

TIMETABLE of EVENTS

09:00	ARRIVE / REGISTRATION / COFFEE and POSTER DISPLAY	
09:45	CHAIRMAN'S INTRODUCTION	Ian Ohnstad The Dairy Group, UK
	Controlling Somatic Cell Counts	
09:55	Progress in mastitis control?	John Sumner UK
10:25	The DairyCo Mastitis Control Plan – where are we?	Andrew Bradley QMMS Ltd, UK
10:35	The DairyCo Mastitis Control Plan – a practitioner user's view	Mike Kerby Delaware Veterinary Group, Somerset, UK
10:50	The DairyCo Mastitis Control Plan – a farmer's perspective	Dan Norris Somerset, UK
11:05	Questions and Discussion	
11:15	COFFEE and POSTERS	
	Research updates	Brian Pocknee DHC, UK
11:45	<i>Streptococcus uberis</i> – are we nearer an effective treatment?	Jamie Leigh University of Nottingham, UK
12:00	PCR technology – is it the future?	Hannah Pearse NMR, UK
12:15	National Mastitis Survey	James Allcock Lambert, Leonard and May LLP, UK
12:30	Questions and Discussion	
12:40	LUNCH and POSTERS	
14:00	CHAIRMAN'S INTRODUCTION and VOTING ON POSTERS	Elizabeth Berry DairyCo, UK
	Milking management	
14:05	What makes a good teat disinfectant?	Alison Clark GEA Farm Technologies Ltd, UK
14:40	Ventilation and its impact on mastitis control	Jamie Robertson University of Aberdeen, UK
15:15	The role of milking machines in mastiti	Dave Reid BouMatic, Wisconsin, USA
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¹ The Institute of Bacteriology and Mycology, Faculty of Veterinary Medicine, University of Leipzig, 04103 Leipzig, Germany; ² Current address; c/o EM Agriton, Molenstraat 10-1, 8391AJ Noordwolde, The Netherlands
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¹ Biobest Laboratories Ltd, 6 Charles Darwin House, The Edinburgh Technopole, nr Milton Bridge, Penicuik, Midlothian, UK; ² The Shepton Veterinary Group, Allyn Saxon Drive, Shepton Mallet, Somerset, UK
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¹ Synergy Farm Health, West Hill Barns, Evershot, Dorset, UK; ² Bakers of Haselbury Plucknett, Rushywood Farm, Haselbury Plucknett, Somerset, UK
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¹ The Dairy Group, **New Agriculture House, Blackbrook Park Avenue, Taunton, UK**; ² BASF Pest Control Solutions, Earl Road, Cheadle Hulme, Cheshire, UK
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CHAIRMAN'S INTRODUCTION

Welcome to the 22nd British Mastitis Conference. This year, as part of our constant effort to improve the conference for delegates, the Organising Committee has decided to depart from the NAC at Stoneleigh and move to more modern conference facilities in Worcester. We believe this venue is more suited to our requirements and hope that you agree.

The Organising Committee has worked hard throughout the year to bring together a group of speakers, both international and home grown, that we believe will prove thought provoking and stimulating presentations.

Our first paper examines the progress that has been made in mastitis control over the last 25 years, focusing on important milestones and then casting a view forward towards future developments.

We then have three papers related to the DairyCo Mastitis Management Plan. Firstly an update on how the Plan has been adopted and accepted, secondly a veterinary surgeons perspective on how he has implemented the Plan and finally, a farm perspective on how the Plan has assisted their business.

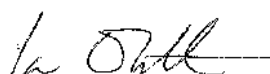
We then have three research summaries examining new mastitis detection technology, treatment of *Strep.uberis* mastitis and a review of the National Mastitis Survey.

After lunch, we will turn our attention to teat, environmental and milking management, with three papers updating our thoughts on the role of teat disinfection, ventilation and the milking machine.

As always we have an excellent group of posters and I would urge you all to make time to review the posters and speak with the authors.

We continue to try to find you the best speakers with the best and most relevant (and latest) information. This is achievable only thanks to all our generous sponsors. This year our sponsors are: DairyCo (at the new Diamond level), Boumatic, Pfizer, Boehringer-Ingelheim, Fullwood, Evans Vanodine, Vetoquinol, Ambic, Lely and Intervet/Schering Plough. As usual the event could not happen without able administration, now provided by Emma Palfreyman at the School of Veterinary Medicine and Science, University of Nottingham.

Finally, as always, thank you for attending and supporting the conference. I trust you will have an enjoyable and worthwhile day.



Ian Ohnstad
British Mastitis Conference Chairman
The Dairy Group

FURTHER INFORMATION

Organised by *The Dairy Group*, DairyCo and University of Nottingham

The Dairy Group

DairyCo



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PROGRESS IN MASTITIS CONTROL

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SUMMARY

The progress in the control of mastitis in the UK over the last thirty years is reviewed against a background of dairy farming industry undergoing political and rapid structural change. Developments in new technology are considered and the progress of cell count levels and numbers of clinical cases tracked within a framework of fewer dairy farmers, increasing herd size and cow yield. The significant drop in cell counts over the period is detailed but concerns are raised over the lack of progress in relation to clinical cases of mastitis.

INTRODUCTION

The terms of reference for this paper is to review the progress in mastitis control over the last 30 years. This period will more than cover the lifespan of the British Mastitis Conference, the first of which was held in 1988. It is not intended to analyse or compare all the scientific progress made in the intervening years, but to assess the impact science, technology, veterinary and husbandry advice has had on the national herd and the progress made in reducing the levels of mastitis.

When assessing the progress made in any aspect of dairy farming, including the progress made in disease control, it is necessary to consider the external influences, such as economic and political circumstances appertaining at any particular time. Evidence from the recent Farm Health Planning Cattle Initiative (6) funded by Defra and delivered by an Industry/Defra Working Group, showed that the likelihood of farmers embracing the practice of proactive health planning was influenced by factors other than the need to improve the health and welfare of their herds.

This paper will therefore track the progress in the control of mastitis within the framework of a changing dairy farming industry.

DAIRY FARMING'S CHANGING STRUCTURES

Dairy Farming in the UK has and continues to experience rapid and remarkable structural change. Table 1 illustrates how the industry has restructured over the past half century.

Table 1 Numbers of dairy farmers (UK)

Year	Producers	Dairy cows (‘000)	Herd size	Milk Yield (litres/cow)
1960	151, 625	3,165	20	3,380
1970	100, 741	3,244	30	3,750
1980	56,247	3,224	51	4,670
1990	41,248	2,846	67	5,145
2000	29,000	2,350	80	5,800
2008	17, 060	2.055	114	7,400

Source: MAFF/Defra, DairyCo

Producer numbers have been declining at around 3% per year throughout the period. Some recent years have witnessed larger number of farmers ceasing milk production providing opportunities for others to expand. It is important to note that this trend is not unique to the UK, but it is common to all of the developed dairying countries as mechanization and automation replaces labour on dairy farms.

The decline in the number of dairy farmers continues, herd size continues to increase and it is likely that at the present time over 80% of the UK’s milk is produced by not more than 7,000 farmers. Better nutrition, improved genetics and good management have all contributed to improve technical efficiency.

POLITICAL INFLUENCES

In this 30 year period under review at least three major political changes have occurred which have presented major challenges for UK dairy farmers. In 1984, the EU’s dairy policy finally conformed to GATT, which had been demanding that milk prices should be at a reasonable level. Furthermore, the EU was at the time producing 20% more milk than it could consume, and in order to reduce the cost of supporting milk product surpluses, a regime of milk quotas was introduced. The controls on farm output and limitations on business growth presented individual farmers with immense management challenges.

Ten years later in 1994, the UK industry underwent radical change when the Milk Marketing Boards (MMBs) ceased their statutory buying of all wholesale milk off farms. The challenge presented to individual farmers should not be underestimated in that for the first time in 60 years, farmers had to decide how to sell their milk, an element of business management completely new to them. It is noteworthy that at the time of deregulation, UK farmers were at the bottom of the EU milk price league table and many had begun to consider that controlled marketing discouraged innovation. However, the safety net of protectionism had been removed

A period of volatility followed. Milk Marque, a voluntary farmers' co-op was soon disbanded the industry reformed again and the process of reforming has continued since. Importantly, the milk price paid to farmers has been extremely volatile, much of the time at a price less than the true costs of production, making forward planning difficult and contributing to the decline in farmer numbers.

Add to the above the continuing reform of the CAP and its proposed demise in 2015, plus the widening of the EU, dairy farmers have had to come to terms with the move from an internal protected market into a free trade world.

When assessing the progress made in the control of any disease, especially mastitis, which demands changing attitudes and human behaviour, it must be remembered that nothing happens in a vacuum. It can, and should be argued that when profitability is poor, reducing costly disease makes good sense, but in many farming situations, the benefits on offer are not always easily recognized.

The external influences described above, and there are others, not to mention outbreaks of BSE, Foot and Mouth Disease, and on-going TB, which occurred during this review period, all influence progress and part of the background in which progress in mastitis control should be considered.

DEVELOPMENT OF MASTITIS STRATEGIES

It is worth noting that developments in mastitis control only really began to make progress in the early 1960s when tools were developed for cytological and bacteriological testing of milk. The International Dairy Federation (IDF) worked on a definition of mastitis in the middle of that decade and suggested classifications for normal udders, subclinical mastitis, and latent infections. It later agreed that 500,000 cells/ml in milk from a single quarter should be the SCC threshold. The debate on SCC thresholds and definitions of normal and abnormal milk continued, and then in the early 1980s, investigations showed that a healthy lactating quarter should have an SCC of less than 100,000 cells/ml (8).

During this time, researchers at Reading (The National Institute for Research in Dairying (NIRD)) and the Central Veterinary Laboratory, Weybridge, (CVL) made major contributions to the understanding of mastitis and methods for controlling the disease. The "five point plan" for the control of mastitis was born. The principles of mastitis control were published in 1973 (11) based on two apparently simple steps; *prevention of new intramammary infections* (reducing the exposure to pathogens) and *reduction of infections* (antibiotic therapy and culling persistent cows).

Although the above brief history is outside the time frame of this paper, the approaches developed at that time are the basis of present day control systems.

PROGRESS DURING 1980s

Against the background of rapid structural change described above, there was increasing interest in implementing mastitis control methods. Progress was encouraged by control schemes, SCC sampling services, free advice and milk payment systems. A summary of the mastitis status in the national herd at the beginning of the decade is outlined in Table 2.

Table 2 Position in 1980 (UK)

Year	Herd size	Milk yield (litres/cow)	Number of cases/100 cows	Ave SCC (‘000/ml)*
1980	51	4,670	54.6	469

Source: CVL/MAFF; Dairy Facts and Figures: * England and Wales

A three year survey (17) to determine the incidence of mastitis carried out at the time in England and Wales showed that the annual incidence of mastitis declined from 54.6 cases per 100 cows in 1980 to 41.2 cases in 1982. *Escherichia coli* was the predominant organism each year with *Streptococcus uberis* and *Staphylococcus aureus* deemed important for part of the year. Culling rate due solely to mastitis was 3 per cent with *Strep. uberis* the pathogen most frequently isolated from clinical cases which occurred in the dry period. Table 2 above shows the annual average SCC for England and Wales with slightly lower results recorded for Scotland and Northern Ireland.

A seminar held at the National Institute for Research in Dairying Reading together with the British Cattle Veterinary Association was held in 1980 to review the progress made in mastitis control. In an introductory paper Dodd (5) stated that the eradication of a particular pathogen should not be a main objective, but that the first objective of mastitis control was economic, to reduce the level of infections caused by the main mastitis pathogens by low cost methods.

The important link between machine milking and udder infection had been recognised previously and considerable research work was directed during this period to the machine’s role in the transfer of organisms during milking. In the early 1980s it was apparent that a change was underway in the types of pathogens causing clinical mastitis. At this time nearly 60% was caused by environmental pathogens compared to around 10% when the five point plan was first implemented. Control methods had clearly reduced clinical mastitis caused by contagious pathogens and greater emphasis was therefore placed on housing and the cow’s environment generally.

Husbandry

At the start of this decade half of the dairy herd was still milked and housed in cowsheds. Just over half of the milking parlours were of the herringbone type, but the number was increasing. The majority of loose-housed cows were in cubicles. However most cubicles had been installed under the Agricultural and Horticultural Development scheme in buildings which, as required by the scheme, should be multi-purpose. Standard recommended cubicle dimensions throughout this period were for a cubicle to measure 2.1m in length by 1.2m in width. Whilst appropriate at the time, due to the greater use of the Holstein genetics and through improved feeding, cow size increased significantly resulting in many cubicles being inadequate for the need. That legacy of inadequate housing remained for many years, and still does for some dairy farms today.

It is worthy of note that the first British Mastitis Conference was held in October 1988 when James Booth reported that over the last 20 years the overall level of mastitis in the UK herd had been more than halved due to the widespread adoption of the recommended control measures (2). He also reported that although progress had been made, some herds still had more than 100 cases of clinical mastitis per 100 cows in a year and that a quarter of herds had an average SCC count of over 500 thousand cells per ml.

PROGRESS DURING THE 1990s

Table 3 shows the structural and mastitis positions at the start of the decade and the improvement made in cell count levels.

Table 3 Position in 1990 (UK)

Year	Herd size	Milk yield (litres/cow)	Number of cases/100 cows	Ave SCC (‘000/ml)*
1980	51	4,670	54	469
1990	67	5,145	45-50	322

Source: CVL/MAFF; Dairy Facts and Figures: * England and Wales

Political and market changes

The early part of the decade witnessed the MacSharry reforms which introduced a range of agricultural production limitation measures including set aside and reductions in market support. These reforms had a knock on effect on the dairy farming industry increasing the need for increased technical efficiency.

The UK milk market was de-regulated in 1994 which was for most farmers a major distraction. There was competition between the milk buyers for supplies which was initially beneficial (it didn't last long) in terms of the milk price paid to farmers. Differing standards were set for milk quality by

the milk buyers, often based on what they perceived their customers required. It is not unreasonable to say that the milk market was in turmoil.

Health and Hygiene Directive

The Health and Hygiene Directive (92/46/EEC) came into force in the same year setting standards for cell counts. Milk intended for the liquid market (drinking) was required to have a cell count of not more than 400,000 cells per ml with 500,000 the limit for milk for manufacture. In 1990 it was reported (16) that one third of the national herd had cell counts in excess of 400,000 cells per ml. Just prior to the introduction of the Hygiene Directive, central milk testing data showed that over 25% of supplies would not meet the liquid standards and 17% would fail the manufacturing requirement (10). The introduction of this legislation together with competition in the milk market encouraged greater attention to reducing cell counts.

It was increasingly recognised that mastitis control methods depend on improved cattle management, and that the multi-factorial nature of mastitis should be considered in controlling the disease (12). Greater focus was placed upon housing management in order to minimise exposure to environmental pathogens.

Technical developments

The popularity of the herringbone parlour continued to increase and as the technology of milking systems became more sophisticated there followed the opportunity to install automatic devices within milking parlours, linked to electronic cow identification and computer management systems.

Pre-milking dipping of teats to reduce microbial contamination was attracting interest and considered as a further important step in the prevention of mastitis, although there was little substantive evidence available at the time. More recently studies at Moorepark (7) compared a range of disinfectants on bacterial counts on teat skin prior to milking. The outcome was that the use of some disinfectant products for pre-milking teat preparation can have beneficial effects on reducing the levels of *Staphylococcus* and *Streptococcus* bacteria on teat skin.

Considerable international effort went into standardizing the methods for SCC testing to improve repeatability and reproducibility as cell counting was widely accepted as a useful indicator of mastitis control measures and herd health.

Automated cow-side tests for mastitis had been of interest for many years but were labour demanding, and difficult to justify economically other than for specific investigations. Such tests needed to be integrated into milking systems if they were to be acceptable. Research on electrical conductivity gave promise that it could possibly be used as a marker system but would require sensing devices integrated into milking equipment. A number of

sensing devices were introduced to the market with limited success at the time. Workers at ADAS Bridgets and the IAH, Compton (3) monitored conductivity levels in cows milked by an automatic milking system. A relationship between conductivity and cell count was noted when milk cell count increases were considerable, but questions on accuracy remained. For most, in spite of the weaknesses, cell count testing remained the key indicator.

PROGRESS FROM 2000 TO THE PRESENT

Production and mastitis positions at the start of the decade are outlined at Table 4.

Table 4 Position in 2000 (UK)

Year	Herd size	Milk yield (litres/cow)	Number of cases/100 cows	Ave SCC (‘000/ml)*
1980	51	4,670	54	469
1990	67	5,145	45-50	322
2000	80	5,800	40-50	191

Source: Various CVL/MAFF; Dairy Facts and Figures: * GB

On the evidence above considerable progress is being made in reducing the national average cell count but less strong evidence that the rate of clinical cases is being reduced.

Political changes

The Agenda 2000 reforms led to a series of reviews of agriculture support brought together under the Mid-Term Review. It introduced a whole new farm support system including the Single Farm Payment, Cross Compliance and Modulation. For the dairy farming industry it resulted in a market in transition as the level of support provided to the dairy sector continued to be in decline.

Farming structures

Dairy farming numbers continued to decline whilst herd size and cow yield increased. Milk price was volatile, but many forward looking dairy farmers who had decided to remain in the industry made a commitment to the future and replaced worn out milking and housing systems.

The economic need to improve efficiency has resulted in the trend of recent years to parlours with more milking units to allow a greater throughput of cows in a given time. Installation costs are significantly higher, but countered by reduced labour inputs. Anecdotal evidence suggests that of herringbone parlours installed in the last 5 years, some 70% have at least 36 stall with 18 milking units. Rotary parlours offer benefits for larger herds

(more than 500 cows) and the number of automatic milking systems is slowly increasing.

Table 5 Current position

Year	Herd size	Milk yield (litres/cow)	Number of cases/100 cows	Ave SCC (‘000/ml)*
1980	51	4,670	54.6	469
1990	67	5,145		322
2000	80	5,800	40-45	191
2008	114	7,400	47-65	197

Source: Various: Dairy Facts and Figures; Datum * GB

Continuing developments in in-line sampling

The possibilities offered by parlour automation and especially in automatic milking, continued to be explored. According to Graeme Mein of Australia, a series of focus group meetings held in all major dairy countries in 2003, listed on-line mastitis sampling as high priority in the control on mastitis (14). He considered that although most farmers will continue to milk in conventional milking systems for the next decade or so, there are increasing possibilities for automation including sophisticated equipment for disease management.

The ability to measure milk flow and record individual milk yield of each cow opens up the opportunity to measure other milk qualities during milking. As a result development work on in-line sensing has hitherto been driven by the need to control mastitis. Mein reported that many research groups have published results using a wide variety of on-line measurement principles related to SCC, conductivity and milk composition changes, but relatively few commercial products have resulted.

At the BMC conference in 2003, Eric Hillerton analyzed the progress made in testing for abnormal milk, or as the title of his paper implied, the search for the Holy Grail (9). A range of new technologies, made possible by automatic milking systems, are explored with some encouragement that they will speed up the early identification of mastitis.

Technical developments

For some time there had been a growing recognition of the importance of teat management in order that teats were always in optimum condition. Keeping housing and teats clean to reduce the bacterial load at the teat end hasn't always had the emphasis required. In response researches and specialists from a number of countries have joined together and formed "Teat Club International" in order to raise the profile of this key area of mastitis control. (15).

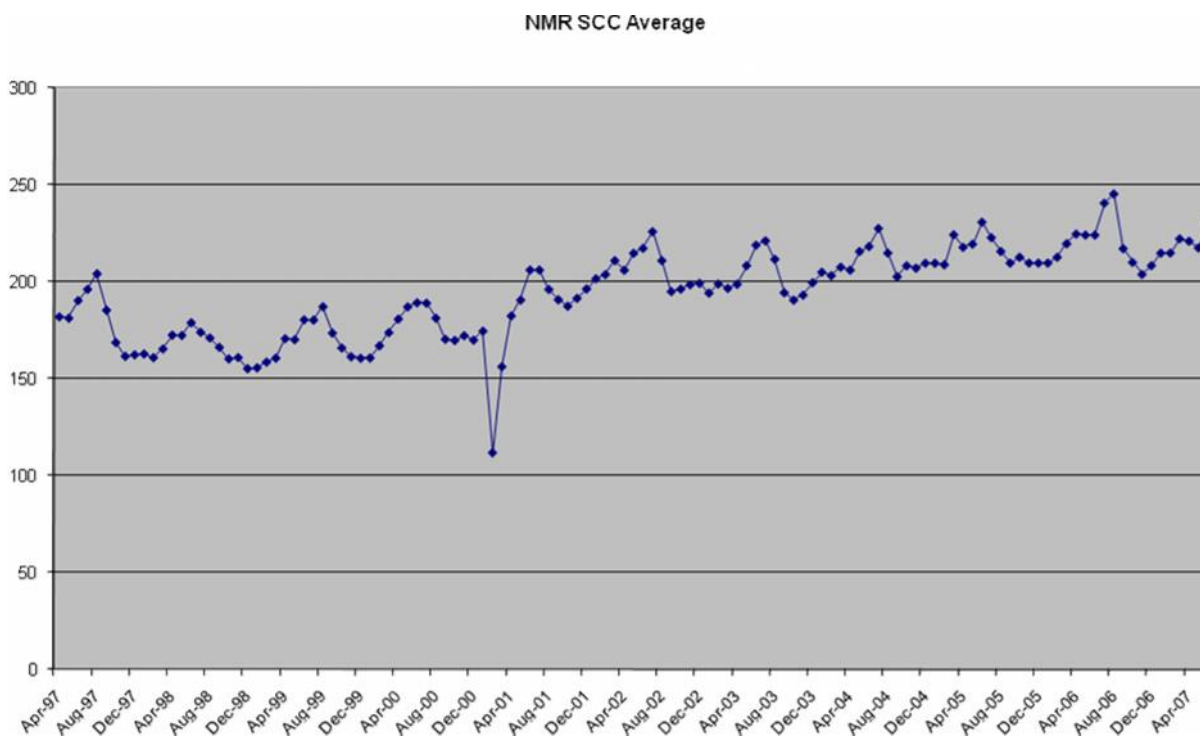
DISCUSSION

The above paragraphs have noted some of the technical developments aimed at early detection on mastitis thus leading to improvements in mastitis control. They have also attempted to track the progress in mastitis control as measured by the numbers of clinical cases and cell count levels against a background of a rapidly changing industry. Scant attention has been paid to the developments in the treatment of mastitis other than those recommended in the “five point plan”.

In terms of assessing progress, the point has to be made that although significant progress was made in the first thirty or so years since the basic control system of the five point plan was introduced, the overall picture today appears that both cell count and clinical case records indicate a deteriorating picture.

