	22	13	26	19

There are multiple reasons for the state of leg hygiene that we see in freestall barns – these include: frequency of manure scraping and removal, stocking density, width of the alley, type of flooring, pen layout, length of the pen and slope of the alley. Unfortunately, there is very little that can be done to influence it once a facility has been built.

In the design phase, we can work towards keeping cleaner cows by recommending two-row pens instead of three-row pens – they have 20% more alley surface area per cow, putting a gentle slope (~1-2%) on the alley so that urine drains, building wider alleyways and creating deeper grooving patterns. Slatted floors may work for smaller farms, but I have yet to be convinced that they work for pens with more than 100 cows – they are simply too traumatic to the cow’s feet. Similarly automatic scrapers and flush systems that operate while the cows are in the pen have not yielded significantly improved leg hygiene overall.

A very common finding however, is for cows in sand bedded herds to be cleaner than in mattress herds bedded with sawdust (Table 3). This finding may be due to the cleaning effect of sand, differences in cow behaviour in barns with the two different types of bedding surface, and less slipping and splash transfer in sand bedded herds.

Table 3. Least squares mean (SE) hygiene scores (Proportion scoring 3 and 4 for each zone) obtained independently by two observers from a minimum of 20 cows in the high group pen on 12 free stall herds (6 sand and 6 mattress) compared using 1-way ANOVA.

Zone	Proportion Hygiene Scores 3 and 4 (%)		SE	P Value
	Sand	Mattress		
Udder	16.7	33.3	4.2	0.02
Lower leg	39.2	74.2	8.6	0.02
Upper leg and flank	1.7	11.7	2.1	0.01

LIMITING MANURE CONTAMINATION OF THE STALL BED

Cows defecate on the stall platform when they stand in the stall and when they are lying down. With cows entering a stall 7 times a day for 14 lying bouts, we must design the stall so that when she is lying, we position her so that her rear end lies over the back edge of the stall or the stall alley, and when she is standing, she defecates and urinates into the alley, not into the stall. This is a difficult challenge!

The cow is positioned in the stall (something we refer to as 'indexing') by several physical and social barriers. We tend to see more diagonal lying and more stall contamination when there are obstructions to the forward lunge and bob of the head, obstructions to the forward thrust of the front leg as the cow rises and when the resting area is of insufficient length.

Forward lunge obstructions may be physical – for example, the stall is simply too short to allow forward lunge, or a divider loop mounting rail is placed in the bob zone at the end of the lunge between 10 and 96 cm above the stall surface, or social – this occurs in stalls positioned in a head to head layout, where the platform is too short. Even though there may be no physical obstructions to front lunge, timid cows will not lunge into the face of a dominant cow when the space is occupied in front.

When cows rise in the stall, they prefer to extend their front leg forward to take weight, before raising the back end. To do this, there must not be an obstruction higher than about 4 inches above stall surface in the brisket area. Many poorly designed stalls have brisket locators that are higher than 4 inches and in these stalls the cow must modify how she rises – unfolding her front legs rather than launching one forward for stability, and some cows lie diagonally, so that their front legs have a little more space when they get up.

The negative consequences of the design flaws already mentioned have been tolerated in the industry because we have used restraint to limit the effects of diagonal lying. Stalls have been built that are too narrow and too short for the cows. While we may be able to keep these stalls clean, we have only recently realized that the reduction in lying time caused by providing insufficient space has cost us dearly in lameness and culling.

Frequently, farmers respond to stall contamination by altering the easiest thing to move in a stall – the neck rail. Neck rails position the cow in the stall when she is standing and they are often moved too close to the rear kerb, making cows perch half in and half out of the stall. Even worse, the neck rail is so far back and too low so that the cow must hit it when she rises in the stall. This is unacceptable.

While a contaminated stall maybe a risk for udder infection, an unused stall is most definitely a risk for inadequate rest, lameness problems and early herd removal. We therefore have to find the right balance between comfort and cleanliness. We have taken the view that we are going to build stalls that provide adequate resting space for the cow – whatever their size, and make adjustments to stall design that help index the cow and reduce contamination of the bedding.

Most mature Holstein cows now measure 1.9m from nose to tail when they are lying down – so we build stalls against a side wall 3m long and we have lengthened the head to head platform to 5.2m, to provide lunge space in front of the cow. In head to head stalls, we provide a deterrent wire, covered with 7cm wide PVC pipe, level with the tops of the cows' heads, to limit problems of cows passing through the open front, while still keeping the front lunge and bob space free. Other dimensions are given in Table 4, with a diagram in figure 2.

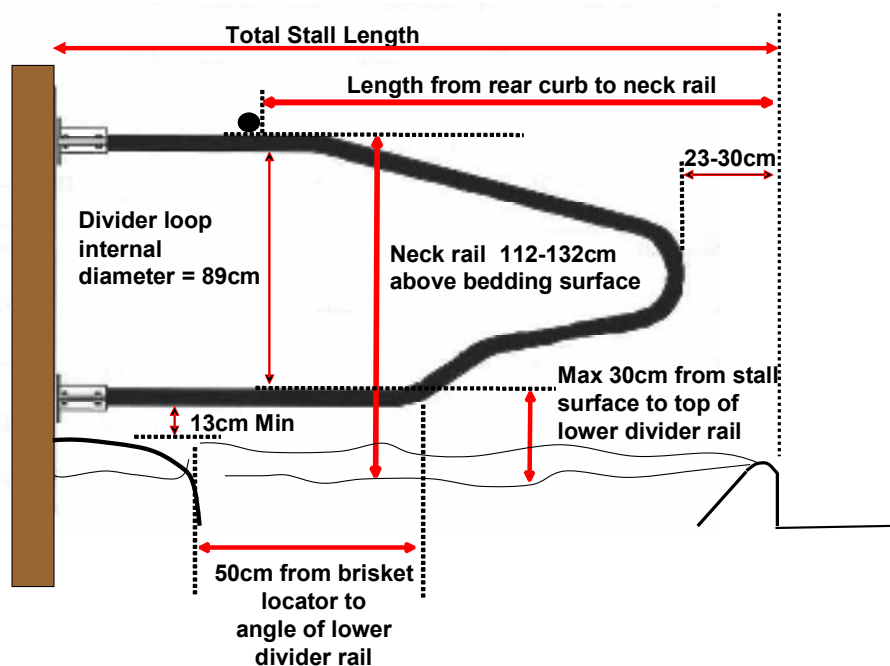
For indexing the cow without causing injury and discomfort, we use a divider loop with the upper edge of the lower divider rail located at 30cm above the stall surface, where the angle of the loop occurs at 50cm behind the brisket locator. The interior diameter of the loop is 89cm, which places the lower edge of the neck rail at 127cm above the stall surface.