Data from National Milk Records (NMR) shows a disturbing upward trend in average cell counts over a ten year period.

Figure 1 NMR SCC averages 1997-2007



Source: NMR

The sudden drop around the end 2000 coincided with the outbreak of foot and mouth disease. Conclusions are dangerous to draw other than the culling of large number of dairy cows had a significant effect.

In terms of clinical mastitis, Bradley and others in 2007 (4) in a survey of 97 dairy farms in England and Wales reported the mean incidence of clinical

mastitis was 47 cases per 100 cow estimated from historic farm records but showed a greater figure of 71 cases per 100 cows from samples collected.

DairyCo have recently reported the results of a 100 herd UK wide survey showing that on average there are between 50 and 70 cases of clinical mastitis per 100 cows per year.

In summary, based on the evidence available, the average herd cell count has been reduced, albeit the trend appears to be in reverse with latest figures above 200.000 cells per ml, but there has been no improvement in the numbers of clinical cases. The current rate of between 50 and 70 cases of clinical mastitis per 100 cows has to be compared with the 120 cases prior to introduction of the five point plan.

At the 2008 BMC Jamie Leigh (13) reported that the species of bacteria involved in the disease have varied little since the implementation of the five point plan, but it had become clear that specialized sub-groups existed within each. It is now widely accepted that in recent years mastitis caused by environmental pathogens has become more common whilst the earlier control strategies targeted cow to cow infection. According to DairyCo, environmental mastitis is now responsible for four out of every five cases of clinical mastitis.

Control drivers and barriers

A major cause of the reduction of cell count levels during the 1980s and 1990s can in part be attributed to financial, legislative and market drivers. Herd bulk milk cell counts had to meet minimum standards if the milk was to find a buyer, and financial incentives were on offer for low cell count milk and good overall milk quality. On the other hand it can be speculated that the cost of clinical cases was either being ignored or underestimated, which is surprising considering the ample evidence available on costs of clinical mastitis.

Kite's Health and Culling Monitor for 2010 shows that clinical cases are increasing with 17% of cows culled for mastitis. They calculate direct costs of over £153 per case with total costs including extra risk amounting to over £255 per case. Kingshay estimate total costs at about £218 per cow treated with 11% of culling due to mastitis, second only to "not in calf" in the list of reasons for culling. The financial implications are significant for all herds and surely an adequate driver to bring about change.

It is easy to make a judgment that the well established basic mastitis control methods are not being applied. It may well be that the pathogens causing mastitis are more difficult to treat than they were. But has the economic need to reduce labour and aim for faster cow throughput during milking become a barrier to implementing good hygiene during milking? Is adequate attention being paid to the condition of teats and teat preparation prior to

milking, is thorough washing and drying of teats being overlooked due to the lack of time and is post milking teat disinfection being done correctly?

A review of the papers given at this conference in recent years plus numerous press articles refer to the need to “keep cubicle beds dry”; a key element of husbandry and management promoted over thirty year ago. Why hasn’t this and other messages got across to the industry?

SUMMARY

It appears that we are at an important crossroad in the progress of mastitis control in the UK. Considerable progress has been made in reducing cell counts but the clinical picture remains disappointing. Whilst clinical cases caused by environmental pathogens are presenting new challenges, perhaps the industry as a whole needs to re-assess if the well recognized control methods are being adequately implemented. If they are not, we should reflect over the reasons why, and why veterinarians, consultants and others must seemingly have to keep banging on the same drum.

Brian Angel of ADAS in paper to the 2007 conference (1) asked if the industry had an approach to communication that was accessible by the target groups, was it believable and related to producers aspirations. These questions need addressing because the lessons of history and the gains of experience should not be lost.

The process of political change and re-structuring will continue. Milk buyers and consumers will show far more interest in the quality of the product and the health and welfare of the cows that produce it. Many dairy farmers are making commitment and huge financial investment in the future, and yet, in the case of this disease, it appears we may not be making the progress we should be.

This brief review of the progress of mastitis control suggests that many tools for controlling mastitis are available, there is sound technical advice on offer, and more new high level technology will come along. Yet, as more than one Prime Minister of this country in recent years has said, there is also perhaps a need to go back to basics.

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NOTES

THE DAIRYCO MASTITIS CONTROL PLAN – WHERE ARE WE?

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SUMMARY

The DairyCo Mastitis Control Plan (DMCP) (formerly known as the MDC Plan) was conceived and tested in an intervention study in 2003-4; which demonstrated a significant reduction in mastitis in herds implementing the Plan compared to those not implementing mastitis control using the same 'focussed' approach (1,3). Following the initial research a pilot study was undertaken to investigate the feasibility of rolling out the control plan on a national basis (2). Subsequently an initiative was launched in early 2009 to facilitate 'roll out' of the Plan across the UK. This paper is designed to update the reader on progress with the national initiative, the concept of which was first outlined at the 2007 conference. The DMCP initiative demonstrates that a large scale, co-ordinated response to improve the control of major endemic disease is possible in the UK.

INTRODUCTION

The principle of the DMCP is that it should be possible, by gaining a better insight and understanding of the mastitis epidemiology on an individual dairy unit, to target mastitis control measures more specifically and thereby assure more cost effective mastitis control. Following development of the Plan an intervention study was designed to test the hypothesis that this well specified plan would result in a reduction in disease incidence in herds with an above average incidence of clinical mastitis. This study demonstrated the utility of the Plan, identifying a significant reduction in disease incidence (3).

Following the initial research a pilot study was undertaken in the South of England where the feasibility of 'roll out' of the Plan was investigated. This culminated in the launch of a national initiative in early 2009. The aim of this national initiative is to train veterinary surgeons and consultants in the principles of the Plan and facilitate implementation of the Plan on a cohort (approximately 750) of UK dairy farms.

In order to become involved, potential plan users undergo two days of training; the first day is spent covering the principles of the Plan and outlining the electronic resources available. Plan Users are then expected to implement the Plan on at least one farm prior to the second training day during which farm specific approaches are discussed using herds in which the plan has been implemented as examples. Thereafter Plan Users are

supported by the authors with the aim of facilitating implementation of the Plan on subsequent farms.

Plan Users can be identified using the Plan website (<http://www.mastitiscontrolplan.co.uk>) and have access to Plan resources contingent on attending an annual 'refresher' day. This involves feedback on the initiative and in depth discussion of specific areas of mastitis control; the aim being to develop a model for sustaining the initiative beyond the initial 3 year phase.

WHERE ARE WE?

Initial Training

The first training course was held in Somerset in late April 2009, with five further courses being held at locations around the UK over the subsequent 13 months. To date 163 Plan Users have been trained, comprising 132 veterinary surgeons and 31 consultants. A further 17 delegates are registered for the seventh course to be held in Somerset in October 2010. In addition a further 50 veterinary surgeons and 8 consultants have expressed an interest in participating in the scheme. It is anticipated that further courses will be arranged to fulfil demand in the coming months. Plan Users are distributed throughout the UK and their locations can be viewed on an interactive map on the Plan website (http://www.mastitiscontrolplan.co.uk/index.php?option=com_content&view=article&id=17&Itemid=71).

The first 'Update' courses have now been scheduled with over 90% of Plan Users currently choosing to remain 'enrolled' in the initiative.

Implementation

As of the end of August 2010 a total of 403 farms had been enrolled in the scheme and were at varying points in the process of implementation.

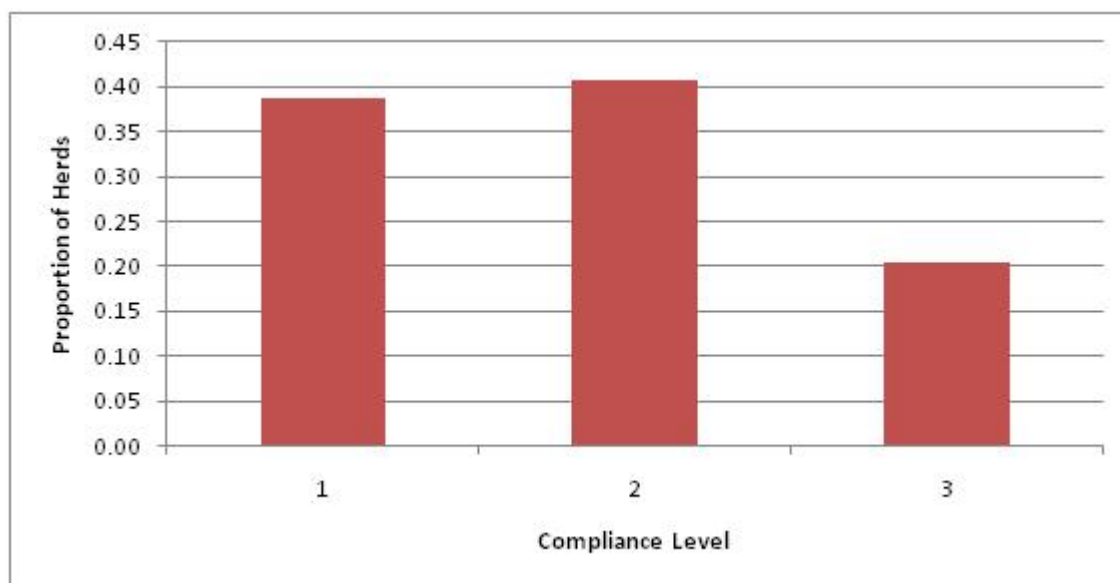
Preliminary Assessment of Progress

Data following 12 months of implementation of the plan has been submitted from a total of 47 herds by the end of August 2010. It is important for the reader to be aware that this data is preliminary and should be interpreted with caution. All figures are based on rolling 12 month statistics.

Herds have been subjectively scored, by the implementing Plan User, according to the level of compliance with the Plan. Herds have been allocated a score of 1, 2 or 3 indicating implementation of less than 1/3rd, greater than 1/3rd but less than 2/3rds or greater than 2/3rds of the recommendations respectively. The level of compliance of herds from which 12 months data has been received, in which compliance level is currently known, is illustrated in Figure 1. Interestingly, only 23% of herds are

currently fully implementing the Plan (*i.e.* implementing >2/3rds of the recommendations) as put in place by the Plan User, illustrating the need for a better understanding of the obstacles to implementation and farmer motivation.

Figure 1. Illustration of the compliance category of herds from which data have been returned after 12 months of Plan implementation



The impact of the Plan on a number of key mastitis parameters are outlined in Table 1.

Table 1. Relative change (%) in rates of key mastitis parameters by level of compliance

Parameter	Mean Impact	Compliance 3	Compliance 2	Compliance 1
BMSCC	2.19	-6.64	1.75	8.19
LIMI	-14.57	-13.72	-13.07	-16.71
FCIR	-9.38	-17.69	-13.18	-0.15
DPNewIMI	-3.44	-19.31	-3.72	6.76
IRCM	-5.20	-5.34	-2.40	-8.76
IRCA	-12.31	-11.60	-11.00	-14.57

Key: BMSCC: Bulk milk somatic cell count
 LIMI: Rate of new infection in lactation (as measured by SCC)
 FCIR: Proportion of cows infected in early lactation (as measured by SCC)
 DPNewIMI: Rate of acquisition of new infection in the dry period (as measured by SCC)
 IRCM: Incidence rate of clinical mastitis
 IRCA: Incidence rate of cows affected by clinical mastitis (proportion of cows affected)

These figures need to be interpreted with care and in light of the fact that implementing the Plan is likely to result in an increase in recording (particularly of clinical mastitis) and therefore have a confounding effect on any benefits of Plan implementation. In addition figures are based on rolling 12 month statistics and it is important to note that it may take some time for certain control measures to impact outcomes. Finally, Plans are targeted at specific areas of mastitis control (e.g. dry period or lactating period) and more in depth analysis, focusing on outcomes related to the Plan implemented would be more appropriate; this will be possible when more datasets are available.

Effects of the Plan on bulk milk SCC, the rate of new infection in lactation, in the dry period and the proportion of cows affected with mastitis are also illustrated in Figures 2, 3, 4 and 5 respectively.

Figure 2 Illustration of Impact of the Plan on Bulk Milk SCC

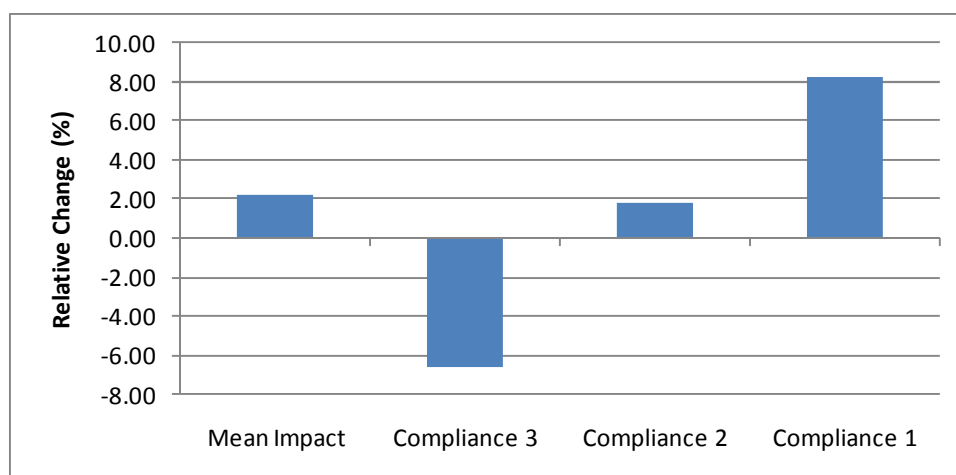


Figure 3. Illustration of Impact of the Rate of New Infection in Lactation

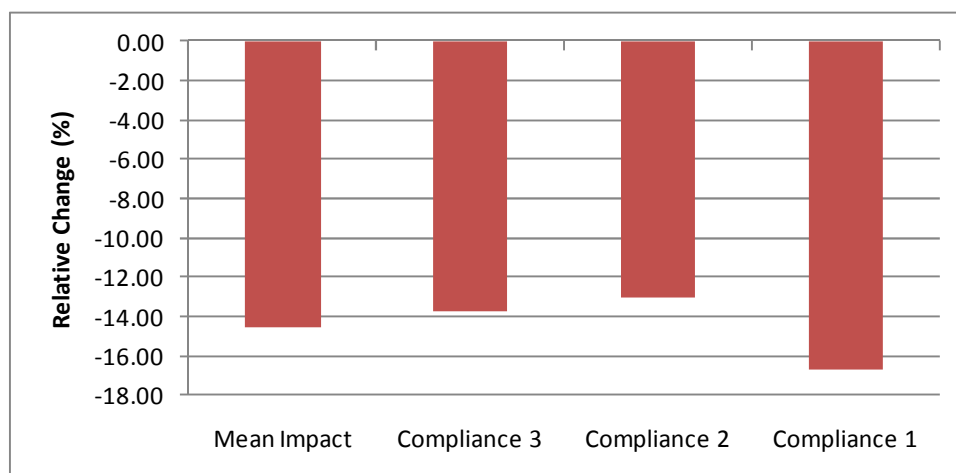


Figure 4. Illustration of Impact of the Rate of New Infection in the Dry Period

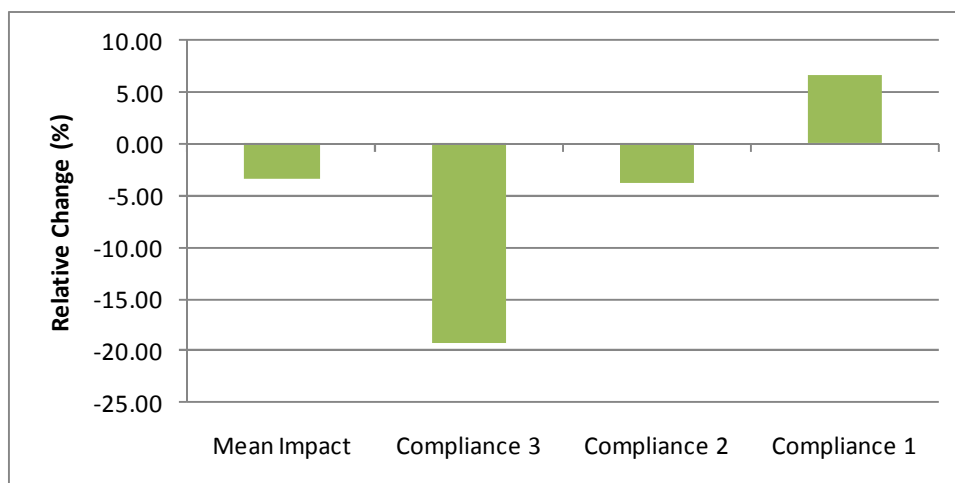
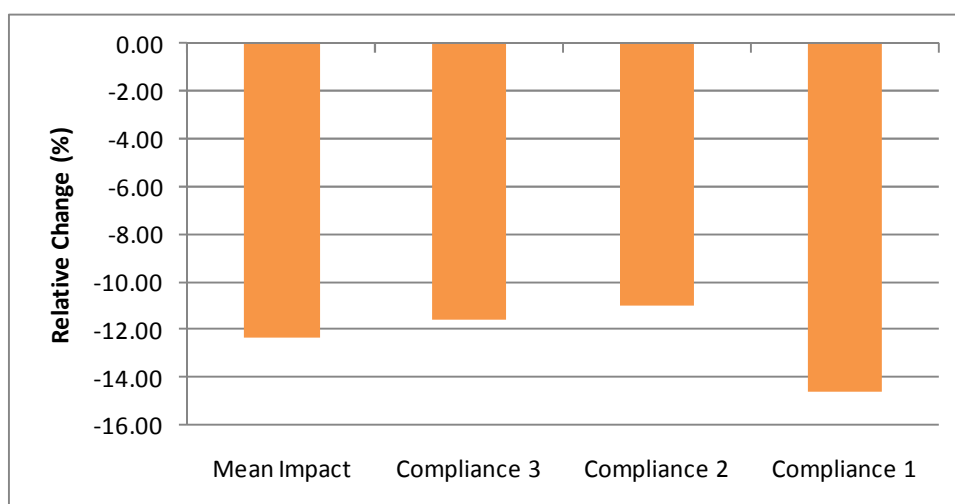


Figure 5 Illustration of Impact of the Plan on the Proportion of Cows Affected by Clinical Mastitis



DISCUSSION AND CONCLUSIONS

It is too early in the national roll out of the DMCP to draw firm conclusions about its effect or success. However, interim analysis appears promising and suggests that the Plan is having an impact, though care should be taken in drawing firm conclusions in the absence of complete data and an ability to undertake robust comparisons.

The number of Plan Users trained and herds enrolled are both currently ahead of target and are progressing well. Inevitably the longer term durability of the initiative will be key and will only become clear over time. It will only succeed with wholehearted industry support!

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ACKNOWLEDGEMENTS

The authors would like to thank DairyCo, funders of the National Mastitis Control Plan and the vets and consultants now enthusiastically engaging in the initiative.

NOTES

THE DAIRYCO MASTITIS CONTROL PLAN – A PRACTITIONER USER’S VIEW

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LIKES

1. The Plan draws together all aspects involved in mastitis (clinical, subclinical/somatic cell count) allowing a structured approach to its definition, differential diagnosis, investigation and improvement.
2. Data analysis – an in-depth view with dynamism allowing evaluation of levels and underlying trends.
3. Flexibility – can focus in on one or more sections where appropriate.
4. Doesn't remove individual clinical judgement 'on Farm'.
5. Allows for appropriate cows to be sampled for bacteriology.
6. Side Benefits – correction of worming programmes, calf health and management, 'early' dry cow management.
7. Cost calculator – allows a sensible look at real costs and the potential savings if improvements made.
8. Website – sharing/seeking opinions especially on non-straight forward situations e.g. mixture of contagious and environmental causes of infection in the dry period.
9. Involvement of young veterinary graduates – “in house” training in gathering information, looking at the whole farm situation and cost benefit analysis.

DISLIKES

1. Must/could/should – can generate strong opinions in discussion (EBFM versus EBVM i.e. 'evidence'- based farmer medicine versus evidence – based veterinary medicine) sometimes making compliance difficult to achieve.
2. Length of Plan – can be daunting to some clients, especially if left to fill in themselves in order to speed up the process/reduce the cost.

3. Amount of Analysis and Data presented – again can be daunting so I tend to concentrate on two or three key graphs/charts.
4. Benchmarking – can be demoralising and lead to dropping off of compliance/interest; I tend to benchmark within farm from period to period.

OBSERVATIONS

1. Still enormous confusion in some farmers' minds about the difference between a clinical case and a new infection on a SCC basis. More education is required here.
2. Different farms require different approaches – there is no one size fits all – there is still room for the 'art' of veterinary practice (often facilitates compliance) amongst the 'science'.
3. Success and improvement depends more on enthusiasm and willingness to try by both vet AND farmer than cost or any other factors.
4. Personally, I tend to use it in conjunction with Interherd/Totalvet/Herd Companion for on-going monitoring.
5. Always worth looking at farms which appear not to have a SCC or clinical mastitis problem – it can be surprising what one finds!

NOTES

Due to unforeseen circumstances Dan Norris is unable to present his paper. We are grateful to Jamie Montgomery, Manor Farm, North Cadbury, Yeovil, Somerset for giving his views on the DairyCo Mastitis Control programme from a farmer's perspective

THE DAIRYCO MASTITIS CONTROL PLAN – A FARMER'S PERSPECTIVE

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My vet mentioned this approach when I sought his opinion about the cost effectiveness of installing a cluster flush system in May 2009.

The herd's somatic cell count was rising steadily from below 200 in 2008 to 309 at the NMR recording of April 2009 and my vet had mentioned the rising first recording infection rate (averaging 28% in the recordings for 2009 at that time). We also had recorded 71 cases of clinical mastitis per 100 cows in the previous 12 months (Interherd records) with many cases being recurrent – *Strep. uberis* had always been the predominant problem on milk sampling of these.

The herd was already InterHerd recorded through the veterinary practice bureau so data collection was already in place. I found the questionnaire only a slight inconvenience as many sections were left to my vet to discover on his own wandering around, inspecting the buildings, milking routine, etc.

Incredibly, we did discover that our worming regime needed modification along the way. However, the main findings were *Strep. uberis* and *Staph. aureus* in 80% of both clinical cases of mastitis sampled and high cell count cases sampled. Additionally, there were issues with both dry cow/transition cow housing and the possible suitability of the dry cow product used: only nine of the previous 17 infected cows at drying off had re-calved uninfected.

The cluster flushing system was installed; transition accommodation was changed to an old cubicle house with a lower stocking rate and longer and wider cubicles for 365 days a year; the dry cow product was changed to a cephalosporin for cows with a SCC of more than 200 at the last recording before drying off; an internal teat sealant was used in combination with the dry cow products; cows with SCC's greater than 200 in the two or three recordings prior to drying off are sampled for bacteriology one week ahead of drying off and selective antibiotic therapy given by injection at drying off if *Staph. aureus* isolated; A three day course of antibiotic injections was to be used on all recurrent cases of mastitis in addition to intramammary tubes.

The herd's cell count has been below 200 since September 2009, the average first recording infection rate is 10% and the clinical case rate has dropped to 49 cases per 100 cows.

My vet continues to monitor the herd's "udder health" performance every three to six months via Interherd and Totalvet as well as via the monthly Herd Companion reports from NMR, highlighting specific cows to sample either prior to drying off or because of persistent high SCC without a clinical case of mastitis.

While the herd has shown an encouraging improvement, we cannot rest on our laurels and still need to reduce the clinical case rate further.

NOTES

STREPTOCOCCUS UBERIS – ARE WE NEARER AN EFFECTIVE CONTROL?

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SUMMARY

Various approaches have been used to generate experimental vaccines against *S.uberis*; these have often shown promise but have failed to progress to a useful, commercially available product due to their lack of cross protection against a wide range of strains. Recent research has identified three proteins on the surface of *S.uberis* that appear to play a major role in the pathogenesis of mastitis. Genetically manipulated strains unable to produce these proteins were markedly attenuated. Generation of specific antibodies capable of inhibiting the function of these would be predicted to impair and possibly ablate virulence of *S.uberis*. The identification of defined components with a defined role in virulence allows prediction of the likelihood of cross protection against the range of strains present.

Determination of the genes required by *S.uberis* for virulence will also allow identification of field isolates in which these are absent. These strains may be equally able to colonise the gut and survive in the environment, but would be predicted to be less able to infect the mammary gland. It is interesting to speculate if such strains could form the basis of a disease preventing probiotic.

INTRODUCTION

The control of mastitis arising from infection of the bovine mammary gland by bacteria can be achieved at various levels. These include preventing - exposure to the pathogen, penetration of the teat canal, colonisation of the mammary gland or the events *in vivo* that induce the signs of disease.

If we consider the whole processes involved in the case of *S.uberis*, we can assume that the organism must be seeded into the environment (most likely via faeces), it must survive long enough within the environment to contaminate the teat end (12), from there it must penetrate the teat canal, multiply within the secretion (and/or tissues), avoid an array of pre-existing and induced innate host defences and ultimately induce (or cause the host to induce) the signs of disease (9). Intervention at any one of these stages may be sufficient to reduce or prevent mastitis due to this bacterium. Given the how disease patterns have changed following implementation of the five-point control plan, it can be assumed that ability to successfully complete all of these tasks on a regular basis is limited to very few bacterial species.

The different attributes required to achieve this are embedded within their respective bacterial genomes. Genomic sequence data are becoming more accessible on a routine basis. 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 at little cost. 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 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.

S.uberis encodes the ability to successfully achieve all of the necessary stages (gut colonisation, environmental survival, resistance to innate immunity and induction of mastitis) within a single circular chromosome containing a little over 1700 genes (16). It is part of the job of scientists in this area to determine which genes are required for each process and then envisage a way to exploit this information in the control of disease. An obvious approach is to use the information to formulate vaccines directed against colonisation or induction of the disease. I will discuss the advances we have made in this area and how new technologies are being used to enable rapid progress. However, vaccines may not be the only possible solution and I will briefly highlight another, as yet under-developed and under-resourced, area that may offer other possibilities for controlling mastitis caused by *S.uberis*.

VACCINES

A number of attempts have been made to formulate experimental vaccines against *S.uberis*. These have included the use of simple killed bacterins (3), live-antigens (4, 5) and crude sub-unit preparations (8). Although many are effective experimentally against a limited number of strains, none have been deemed sufficiently effective to make their way through the commercialisation process and into the real world. A common problem has been lack of cross protection against the vast number of strains of this bacterium that are present and able to cause the disease (3, 4). The issue underpinning this observation is that such vaccines lack definition of the key, protective components. Consequently, when they fail to protect it is not possible to determine why; similarly, it is not possible to predict the level of coverage that any single crude vaccine may afford.

An additional problem with vaccines against mastitis is that such products have to confer protection without induction of the signs of disease. Since the

disease is an over elaboration, or inappropriate version, of the host response to infection we have to consider if we are asking the impossible.

I recall when joining the Milking and Mastitis Centre at the Institute for Animal Health at Compton in 1987 I was offered one piece of advice from a departing member of staff who had finally thrown in the towel. This was (and I paraphrase), "...there is no solution to this disease; mastitis (the over elaborate host response) is required to prevent mastitis, so find some aspect that you enjoy and carry on with that, but don't try to solve it."

Several observations made during the investigation of experimental vaccines aimed at *S.uberis* have shown the basis of this statement to be incorrect. Most notably, live vaccines and crude preparations of *S.uberis* culture fluid induced protection (and often clearance of the infection) in the absence of any clinical signs (4, 5, 8). It was against this background that an approach was taken to identify the components that were required for *S. uberis* to cause infection and disease in the mammary gland. These studies were conducted under the (reasonable) assumption that if we could induce an immune (antibody) response that was able to inhibit the function of molecules that play a critical role in disease pathogenesis we would substantially reduce the likelihood of infection and disease.

A number of studies have been undertaken which exploit the existence of the genome sequence of *S. uberis*, the ability to create mutations within the genome and access to experimental models of infection (11). This approach has revealed that *S. uberis* needs neither the hyaluronic acid capsule (2) nor the plasminogen activator (PauA) for infection of the mammary gland (15). Mutant strains in which these putative virulence genes were inactivated were equally virulent as the genetically intact strain. Conversely, a protein (MtuA) responsible for uptake of manganese in environments in which the concentration of this metal ion is low was essential for infection of the lactating mammary gland (13). Unfortunately, the MtuA protein is not suitable as a vaccine as it is located beneath the cell wall, thus rendering it inaccessible to antibody molecules (6). Therefore, even if an antibody was raised that was able to prevent the biochemical function MtuA it would be unable to bind to this protein in intact bacterial cells.

Recently, a study has been undertaken that has investigated the requirement by *S.uberis* for a number of different proteins that are attached to the cell surface. This study was instigated by the observation that a mutant strain carrying a mutation within the gene encoding the enzyme sortase (SrtA), responsible for anchoring proteins to the outer surface of the bacterial cell wall, was less virulent than the otherwise identical strain in which the gene was intact (10). Sortase is responsible for attaching a discrete subset of proteins, with a variety of different functions, to the bacterial surface. An investigation of the surface proteins present on *S.uberis* identified ten that were anchored through the activity of SrtA [1]. Detection of these proteins enabled identification of their encoding genes and in turn permitted production of ten corresponding mutant strains each

lacking the ability to produce one of the SrtA anchored proteins. These strains were used to challenge dairy cattle to determine which, if any, were less able to infect the mammary gland and induce mastitis (10).

Three sortase-anchored proteins were identified that appeared to play a role in the disease process. Their likely function was identified from each amino acid sequence. This indicated that the protein encoded by the gene sub1154 is a protease similar to those encoded by other streptococci that degrade proteins involved in the innate immune response. The protein encoded by sub1095 is a collagen like protein, and that from sub0145, a protein with similarity to another shown previously in *S.uberis* to bind lactoferrin.

Whilst the search for a greater array of bacterial molecules with a role in the infection and disease process will continue, the fate of the proteins identified so far is about to diverge. There is now enough information to conduct a series of proof of concept studies, in which the induction of an immune response to these molecules can be optimised, and subsequently the protective nature this response determined.

On their alternate route, within the laboratory, the precise function of these proteins will be determined, the functional regions of the protein identified and this information will be cross-referenced to the sequence variation that is seen in other isolates worldwide. In so doing, we will extend our knowledge of the interactions between *S.uberis* and the dairy cow that result in infection and disease. It will also be possible to start to predict whether the immune responses obtained are likely to protect against the whole range of *S.uberis* that are present.

It is possible that none of the proteins identified to date actually make it as vaccine components. This might be a consequence of factors totally outside their utility as a protective antigen (e.g. cost of production, stability, sequence variation amongst strains etc). However, it is clear that technology has now been developed that will enable the rapid identification of proteins of *S.uberis* that have a definite role in disease pathogenesis and which are therefore likely to be useful as vaccine antigens. It is also clear from the standpoint of a researcher that even more powerful, precise and rapid technologies to enable the same are in the pipeline.

Consequently, we have moved from an empirical and rather slow approach to the identification antigens for inclusion in vaccine formulations, to an era of strategic, rapid approaches to identify defined vaccine components. In so doing it is hoped that we will be able to make predictions of the outcome of a given immune response and therefore what combinations of components may work well together and how these fit with the underlying genetics of the pathogen. How quickly we get from our current position to one of having a whole range of different and effective vaccine candidates entering this pipeline is now, more than ever before, dependant on the commitment of those that perform, and those that fund, such research.

OTHER APPROACHES

The ability to acquire DNA sequence data rapidly and cheaply enables cross referencing of data for a huge array of genes from a large number of isolates. In the case of *S.uberis* I will briefly outline one possible approach where this could impact disease control in the future.

As mentioned previously, *S.uberis* undergoes a tortuous route to reach the bovine mammary gland and cause mastitis; faecal shedding into the environment, survival in this niche, contact with the teat end, penetration of the teat canal, growth in milk (not a particularly good growth medium for *S.uberis*), resisting innate chemical and cellular host defences and finally reaching a level of colonisation capable of inducing a severe inflammatory disease in the host. The difference in the respective environments in which *S.uberis* will find itself is so great that it is inconceivable that the same genes are required for all the different interactions that must be involved. Therefore, it is reasonable to assume that there is a specialised requirement for different sub-sets of genes in each niche (16). Thus it can be hypothesised that loss of a gene specifically required for infection of the mammary gland would not necessarily have a consequence to *S.uberis* residing in the gut of a cow. Indeed we have shown experimentally that loss of function of such genes has little consequence to the viability of the strain (10). Certain genes identified as important, or essential, for infection of the lactating gland have been shown to be absent from some field isolates. For example, a gene essential for the function of the manganese uptake system (Mtu), was lacking from some strains of *S.uberis* (7). Similarly, one of the genes identified recently as important for pathogenesis, sub1095; encoding a collagen like protein was shown to be absent in a collection of strains obtained from asymptomatic cows (14, Tomita; unpublished). Therefore, not all strains that exist would be predicted to be equally able to follow the route from gut to the mammary gland and some would be predicted to simply cycle through the gut and environment and infection of the mammary gland, if it were to happen, would be inconsequential.

Following on this line of argument, it can be assumed that the isolates of *S.uberis* that fill the freezers of researchers around the world, which were largely obtained from the clinically and sub-clinically diseased mammary glands of cattle (and sheep), represent only the sub-set of strains that made it through this whole process. It can be hypothesised that the gut also contains strains of *S.uberis* that are avirulent with respect to the mammary gland.

The same scientific procedures and approaches that have enabled us to start to identify genes and proteins responsible for colonisation and disease in the mammary gland can also be applied to determine which genes are required for other stages of this journey. This would enable us to configure treatments to prevent colonisation of the gut by *S.uberis*, thus breaking the cycle of shedding, environmental contamination, etc. Alternately, combining

the information from these data sets would enable us to identify which strains are able to colonise the gut well, but unable to cause disease in the mammary gland. It is then not a wild leap of imagination to assume that a herd shedding only that type of strain would suffer considerably less mastitis due to intramammary infection by *S.uberis*.

CONCLUSIONS

Modern genetic technologies have been applied to *S.uberis* and these have been exploited to identify proteins that appear to be required for infection of the lactating bovine mammary gland and induction of mastitis. Some of these are suitable candidates for pursuing as potential vaccine antigens. The rapid progression of genetic technologies and their application in suitable models of disease is likely to permit identification of further vaccine candidates from this bacterium. Similar technology is likely to increase our knowledge of the underlying genetics of this pathogen that impact all stages of its commensal existence and pathogenic behaviour. Strategic exploitation of these data may ultimately allow alternative/additional approaches to control of intramammary infection by *S.uberis* by controlling exposure of the dairy cow to pathogenic strains.

So in answer to the question, "...are we nearer an effective control?"; we are, and we think we know how to get to where we want to be, but we are not there yet.

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ACKNOWLEDGEMENTS

The author would like to acknowledge the financial support of Defra and BBSRC in support of this research through the past 20 years and would like to thank all those co-workers and collaborators that have been in the past and are at present active in this research effort.

NOTES

PCR TECHNOLOGY – IS IT THE FUTURE?

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SUMMARY

Mastitis is the most frequently occurring and economically most important infectious disease in dairy cattle (2). It is estimated to cost the GB dairy industry in the region of £200 million per year.

Recent developments in diagnostic technology mean there are new tools available for the detection of mastitis pathogens in milk using real-time Polymerase Chain Reaction (rtPCR). Whilst there is still much to learn about this technology it provides a genuine alternative to traditional culture methods for the detection of mastitis causing pathogens in milk samples.

Using new techniques producers and their vets can look to tackle this disease in a timely, targeted and cost-effective fashion leading to a reduction of the incidence of mastitis in the GB dairy herd.

INTRODUCTION

Mastitis refers to infection and inflammation of the mammary gland and is a major endemic disease of dairy cattle in Great Britain. *Streptococcus uberis*, *Escherichia coli* and *Staphylococcus aureus* are probably the most common mastitis pathogens in the GB dairy herd, however there are upwards of 150 different pathogens which have been identified as potential causes of mastitis in dairy cattle.

The majority of mastitis cases are caused by one primary pathogen (6) with just ten bacterial species or species groups accounting for over 95 percent of all clinical and subclinical infections (3).

Understanding the pathogens responsible for the cause of mastitis in the dairy herd is of paramount importance when tackling this disease.

Traditionally, mastitis detection has been carried out using bacterial culture methods. More recently rtPCR has been identified as a method to complement or even replace these culture methods.

INDICATORS OF MASTITIS

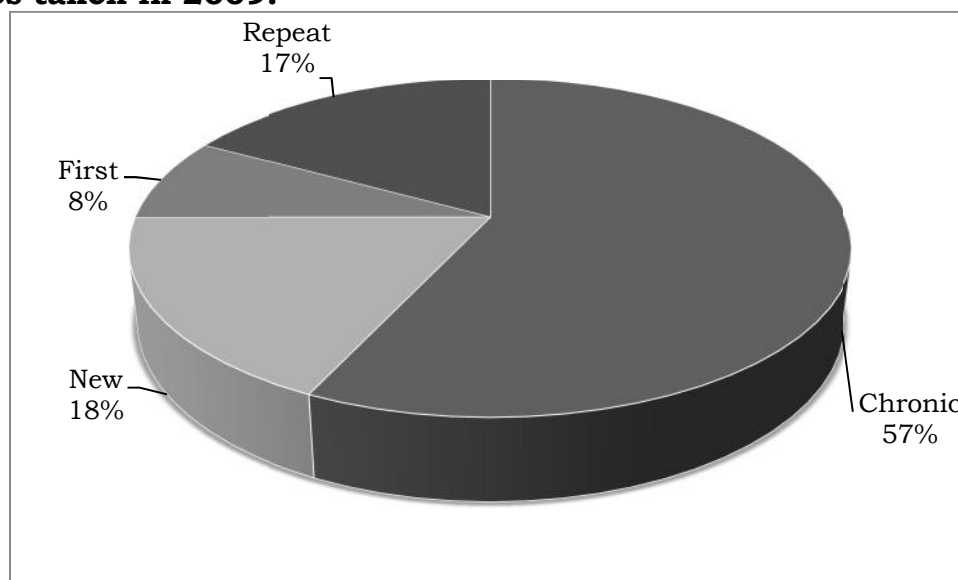
Bulk milk somatic cell counts (BMSCC) provide a broad indication of the general level of udder health. In the 1950s average bulk milk cell counts

were in excess of 600,000 cells/ml, falling to around 200,000 by the mid 1990s. The average NMR somatic cell count was 236,000 at the beginning of August 2010 compared with 202,000 in August 2009, suggesting that perhaps cell counts are on the rise again.

A study into the effect of individual cow cell counts on the BMSCC (Hanks & Watson, 2009, unpublished data) showed that if the level of chronic cows (cows with two consecutive milk recordings over 200,000 cells/ml) in the herd exceeded 10%, the bulk tank cell count would almost certainly be above 200,000. The mean percentage of chronic cows in NMR recorded herds is 14 percent suggesting most herds just have too many chronic cows to maintain BMSCC below 200,000.

This is further supported when analysing the number of cows falling into the chronic category at the monthly milk recording (Figure 1).

Figure 1. Distribution of high SCC categories from all NMR milk samples taken in 2009.



MASTITIS DETECTION IN THE GB DAIRY HERD

Traditionally the collection of sterile milk samples for bacteriological culture has been the method through which to definitively diagnose infectious mastitis and identify the causative organism.

Data has been published over the years providing an estimate of the prevalence of pathogens in the GB dairy herd (Table 1). Data indicates that *Streptococcus uberis*, *Escherichia coli* and *Staphylococcus aureus* are the most common mastitis causing pathogens in the GB herd.

More recently PCR technology has been developed to provide a commercially viable alternative to culture testing for the detection of mastitis pathogens.

These tests are designed to detect the DNA of specific mastitis pathogens in the milk sample.

Table 1. All incidents of mastitis in cattle in Great Britain as a percentage of total mastitis diagnoses in 2002 (n = 6517)

Pathogen	Prevalence
<i>Streptococcus uberis</i>	22%
<i>Staphylococcus aureus</i>	19%
<i>Escherichia coli</i>	21%
<i>Streptococcus dysgalactiae</i>	6%
<i>Streptococcus agalactiae</i>	1%
No growth	19%
Other	12%

Source: VIDA annual report 2002

PCR testing is available from two companies in the UK (National Milk Records Plc and Biobest Laboratories). The services offered by these laboratories vary, however the test assay is based on that supplied by Thermo-Fisher (formally Finnzymes) in both instances. This test system is now used in over 10 major dairy countries world-wide including Canada, USA, Scandinavia and Australia.

rtPCR technology has a number of significant advantages over traditional culture methods (Table 2) and as such should be considered carefully as a tool to for the purpose of mastitis detection.

The high level of ‘no growths’ seen in cultured samples is frustrating and costly to producers and vets. rtPCR is estimated to provide a bacteriological diagnosis for almost half of the cases where conventional culture results are negative (1, 4) which provides producers with more confidence in being able to address issues through PCR testing rather than culture.

Table 2. Benefits of PCR over traditional plate culture methods

rtPCR	Culture
Rapid diagnostic method. Start to finish <4 hours in the laboratory	Slow growth - takes >24 hours to obtain a presumptive result
Identifies 12 targets and penicillin resistance in one suite	Most culture suites identify just a few pathogens
Able to use preserved milk and growth inhibited /dead bacterial cells	Fresh sample required to grow live cells on agar plate
Very high sensitivity and specificity	Subjective nature of culture method can lead to ambiguity in results
Very few ‘no growths’ with detection of DNA in milk samples	Up to 50% of clinical mastitis samples present as ‘no growths’

The Pathoproof assay has an analytical sensitivity and specificity of 100% (references). The test suite comprises *Staphylococcus aureus*, Coagulase negative *staphylococci* (CNS), *Streptococcus agalactiae*, *Streptococcus dysgalactiae*, *Streptococcus uberis*, *Escherichia coli*, *Corynebacterium bovis*,

Enterococcus faecal & *E. Faecium*, *Klebsiella*, *Serratia marcescens*, *Arcanobacterium pyogenes*, *Peptostreptococcus indolicus* and the *Staphylococcal* β -lactamase gene responsible for penicillin resistance.

Presence or absence of each pathogen is provided in reports along with a semi-quantitative level for pathogens present of low, medium or high (+/++/+++).

PRELIMINARY PCR DATA

There is a growing data set for samples tested using PCR and an overview of the data to date is reported below. In the future it is hoped these data can be used to provide surveillance figures for the prevalence of pathogens in the GB dairy herd with particular emphasis on seasonal or regional variance.

Based on data for 1194 individual cow milk samples, the most common pathogen identified using PCR technology was CNS, present in 62 percent of samples. However this was at a low level in 40 percent of samples suggesting that because CNS is found on teat skin and in the streak canal, they are a common contaminant of milk samples.

In terms of pathogenic bacteria, *Strep. uberis* was found in 42 percent of individual milk samples, *E.coli* in 25 percent of samples and *Staph. aureus* in 27 percent of bulk milk samples.

Table 3. Prevalence of pathogens identified at a medium & high level using PCR technology. (Figures in parenthesis show percentage prevalence of pathogens identified at a low, medium and high level)

Pathogen	% Prevalence Individual Cow (n=1194)	% Prevalence Bulk Milk (n=1625)
<i>Staphylococcus aureus</i>	12 (27)	6 (61)
of which +ve for beta-lactamase	(77)	(86)
Coagulase negative staphylococci (CNS)	22 (62)	27 (69)
of which +ve for beta-lactamase	(59)	(80)
<i>Streptococcus agalactiae</i>	<1 (1)	1.5 (3)
<i>Streptococcus dysgalactiae</i>	7 (17)	16 (70)
<i>Streptococcus uberis</i>	29 (39)	61 (89)
<i>Escherichia coli</i>	15 (25)	11 (32)
<i>Corynebacterium bovis</i>	4 (43)	<1 (69)
<i>Enterococcus faecal</i> & <i>E. Faecium</i>	3 (21)	3 (51)
<i>Klebsiella</i>	3 (6)	1 (8)
<i>Serratia marcescens</i>	1.5 (2)	<1 (1)
<i>A. pyogenes</i> & <i>P. indolicus</i>	2 (17)	3 (57)

N.B. Multiple pathogens are often found in any given sample. The data has not been analysed to remove these multiple pathogens and therefore figures do not represent main or causative agent merely pathogens identified in all samples.

The level of *Staph. aureus* is perhaps significant given its highly contagious nature. Data from the VIDA showed 47% of *Staph. aureus* isolates cultured were resistant to penicillin using the disc diffusion method (5). Our data shows that the beta-lactamase gene was present in 77 percent of samples which is significantly higher than the VIDA data suggests. This is valuable information for producers and vets in making treatment decisions for these cows. Further analysis and investigation is required to determine the reason behind these discrepancies but much will be due to the difference in the nature of the test method used.

Aggregate data from bulk milk samples shows a significantly higher level of pathogen than observed at the individual cow sample level. This is to be expected given the nature of the PCR test however further data analysis is required to identify trends in pathogen groups regularly occurring on farm.