While some farmers have removed brisket locators that are too high or too close to the rear kerb, we believe that we need something to help position the cow when she is lying in a wider stall. Smoother more rounded brisket locators that are 12cm high or less have become available and are moderately effective, although some cows choose to lie over the top of them. We have developed a concrete brisket slope design which appears to position the cow effectively and enable her to rise without obstruction to her front leg movement – by sloping the concrete gently to allow the foot to be placed on the slope when rising.

Table 4. Target stall dimensions for dairy cows with a range of different body weights

Stall Dimension (cm)	Body Weight Estimate (kg)				
	455	545	636	727	818
Total stall length facing a wall	244	244	274	305	305
Kerb to kerb distance for head to head platform	488	488	518	518	549
Distance from rear kerb to brisket locator	163	168	173	178	183
Centre-to-centre stall divider placement (Stall width)	112	117	122	127	137
Height of brisket locator above stall surface	7.6	7.6	10	10	10
Height of upper edge of bottom divider rail above stall surface	28	28	30	30	30
Height below neck rail	112	117	122	127	132
Horizontal distance between rear edge of neck rail and rear kerb for mattress stalls	163	168	173	178	183
Rear kerb height	20	20	20	20	20
Rear kerb width (loose bedded stalls)	15	15	15	15	15

Figure 2. Configuration of freestall divider loop, brisket slope and rear kerb.



Neck rails are located in mattress stalls directly above the correctly located brisket locator – so that the cow is able to stand squarely in the stall. In

deep bedded stalls, where the neck rail is at least 122cm above the surface, we move the rail back a distance equivalent to the width of the rear kerb, so that cows take a step back and perch half in and half out of the stall. While we will not tolerate this behaviour in a flat, mattress stall, we are prepared to tolerate it in a deep loose bedded stall, because the front foot elevation is much less and the problems of managing a deep bed soiled with urine and faeces are too great.

MAXIMIZING LYING TIME

Cows lie down for longer in stalls with softer surfaces, and in the past four years we have seen the marketing of softer foam filled mattresses with the aim of providing more cushion. Attempts to provide increased cushion with organic bedding are usually met with disaster due to the retention of material with a high bacterial count. I maintain that cushion must be provided by the mattress itself and organic bedding used on top of the mattress is merely there to absorb moisture. As such it should be removed and replaced every 24 hours.

Many larger freestall dairies with anaerobic digesters have persisted with the approach of using digested manure solids in a deep loose bed, which is very comfortable and soft, but very few have managed to control udder health in the upper Mid-West. It is likely that hot dry climatic conditions with low humidity are required to make this option successful.

We have observed the greatest success in maximizing resting times for both lame and non-lame cows and improving udder health status using sand bedding – either deep beds, or 2-3 inches of sand over a mattress fitted a few inches below a raised kerb. This latter design, called the Pack Mat™ has the advantage of using half as much sand as a deep bed, while still maintaining the behaviour advantages for lame and non-lame cows that we have observed in deep sand stalls.

Sand stall management has not been without its challenges. Fresh sand has to be added to the stalls 1-3 times per week and the surface needs to be cleaned of manure and levelled frequently. We prefer systems that redistribute sand from beneath the divider loops over rakes that stir sand up from deeper in the stall. Sand particles must also be cleaned from the teats prior to milking which often requires inserting an extra dry wipe into the teat preparation routine. In a database of over 70 herds that we store for mastitis herd investigations, sand bedded herds appear to average ~80,000/ml lower bulk tank somatic cell count (BTSCC) than herds using organic bedding and almost one third fewer clinical mastitis cases. These udder health savings are worth approximately \$60 per cow per year.

Mastitis outbreaks do occur in sand bedded facilities, and while fresh sand may be inert and have a very low bacterial count, sand contaminated with milk, urine and faeces may grow large populations of bacteria. Commonly,

sand bedded facilities start to see contamination problems in their third year after construction, and clinical *Klebsiella* mastitis is a common presentation in the hot summer months in Wisconsin. Table 5 shows the median and upper and lower quartile bedding counts for coliforms and streptococci for 82 sand bedding samples collected from 23 farms. These counts would suggest that coliform mastitis would not be a risk, but streptococcal infection would be a major problem. In fact, in these 23 herds, the proportion of mastitis due to gram negative pathogens averaged 75%, and mastitis due to streptococci was rarely a major problem.

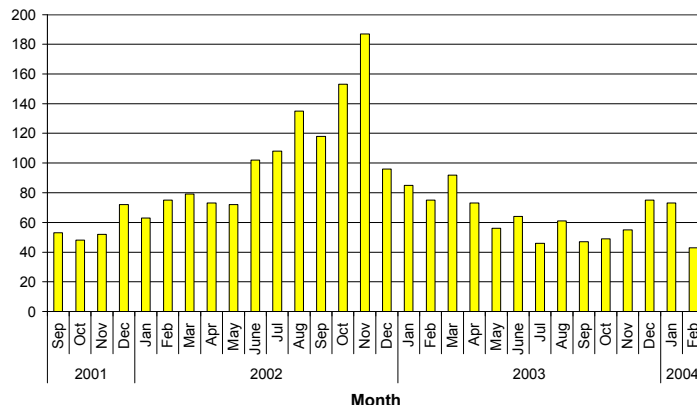
Table 5. Coliform and streptococci counts (CFU/ml) for 82 used sand bedding samples collected from random stalls from 23 herds.