THE USE OF PCR DATA FOR MASTITIS CONTROL

The NIRD/CVL Five Point Plan is probably the most recognised and widely used approach to mastitis control in the GB dairy herd. Three major areas of focus in the plan are:

- Prompt identification and treatment of clinical cases
- Dry cow management and therapy
- Culling of chronically infected cows.

The use of PCR technology for mastitis detection proves a valuable tool in implementing these areas of the Five Point Plan.

If producers and their vets can accurately identify problem cows and the specific pathogens responsible for mastitis, a more targeted approach can be taken to treatment. Excessive use of antimicrobials in the dairy herd is of concern to the industry as well as consumers and PCR has the potential to significantly improve the efficacy of such drugs through a more informed approach to treatment.

In addition to this, the use of PCR for surveillance of farm specific pathogens enables accurate and effective management and treatment of dry cows on the farm.

Traditional culture methods identify the most common pathogen found in the milk sample but often overlook other issues which may be lurking on the farm. By using PCR, producers and their advisers are able to employ a more holistic approach to mastitis management. Taking into account all pathogens found in the samples, be they causative or contaminant, enables a better understanding of the risks to which the herd is exposed.

Bulk milk testing provides a starting point for any investigation into raised BMSCC in the herd. In some instances bulk milk samples contain a significant level of multiple pathogens. Results can be analysed to identify the likely origin of each pathogen and where possible steps taken to reduce the exposure of cows to these pathogens on the unit. For example, a reasonable level of *Streptococcus dysgalactiae* in the bulk sample, whilst it may not be responsible for a mastitis outbreak, may spark a discussion on teat condition and parlour functionality and this is valuable information which may previously have been over looked in a more traditional bacteriology investigation.

There are of course some limitations to PCR in that it will not currently detect yeasts & moulds, Bacillus, Pseudomonas or Pasteurella. Further investigative work can be carried out on samples producing 'no growth' results on PCR using alternative methodology.

CONCLUSIONS

Culture-negative milk samples represent a large proportion of analyses in conventional bacteriology and are frustrating for the laboratory, the producer and the vet. It is therefore unsurprising that bacteriology is not routinely used by more producers.

New PCR services provide a genuine alternative to bacterial culture however there is still much to be learnt about the use of the results in practice. A growing data set will enable service providers and the industry to tailor the use of samples appropriately and to ensure effectiveness of the information provided to the producer.

Routine use of PCR technology for the detection of mastitis in milk samples has the ability to enable a more focused and motivated approach mastitis control in the GB dairy herd.

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NOTES

THE NATIONAL MASTITIS SURVEY

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SUMMARY

This paper considers the outcome of two postal surveys of dairy farmers. The survey response represents around one tenth of the dairy farms in the UK. Many of the responses provide confirmation of the generally accepted and often quoted 'normals' of management on dairy farms. However new light is cast on some important aspects of mastitis management. In particular it is clear that certain important health control messages are still to become widely adopted.

INTRODUCTION

Anyone interested in quality milk production and who spends much of their time on farm will know that each unit operates very differently. There is no 'standard' routine and what works for one may not be practicable for another. Advisors could be forgiven for assuming that many of the principles of best practice are well established on UK dairy farms but there is little hard evidence available regarding what management practices are being employed in reality across UK dairy farms.

It was against this backdrop that Intervet Schering-Plough Animal Health undertook the first ever Cobactan National Mastitis Survey in early summer 2009. Responses to the questionnaire were incentivised and mailed with the June & July issues of Dairy Farmer magazine. This publication is mailed to around 12,000 farmers throughout the UK.

The survey aimed to provide a snapshot of what really happens on as many UK dairy farms as possible. A simple tick box survey was developed that covered areas ranging from yield to mastitis rates and from parlour routine to drying off approach.

Surveys such as this frequently obtain very low response rates so the organisers were very pleased when 1,322 responses were received and processed. In 2010 the survey questions were modified slightly and the data collection process was implemented using only the June issue of Dairy Farmer. A further 8,000 dairy farmers were mailed using Intervet Schering Plough's own mailing database. An online option was also made available but disappointingly received just 42 responses. In 2010 a total of 962 responses were received and included in the analysis.

In June 2010 there were 11,256 dairy producers in England and Wales (DairyCo datum). The number of responses to the Intervet-Schering Plough National Mastitis Survey (NMS) is therefore approximately representative of an impressive ten percent of the total number of dairy farms in England and Wales.

VALIDITY OF THE INFORMATION

It is important to recognise that survey respondents are not randomised but were readers of a specific dairy farming publication and responding to an incentivised data collection campaign. This will impose a rather inevitable degree of bias within the dataset.

Questions will always be raised regarding how truly robust data can be when gleaned via surveys of this nature. A component of this discussion relates to the quality of the questions used. The 2010 survey was modified and developed in an attempt to accommodate some of the areas of uncertainty that became apparent following analysis of the initial 2009 survey.

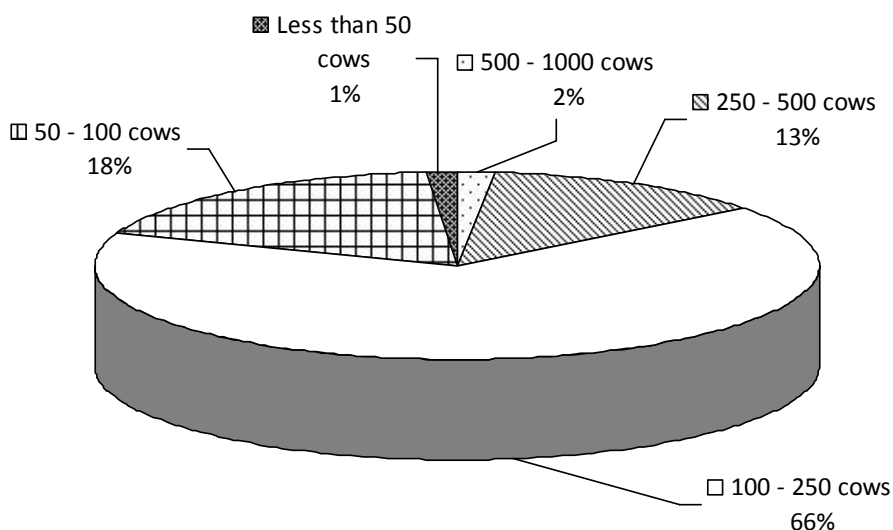
Closed questions relating to intramammary tube use can be compared with National tube sales data to provide some indication of how well the survey data represents the real field facts. The 2010 survey showed Tetra Delta being the tube of 1st choice for 36% of respondents and the national market share information for the twelve months between the two surveys shows Tetra Delta to represent 36% of sales volume (source GfK national sales data MAT June 2010). Cobactan is the reported first preference for 33% of respondents and the same sales data shows a market share by total volume of 27%. For dry cow therapy the survey data shows the first choice tube for 46% of respondents is Cepravin while sales data shows that Cepravin tubes represent 40% of antibiotic dry cow therapy sales by volume.

These comparisons take no account of the influence of farm size but do help provide a degree of response validation.

Herd size

In 2009 one in eight herds had more than 250 cows and one in four herds had less than 100 cows. In 2010 only one in five herds had fewer than 100 cows the general trend being to larger herds. Three in every ten respondents reported that they had bought in cows within the last twelve months.

Herd Size Bands

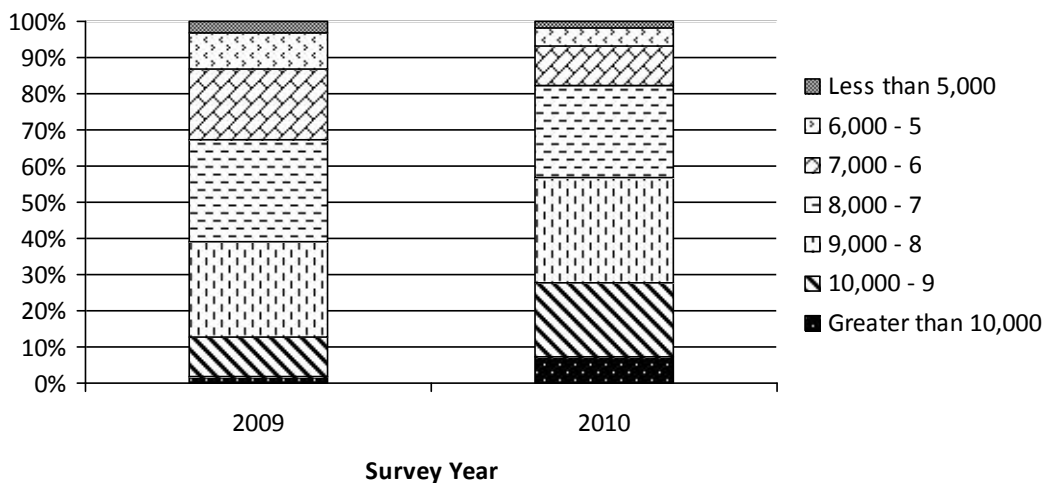


Herd Yield

There appears to have been a significant rise in reported herd yields between the two survey years. In 2009 only one in fifty respondents reported a herd yield of over 10,000 litres but in 2010 one in 14 herds reported yields greater than 10,000 litres.

In the 2009 survey one in three herds reported yields of less than 7,000 litres whereas in 2010 farmers reporting an average of less than 7,000 litres per annum had fallen to fewer than one in six farms .

Annual Herd Milk Yields



At the farm level one in four of respondents reported annual farm milk sales of less than 750,000 litres in 2009. In 2010 the proportion of farmers reporting milk sales of 750,000 or less was down to fewer than one in six. The proportion of respondents reporting milk sales of over two million litres rose from one in eight in 2009 to more than one in six in the 2010 survey.

There is a general tendency for the larger herds to be associated with higher yields. A 'rule of thumb' based on the 2009 responses is that herd yield in litres will be roughly equivalent to $7,500 + 1.5 \times \text{herd size}$. This trend is less apparent across the slightly smaller number of 2010 responses.

In the 2010 survey respondents were asked how many times per day the cows were milked. Just 3% milk three times per day with the balance on twice daily milking intervals. Four farms reported milking just once each day.

Health status

Capturing the number of clinical cases of mastitis proved a challenge in design of the questions. In 2009 respondents were asked how many cases the farm treats in a year. In 2010 the question was changed to survey how many cases are typically treated each month. It was thought that this may have more immediate relevance for dairymen and perhaps would produce more revealing and accurate data. The treatments per month were then compared against the number of cows milked in the herd to calculate a reported number of quarter clinical cases per 100 cows per year.

Just over half of all respondents reported a case rate of 20 to 50 quarter cases per 100 cows per year in the 2009 survey. 30% reported fewer than 20 quarter cases per 100 cows per year and 18% from 50 to 100 quarter cases per 100 cows per year.

The 2010 survey saw very little change with a very slight increase in the proportion of respondents reporting fewer than 20 quarter cases per 100 cows per year.

These figures appear to be a slight under-estimate of clinical mastitis rates when compared alongside typical figures calculated by advisors and published elsewhere. A large UK dairy practice, providing veterinary care for over 36,000 adult dairy cows, used tube sales per cow over two years of age to rank 138 dairy herds by mastitis case rate. Based on an assumption that an average of six tubes were used per clinical case the median number of clinical cases per 100 cows per year was 46 and only the upper quartile of dairy farms had fewer than 25 cases per 100 cows per year. A quarter of this group had a case rate of 76 cases per 100 cows per year or greater. If fewer tubes per case were assumed then the case rates would be proportionally higher. (source: James Allcock)

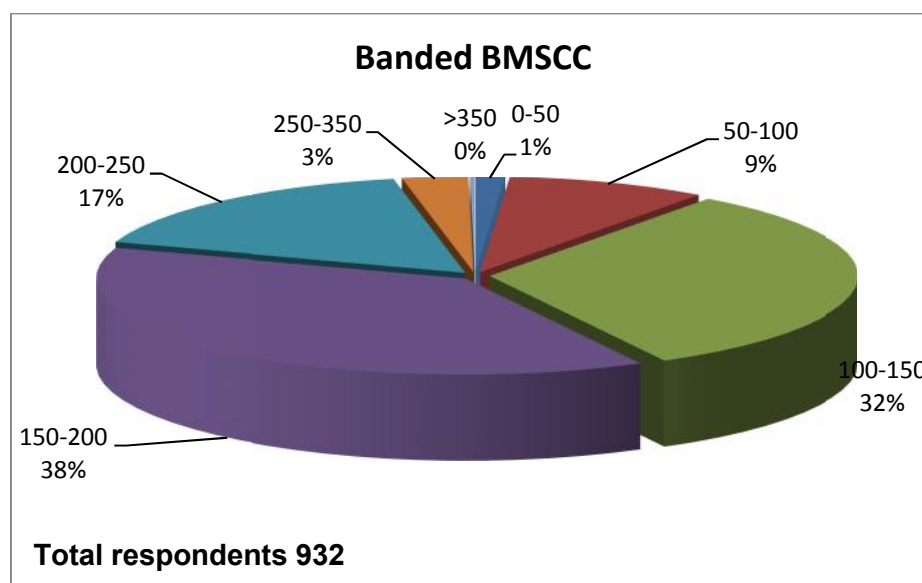
138 dairy herds assuming 6 tubes per clinical case	
Median cases per 100 cows per year	46
Cases per 100 cows per year on the upper quartile of farms	25
Cases per 100 cows per year on the lower quartile of farms	76

The survey asked respondents to choose an option regarding when most cases of mastitis occurred in their herd. In both years over three-quarters of cases were reported to occur in the first 100 days of lactation. However over one in five respondents quoted mid lactation as the main risk stage in their herd. In 2010 the options allowed respondents to specify if cases occurred in the first month of lactation or later and half of all clinical cases were reported to occur within the first 100 days but after the first month of lactation; i.e. around peak yield.

In 2009 18% of farms reported that more than one in ten heifers suffered mastitis during their first lactation. In 2010 this figure had dropped to 16%. In both years a little over half of respondents said that fewer than one in twenty heifers suffered mastitis during their first lactation.

Sub-clinical mastitis was surveyed by asking for the farms usual bulk milk somatic cell count. In 2009 42% of respondents claimed to have a cell count of less than 150,000 cells per ml with no farm reporting a cell count of over 400,000 cells per ml. Five out of six (83%) of farmers said their cell count was less than 200,000 cells per ml. In 2010 respondents were asked if they carry out individual milk recording. Three out of four said that their cows were tested monthly and one in eight said they tested occasionally.

In 2010 the survey asked farmers to select one of seven categories of typical herd cell count. Four of five farmers selected a band less than 200,000 and only two respondents out of the 932 reports (0.2%) admitted bulk milk cell counts above 350,000 cells per ml. These responses would appear to be a little more positive than other data sources such as National Milk Records data would suggest. This may perhaps be partly because recorded bulk cell count is often higher than the bulk cell count reported by the milk processor and it is on the latter that the farmer is usually most focussed.



46% of respondents said that their usual bactoscan was less than 20,000 with a further 41% saying that their bactoscan was between 21 and 40,000. Only ten percent reported a typical bactoscan of 41,000 or above.

Respondents were asked how frequently they and their veterinary advisors undertook bacteriological tests on milk. In both years around one in three respondents said they never tested while just over half of all respondents reported that they tested one or two times per year. Around one in six farmers reported tested three or more times per year.

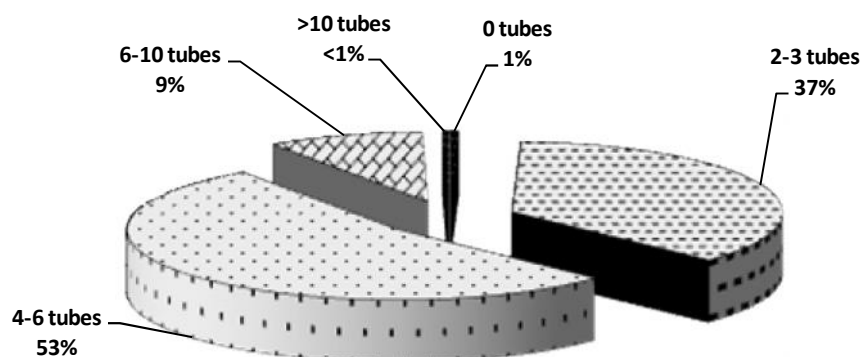
The typical range of bacteria reported to be found on respondents farms were *Strep. uberis* (36%), *Staph. aureus* (31%) and *E. coli* (23%) These proportions were almost unchanged across the two survey samples.

It is interesting to observe that nearly one quarter of all respondents said that they didn't carry out any milk bacteriology but then were able to list which bacteria were involved on their farms.

Mastitis treatment and control

In both years farmers were asked to select one of a range of numbers of lactating cow tubes used to treat a typical case of mastitis. This was intended to estimate the duration of intramammary therapy. Around two thirds of respondents report that their normal period of therapy extends beyond the typical manufacturer's recommendation of three tubes. In both survey years over half reported that a typical course consisted of four to six tubes per clinical case. In 2009 one in seven respondents (14%) reported that a typical case was treated with a course of 6 tubes or more while in 2010 around one in ten respondents reported typically using 6 tubes or more.

Average tubes per case



This information raises serious questions about the relevance and usefulness of manufacturer's packaging information surrounding appropriate use of their intramammary antibiotic tubes. Of more concern is the fact that there is no useful information available regarding the appropriate milk withdrawal periods that should be used for the products on most of the occasions when they are used.

Tetra Delta was the principle choice of lactating tube used at 36% of all tubes mentioned. Cobactan was a close runner up at 33% in 2010. Both these brands had profited from the non-availability of Ubro Yellow since 2009 when 17% of respondents had reported this brand as their first choice. Tetra Delta was 34% and Cobactan 27% of first choice tubes in 2009. Interestingly there were some significant regional differences in the preferred tube choice with the Norbrook brands, Multiject and Noroclav, being much stronger in Northern Ireland and Tetra Delta being much less dominant in Northern Ireland, Scotland and Northern England.

43% of respondents who nominated Tetra Delta as their first choice tube named Cobactan as their second choice. 18% of the same group chose Synulox and 13.5% chose Ubro Yellow as their second choice tube.

For those one third of farms who use Cobactan as their first choice 36% use Tetra Delta, 19.8% Synulox, 11.1% Ubro Yellow and 10.6% Ubrolexin as their second choice tube. When first and second choice tubes are combined both Tetra Delta and Cobactan represent 30% of the total lactating tube market volume with Synulox at 10%.

In 2009 nearly 10% of respondents reported using *combination antibiotic* therapy for over three-quarters of their mastitis cases. The survey did not specify what was meant by this term. One in six respondents reported using a combination approach for more than half their clinical cases. In 2010 the

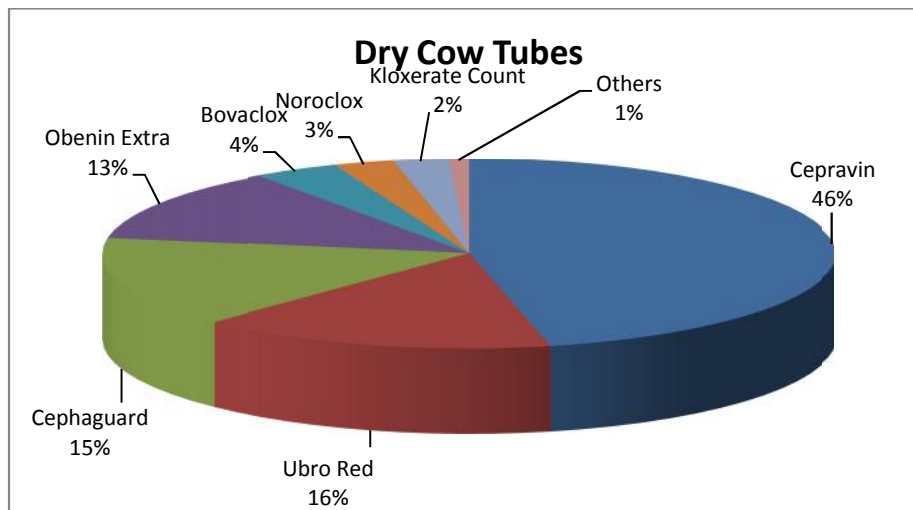
question was more specific and asked what *proportion of cases receive tubes plus antibiotic injections*. Nearly one in five respondents said this technique was employed for more than half of the cases they treated. Over one third of farmers use combination therapy for more than a quarter of the mastitis cases that they treat.

Examination of links between other parameters and the farmers using combination therapy show trends that suggest that farmers with lower reported mastitis case rates and longer courses of tube therapy also use combination therapy more frequently. Perhaps this subgroup of farmers are the ones for whom clinical mastitis control is of especially high priority.

In the 2010 survey farmers were asked if they treat high cell count cows and 53% replied that they did.

Just over half of all respondents said they used a teat sealant in all cows at dry-off and a further 13% said they used teat sealant selectively in some cattle. One third of respondents said that they do not use a teat sealant. Respondents were also asked if they used teat sealant in combination with antibiotic dry therapies and although the distribution was very similar, just over one in six replied that they used it on its own.

Cepravin is the preferred antibiotic dry cow tube on 46% of farms. Ubro Red and Cephaguard DC were the dry tube choice for 16% and 15% each.



Using Cephaguard DC as first choice dry cow tube appeared to change the farmer attitude to first choice tube. Cobactan became first choice on 46% of farms whereas Tetra Delta fell to first choice in just 35% of farms. A similar redistribution was not observed when Cepravin or Ubro Red were the dry therapies of choice. Until 2008 Cobactan and Cephaguard DC were both promoted and distributed by Intervet Schering Plough. This may help provide a partial explanation for any apparent association between these two brands.

Housing and milking facilities

In 2009 seven out of eight farms housed their cows in cubicles. In 2010 9% of respondents reported housing cattle in straw yards with 12 % saying they had both straw yards and cubicles. One in seven farms now house cattle all year.

85% of farms have a herringbone parlour with slightly more respondents having abreast parlours (5%) than rotary parlours (4%) Only 1% of respondents were using robot milking installations. Only six respondents reported using a herringbone parlour for more than 500 cows whereas one quarter of rotary installations were milking more than 500 cows.

Half of all installations had between 9 and 16 milking points with a third of all parlours having more than 16 milking points. Two thirds of all parlours had between six and ten milking points per 100 cows in the herd.

Based on reported herd yields and the number of milking points two thirds of herds have sufficient units to harvest up to 100,000 litres per milking unit per year. Over a third of farms are harvesting over 100,000 litres per unit per year.

Milking routine

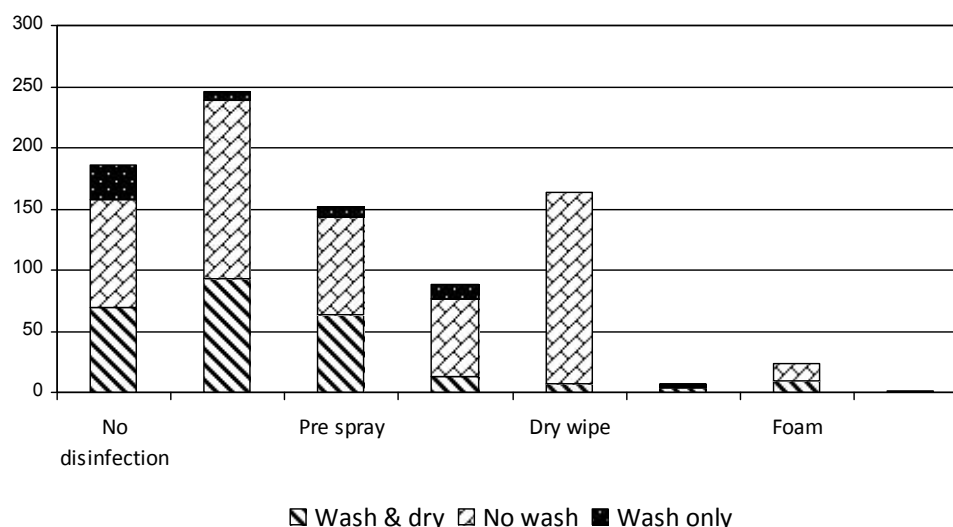
At milking half of all respondents reported that all quarters are stripped before milking. Interestingly 30% say that gloves are not used. It is possible to speculate that farmers may be inclined to respond positively when asked if they foremilk their cows since this practice usually remains a requirement of most milk buyers' contracts. Use of gloves in milk harvest is not any kind of statutory requirement but perhaps should be!

One third of farms wash cows before units are applied but only one in six of those washing teats then dry them!

Half of the farms reporting teat washing only with no drying (28 of the surveyed farms) also do nothing else. They report using water only to wash with no disinfectant. After many years over which advisors have discouraged use of water without thorough drying this data seems particularly depressing.

Fortunately the majority of farms do carry out a more thorough teat preparation. 28% use a dry wipe only, 27% use a pre-dip preparation, 15% a pre-spray and 12% a medicated wipe. However of the two thirds who don't wash one in six (over 90 farmers) don't do anything other than attach the units with no preparation at all.

Teat Preparation

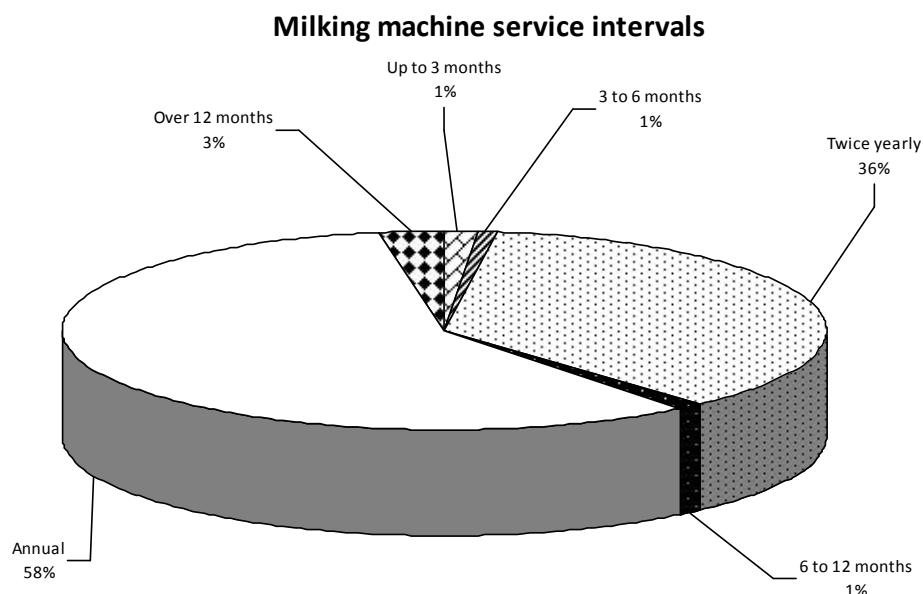


Just over four out of five parlour installations employ automatic cluster removal (ACRs) and 44% of respondents now report that they carry out cluster disinfection. Nearly two thirds carry out cluster disinfection by dipping (39%) or spraying (25%) and around one in three now have some form of automated cluster flush facility.

Of the farmers not wearing gloves around three times as many were not cluster flushing as were flushing clusters. Around half of those wearing gloves flushed clusters. Perhaps this implies that non-gloved operatives were particularly averse to any additional hygiene routines.

Automated post-milking teat disinfection (via ADF) is carried out in one in 25 responding herds and post-dipping of any kind is not carried out at all in one in 100 reporting herds. The balance of farmers are fairly evenly divided between those that post dip (49%) and post spray (46%) This seems a surprisingly high report of the use of dip cups and is probably encouraging in terms of effective post milking teat disinfection.

58% of farms reported that they have their milking installation serviced annually and over a third say that the machine is serviced twice per year.



Two thirds of farms report a liner change interval of between three and six months. However, when liner change interval is calculated via cow numbers, milking frequency and the number of milking units a large proportion of respondents should be changing liners more frequently than they are reporting. When the farm reported a frequent liner change interval of three or four months then this was generally more closely aligned with the calculated frequency.

CONCLUSIONS

The information captured from this large and up to date survey of dairy herds provides an unrivalled and very valuable insight into the habits and management practice prevailing on UK dairy farms. There is clear evidence of the expansion of farms but some concern that much of the now 'standard' advice regarding good health management for milk producers still fails to be adopted. It should be of serious concern to the industry that one in three personnel involved in producing milk for human consumption, do not wear gloves. However at the same time the recent popularity of cluster disinfection seems to have gathered considerable momentum.

This dataset should place industry advisors and educators in a much better position to prioritise learning objectives and measure results as the survey is repeated in years to come.

More information on the 2010 Cobactan National Mastitis Survey is available from Intervet/Schering-Plough Animal Health on 01908 685685.

NOTES

WHAT MAKES A GOOD TEAT DISINFECTANT?

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INTRODUCTION

Teat disinfection is a widely accepted practice known to help prevent new IMI in dairy cows. The advent of the 5 point mastitis control plan in the UK in the 1960s saw post-milking teat disinfection (PMTD) become one of the most important management tools to help reduce the new infection rate of contagious pathogens such as *Staphylococcus aureus*, *Streptococcus agalactiae* and *Streptococcus dysgalactiae*. Pre-milking teat disinfection was introduced to the UK in 1991 and has been adopted as an addition to good udder preparation to help remove environmental mastitis pathogens including *Streptococcus uberis*, *Escherichia coli* and *Klebsiella pneumoniae*, prior to unit attachment.

Today there are hundreds of product options available and the market is extremely dynamic which means milk producers need to be able to sort fact from fiction to compare teat dips properly and make an informed choice that truly benefits the herd health and milk quality on their farm. The aim of this paper is to provide insight into the ingredients, the guidelines for teat dips and to help dairy farmers with the all important buying decision.

EFFICACY AND REGISTRATION

Only products known to be safe and effective should be used and this should involve registration with the relevant authority. There are three main product categories: VM Licensed, Approved biocide or unapproved product.

When manufacturers or suppliers of a teat dip make medicinal claims then the product must be registered with the Veterinary Medicines Directorate (VMD) and is classified as a medicine having first demonstrated that it is safe and effective in the control of mastitis. If manufacturers do not make medicinal claims on the label or on any other sales literature, the biocide will need to be registered with the HSE under Biocidal Products Directive (98/8/EC) and the active listed under Annex 1 PT3 (Veterinary biocidal products). To register products or an active biocide, supporting efficacy data must be submitted as part of a dossier. For teat dips the European suspension test for teat dip disinfection efficacy is EN1656 – to pass this test the biocide must achieve a log 5 reduction of mastitis pathogens (*E coli*, *Staph. aureus* and *Strep. uberis*) within 5 minutes at 30°C with milk solids present. Log 5 equates to about 99.999 percent kill.

The Commission Regulation (EC) No 1662/2006 of 06/11/2006 clearly states that “that teat dips or sprays are used only after authorisation or registration in accordance with the procedures laid down in Directive 98/8/EC of the European Parliament and of the Council of 16 February 1998 concerning the placing of biocidal products on the market (1)”. This means that if a product has no VM license or is not registered with the HSE under the BPD (08/8/EC) it must not be sold as a teat dip or spray.

Good Manufacturing Practice

The VMD (previously this was the MHRA – Medicines and Healthcare Regulatory Authority) regularly inspect manufacturing premises and issue GMP certificates. Without GMP status manufacturers are not permitted to produce veterinary medicinal products.

GMP is a European requirement and is laid out in the Orange Guide (which despite its name is a legislative document not a guide) or to give it its full name: “Rules and Guidance for Pharmaceutical Manufacturers and Distributors”.

GMP is defined as “the part of Quality Assurance which ensures that products are consistently produced and controlled in accordance with the quality standards appropriate to their intended use.” The principles and guidelines for GMP are set out in Directive 91/412/EEC, as amended, concerning veterinary medicinal products. Compliance with these principles and guidelines is mandatory within the European Economic Area.

To protect the end user, samples of batches are kept in the event of an adverse reaction or complaint about the product.

Labelling

In addition to the VM number, although most teat dips are classified as non hazardous, there should be safety information on the label which should include a 24 hour telephone number in case of ingestion, contact with eyes or spilt on the public highway.

The label should include the manufacturer’s suggested application method, whether the product is ready to use or concentrated, what the levels of active ingredients are and whether the product is pre- or post-milking or both. It should state active ingredients and their levels and for traceability purposes the batch number and expiry date must also be clearly visible.

Shelf Life

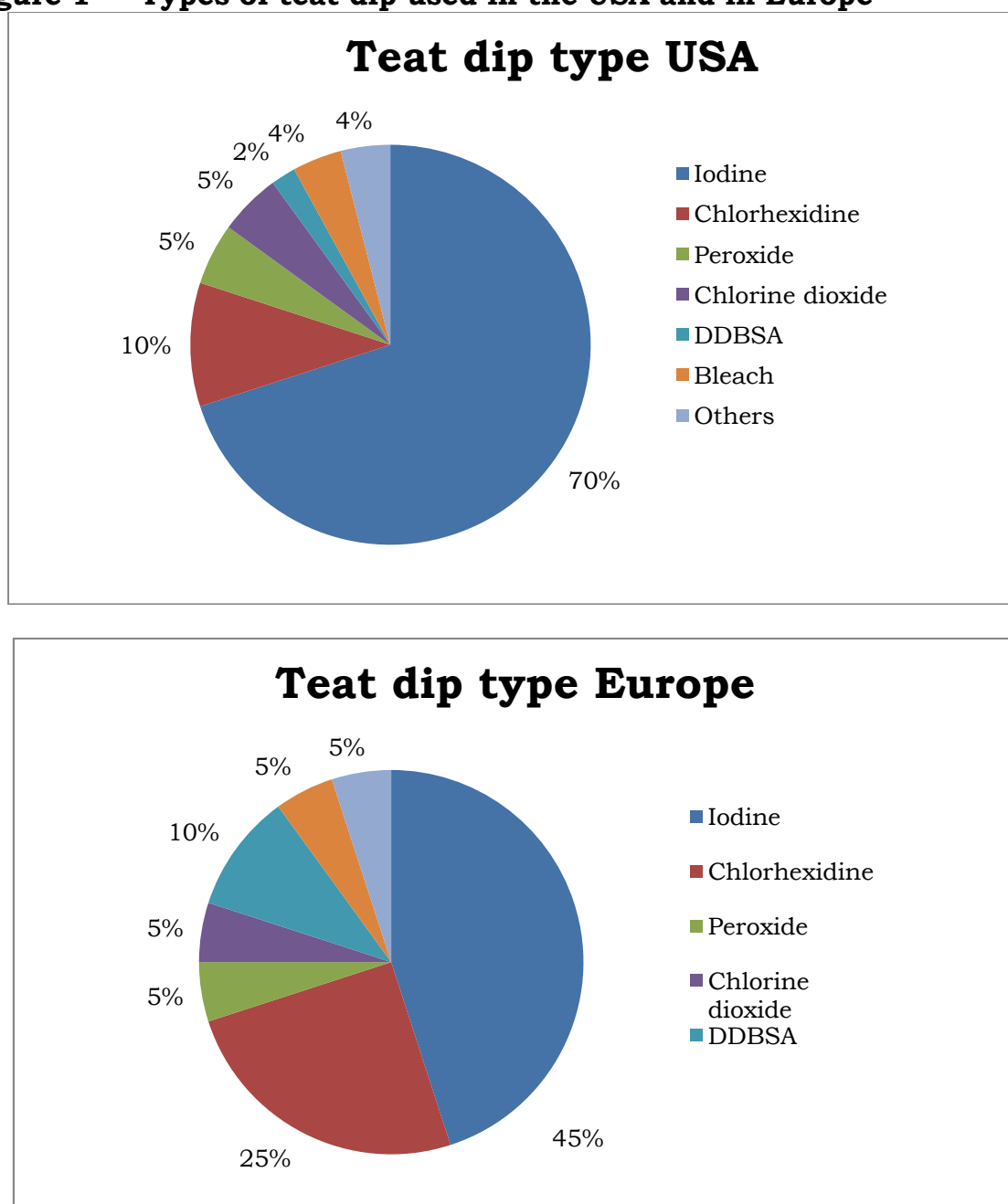
Originally, the products could be tested at room temperature but the rules have now strengthened to the VICH standard of 25°C/60% relative humidity. This means that for Veterinary registered iodine based products

shelf life may now not be more than 12 months but chlorhexidine products may have a shelf life of up to 2 years.

ACTIVE INGREDIENTS

A wide range of biocides are formulated into teat dip products including Iodine, Chlorhexidine gluconate, Lactic acids, Fatty acids, Hydrogen peroxide, Chlorine dioxide and Dodecyl benzene sulfonic acids which destroy bacteria by chemical or biological action and disruption of cell membranes.

Figure 1 Types of teat dip used in the USA and in Europe



Hemling, T.C (2002) Teat Condition – Prevention and cure through teat dips. Proc. BMC 2002

Iodine

Iodine has by far the largest market share in both the UK and USA due mainly to the supporting data to support the efficacy along with feedback from consumers and is often referred to as 'the gold standard'. It is only soluble up to 300ppm so has to be solubilised or complexed to make an iodophor. An iodophor is a complex of iodine in a surfactant or polymer such as polyvinylpyrrolidone (PVP). It is compatible with a wide range of emollients, there is a neutral impact on the environment as it breaks down to iodide, and it is naturally staining for high visibility on teat skin although more modern formulas stain less well. Some iodophors are effective even at low concentrations and can persist for up to eight hours on the teat (1). Its disadvantages are that it can have a loss of efficacy when affected by organic matter and often has a low pH (2.5 to 5) depending on the formulation of the iodophor. Levels vary from 0.1% (1,000ppm) to 0.5% (5,000 ppm) available iodine in the UK and up to 1% (10,000 ppm) available iodine in the USA.

All iodophors release small amounts of free iodine typically 50 ppm and the more loosely bound the complex is the more free iodine is released to kill bacteria which continues to release free iodine until all the available (titratable) iodine is used.

Chlorhexidine-Digluconate – CHG

Chlorhexidine-Digluconate is the second most popular teat disinfectant and since being discovered by scientists in the UK and has been widely used in teat disinfectants since 1970. It has a mild pH of 6.5 and works by penetrating the cell wall and disrupting the metabolic process in the bacteria. As it adheres well to teat skin can provide up to eight hours microbial action on the teat (1). It is also compatible with a wide range of emollients and fly repellents and has a long shelf life.

It is not as effective against gram negative bacteria, pseudomonas or spores as iodine and as it cannot be mixed with anionic surfactants it is more difficult to produce barrier type products.

Chlorine Dioxide

Chlorine dioxide and Chlorous acid products are extremely effective at killing bacteria and are now widely used in the UK and USA. Most formulas have to be mixed on a daily basis as once mixed they rapidly deteriorate although there are now some ready to use products on the market.

Dodecyl Benzene Sulfonic Acid – DDBSA

Usually used at 2% inclusion rate DDBSA and is an anionic surfactant which is widely used in household detergents. They are effective against gram positive bacteria and gram negative bacteria but have poor persistency

on the teat (1). It works by disrupting cell membranes and is easily formulated into teat dips. It has been licensed as a veterinary medicine since 1981/2.

Lactic Acid

Lactic acid is a safe, water soluble biocide effective against both Gram-positive and Gram-negative organisms (2). It is easily formulated with glycerine to create products with good skin care characteristics. It is also used in formulations combined with other biocides with which it can appear to have a synergistic effect. Lactic acid is widely used as a teat conditioner within chlorine dioxide type products. It also has defoliant capabilities.

Fatty Acids

These are not listed as biocidal ingredients under the BPD. Capric acid is the most common type of fatty acid to be used in teat disinfectants and behave in a similar way to iodines as they disrupt cell membranes and prevent bacterial growth. They are not water soluble and have to be emulsified to allow them to be formulated. Being more expensive they are often mixed with other biocides to ensure that the formulation is cost effective in use. Fatty acids could not be used alone as they are not listed biocides.

Hydrogen Peroxide

Hydrogen peroxide is becoming more widely seen in formulations as it is an extremely good oxidizing agent and is often combined with lactic acid to form alpha hydroxyl acids often seen in human cosmetics. It has a broad spectrum kill and there is anecdotal evidence that teat skin improves with use as the active removes dead skin cells and this helps to prevent bacteria from colonizing the teat skin surface.

Table 1 Summary of Microbial Efficacy of Various Active Ingredients

Active Ingredient	Micro-biological efficacy	Skin tolerance	Advantages	Disadvantages	Stability
Iodine	+++	Fair	Broad spectrum Commonly available	Can cause allergies or dry skin	Good to fair
Chlorhexidine	++	Good	Long lasting Sticks to skin	Lower spectrum than iodine	Very good
Chlorine dioxide	++++	Fair to poor	Broad spectrum Fast kill High efficacy	Has to be mixed - two component system Affects skin condition	Good before mixing, poor after
Hydrogen peroxide	+++	Fair	Broad spectrum Effective No residue	Can irritate skin Can be inactivated	Fair
Peracetic acid	++++	Fair	Broad spectrum Very effective No residue	Irritant Dangerous to work with at high concentrations Pungent odour	Fair
DDBSA	++	Good	Fair efficacy Widely used	Poor persistency	Good
Lactic acid	-	Good	No residue Safe	Better effect when used in combination	Good
Fatty acids	++	Good	Some quite effective Safe and non-toxic	Not soluble in water Expensive	Good
Nisin	++	Good	Very effective against gram positives Safe	Expensive Not effective against gram negatives	Good
Chloramine T or Halamid	++	Fair	Wide application Low residue Mild	Not advised for long-term skin contact	Fair
Chlorine	+++	Poor	Broad efficacy Inexpensive	Irritates skin Efficacy affected by organic matter	Poor
Phenolics	++	Fair to poor	Available Used as preservatives	Some carcinogenic Persistent in nature Can taint milk	Good

SKIN CONDITONING AGENTS

Skin conditioning agents (commonly called emollients) are used to offset the potentially negative effects of actives, detergents and the low pH of some formulations and help improve or maintain teat condition, combat the harsh effects of the weather and other influences on teat skin.

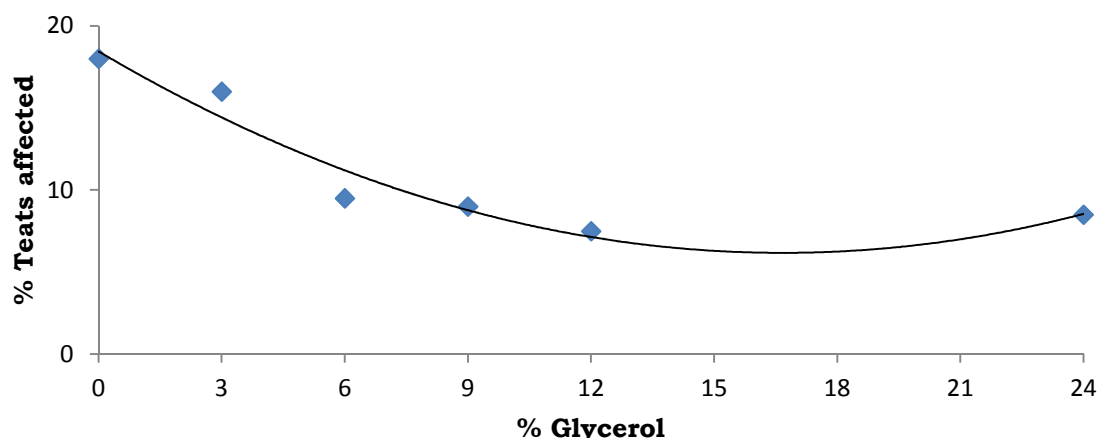
Good smooth skin helps to prevent bacterial colonization and thereby reduce mastitis. There is also evidence that by improving teat skin condition teat preparation time is reduced as teats are easier to clean and milk let down improves which can lead to quicker milking times(3).

Table 2 Summary of Emollients

Emollient	Type	Availability/cost
Glycerine	Humectant, emollient	Widely available in natural and synthetic, swings in prices, only vegetable-derived glycerol can be consumed in foods
Propylene glycol	Humectant, Emollient	Widely available, works with fatty acids, is synthetic, swings in prices, has some antimicrobial properties, is food-grade or GRAS (generally recognized as safe)
Lanolin	Occlusive agent, emollient	More expensive than other emollients, available both natural (sheep wool) or synthetic
Aloe Vera	Humectant, healing properties	Natural derivative of the aloe vera plant
Allantoin	Keralytic agent, healing properties	Natural extract of comfrey plant, also has wound healing and anti-irritating properties, softens skin, stimulates growth of healthy tissue
Sorbitol	Humectant, skin conditioner	Is used as a sugar substitute, present in fruits and vegetables. Commercially is derived from com syrup.
Polyvinylpyrrolidone	Skin conditioning agent	Expensive, also used for complexing iodine

There are many types of skin conditioning agents with some acting as humectants (glycerine) i.e. draw the moisture into the teat skin and others acting as emollients (lanolin) which seal moisture into the teat skin and prevent moisture loss or exfoliates (lactic acid) which remove dead skin cells. Teat skin conditioners are usually included at up to 10-12% as at above this level the effectiveness of the biocide can decrease (4).