Used Sand Bedding Samples	Count CFU/ml	
	Coliform count	Streptococci count
Median	50,000	6,650,000
Upper Quartile	10,000	2,612,500
Lower Quartile	134,250	14,787,500

We have found significant gram negative mastitis problems (*Klebsiella spp* in particular) at a coliform threshold of ~100,000/ml, and this is typically used as our intervention level rather than the 1 million CFU/ml widely quoted in the literature. Even lower counts have been used in very cold weather during the winter. Streptococcal counts in sand can rarely be kept below 1 million /ml, and high counts usually reflect the duration of sand retention in the beds – i.e. we can keep the count lower by increasing the turnover rate of the material. In my experience, associated streptococcal mastitis problems are a good indicator of poor teat end cleaning procedures in the parlour.

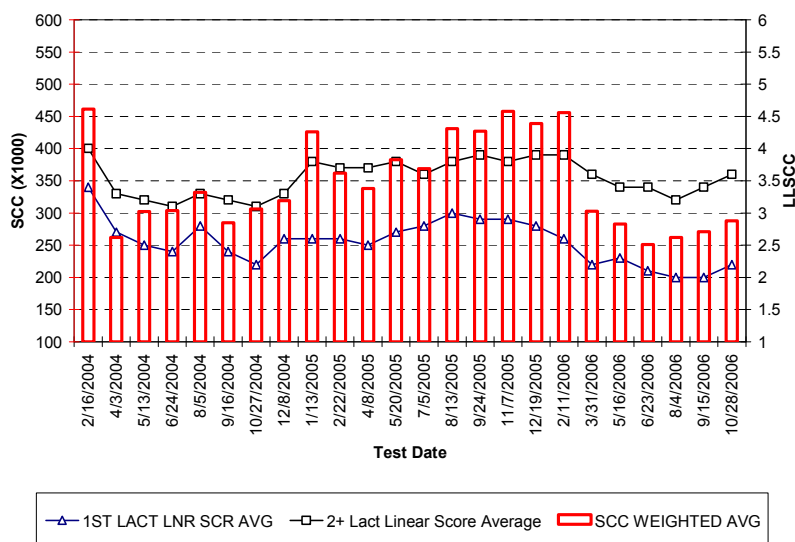
We have successfully improved the rate of clinical mastitis and lowered herd BTSCC by removing contaminated sand bedding and changing to coarser screened sand rather than fine sand (6). The 1314 cow dairy shown in Figure 3 was suffering an extremely high gram negative clinical case rate in the summer of 2002. Following sand removal and replacement, the case rate was halved in 2 months and returned to target levels within 6 months.

Figure 3. Clinical quarter cases of mastitis by month before and after sand removal from the free stalls in a 1314 cow dairy in November 2002.



A 1400 cow dairy was visited in February 2006 with contaminated compacted sand stalls. The sand was removed and replaced with coarse washed mason sand. Not only did cow comfort improve, but clinical treatment rate and bulk tank SCC was halved within one month (Figure 4). In these herds, we now keep the coliform counts in the bedding less than 10,000/ml and see many fewer gram negative clinical cases.

Figure 4. Somatic Cell Count response (monthly average weighted SCC, first lactation and mature cow linear score) at a 1400 cow dairy where sand was removed from stalls in February 2006 and the fine sand replaced with coarse washed mason sand.



It is now commonplace for our sand bedded herds to completely remove the sand from the rear third of the stall every 6-12 months and start afresh.

Correlations between bedding counts and teat end contamination have been made (13) and confirm that higher correlations are generally made for organic bedding than for sand. In particular, the correlation between

streptococcal sand bedding counts and teat end counts is low ($r=0.28$, $P=0.06$), compared with that for *Klebsiella spp* ($r=0.40$, $P<0.05$). More research is needed to fully understand the transfer mechanisms of pathogen groups from the bedding to the udder, but in the mean time, these anecdotal reports confirm that dramatic improvements in udder health can be achieved by lowering the teat end challenge from contaminated bedding.

The secret to the success of certain types of sand, including recycled sand may be in the particle size as it is apparent that there are few differences in bacterial load (8). Note the differences in particle size between the three types of manufactured sand in table 6. While we associate torpedo and mason sand with relatively few udder health problems, we have encountered mastitis issues with the use of #8 sand that have been reduced by switching to mason sand. The large amount of fine particles in the #8 sand may create a different microenvironment in the sand bedding, may lead to greater compaction and reduced cushion and surface drainage, or it may simply be that the finer sand is more difficult to clean off the end of the teat than the more coarse sand particles, leading to greater teat end contamination.

Table 6. Sieve analysis for different grades of sand. Proportion of the sand passing through each screen size is listed, where size 4 is the largest opening and size 200 the smallest.

Screen Size	Torpedo Sand	Mason Sand	#8 Sand
4 (largest)	99	100	100
8	84	99	100
16	67	93	99
30	50	80	96
50	21	15	47
100	4	0	9
200 (smallest)	0	0	1.5

CONCLUSIONS

There remains ample evidence in the field that clinical mastitis relates to the exposure of the teat end to large numbers of bacteria. Udder hygiene and bedding management remain the cornerstones of limiting this bacterial exposure to environmental pathogens, and well managed sand bedded stall facilities continue to outperform other systems of management with regard to udder health.