Figure 2 Relationship between % teats with lesions in 30 herds and glycerol concentration in an iodophor teat dip



Bramley, A.J. (1980) In: Mastitis Control and Herd Management, Technical Bulletin 4, National Institute for Research in Dairying, Reading, England, pp 60-66.

APPLICATION METHOD

Post-Milking Teat Disinfection

Application of the post milking product is often determined by the type of product used e.g. If a very viscous product (usually a barrier) is used then it will have to be applied by teat dip cup. Conversely a non-viscous product may be dipped or sprayed. Also, if a true barrier type product is used then some form of wet teat preparation will be needed to properly remove the strong film at the next milking. If this film is left to build up then problems with hyperkeratosis may be encountered.

Vets and advisors generally recommend that teats should be dipped using a teat dip cup to fully cover teat skin and to allow penetration into the teat orifice but if teats are sprayed correctly and the teat skin is fully covered then that is also acceptable. The consumption for teat dipping is approx 10ml/cow/milking and for manual teat spraying approx 15ml/cow/milking. In automatic walkover type systems consumption increases dramatically to between 26 to 40ml/cow/milking. As they are usually situated on the parlour exit race an increase in the prevalence of *Corynebacterium bovis* has been seen. There are now several other automatic systems that are

mounted in parlours which spray teats as soon as units are removed. These tend to need refilling often and perform quite well, provided complete teat coverage is maintained.

There is also an automatic dipping system available in the UK but as with all automated systems, they need to be well maintained and rigorously monitored at every milking.

Care should also be taken to avoid adverse reactions with bedding materials or bedding conditioners, especially where products with extremely high or low pH are used.

Above all the objective is to ensure that teats are disinfected as soon after unit removal as possible and that teats are fully covered.

Pre-Milking Teat Disinfection

Pre-milking teat disinfection and application methods should require the same attention as PMTD and should only be considered in addition to PMTD alongside good udder preparation. It can be used to encourage foremilk and its use negates the argumentation about spreading contagious bacteria by the action of foremilk.

Often routines are performed less well if teats are dipped in full rows as the time taken between dipping and unit application becomes so lengthy and way over the recommended 60 to 90 seconds prep-lag time that cows “forget what they came into the parlour for”.

It is recommended that in a herringbone parlour cows are prepared in batches of 5 or 6 or when two operators are milking the first sets off with the dipping and stripping and when he reaches cow five then the second operator should begin to wipe with single service wipes and apply units. This helps to avoid bimodal milk letdown and can reduce the length of milking time. There is also evidence that pre-milking teat preparation, with correct prep-lag times, increases milk yields by 500kg per cow per lactation (5). The product should have enough contact time to penetrate the soiling and allow the active to work and this should be according to the manufacturer's instructions. The order of the process should be strip, dip, dry, apply although some operators prefer to dip, strip, dry, apply and both routines are acceptable if performed well. What is not recommended is to dip, dry, strip, apply as this can lead re-contamination of teats.

To enable an iodine product to be sold as a pre dip it must contain below 0.3% (3,000ppm) active iodine or 0.3% CHG with some formulations available as combined products for pre and post milking disinfection. These products do not always mean compromising on efficacy as there are now many post milking iodine formulations on the market below 0.3% active iodine or CHG that have been tested and proven to give a log 5 reduction.

Table 3 Comparison of the effectiveness of teat preparation methods

Dry Towel	Wet Towel	Pre-dip	Manual Dry	Effectiveness
✓				4%
	✓			40%
	✓		✓	77%
		✓	✓	85%

Extracted from Cook, C, Dairy Hygiene Inspectorate (proceedings of the British Mastitis Conference 2002)

HANDLING, PREPARATION AND STORAGE

Reasonable care should be taken to handle teat disinfectants safely and it is advisable to read labels before using a product. Dip should be stored above 2°C as they deteriorate if frozen. Drums should also be stored properly closed to avoid contamination and away from children, animals and animal feeds.

Although there is a move in the UK towards ready to use products as producers and advisors see the benefit of well formulated products there are still a great many concentrated products widely available. Problems can occur with mixing these types of products, e.g. a 3:1 concentrate can be mixed up incorrectly as a 4:1 product and microbial action is impaired or alternatively the mixed product is too strong and can have an adverse affect on teat condition.

Veterinary medicinal concentrate teat dips must only be mixed on a daily basis according to the requirements of the VMD and the Irish medicines Board (IMB). Problems can occur if borehole water or spring water is used to mix concentrated dips and some products can settle out after a period of time. It is also extremely difficult to incorporate high levels of emollients and humectants into a concentrated teat dip with a maximum of 5% teat skin conditioning ingredients often seen. There are also practical difficulties when mixing products with high surfactant levels and a nice foam bath can be produced!

WHEN THINGS GO WRONG

If farmers suspect that problems they have encountered on farm may be due to their teat dip there are a number of possibilities open to them. They should contact the company that supplied the product or the manufacturer who can investigate the claims and also offer support and advice to the end user, and at the same time seek expert advice. Some manufacturer's have

field support staff who have trained to implement the DairyCo Mastitis Control Plan and are able to offer mastitis control and dairy hygiene practices.

In the case of veterinary medicines, if an adverse reaction is found this must be reported immediately to the supplier and licensing authority.

SUMMARY

When selecting a teat dip dairy farmers should take into account all the following factors:

Select a proven product that effectively kills mastitis causing organisms, maintains healthy teat skin.

One that is supported by good service and advice, related to management factors such as the milking and housing system, weather conditions, the type of mastitis pathogens isolated from clinical and sub clinical cases, and the stage of lactation of new infections.

The general recommendation in the UK by Dairy Hygiene Inspectors and dairy husbandry advisors is to use an iodine teat dip with 0.5% (5000 ppm) available iodine and 10 % glycerine that has been proven.

Since 1980, 31 peer-reviewed research papers have been published with iodophor as the active ingredient. In the same time there have been 7 papers using chlorhexidine and 8 papers using chlorine dioxide. 18 other projects covered various other dips.

Iodophor dips are used as the reference dip in most research work and still are most popular on farms.

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NOTES

VENTILATION AND ITS IMPACT ON MASTITIS CONTROL

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SUMMARY

The built environment is one that we perceive as being designed to maximise the efficiency of the productive processes contained inside. In the case of livestock production the industry might expect that the built environment is one that is designed to suit the needs of the livestock housed. The reality is substantially short of this expectation; previous work has shown that approximately 50% of naturally ventilated buildings are not suited to the housed livestock, whilst other studies demonstrate that the design and maintenance of floors, cubicles, feeders and drinkers do not always meet design needs.

Whilst building design quality can be viewed as a weakness in the industry, such a view ignores the potential for improvement and subsequent impact on cost efficiencies. There also lies a mostly hidden benefit from good design for livestock buildings; that good design will not only be supportive of animal needs but can also have a catastrophic effect on the survival of certain pathogens. The role of ventilation is essential because it can directly influence the survival rates of *E.Coli* and *Streptococcus uberis*.

INTRODUCTION

The suggestion that various animal health problems can be influenced by improved housing is hardly new. Ten years ago Hughes (6) stated that in spite of many improvements in knowledge and available veterinary preparations, the prevalence and severity of mastitis is still a serious problem at a national level. The contribution of environment was acknowledged as relevant and promoted via expression of the need for 'good ventilation'. Allan (1) and Pocknee (8) have both stressed the need for improved environment in previous conferences. The hypothesis of the current presentation is that we are in a position to move forward from generalities in specific areas of building design, and that the answers lie in established research work that needs to be taken directly to the farm business.

The need for adequate ventilation in naturally ventilated cattle buildings is described in BS5502 part40 (2005), but only provides data as ventilation rates per kg LW. Useful data was available in 1975 (2) and later but the key fact is that the thermal dynamics of natural ventilation in cattle buildings was tried, tested and published to universal acceptance in 1982 (3). Since that time the use of the resultant ventilation model has demonstrated that

approximately 50% of naturally ventilated cattle buildings are not adequately ventilated for the stock housed (7). Later work related to the relationship between environmental factors and calf pneumonia found similar levels of inadequacy (9). There has been no published data on the competence of ventilation design in more recently constructed cattle buildings but it is the author's opinion that the situation has not improved at all. The tragedy is that recent investment in buildings made by the dairy sector show similar levels of incompetent design and construction as before.

DESIGN FOR HEALTH

The pathogens associated with mastitis and environmental concerns are *E. coli* species and *Streptococcus uberis*. Both organisms are probably commensals in UK dairy herds which leads to the question as to why one or both of these organisms can be a severe problem in some herds and not others. Explanations will include differences in susceptibility of stock over time and between herds, the prevalence and virulence of the specific pathogens and other specific local stress factors, but the contribution of the environment outside the parlour is usually limited to concerns about hygiene and moisture levels.

The knowledge of environmental conditions that promote the survival, replication, or decay of specific pathogens is broadly understood, which means that it should be possible to produce a set of environmental criteria that need to be met by adequate building designs. The environmental design targets should be of greater importance than the visual or aesthetic aspect of a building, but our current system of building planning and control does not support such an approach.

Aerobiology

The role of air quality can be critical in the survival rates of both *E. coli* and *Strep. uberis*. The physical factors relevant to this association are relative humidity (RH), temperature, and open air factor (OAF), and all determine survival and decay rates of a number of bacteria and viruses involved in animal health problems. The aerial environment can produce surface inactivation of bacteriophages during aerosolisation of particles, and whilst the damage may be temporary, the effect will be to reduce infectivity rates. In addition, the ability of a bacteriophage to survive and return to original infectivity depends on the surrounding nutrients in the environment and the broader picture of air quality. The hypothesis presented is that exposure to air circulation is vital to health when either *E. coli* or *Strep. uberis* are present within a straw bedded structure or on the surface of cubicles.

The contribution of all the design features that contribute to the viability of organisms within animal houses will not be addressed in this presentation. Competent flooring, drainage, roofing and management all impact on moisture levels within a building, and the design, use and management of

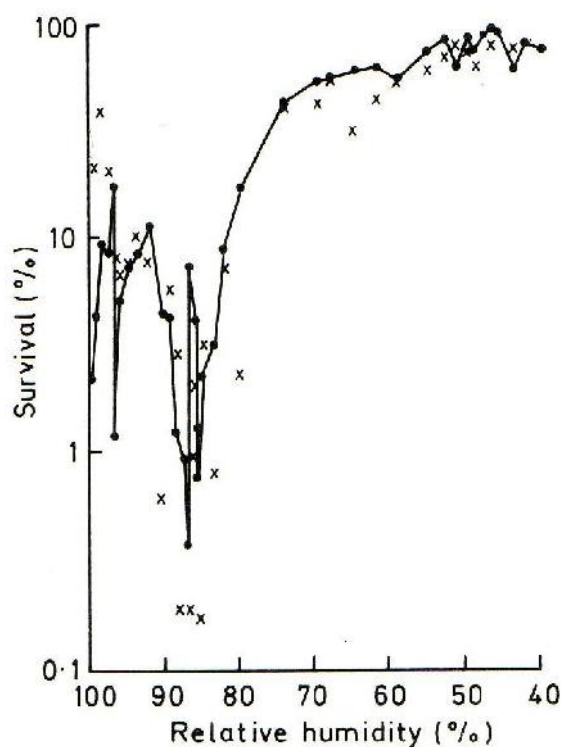
materials can all effect the competence of hygiene practices. However, the discussion will focus on the impact of ventilation and the design requirements to ensure competent operation.

Moisture

When a bacteria is aerosolised (as with airflow across a straw bed or cubicle) the loss of water molecules by evaporation may cause loss of microbial activity. The effect of a rapid change in RH may also be compounded by impact on the stability of the organism due to failure of various biological functions. Cox (4) suggests that this may be related to the stability of organisms in solutions of high concentrations of a wide variety of solutes. Dehydration of bacteria from a cow bedding environment will cause exposure of bacteria to high concentrations of solutes as the moisture concentration of the surrounding environment decreases rapidly.

The work covered by Cox describes the viability of *E. coli* B as virtually complete at lower RH and at 100% RH (Figure 1), with the latter the equivalent of being in solution. Figure 1 describes the aerosol survival of *E. coli* sprayed from a suspension in distilled water into nitrogen as a function of RH, with the RH range of 80 to 100% being more traumatic to survival of the organism. Most cattle buildings will have relative humidity at or above 80% RH. The design aim therefore is to expose as many of the pathogens to the aerial environment as possible.

Figure 1. Aerosol survival of *E. coli* B as a function of RH



From Cox (4)

E. coli or *Strep. uberis* in bedding or cubicle surfaces will mostly be in a saturated atmosphere in terms of relative humidity. The microscopic environment of the bed will also include a variety of nutrients in and out of solution that may have a supportive role in bacterial survival. In experimental work the presence of polyhydric compounds such as dextran and glycerol protect *E. coli* at higher RH, which suggest that *E. coli* in a dirty bed may be more resistant to degradation by changes in RH than if in a clean bed where such compounds are less prevalent.

Cox states that “To summarise the findings with *E. coli* strains following aerosolisation into inert atmospheres, they suffer damage to their surface structures. This damage results in leakage of ions, reduced DNA, RNA and protein synthesis, impaired active transport and greatly decreased oxygen consumption. Surface damage arising through denaturation, possibly of proteins or lipoproteins, occurs to the greatest extent at high RH.” The reduction in oxygen availability at cellular level will severely reduce the competence of repair mechanisms, whilst any denaturing of virus surfaces will eliminate their infectivity.

Temperature

Temperature has an obvious link to bacterial survival and infectivity within broad limits. There is an expectation of relationships based on the temperature sensitivity of biological processes within cells, and also of interactions between temperature, RH and cell survival and infectivity. The impact of temperature on infectivity is crucial in the livestock environment, as clinical disease is a function of organism dose at host level, all other factors being constant.

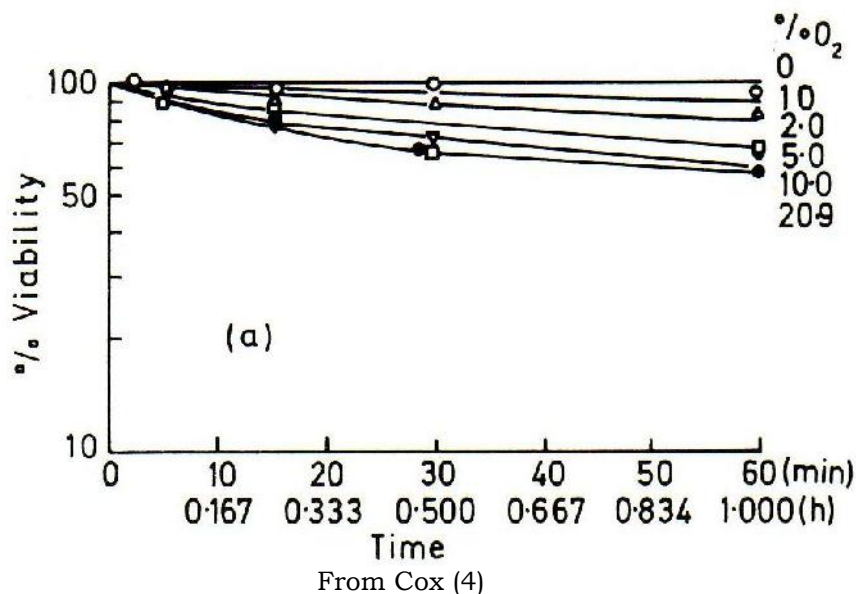
In a study of survival and replication of different *Strep. uberis* isolates, generation times in milk were calculated as 2.7 ± 0.1 h, 2.1 ± 0.1 h, and 1.0 ± 0.1 h for 116-520-S-1 isolates and 1.8 ± 0.4 h, 1.3 ± 0.3 h, and 0.8 ± 0.1 h for 116-520-S-2 isolates at 21, 25, and 32°C, respectively (5). Whilst there is no indication of survival and replication outside a milk environment, it is suggested that *Strep. uberis* prefers an environment that is warmer rather than cooler. Similar results occur for *E. coli*, with replication low below 20°C and rising to an optimum at 37°C. One implication is that, as environmental temperatures rise, the survival rate of both of these organisms increases. The impact of raised temperature directly on cow comfort and immune competence is also highly relevant (8).

Oxygen

The impact of oxygen on any specific bacteria during aerosolisation is difficult to separate from the effect of RH, as dehydration-rehydration stress will be simultaneous with oxygen-induced damage. For some organisms survival rates reduce with increasing oxygen concentrations of 1 to 20% at the same RH (Figure 2). The rate of decay is much higher at lower RH than

higher RH, and Cox describes work with *Serratia marcescens* 8UK where the loss of viability due to oxygen toxicity at RH values above 70% is 1/300th of losses at 20% RH. For *E. coli* B oxygen toxicity disappears at high RH, which would be the normal RH in and around the biological matrix that forms the upper boundary layer of the cubicle/bedding environment. The design requirement is for a dry bed.

Figure 2. Aerosol survival of *Serratia marcescens* 8UK as a function of O₂ concentrations at 60%RH



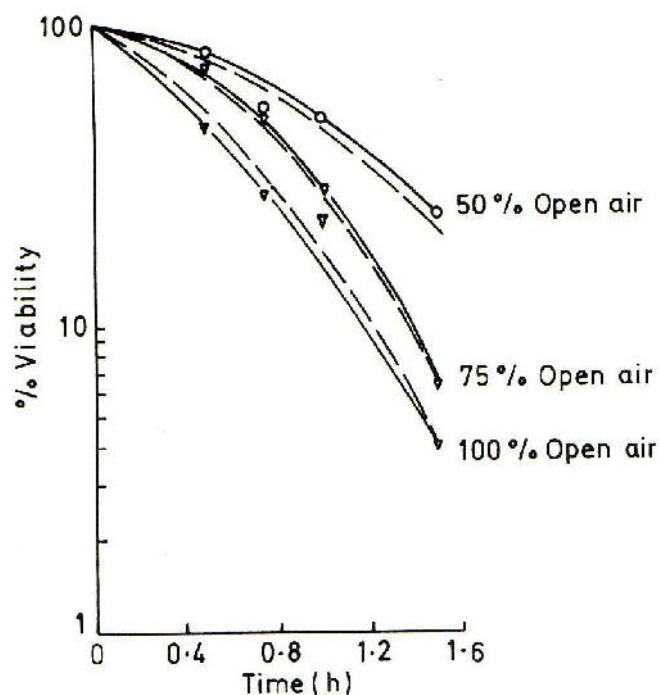
OAF

The final vital ingredients of clean, fresh air that are damaging to relevant pathogens are communally described as 'Open air factor' (OAF), simply described as ozone and olefin products, pressure changes at microscopic level, and ion concentrations. Group C streptococcus and *E. coli* show similar sensitivities to OAF. The impact of OAF on bacterial and viral survival rates can be catastrophic with viability and infectivity showing scalar reductions. Figure 3 describes the change in viability of *E. coli* with exposure to different levels of OAF.

Ozone increases damage to the amino acid/protein coat of bacterial phages, particularly cysteine, tryptophan and methionine. Ozone has also been shown to modify the pyrimidine base on the nucleic acids of *E. coli*, and *E. coli* B cannot fully support colony formation in the presence of ozone.

OAF does not transport easily without loss of efficacy, and the damaging components of OAF show a time-dependent degradation of bacteria and viruses that suggest continued exposure is the optimum strategy for good hygiene. A constant supply of OAF is a design requirement; a constant supply of clean, fresh air is the target.

Figure 3. Aerosol viability decay curves for *E. coli* B as a function of different OAF dilutions



From Cox (4)

Summary

The difficulty of maintaining a dry bed or a reduced RH at the cubicle/bedding boundary layer is that the source layer is in a dynamic condition. The cubicle surface will be influenced by the continuous addition of energy via sensible heat from cows lying, from the metabolic activity of commensals at the cubicle surface, and of moisture from respiration, defaecation and urination. In a bedded court the activity described above is added to by the thermal and moisture gradients within the bed pack that all show a dynamic shift towards the surface layer.

VENTILATION

If the design requirement is to get a constant supply of clean, fresh air into all parts of a livestock building, the next question must be, how? The clear answer is that the knowledge of how to provide 'adequate' ventilation into a naturally ventilated building has existed for 30 years. The failure has been to apply that knowledge in a systematic manner.

The thermal dynamics of naturally ventilated livestock buildings is described by Bruce (2) and a model published (3). Subsequent use of the model demonstrated the high frequency of cattle buildings that were inadequately ventilated for the stock housed (7, 9). The information derived from the model allows the description of the open areas required around the roof and

walls of that building to allow the building to ventilate due to stack effect. The key is that the stack effect will ventilate the building even when the wind does not, providing a constant flow of fresh air across the cattle housed. The efficacy of the stack effect in delivering fresh air throughout a building is not consistent above building widths greater than 25m (80ft). The difficulty of getting fresh air into building spans greater than 25m or into multiple span building will be discussed later.

Stack effect

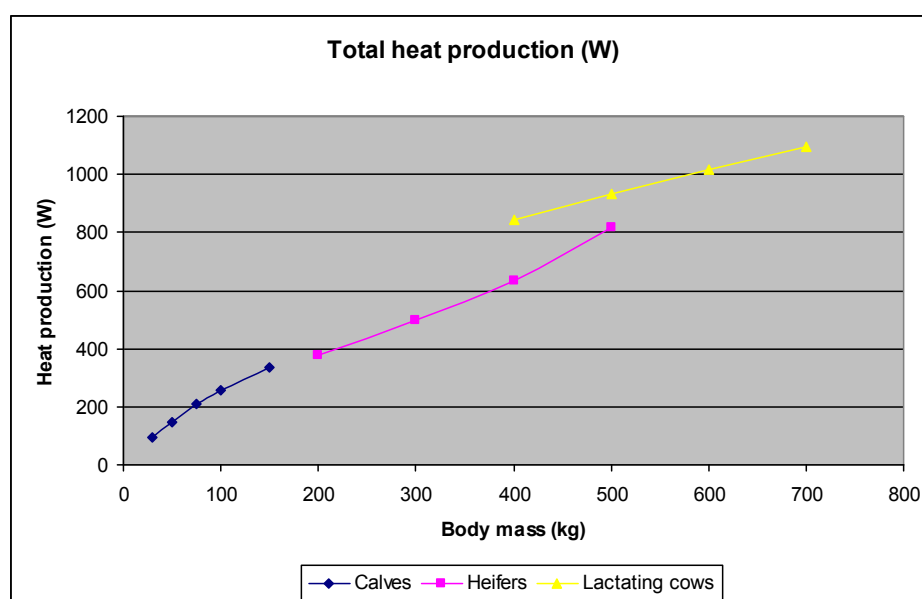
The principle of stack effect is that the combined sensible heat production from cattle in a building transfers to the surrounding air, and that warmed air rises through thermal buoyancy. The complimentary factors of animal liveweight, stocking density, roof slope and the area and height of any openings determine the ability of the building to ventilate 'adequately'. The difference between the thermal model and other descriptors of ventilation 'adequacy' is that the model defines requirements in discrete numerical terms. The use of the term 'adequate' should be secondary to a quantitative description of ventilation openings, not vice versa.