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THE NATIONAL COHORT OF DAIRY FARMS – A RESEARCH PLATFORM FOR MASTITIS MANAGEMENT, PLANNING AND CONTROL IN CANADA AND BEYOND

K. Reyher¹ and D. Scholl²

Canadian Bovine Mastitis Research Network

¹ Atlantic Veterinary College, University of Prince Edward Island, PEI, Canada

² Faculty of Veterinary Medicine, University of Montreal, Quebec, Canada

Individual mastitis management, planning and control research projects are often limited in impact and scope by the high costs of extensive field data collection and processing. To mitigate this, the Canadian Bovine Mastitis Research Network (CBMRN) has created a single mastitis resource platform to optimise data collection for several different research endeavours simultaneously. The platform is multi-institutional and national in scope and supports the collection, archiving and distribution of data for the applied and fundamental mastitis research projects currently forming the CBMRN research program. Various other projects focusing on antimicrobial resistance, immuno-genetics, parasite loads in pastured cattle, and selenium levels in cattle are also able to collect and use data.

The CBMRN is an innovative partnership between scientists, the Canadian dairy industry, transfer organisations, and animal health pharmaceutical companies all working together toward the common goals of decreasing the incidence of mastitis, reducing associated financial and milk quality losses and enhancing animal welfare. The CBMRN seeks to mobilise resources to reduce the impact of mastitis through concerted research and transfer of research results to users. Discovery of farm management conditions and strategies by participants in the Network will serve to enable successful implementation of mastitis prevention knowledge and biotechnology on commercial dairy farms across Canada and beyond.

CBMRN's strategy is one of cooperation: the deepest and broadest assemblage of mastitis experts in Canada have come together to exploit the resources, knowledge, skills and expertise that exist among such a diverse group of scientists and partners. This pooling of financial and site-specific personnel resources has allowed us to reduce data collection costs for individual research projects as well as enable researchers to study a national-level target population. Coordination of a common data collection for multiple research lines was developed to ensure a number of things, among these uniformity and rigour of data quality. "Shareability" of data among collaborators from different research domains is also a focus, and the suitability of collected data for all the research purposes intended was given much thought before the creation of the data collection platform.

The core research platform of the CBMRN consists of three parts, the first of which is the National Cohort of Dairy Farms (NCDF). The NCDF serves to reduce the incremental costs of epidemiological studies by providing national-level research on commercial dairy farms. Researchers are able to capture information on the diverse husbandry practices in major dairy

centres of the country in order to address the need for greater knowledge of the diversity of the Canadian industry. In this way, regional discontinuities in mastitis epidemiology research are overcome. The second part of the core research platform, the Mastitis Laboratory Network, serves as a group of coordinated mastitis bacteriology labs across the nation. Thirdly, the Canadian Mastitis Pathogen Culture Collection provides storage for geographically representative and epidemiologically referenced intramammary pathogens as a resource for researchers to identify sub-populations of dairy farms with particular features.

Taken together, these three components benefit from the multi-institutional support and coordination of the entire CBMRN. The data collection platform - an industry-linked durable repository of biological and material data - makes possible the recording and storage of this huge amount of data (be it managerial, bacteriological or genetic) for further investigation.

In the planning stages of the CBMRN, preliminary classification of the Canadian dairy industry was undertaken to aid in Cohort farm selection. Coordinating universities were identified in each of the four regions (Atlantic, Quebec, Ontario and western Canada). Primarily Holstein farms participating in Dairy Herd Improvement data collection were selected within strata of high, intermediate and low 12-month rolling average bulk tank somatic cell counts (SCC). Herds were also selected to achieve a proportion of freestall systems to reflect their regional freestall percentages.

Ninety-three commercial dairy farms in six provinces were enrolled in the NCDF early in 2007 for a two year period of data collection. Uniform protocols are implemented for repeated quarter and composite milk samplings on clinical mastitis cases, on fresh and randomly selected lactating cows, and on a selection of cows at dry-off and after calving. Milk bacteriology results are recorded in a central database and bacterial isolates are archived in the Culture Collection. Management, demographic, health, treatment and production data are collected at the individual cow and farm levels and stored in the central database as well as cross-referenced with the Culture Collection database. Innate host resistance data and host DNA from a sub-population of cows are also archived.

These data and biological materials provide a nationally uniform and comprehensive data set enabling interlinked applied and fundamental research leading to mastitis solutions at the cow, herd and regional levels. A strong team of technicians and regional coordinators has been established and has led to increased communication amongst Canadian mastitis research workers, as well as interest from researchers worldwide. International scientists are encouraged to make use of this extensive archive of data and material to enhance their own mastitis research projects.

A REPORT ON A TIME AND MOTION STUDY OF THE AUTOMATED DIPPING AND FLUSHING (ADF) SYSTEM

J. Duke¹ and B. Van Santen²

¹ Research Development and Innovations Ltd, Ham Road Sidlesham, Chichester, West Sussex, PO20 7NY

² The Dairy Group, New Agriculture House, Blackbrook Park Avenue, Taunton, TA1 2PX

A time and motion study involving five farms measured milking routine efficiency before and after the Automatic Dipping and Flushing system (ADF) had been installed. Results show that overall milking time fell by a minimum of 15 minutes per milking and was worth an annual saving of £7 - £31/cow/year. The system releases operator time which can be spent on other tasks in the milking routine, reducing overall milking time.

Five farms were studied in autumn 2007. Two had new ADF installations, the other three were existing users. All farms were observed for two consecutive milkings with and without the system (existing users disabled the setup and reverted to previous practices for two consecutive milkings). The total time taken to carry out each task in the milking routine was recorded.

Although every farm had a shorter overall milking time after the installation of ADF, it was interesting how the adoption of technology affected other elements of the work routine. Less time was often spent on loading the parlour because the operator was available to assist cows as they came in. Before, they would have been busy dipping teats and cleaning clusters.

Automating certain parts of the milking routine should release time for the operator to adopt a more structured, efficient routine. This is where the ADF system ensures quick but good teat coverage of disinfectant after milking and replaces the time-consuming chore of manually dipping clusters between cows.

In addition, milking routines were more structured and less erratic for all farms with the ADF system.

ACKNOWLEDGEMENTS

The co-operation of the five test farms is gratefully acknowledged.

A REVIEW OF A CLIENT SURVEY FOLLOWING THE INTRODUCTION OF A NEW INTRAMAMMARY MASTITIS TUBE

A. Davies¹, R. Booth²

¹ Southfield Veterinary Centre, Dorchester, DT1 1DU

² Intervet/Schering-Plough Animal Health, Milton Keynes, MK7 1AJ

New products receive significant testing pre-launch, but once on-farm it is common to hear only negative feedback. Following the launch of Mastiplan LC in September 2007, a survey of a selected group of clients was undertaken. Seven farms were selected and 49 treated cows formed the sample group. Bacteriological testing was undertaken in 31 cases with results showing 10 *Streptococcus uberis*, six *E. coli* with the rest spread between no growths, *Streptococcus dysgalactiae*, CNS and *Bacillus*.

Because many farmers' mastitis treatment regimes remain unchanged over the years, new and/or different products must be perceived as equally effective and as easy to use as their 'normal' product. Therefore, respondents were also asked for feedback to ascertain their overall impression of the treatment and how easy it was to administer.

Type of mastitis was graded 1-3 as per conventional classification. Ease of administration was scored 1-5 with 1 being low, and 5 high. The average response was 4.2. Users' overall impression of the effectiveness of the product – which covered perceived clinical cure rates, speed of resolution of signs of infection, improvement in cow comfort, and return to saleable milk – was also scored 1-5, with the mean rating being 3.4. Overall impression of the product averaged 3.1

Further analysis of cure rates, bacteriology and recurrence rates is ongoing.

ACKNOWLEDGEMENTS

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All our clients and in particular the dairymen at the herds involved for their work in completing the questionnaires and taking milk samples.

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EVALUATION OF A CLUSTER DISINFECTION BACKFLUSH SYSTEM

T. McKendrick¹, J. R. Baines²

¹ The Dairy Group, New Agriculture House, Blackbrook Park Avenue, Taunton TA1 2PX

² Fullwood Ltd, Ellesmere, Shropshire SY12 9DF

BACKGROUND

Even after thorough pre-milking teat preparation, milking liners showing evidence of soiling are sometimes applied to the teats of cows.

In addition to soiling from the external surfaces of the teat, the liner can come into contact with milk from quarters infected with pathogenic bacteria. The presence of such bacteria on the teatcup liner surface is likely to be a source of infection for subsequently milked cows.

OBJECTIVE

To assess the effectiveness of a Fullwood automated backflushing system in reducing bacterial contamination on teatcup liners.

DISCUSSION AND RESULTS

The evaluation was carried out on liners which had already carried out 1900 cow milkings and repeated after the liners were changed. A full CIP was carried out prior to the new liners being used.

The results clearly demonstrate the efficacy of the Fullwood backflush system in reducing bacterial contamination of the liner.

The farm has subsequently reported a reduction in mastitis incidence.

REGULAR MONTHLY USE OF CELL COUNT DATA IN VETERINARY PRACTICE TO INFLUENCE MASTITIS MANAGEMENT ON FARM

J. Allcock¹, W. May¹, A. Corbishley² & J. Hanks³

¹Lambert, Leonard & May, Old Woodhouses, Broughall, Whitchurch, Shropshire SY13 4AQ

²Towcester Veterinary Centre, Burcote Road, Towcester, Northamptonshire, NN12 6JW

³Veterinary Epidemiology & Economics Research Unit (VEERU), School of Agriculture, Policy & Development, The University of Reading, PO Box 237, Reading RG6 6AR

Lambert, Leonard & May, a large farm-only dairy veterinary Practice in North Shropshire has been using cell count information from monthly milk recordings for regular monthly surveillance of milk quality management for over 2 years. The NMR Companion tool provided the basis for cell count interrogation and this has now been developed further to allow extensive performance comparisons to be made between farms. The benchmarking of farmers against their local peers has proved extremely effective in motivating change.

Sold as a regular 'Cell check' service to farm clients, reports are generated by technicians with desk veterinary surgeons providing technical interpretation. The aim is produce simple action lists each month that allow maintenance of optimal milk quality and gradual resolution of problem areas. Clinicians visiting the farms for regular routine fertility work are supplied with copies of the reports that have previously been mailed to participating farmers. Commonly recommended actions include the following:

- Cows that have been recently infected and which were a problem for several of their recordings in their preceding lactation are identified for CMT and early therapy
- Cows suspected to be recently infected and which have failed to resolve in the preceding month are listed for CMT checks and possible treatment in lactation.
- Cows that have yielded cell counts above 200,000 for the last 4 to 6 months or more are listed for aggressive dry therapy techniques.
- Cows that are over 400 days in milk and have a cell count of greater than 200,000 are listed as possible candidates for early dry-off.

Central to the success of the program has been the creation of a Cell Check 'Club' within the Practice. The 'club' currently involves around 40 farm clients. Each month key subclinical cell count parameters are benchmarked across the group with league tables being the latest innovation to motivate change on farm. The group also meet every 3 months on farm to focus on certain aspects and learn from each other.

Key criteria for cows in milk that are compared across participating farms include:

- The proportion of the herd with a cell count above 200,000
- The proportion of cows at first recording that have a cell count above 200,000
- The proportion of the recorded cows that have recorded above 200,000 having been below for all previous recordings in the current lactation
- The proportion of the recorded cows that have recorded above 200,000 having been below at their previous month's recording but above on one or more occasions earlier in the current lactation
- The proportion of the recorded cows that have recorded above 200,000 having recorded above 200,000 in the previous month's recording in the current lactation.