For cubicle or dry cow housing, the principle weaknesses of building designs that create a lack of ventilation are:

- A lack of or inadequate outlet area in the roof,
- A lack of or inadequate area of inlet, or
- An inappropriate distribution of adequate outlet and/or inlet areas.

The primary aim is to get the stack effect to do all the work of ventilating a building, where possible. Dairy cattle produce substantial quantities of sensible heat (Figure 4) and even after heat losses to wet floors and structural components of a building there will be sufficient thermal energy to drive the stack effect, if there are sufficient outlet areas in the roof and inlet areas in the walls. And therein lies the scale of the problem; very few cubicle houses, old and new, have sufficient outlet area in the roof to realise the maximum flow of fresh air into the cubicle area. Inadequate inlet areas have the same effect.

Figure 4. Total heat production from dairy cattle



The sequence of events that lead to a significant contribution of a poorly designed environment to elevated incidence and severity of mastitis can be as follows:

1. Heat and moisture production, 24 h/d
2. Presence of viable *E. coli* and/or *Strep. uberis* populations 24 h/d
3. Removal of excess heat and moisture from the bed/bedding across a diffusion gradient created by wind driven ventilation that removes metabolic products from the building.
4. The aerosolisation of pathogens at the bedding/air interface, with bacteriocidal benefit
5. The wind abates (or the building has internal areas untouched by wind driven ventilation).
6. The concentration of moisture and heat within the building increases.
7. The diffusion gradient from bedding to air decreases, and concentrations of moisture and heat increase within the bed
8. The bacteriocidal properties of fresh air are absent
9. Bacterial populations are no longer limited by RH%, oxygen, temperature or OAF

The hypothesis does not diminish the beneficial impact of regular bedding with quality dry materials, alkaline materials or dry disinfectants, or the regular removal of organic debris. However, the effective distribution of fresh air throughout a cattle building has many benefits over any bacteriocidal properties and the solution is routinely cost-effective.

Solutions

The routine analysis of the ventilation competence of cubicle housing demonstrates that ventilation outlet areas are inadequate in too many cases. A common misconception is that, because a building has some vents in the

roof it will ventilate adequately. A building with only 50% of the required outlet area in the roof will only be able to provide adequate stack effect ventilation for 50% of the stock housed.

A further misconception is that if a building has a roof and a surfeit of inlets (a current trend in cubicle housing design) the impact of the outlet component in the roof is negligible and therefore not necessary. The reality is that when wind speeds drops to calm, the only way that fresh air can enter the building is when air leaves the building. With no outlets or inadequate outlets in the roof, there will inevitably be an increase in heat and moisture within the building under still air conditions.

Any opposition to putting holes in the roofs of cattle buildings is misplaced. Concern about the ingress of water on the cubicles or feed below is correct, but is managed by the use of covered open ridges. The simple design of upstands either side of a ridge opening not only keep the majority of rain out of the building, but increase the efficiency of the stack effect by adding further negative pressure at the roof ridge. The effect is created by the normal movement of air (wind) that has to accelerate over the raised opening on the outside of the ridge. The majority of potential rain ingress is however prevented by the warmed air leaving the building driven by thermal buoyancy.

Outlet designs

It is the area and the distribution of that area along the ridge that dictates the functionality of the outlet. The actual design will depend on cost, expected rainfall, building exposure and other building uses. Typical solutions include open ridge, open ridge with upstands, covered open ridge, slotted roofs and a cranked crown ridge or roof cowl. For cubicle housing at normal stocking densities the calculated open ridge area is seldom less than 200mm for the whole ridge length, and is often wider.

The ventilation and stack effect requires a basic matching of inlet and outlet areas to be able to work to maximum efficiency. With exact computation it is possible to counteract minor restrictions in either inlet or outlet areas by compensating with changes in the other area component. The exact relationship between inlet and outlet areas is building specific, but a rough rule of thumb is to require an absolute minimum inlet to outlet ratio of 2:1, and typically 4:1.

Inlet design

The design aim is to provide no restriction on the pressure-driven competence of the stack effect, and the delivery of a stream of clean fresh air around the perimeter of the animal space, above animal height. The design should also aim to limit excessive air speed at animal height.

The design and materials used successfully are many; the principle requirements are the total area of inlet area and the subsidiary factors of cost, durability, suitability to the prevailing weather conditions and protection from elevated wind speeds. Inlet designs include solid wall sheets above a solid wall but offset to leave a horizontal gap, perforated cladding of various materials, space boarding, Yorkshire boarding, and variable height screens.

Large spans/multiple spans

Where two buildings are joined along adjacent walls, it is inevitable that the stack effect is not able to deliver a supply of clean fresh air along the joint boundary. Short buildings may be successfully ventilated from inlets on the gable ends, but otherwise the only robust delivery mechanism is to use fans and ducts suspended from the roof for the length of the building. Fans without ducts can be effective when they are used to move large volumes of air. Typical fans can move 4m³/s or more and are very effective at improving the rate of heat and moisture loss from cows and cubicles, but they do not deliver clean air along the length of the air trajectory.

There is no experimental data to support the detail of the current hypothesis. However, a number of practical interventions in cubicle housing and dry cow accommodation where mastitis was a significant problem has been associated with improvements in the severity and prevalence of mastitis cases. The principle change has been to apply the design criteria of Bruce to the ventilation of the buildings and to maximise the available stack effect. There are no building designs that cannot be ventilated correctly. The effect is particularly easy to achieve in low volume building designs such as wooden kennels and Atcost© housing erected in the 1960s and 1970s. The thermal energy density of these relatively low structures is high, which means that once the correct area of apertures is created in the roofs and sidewalls the air flow rates within the buildings are as high as any cubicle house design.

CONCLUSIONS

Mastitis has many contributory factors that support survival, replication and decay. The proposal is that many examples of existing cattle housing have the ability to improve the removal of excess moisture and thermal concentrations to the benefit of cow comfort and the detriment of bacterial survival, as well as the delivery of clean fresh air that can be catastrophic to the survival rates of *E. coli* and *Strep. uberis*. The solution is to provide designed ventilation openings based on competent existing knowledge.

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ACKNOWLEDGEMENTS

This work is supported by LMS ltd.

NOTES

THE ROLE OF THE MILKING MACHINE IN MASTITIS

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MILKING MACHINES & MASTITIS:

There is considerable debate in the industry of just how much influence the milking machine and its function have on the new infection rate for mastitis. In 2004, a paper was presented at the NMC, which discussed the research behind what is known about milking machine and other factors of the new infection rate. Most new mastitis infections are caused by factors other than the milking machine.

A survey of research shows a range of 6 to 20% of new infections is directly or indirectly caused by the milking equipment. There certainly is much discussion about these percentages in the dairy industry. These influences can and do exert potent effects on the mastitis incidence on real farms under field conditions, and many in the industry will state that the machine is a very important factor far above what the research information may state. The four major areas affected by the milking machine are:

- Facilitate the transfer of bacteria between cows or quarters on the same cow.
- Aid in the multiplication of bacteria at the teat end.
- Increase bacterial penetration of the teat during milking.
- Modify the teat/intramammary environment to enhance bacterial infection or impair host defences.

The key factor to reducing and/or maintaining acceptable udder health is the number of bacteria present on teat skin when units are attached. A major goal should be to bring the cows to the parlour as calm and as clean as possible at every milking. Clean, calm cows will improve the overall parlour efficiency, performance and enhance milk quality. Cornell University data has determined that even the best udder preparation will only result in a reduction of between 80 to 85% of the number of bacteria on teats when they enter a parlour. When the new infection rate goes up, the number of bacteria on teats is the main factor.

Bacterial Transfer

If a milking machine is used on a cow infected with contagious bacteria, such as *Strep. agalactiae* or *Staph. aureus* then these bacteria can easily be moved to the next cow milked with that milking unit. Another common method of bacterial transfer is if a unit is contaminated at detach with environmental bacteria then these bacteria can be moved onto the teat skin of the next cow milked with the unit.

Bacteria can also move from one quarter to another during the milking process when the unit is attached to a cow. This transfer can be reduced by using newer style claws that have adequate volume and the ability to remove milk quickly from the claw into the milk hose away from the cow. Increasing the diameter of the short milk tube of the liner and the use of vented liners will also reduce this cross contamination effect. The use of vented liners will also improve milkability and pulsation in fast milking cows due to better removal of milk from the area below the teats.

Bacteria Multiplication

The milking machine can influence the overall teat end and teat skin condition. Smooth teat ends and teat skin reduce the ability of bacteria to multiply and develop on these surfaces. Commonly observed issues in the field are teat end hyperkeratosis and chapped skin. Both of these will allow bacteria to grow more efficiently on the teat skin and they both make teat end disinfection during udder preparation more difficult. When more bacteria exist on the teat just before milking the new infection rate will increase. Milking machines are also capable of causing small haemorrhages on the teats, which blister, open and become great locations for bacteria to easily multiply.

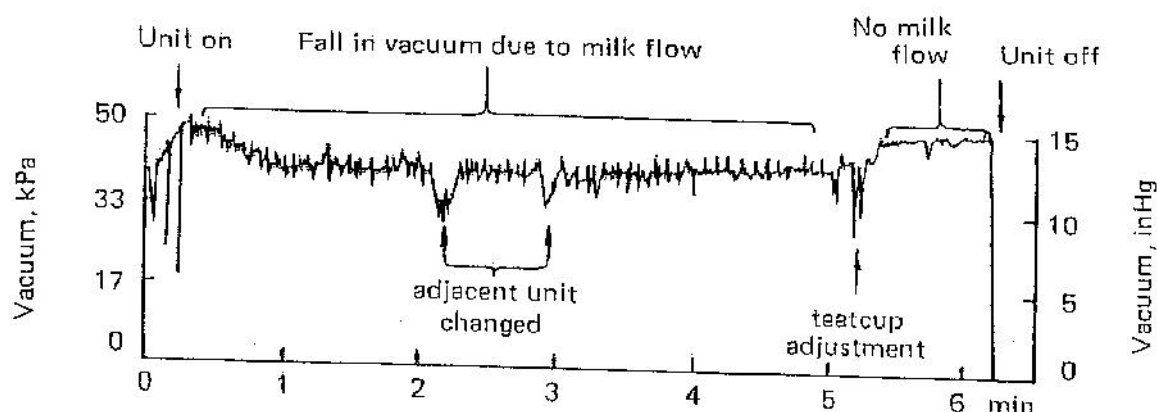
Teat end and teat skin condition are directly related to milking duration. Reduce the milking duration and these both improve. The principle of removing the available milk gently, completely and quickly will reduce the new infection rate. The consistency of cow handling and the udder preparation steps are also very critical to reducing the new infection rate and minimizing the average milking duration. Using quality pre- and post-dips applied in clean functional dippers will help maintain teat end and teat skin condition at appropriate levels.

Optimizing equipment settings, including a system vacuum level appropriate for the liner being used, the detach thresholds and the pulsation system can also reduce milking duration and improve the overall teat end and teat skin condition. Attention to scheduled service parts replacement and testing of the dynamic performance of the milking equipment on a regular basis is necessary to maximize the overall udder health of the herd. The stability of the system vacuum level, the pulsator performance graphs and the average peak milk flow claw vacuum between the first and second minute after unit attachment on high producing cows are key areas for regular scheduled analysis.

Teat Impacts

The action of the milking machine can cause bacteria to be propelled directly at the teat end and, in some cases, directly into the teat sinus during milking. A normal milking will have two types of vacuum fluctuation. Cyclic fluctuations are associated with each pulsation cycle and the

movement of the liner as it opens and closes. Cyclic vacuum fluctuations are also influenced by the ability of the milking system to remove milk in an efficient manner from the claw to the milk line. Irregular vacuum fluctuations are the result of sudden air admissions into the claw generally by leaks around one of more teats. These can be both audible and inaudible to the operator and are referred to as liner squawks. The penetration of bacteria into the teats most often occurs towards the end of milking. During this time there is little or no milk coming from one or more of the teats. When bacteria are moved into teats at this time the likelihood of flushing them out with additional milk is minimized so the new infection rate is increased. Close to or just before the unit detaches, the teat sinus and the gland sinus are often under a partial vacuum relative to the atmospheric pressure, which can allow better higher penetration into the gland and result more new infections. The following diagram illustrates how a normal complete milking graph can appear. This graph was taken from the textbook, "Machine Milking", and can be found on page 363 of this excellent text on milking machine function.



The drop in vacuum labelled "teatcup adjustment" indicates the typical time when liner squawks capable of causing teat end penetration occur. Based on the increase in the average milking immediately after this event there would be very little or no milk flow from this cow so any bacteria are now in an environment where they could grow rapidly with a food supply –milk– and an almost ideal temperature for growth.

Fine-tuning equipment settings, improving unit alignment, and attaching units to properly prepped teats will all decrease the incidence of liner squawks.

Host Defences

Many of the conditions that have been described in the first three sections can cause pain for the cow. Pain results in the release of various hormones into the cow from the adrenal gland and other organs. Adrenalin can cause constriction of blood vessels and will block the milk letdown of the hormone

oxytocin resulting in incomplete milk out and more time in low flow periods during milking. The more issues with the milking system, the greater the pain to the cow and a higher level of new infections. The goal is to test the system often enough to reduce the likelihood of machine malfunction that can lead to new infections.

While research indicates the machine plays only a small part relative to the new infection rate, many in the real world are telling producers the opposite, that the machine is a primary cause. This may be true in milking systems that have faulty pulsation systems, over used liners and hoses, short pulsation tubes in poor condition, improper takeoff settings that allow long milking durations after all visible milk flow has ceased from the cow, and using improper liners and system vacuum levels. Poorly maintained systems will have more irritated teat ends and more liner squawks so in these herds the machine will play a larger role in the new infection process. Some herds with very poor scheduled service program and many defective equipment issues will not experience increased mastitis or somatic cell levels. The reason is these herds have very low bacterial numbers on teats when units are attached. While these herds will have minimal mastitis issues, they will, in most cases, experience lower production due to the machine issues. Remember to “always complete the physical examination before making a diagnosis”. Look at production trends as well as the milk quality history of the dairy to fully understand the costs of a poor maintenance program.

Producers that have a full scheduled service program along with dynamic testing on a scheduled basis will have fewer new infections related to or primarily caused by the milking equipment. These two programs are good for the cows, good for the dairy producer, and good for the milking equipment dealer. There is no substitute for being in the barn (cowshed) or parlour on a scheduled basis for the purpose of testing the dynamic performance of the milking equipment, observing cow behaviour during milking and evaluating both cow handling and milking protocols. An evaluation of these areas, followed up with making recommendations to producers can and will lead to better overall satisfaction with the milking equipment and often an improvement of the herd udder health while maximizing the ability of the system to harvest milk.

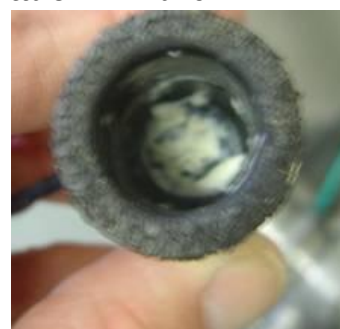
Let's look at a typical situation that I experienced several weeks ago. At the time of this investigation the sprinkler systems in both the holding pen and at feed lanes have been operating for a few weeks. The customer is concerned about an increase in both the SCC and clinical mastitis cases but tells me that nothing has changed with either the parlour maintenance or the milking procedures. When I visit the dairy at milking time, I find the equipment is working very well and all the dynamic graphs show minimal or no change from the last testing. I observe more manure splash and I see cows dripping water off their sides in the parlour. Due to this excess water, some cows are having units attached to wet teats, even with unchanged drying procedures. Some of this water is running down the sides of the

udder pooling on the mouthpieces of liners and then “magically disappearing” at the least liner squawk. Herds like this will typically also have increased total bacteria counts as well as increased coliform counts if these are routinely being run on the shipped milk. The fix in this case is to adjust the sprinklers at the feed alleyways and in the holding pen to prevent most cows from dripping water onto the sides of the udder.

Look for obvious maintenance issues on the milking facilities when you are visiting a dairy for an evaluation or just make a policy of developing a routine to just look at the parlour every day. Pointing out these issues will lead producers to see the value of a scheduled service program. The following are a few examples of a recent dairy visit I made with several veterinary colleagues at a parlour during a training session.



This last picture is the inside of a plug found on the milking line close to the receiver jar on a straight nipple that extended off the line about 2.54 cm and was likely installed to facilitate testing slug formation in the milk lines. When I found these, I asked the herdsman about their bacterial counts and suggested they were fluctuating on a regular basis. He wanted to know how we knew the counts were not good! No testing was performed on this dairy, only observation, and we easily convinced this producer to re-evaluate his decision to cut costs by reducing scheduled service program.



Emphasize the calculation of cows milked per stall per day or how many cows is the producer willing to put at risk per day when explaining the benefits of a scheduled service program. Many dealers also calculate the total cost of normal parts replacement including labour and then present this on a cost per cow per day. Everyone wins with scheduled service. The dairy producer will have a program designed to minimize mechanical issues relating to milk harvest, mastitis will be minimized, milk quality will be improved, milk production capability will be maximized and there will be fewer emergency calls for service of the milking system which result in less down time. As Mr. Goodwrench says in the General Motors commercials in the US about scheduled service for their cars and trucks, “you can pay now for maintenance or you can pay later when the vehicle breaks down along the road”.

Have you ever heard a veterinarian state that it must be the milking equipment, or some other factor such as the teat dip or the liner choice affecting a certain herd’s level of new infections? Veterinarians will base this opinion because they are looking at the how cow’s respond to their treatment regimens for clinical mastitis. They use the same or very similar protocols for two or more herds that have very similar milking procedures and cow handling, but one of the herds does not respond to the therapy very well compared to the other herds on similar protocols and management levels. They conclude that it must be something unique to the equipment or some of the products such as teat dips being used on the farms.

These veterinarians are violating one of the major rules of veterinary practice. They are “making a diagnosis without completing the physical exam” of the dairy. One of the most common sources of low response to treatment is sub clinical acidosis. Cows that have repeated bouts of rumen acid increases will have impaired immune systems and therefore they will not respond to treatment in a predictable fashion. Many very well managed herds have subclinical rumen acidosis in a surprising number of cows all during the calendar year. Some common causes are lack of adequate fibre in the ration, feeding to eliminate weigh backs so cows are out of feed for longer than 1.5 hours per day, lack of consistent feed push up or excessive sorting of the ration in the feed bunk. If cows, especially high producing groups or individual cows, have not had access to a Total Mixed Ration for more than 3 hours they will slug feed on the TMR and they can and do develop subclinical acidosis on a regular basis.

Production can still be very good in these cows but they typically will show reduced milk fat levels and sometimes even reversed milk fat to milk protein ratios. In herds with meters, these cows often show more than a 2-liter milk production swing from milking to milking or maybe even more for 24-hour milk production. Herds with a high level of subclinical rumen acidosis will also show longer toes and toes that begin to cross and touch on many cows. If most or many of the cows in the herd exhibit these hoof changes

then “the abnormal becomes normal” syndrome sets in and people do not make the association between the hoof lesions and the basic cause.

The months of July and August in the US are the times when many herds have a higher incidence of sub clinical rumen acidosis due to the additive affects of heat stress and other related feeding issues. Cows can easily increase their core body temperature by over 1 degree centigrade if the holding pen/parlour does not have adequate fans and sprinkler systems. Herds housed under these conditions will see a very pronounced spike in claw lesions such as sub solar abscesses and white line disease in cows between mid August through the end of October as reported by their hoof trimmers. These occur between 6 to 8 weeks after the first exposure to repeated bouts of holding pen heat stress.

The secondary effect of ration fibre issues, slug feeding, sorting, or heat stress are increased levels of clinical mastitis. Mastitis is directly related to the number of bacteria on teats when units are attached but the new infection rate is also related to the overall immune response of the individual cow. Data from many parts of the US shows an increase in the overall SCC level in shipped milk in the period between July and October. This is related to these two factors: the number of bacteria on teats when units are attached and the overall immune response of the cow.

If herds have some of these hoof lesions along with increased mastitis levels, these are not caused by the milking equipment, but the equipment, liners or teat dip often get the blame. If you are dealing with problem herds, look at these issues to determine if they may be impacting the new infection rate for the herd. Remember to always **“complete the physical exam before making a diagnosis!”** Be sure to look at all issues as you are called to problem farms. Involve the local veterinarian and work with him or her as a problem herd is investigated. The results can be dramatic and profitable for the dairy.

NOTES

POSTERS

AN EVALUATION OF BACTERIAL COUNT IN A LINER BEFORE AND AFTER SPRAYING WITH PERACETIC ACID SOLUTION

Richard Hiley¹, Richard May¹, Colin Kingston¹ and Ian Ohnstad²

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INTRODUCTION

The milking cluster is a well known source of transmission of mastitis pathogens from cow to cow. Prior scientific research has demonstrated that once an infected cow has been milked, the next 6 to 8 cows milked through that same cluster are at risk of contamination, the first 1 or 2 cows being exposed to a particularly high level of risk.

Sanitisation of the cluster between cows is one effective method of reducing this risk by killing any pathogens present in the cluster before it is re-attached to the next cow. Peracetic acid (PAA) (also commonly referred to as peroxyacetic acid) has been found to be a particularly effective means of cluster sanitisation.

Methods of cluster sanitisation vary in capital intensity and level of automation ranging from manual cluster dipping through semi-automated cluster spraying to fully automated cluster back flushing.

EVALUATION METHOD

An evaluation was undertaken to assess the level of bacterial soiling on the internal surfaces of a liner before and after spraying it with a 0.5% (5,000 ppm) concentration of peracetic acid solution using a semi-automated Ambic PeraSpray system. Selected liners from 30 cows were swabbed after the cluster was removed from the cow. After swabbing, the liner was subjected to a 3 second spray of disinfectant solution and left to drain for a further 10 seconds before a second swab was collected.

LABORATORY METHODS AND STATISTICAL ANALYSIS

Growth was evaluated with the aim of allowing counts of a number of 'putative' bacterial populations to be made. Samples were evaluated using a standard pour plate technique using 1ml of hygiene swab solution and 2 or 3 dilutions according to the media used. When necessary, and where appropriate, further dilutions were undertaken to allow an accurate enumeration of colony forming units (cfu) to be determined. This technique allowed a threshold of detection of 10cfu per swab.