The average of all the above parameters for the last 6 months recording dates are also benchmarked. The report for each farm shows the individual farms' average performance figures over the preceding 6 month period. It also shows figures for the 'best' and 'worst' performing herd in that criterion over the preceding 6 months. An average value is also illustrated. Target and interference figures are expressed and compared to the farms own figure which is then highlighted in red, amber or green to emphasise areas for greater focus.

Dry period performance of cows is compared according to the following key criterion:

- The percentage of heifers that calved in the 365 days starting their lactation with a recording of >200,000 cells/ml
- The percentage of all cows that calved in the 365 days ending their lactation after a recording of >200,000 cells/ml
- The percentage of cows that calved in the 365 days that ended their previous lactation after a recording of <200,000 cells/ml and started this lactation with a first recorded cell count of <200,000 cells/ml
- The percentage of cows that calved in the 365 days that ended their previous lactation after a recording of >200,000 cells/ml and started this lactation with a first recorded cell count of <200,000 cells/ml.

Finally the proportion of cows calved in the 365 days that have been above 200,000 for the first time at any recording that then return below 200,000 at the subsequent recording is compared across all farms.

This service provides a solid basis for strengthening contact with veterinary health advisors and for effective herd health planning. The 2008 BMC poster illustrates the benchmark tables involved for all farms and illustrates performance league tables. An individual farm's progress is chronicled as an illustration of the power of benchmarking against a local peer group.

THE NEW IP – V, EUROPEAN VENTED IMPULSE SYSTEM, A NOVEL WAY OF REDUCING MASTITIS DUE TO CROSS CONTAMINATION.

D. Boast

Avon Milkrite, Hampton Park West, Semington Road, Melksham, Wiltshire, UK

A new dairy liner has been developed that reduces mastitis caused by backflow or impacts during milking (this is also sometimes called respray). Backflow is the flow of milk back on to the teat from the short milk tube when the liner opens. Contaminated milk can then cause infection as infected milk contacts the teat.

Conventional milking machines are vented at the claw. Some US liners have vents in the short milk tube. Both of these types of vent encourage backflow and impacts, by increasing the pressure difference across the short milk tube.

The new liners have a vent in the mouthpiece. Because air is entering at the mouthpiece and flowing past the teat end there is little force to drive the backflow and impacts. At the end of milking the teats are dry. The rate of milk flow away from the teat is improved.

Farm trials show a reduction in mastitis. Additionally the high levels of vacuum in the mouthpiece are automatically reduced. Ringing and swelling can also be reduced.

PROBLEMS ENDANGER MILK PRODUCTION IN EGYPT

Abd El-salam El-Sawy

Faculty of Veterinary Medicine, Alexandria University, Egypt

Milk production is a business with profit as the motive. However, there are numerous clinical affections of the udder and teats that result in high economic losses. Although mastitis and/or teat affections occur sporadically in all animals, they assume major economic importance only in dairy cattle.

The present work was carried out on more than 500 lactating cows and buffaloes showing udder and teat affections during the period 2003–2008 in El-Behera Province, Egypt. Bacteriological examination revealed *Staphylococcus aureus* in milk samples of 11 animals showing signs of peracute mastitis (2 cows showed signs of gangrenous mastitis). Clinical signs include fever $41.7^{\circ}\text{C} \pm 0.3$, weakness, slight dehydration, off-feed and false lameness. The affected quarter was very large, hard, hot, swollen, painful and secretes a reddish-yellow discharge. Another cow showed signs of peracute Staphylococcal mastitis for 3 successive lactations, the last one terminated with abscess. Animals affected with acute clinical mastitis constitute 85% and teat affections 10%. They showed a slight but insignificant rise in body temperature and were normal in all other aspects. Udder oedema (3-4%) produce no systemic signs and milk was apparently normal.

The aim of the present work was to demonstrate photographically the main problems which endanger milk secretion and production in lactating cows and buffaloes in Egypt. Moreover, the possible lines of treatment were also taken into consideration.

The obtained results demonstrated that clinical mastitis, mammallitis, severe form of physiologic udder oedema, blood in the milk and udder impetigo and/or udder acne constitute the major problems in lactating cows and buffaloes. The gross pathology and successive changes of gangrenous mastitis (in one of two cows) were recorded step by step during the period of treatment until healing of the wound.

Mastitis is a multi-factorial disease. Acute mammallitis recorded in this work could be attributed mainly to trauma of the teat and/or over stretched teat in 1st calving buffalo heifers. Similarly, trauma of the udder results in blood in the milk. Udder impetigo or udder acne is usually due to bacterial infections. The site of udder is a predisposing factor that facilitates these affections. Severe form of physiologic udder oedema is known to be due to accumulation of lymph between the skin and the mammary tissue. It is well known that partial damage of teat wall interferes with milking and results in failure of some natural defence mechanisms of the udder, while, severe damage (as recorded in the present work) results in failure of all functions of the teat and mastitis. That was concluded because the mammary glands are composed of delicate tissues which are severely attacked by bacteria

following teat damage. The environment inside the udder is warm and moist with plenty of available nutrients, so bacteria multiply rapidly leading to mastitis.

In this case, chemotherapy must be started without waiting for bacteriological examination. Therefore, isolation of the microbial agents and sensitivity testing is not carried out for acute bacterial mastitis because these tests delay chemotherapy and recovery, are time consuming and expensive although they do give accurate information for diagnosis which is achieved after 48 hours while immunity of the udder is usually poor. In this case, successful chemotherapy for acute mastitis was applied as in the present work.

Again, for rapid relief and recovery, to save time, to reduce cost of treatment and for rapid interference, some successful pharmacological lines for treatment of acute or peracute clinical mastitis and mammallitis were applied. These include a combination of bactericidal antimicrobials, soothing and emollient anti-inflammatories, an anti-histaminic and analgesic agents. The same lines were applied for udder impetigo or udder acne in addition to topical antibiotic and anti-inflammatory.

Udder oedema interferes with milking, suckling and exposes the udder and teats to trauma. A mild rubefecient for short intermittent periods was used for treatment of severe forms of physiologic udder oedema concurrently with i/v or i/m furosemide or bumitanide.

Blood in the milk makes the latter unfit for humans consumption. Calcium borogluconate was given i/v with topical vasoconstrictor fomentations for treatment of severe cases of blood in the milk. To avoid excessive blood in the milk, trauma and damage of the oedematous teats, only gentle hand milking with cooking oil as a lubricant applied topically, or soothing and emollient anti-inflammatory agent are recommended for 2-3 days. Moreover, suckling is contra-indicated for 2-3 days during both affections.

It could be concluded that clinical mastitis combined with teat injuries continue to be a major cause of economic loss to the owner, the consumer and the national income. That was concluded because these affections produce many adverse effects on milk synthesis and production. Veterinarians and dairy producers must know that gangrenous mastitis alone, acute mammallitis combined with acute mastitis and/or complete damage of teat wall terminate the function of the quarter, reduce the sale value of the animal and encourage fattening and slaughtering of the animal. Treatment of peracute Staphylococcal mastitis needs a special pharmacological program. Although the obtained results concerning this point are satisfactory, this program needs evaluation in a further study to be in common use. Treatment of mammallitis is very expensive and prolonged especially in buffaloes. Teat lesions must be treated immediately.

THE ADVERSE EFFECTS OF TEAT LESIONS ON MILK PRODUCTION IN BUFFALOES

Abd El-salam El-Sawy

Faculty of Veterinary Medicine, Alexandria University, Egypt

Each year, a number of winter dairy conferences are held dealing with such subjects as mastitis, milk quality and management. Although mammallitis and/or teat wall damage are more dangerous conditions than mastitis especially in buffaloes, little or no attention for such cases is observed. Therefore, veterinarians and dairy producers have been required to deal with teat lesions for many years. The teat is a very sensitive structure and if it is to function correctly and acts as a reasonable barrier for the entrance of bacteria it must be maintained in a healthy state. Injury to the teat is important in itself as a cause of poor milk, but of greater importance as a means of providing a focus for the development of pathogenic bacteria. Teat lesions are a troublesome problem for the veterinarian as well as the dairy producer. The present work was carried out on more than 300 lactating buffaloes and 50 cows showing signs of mammallitis and/or other teat lesions during the period 2003–2008. The animals were normal in all other aspects. The aim of the present study was to demonstrate clearly the adverse effects of mammallitis and/or teat lesions in the lactating buffaloes and cows in Egypt. Moreover, the possible lines of treatment of these affections are discussed.

The obtained results demonstrated that mammallitis, physiological teat oedema, teat wall damage and teat end damage constituted the main problems of teats in these animals. Two chronic affections of the teat wall of unknown diagnosis were recorded photographically although milking and suckling are normal. Mammallitis and/or severe damage of teat wall usually result in failure of some or all of the natural defence mechanisms of the udder thereby leading to clinical mastitis. In addition, acute mammallitis in buffaloes induces dangerous effects on milk synthesis and production compared with mastitis.

Treatment of mammallitis in buffaloes is difficult, prolonged and very expensive. This could be explained mainly due to difficult handling of these animals, teat lesions are readily colonized by bacteria and thus serve as an important reservoir of infection, and hyperactivity of nervous parts in the teats which make the animal highly irritable and excited. Because teat lesions are generally very painful and the animals resist preparation and milking procedures, they are difficult if not hazardous to milk. Kicking, restlessness, failure of milk let-down reflex, retention of milk, difficult topical and intra-mammary therapy and mastitis are common clinical findings met with acute mammallitis thereby resulting in aggravation of the existing lesion. It is well known that the teat contains the terminal part of the duct system of the udder. Moreover, healthy teats represent the natural defence mechanisms of the udder and separates the alveoli from the external environment. Therefore, any degree of teat damage affects milk

synthesis, let-down and production. Histologically, the teat contains an erectile tissue similar to that of the penis. The teat wall contains an abundance of elastic connective tissue which provides for expansion and contraction of the teat as it fills and evacuates milk in the lactating cow. The near constant movement associated with these physical dynamics of the teat combined with milking preparation procedures, and milk collection complicate the normal healing process. Disturbances of the function of teat facilitate bacterial growth in the quarter leading to mammallitis combined with acute mastitis. Due to prolonged periods of treatment, additional infections must be taken into consideration. Therefore, isolation of the microbial agents and sensitivity test are not carried out because these tests delay chemotherapy and recovery, and are time consuming and expensive although they give accurate information for diagnosis. The latter is usually achieved after 48 hours while immunity of the udder is usually poor. In this case, chemotherapy is given immediately as in the present work. During the course of viral stomatitis and lumpy skin disease, teat lesions represent a part of the general symptoms and are easily identified and diagnosed. Topical prophylactic chemotherapy is recommended. For rapid relief, to save time, to reduce cost of treatment and for rapid interference, successful pharmacological lines for treatment of bacterial mammallitis were adopted. These include a combination of bactericidal antimicrobials with soothing and emollient anti-inflammatory, anti-histaminic and analgesic agents.

It could be concluded that the natural defence mechanisms of the udder, protection and separation of alveoli from the external environment, milk let-down reflex, transfer of milk to the newly born calf and harvesting the milk crop constitute the main functions of healthy teats. Additional functions include infusion of drugs and evaluating their efficacy during mastitis, removal of mastitis waste products, removal of blood clots during cases of blood in the milk, evaluating udder secretion during drying-off and intra-mammary infusion of dry cow therapy. These important functions are usually interrupted or fail during acute mammallitis and severe damage of the teat wall. Mild or moderate lesion of the teats interferes with milking and exposes the teat suddenly to acute mammallitis. Persistent lesions reduce the sale value of the animal. Complete damage of the teat wall in buffaloes results in slough, amputation of the teat and loss of all teat functions which encourage fattening and slaughtering of the animal. Moreover, a prolonged treatment period (3–12 weeks) is required for complete healing of severe mammallitis in buffaloes. The adverse effect of mammallitis on milk production in buffaloes embraces that of mastitis. Over-stretched teats in buffalo heifers terminate the productivity of the animal.

OVER-VIEW ON MASTITIS AND MAMMALLITIS, NEW ETIOLOGICAL AGENTS (IN EGYPT)

Abd El-salam El-Sawy

Faculty of Veterinary Medicine, Alexandria University, Egypt

The mammary glands are skin glands, albeit large ones held exterior to the body cavity. Therefore, the mammary tissue forgoes the potential advantage of rigid skeletal support. This brings its own problems. Moreover, the udder of the present day dairy cow is very large. Because when it is full of milk it is so heavy, damage of the udder and teats is very common. Because of these factors and also because its position exposes it to traumatic influences it is frequently the site of the disease.

The present work was carried out on more than 1000 lactating cows and buffaloes with signs of acute and peracute clinical mastitis, acute mammallitis and/or udder and teat lesions during the period 2003–2008.

The aim of the present study was to demonstrate clearly the possible causes of these affections. Mastitis means inflammation of the udder regardless of the cause. It is characterized by abnormal size and secretion. Based on the latter statement and the well known cardinal signs of inflammation, the obtained results demonstrate photographically numerous causes of these affections. These are:

- Infection with micro-organisms, e.g. bacteria and viruses
- Bad management, e.g. over-stretched teats
- Teat injuries and ulcer lead to mammallitis and mastitis
- Acute mammallitis usually results in acute clinical mastitis
- Mechanical causes, e.g. blood in the milk and udder haematoma
- Physical causes e.g. burns
- Poor medical management of microbial udder infections. Cold water fomentations produce vasoconstriction and hardness of the quarter
- Immunological disturbances e.g. udder and teat lesions were reported during the course of food poisoning (urticaria in lactating animal and even in a young heifer (9 months old))
- Auto-immune, e.g. hyperglobulinemia during colostrum secretion (plasmacytosis, first record 1938). The lesion was reported in a multiparous buffalo (2006) after 8 hours from parturition. Again, it appears in 2007 in the same animal after 6 hours from parturition
- Chemicals: a) treatment of teat lesion with glacial acetic acid, and b) through ignorance, lack of experience and bad diagnosis, a traumatized quarter of the udder is treated topically with iodine ointment resulting in severe swelling of the quarter
- Faulty diagnosis and treatment. Many non-infected oedematous udders are treated with antibiotics via intra-mammary infusion. Frequently, additional infections are introduced into the quarter by careless drug administration. Similarly, many non-infected oedematous udders are

treated topically with pustulants (iodine ointment) to treat physiologic oedema thereby resulting in mammallitis and mastitis

- Mastitis and mammallitis are common sequels to operations on the udder and teat such as spiders or obstruction in the teat canal.

The udder is composed of a delicate mammary tissue. The environment inside the udder is warm and moist with plenty of available nutrients, so bacteria multiply rapidly leading to mastitis.

These factors combined with mammallitis facilitate many types of bacterial infection of the udder. Isolation of bacteria and sensitivity testing is not carried out because these tests delay chemotherapy and recovery, are time consuming and expensive, although they do give accurate information for diagnosis which is achieved after 48 hours while immunity of the udder is poor. In this case, chemotherapy was started without waiting for the result. Udder and teat lesions due to viral stomatitis and lumpy skin disease represent a part of the general symptoms. Acute clinical mastitis, mammallitis and udder and teat lesions caused by bacteria can be controlled. For rapid relief, to save time, to reduce cost of treatment and for rapid interference, some pharmacological lines for treatment of mastitis and mammallitis were adopted. These included a combination of bactericidals, soothing and emollient anti-inflammatory, anti-histaminic, analgesic agents and counter irritant therapy. Pain induced by clinical mastitis produces central vasoconstriction. Most veterinarians used oxytocin to evacuate the udder before therapy of mastitis and recommend cold water fomentations in therapy of acute and peracute microbial mastitis. However, in the present work I never used oxytocin or cold water fomentations in therapy of these cases, in more than 3000 animals with 20 years of experience. Cold water fomentations (topical vasoconstrictor) are highly recommended only for treatment of traumatic mastitis such as blood in the milk and udder haematoma, plasmacytosis, and udder and teat lesions due to hypersensitivity induced by food poisoning. Cold water fomentations intensify hardness of the gland, minimize drugs access (oxytocin) to the udder, delay recovery and expose the quarter to gangrene during peracute mastitis due to added vasoconstriction. Hot water fomentations induce vasodilatation and hyperaemia thereby resulting in analgesia, hyperaesthesia (via local axon reflex), increase drugs access to the udder, reduce swelling and enhance recovery with 100% efficacy in peracute and acute microbial mastitis. It could be concluded that clinical mastitis and mammallitis are complex diseases of multi-factorial causes. Moreover, udder affections are easily established in lactating animals. The site of the mammary gland facilitates injury of the udder and teats. That was concluded because approximately 7-8 causes of clinical mastitis and mammallitis act through this site. Therefore, the presence of a ready, well designed pharmacological program is imperative to save the productivity of the udder and functions of the teat, a point which represent great advances in chemotherapy of clinical mastitis and mammallitis. This program will appear in full details for publication by the author.