RESULTS

The results of the swab counts before and after treatment with the

PeraSpray system are shown in the Tables 1 and 2 below:

Table 1 Summary of findings of Pre- and Post disinfection swab counts - mean values

Parameter	Pre-Flush	Post-Flush	% change
Total Viable Count (cfu)	30562	268	99.1%
Staphylococcus spp Count (cfu)	996	22	97.8%
Streptococcus spp Count (cfu)	4709	46	99.0%
Coliform Count (cfu)	6	1	83.3%

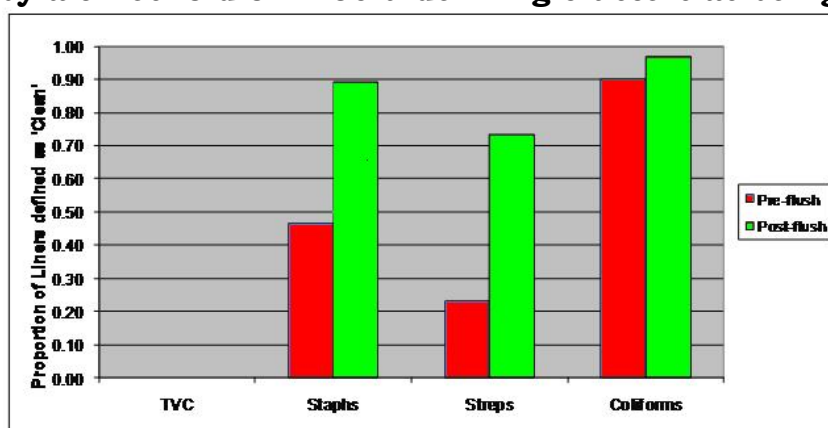
Table 2 Summary of findings of Pre- and Post disinfection swab counts - median values

Parameter	Pre-Flush	Post-Flush	% change
Total Viable Count (cfu)	12425	95	99.2%
Staphylococcus spp Count (cfu)	10	0	100.0%
Streptococcus spp Count (cfu)	455	0	100.0%
Coliform Count (cfu)	0	0	-

The results of the mean values are also illustrated in Figure 1 and can be summarized as follows:

- Total Viable Counts were significantly reduced
- Streptococcal (Step) counts were significantly reduced
- Staphylococcal (Staph) counts were significantly reduced
- Insufficient coliforms were identified to allow meaningful analysis

Figure 1 Illustration of the efficacy of the PeraSpray process as measured by a threshold of <10cfu defining clusters as being 'clean'



CONCLUSION

The PeraSpray system significantly reduced the bacterial count of liners following its use.

While the reduction in bacterial loading may not be as great as seen with fully automated systems, there is a significant cost advantage to be offset against a slight reduction in performance compared with automated systems

TO CMT OR NOT TO CMT – WHAT’S THE VALUE?

Judith Roberts and Malcolm Joyce

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The California Mastitis Test (CMT) is widely used as a qualitative cow side test for the detection of subclinical mastitis in dairy cattle. The threshold for this test is quoted as being between 200,000 and 400,000 cells/ml, depending on the source and is based on human observations of the reaction rather than scientific measurement of the gel that is formed. The viscosity of the milk-CMT gel reaction has been characterised (2) but at present no successful attempt published determining whether the reaction can assess quantitatively the somatic cell count (SCC) in milk samples and the threshold at which the gel reaction occurs. The objective of this research is to fully characterise the CMT reaction at various SCC's, to establish the threshold of the reaction and the quantitative relationship between the CMT reaction and SCC in milk.

Foremilk samples were collected from 35 quarters from 21 Holstein-Friesian cows on a commercial dairy herd in the UK. Viscosity measurements were made using a Brookfield DV-II+ Viscometer (Brookfield, UK) at constant shear rate of 30rpm at 25°C for 3.5ml milk with 3.5ml CMT reagent. A proportion of each quarter milk sample was sent away for external SCC measurement at a commercial approved laboratory (QMMS Limited, UK).

Figure 1 shows the typical apparent viscosity curve for milk samples above the threshold for detection. The maximum viscosity (μ_{max}) and time to reach maximum viscosity ($t_{\mu_{max}}$) are defined at the turning point of the curve. Figure 2 shows the typical patterns for low, medium and high SCC samples using data fitted by the least squares method to the equation $\mu = a/t(1/(e^{b/t} - c))$. The low SCC samples do not have a maximum viscosity and therefore $t_{\mu_{max}}$ is zero. The correlation between SCC and μ_{max} is shown in Figure 3. The correlation coefficient is 0.92. The threshold for the CMT reaction can be clearly demonstrated by plotting log SCC against $1/e^{t_{\mu_{max}}}$, as shown in Figure 4. The time to reach maximum viscosity was taken from the differential equation of the fitted data set for each milk sample. This threshold lies between 5.54 and 5.64, correlating to a SCC of approximately 390,000.

Figure 1 Graph to show a typical curve for apparent viscosity against time

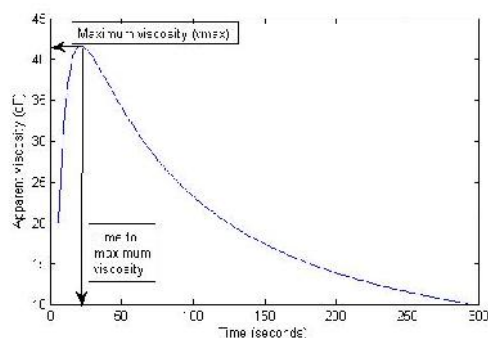


Figure 2 Graph to show typical curves for apparent viscosity against time for low, medium and high SCC samples

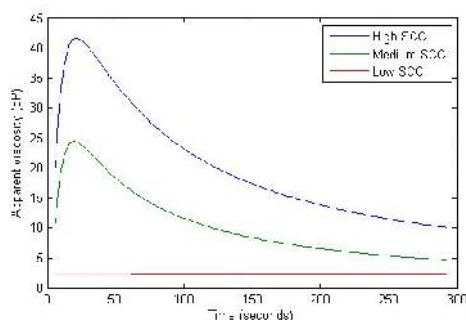


Figure 3 Graph to show SCC against μ_{max}

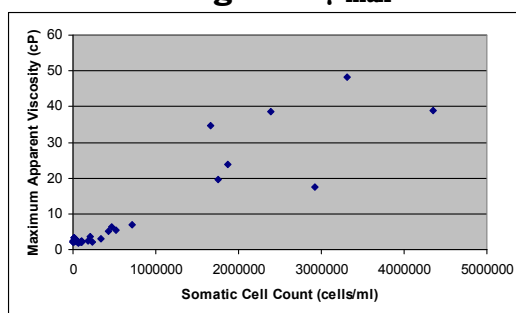
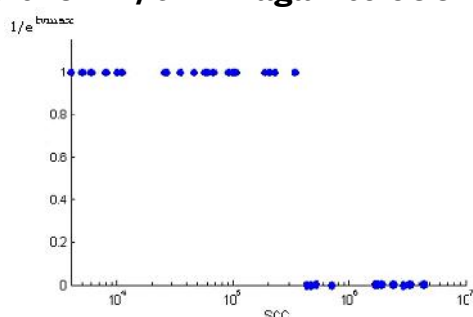


Figure 4 Graph to show $1/e^{-t_{\mu_{max}}}$ against SCC



The CMT reaction has a defined viscosity curve for each sample and there is a good correlation between SCC and μ_{max} when samples are compared. The threshold at which the gel reaction occurs for the CMT can be clearly demonstrated by plotting log SCC against $1/e^{-t_{\mu_{max}}}$, with the threshold being approximately 390,000 cells/ml.

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ACKNOWLEDGEMENTS

Judith Roberts would like to acknowledge the support of the RCVS Trust with their Small Project Grant to assist in this research project.

THE EFFECT OF EM™-TEAT DIP VERSUS CONVENTIONAL IODINE DIP

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EM-Teat Dip is a teat care product based on the fermentation of a natural biopolymer with EM-1, (Effective Micro-organisms). It includes lactic acid bacteria, which has been shown to have antimicrobial and bactericidal activity (1). It provides an active protective film on the teats post milking.

In a field study the effect of EM-Teat dip was compared with a conventional iodine and glycerol post-milking dip as a positive control. Ten Holstein Friesian cows in the herd from the Faculty of Veterinary Medicine, University of Leipzig, (average 30L/day , at 4.5% fat and 3.5% protein) were selected for a within cow diagonal dipping trial of two periods of 14 days separated by one week of dipping with iodine teat dip. Alternate teats within cow were dipped with either of the products for weeks one and two, in week three, all teats were dipped with iodine and then in weeks four and five the teats were dipped with the different dip from the first period (Figure 1). Five cows were in early lactation (0-60 days average age of 3 years) and the other five cows were in late lactation (150-250 days average age of 5 years).

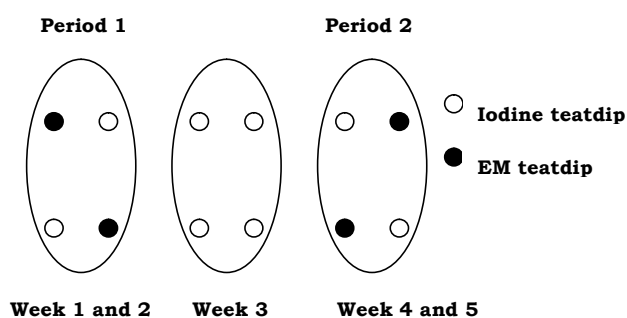


Figure 1: Application of the two products during the dip trial

Teat end and teat canal swabs and milk samples were taken after morning milking on day 0, 14, 21 and 35.

The milk samples were assessed immunologically and bacteriologically. Lactoferrin (Lf) and C-reactive protein (CRP) were measured by ELISA. Somatic Cell Count (SCC) and urea were measured electronically.

The teat end and teat canal swabs and the milk samples were incubated aerobically for 24h, micro-aerophilically for 24 or 48h and anaerobically for 72h at 37 °C. Selective media were used for quantitative and qualitative bacterial analysis. The types of organisms were categorised as in Table 1.

Table 1: Micro-organisms divided in categories

Category 1	<i>Lactic acid</i> <i>Corynebacterium bovis</i> <i>Micrococcus luteus</i>
Category 2	<i>Streptococcus spp.</i> <i>Enterococcus spp.</i> <i>Coagulase Negative Staphylococcus</i> <i>Bacillus spp.</i>
Category 3	<i>Staphylococcus aureus</i> <i>Staphylococcus intermedius</i>
Category 4	<i>Escherichia coli</i> <i>Actinobacillus spp.</i> <i>Citrobacter spp.</i>
Category 5	<i>Yeast</i> <i>Mould</i>

The Shapiro-Wilk-Test was used to test for normality of the results.

Within time urea concentrations were analysed by the Paired Sample T-test and between the two dips with the T-test. Both analyses showed no significant differences. The other immunological and bacteriological parameters were analysed within time with the Wilcoxon test. The Mann Whitney U test was used to analyse the results between the two products. No significant differences were found in time and between the two test groups for the SCC, Lf and CRP concentrations in the milk.

Between the treatment groups no significant differences were found. In the iodine based dip group there was a significant ($p = 0,001$) difference of category 3 type of organisms found on the teat ends. In the teat canals there were significant differences of types 2 ($p = 0,022$) and 3 ($p = 0,015$) organisms within time. The results also showed significantly fewer category 3 ($p = 0,001$) organisms in the milk after using iodine dip. Within time there were significantly ($p = 0,002$) more anaerobic CFU's on the teat ends after using EM teatdip. Significantly fewer micro-aerophilic CFU's were found on the teat ends after using iodine dip. Under anaerobic conditions significantly more CFU's were found within time on teat ends ($p = 0,002$) and teat canals ($0,001$) after using EM teat dip. There were also more CFU's in the teat canals ($p = 0,001$) after the iodine dip, but fewer CFU's were found in the milk ($p = 0,001$). Under micro aerophilic conditions and within time significantly fewer micro-aerophilic CFU's were found on the teat ends ($p = 0,007$) as well as in the milk ($p = 0,001$) after using iodine dip. Between the tested dips, no significant differences were found

ACKNOWLEDGEMENT

The study was supported by EM-Agriton BV Netherlands

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COMPARATIVE TRIAL OF BACTOPROOF™ QPCR AND CONVENTIONAL CULTURE FOR MASTITIS TESTING.

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Identification of the pathogen involved is a key step in the control of mastitis; it should be used to help improving control measures, fine tune treatments and help with decision making for problem cows. Traditionally, this has been done by bacterial culture, but recently a Polymerase Chain reaction (PCR) test has been developed which allows a more rapid analysis for a range of bacteria. Additional advantages of PCR testing are that samples can be preserved immediately after collection and posted at ambient temperatures.

PCR routinely tests for *Staph aureus*, Coagulase Negative Staphylococci (CNS), *Strep. uberis*, *Strep. dysgalactiae*, *Strep. agalactiae*, *E. coli*, *C. bovis*, *Klebsiella*, *Enterococcus*, *Serratia marcescens*, *A. pyogenes* and *P. indolicus* and the Beta-lactamase gene. *Mycoplasma bovis* can be identified using an additional PCR test.

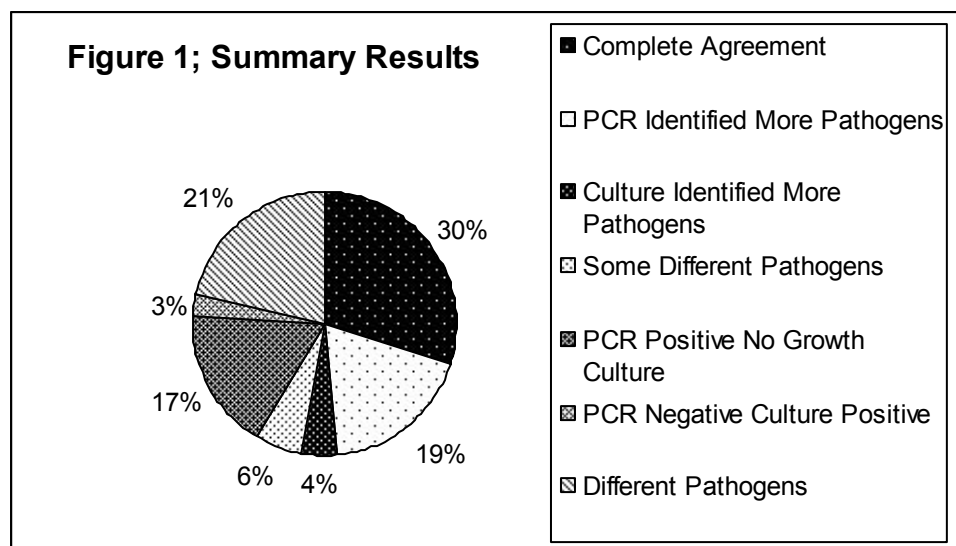
MATERIALS AND METHODS

Duplicate milk samples were collected from 70 cases. These were from high cell count and clinical mastitis cases from vets or clients of the Shepton Veterinary Group. All samples tested using PCR and conventional bacteriology. For conventional bacteriology, clinical samples were frozen and sent as a batch while high cell count samples were posted fresh and sent with ice packs.

RESULTS

Overall PCR yielded a positive result in 64 of the 70 samples tested (91%). Culture gave a positive result in 52 (74%) of the samples tested. Contaminated cultures were not observed in this trial, perhaps reflecting the attention to sampling technique and transport conditions of the samples. In this study 54% of *Staph aureus* and 24% of CNS isolates had the β -lactamase gene present, penicillin resistance was not tested in culture.

There were differences between the results obtained using PCR and conventional culture (figure 1).



DISCUSSION

Conventional culture and PCR are different diagnostic techniques and in 40% of samples tested yielded substantially different results.

With a few exceptions PCR detected substantially more pathogens than conventional culture. No growth results are a significant problem in conventional culture and can be a factor in the reluctance of vets and farmers to test for mastitis pathogens. In culture negative samples which had positive results by PCR, over half of the bacteria detected were CNS; in the remaining samples *Strep. uberis*, *E. coli*, *Staph. aureus* and *C. bovis*. This highlights a major potential advantage of using DNA technology and supports the view that it may be more sensitive and accurate than bacteriology. It should be noted that in some cases conventional bacteriology identified pathogens which PCR testing cannot currently identify including yeasts, *Proteus* spp., *Bacillus* spp., *Pseudomonas* spp., and *Pasteurella* spp. Negative test results by either method should be interpreted with caution. The high rate of identification of β -lactamase gene for *Staph aureus* (56% from 13 cows) and moderate levels for CNS (24% from 42 cows) by PCR provided useful information for treatment decisions. Rapid identification of the β -lactamase gene responsible for penicillin resistance is a significant advantage compared to conventional culture.

Farmers liked the fact that they could get their PCR results back quickly and that PCR testing allowed easy sample despatch. The benefit of sampling non-responsive cows under treatment was also appreciated. The greater percentage of samples returning a positive result was also seen as important. Laboratory work is seen as being expensive, so the more positive samples returned the better. Although no diagnostic test is perfect for all situations, PCR technology for mastitis diagnosis has some significant advantages, and used correctly has the potential to improve mastitis control in the UK.

LARGE HERD MASTITIS: HOW LOW CAN YOU GO?

¹J D Reader, J D, ²N Baker, ²J N Womack, and ²R Y Goodwin

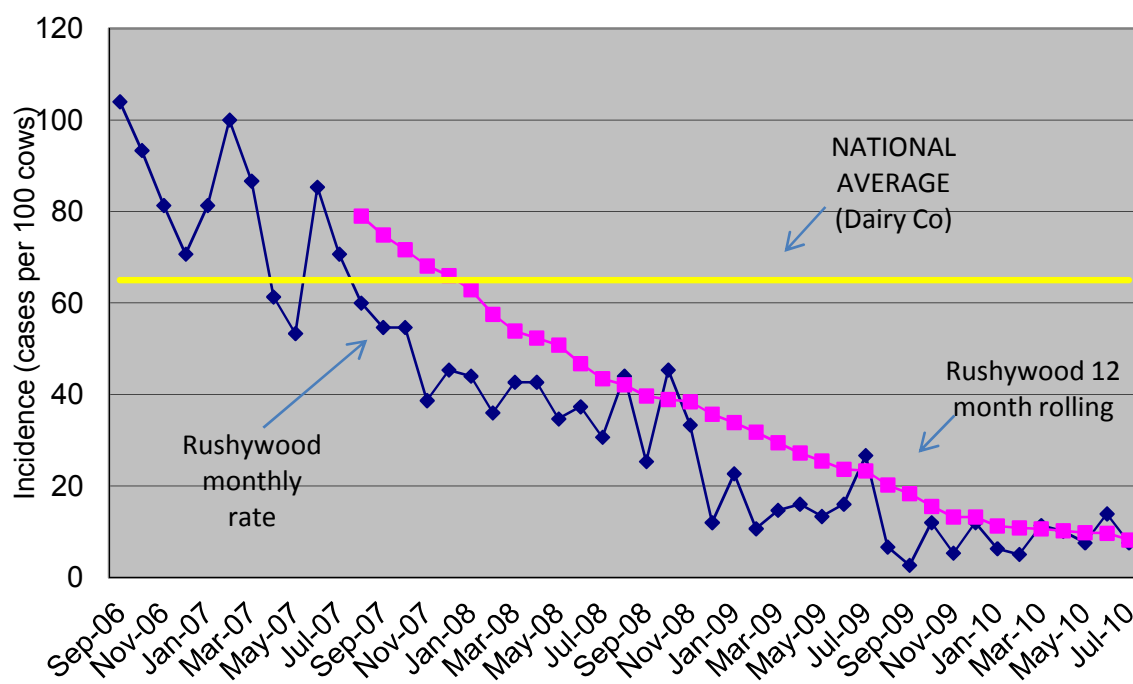
¹Synergy Farm Health, West Hill Barns, Evershot, Dorset, DT2 0LD, UK; office@synergyfarmhealth.com ; ²Bakers of Haselbury Plucknett , Rushywood Farm, Haselbury Plucknett, Somerset, UK. www.bakerscheddar.com

Large herds have come under increasing scrutiny due to the perception that an increase in herd size can lead to poor health and welfare. This case study demonstrates excellent udder health in a large herd under UK conditions.

Key Herd Parameters

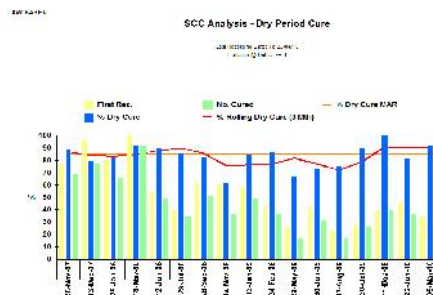
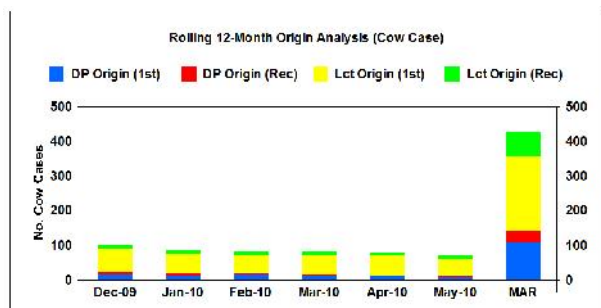
- 1000 cow dairy herd
- 9,800 kg 305 d yield
- 80 point external rotary
- 3X Milking
- Dump parlour for all fresh calved cows and cows with antibiotic residues
- 'Just in time calving'
- Traditional Mature cheddar production
- No Orbeseal permitted
- 2 Farm Managers
- 9 Polish Milkers working in shifts of 5
- TMR, Fresh and High Group Zero Grazed

Rushywood Farm - Mastitis Incidence Rate



Mastitis Data

- SCC (3 month) 131
- Rolling 12 month incidence: 8 cases per 100 cows per year
- Dry cow new infection rate (SCC): 9% (Target <10%)
- Dry cow cure rate (SCC): 92% (Target > 85%)



Cost of clinical Mastitis (@ £200 case)
2006 £180,000 **2009** £18,600

Key Factors

- Clear vision from owner (Target zero mastitis)
- Clear defined management structure
- Attention to detail
- Good communication
- Deep sand cubicles
- Minimal contact with straw/organic bedding at calving
- Strict Milking Routine (with Polish translated SOPs)
- Regular parlour testing / teat scoring (Dairy Group)
- Modern Building design / Stocking Rate < 90%
- Rapid identification and Combination treatment of cases
- Separate 'dump' parlour
- Monthly data review
- Routine use of J5 vaccine

CONCLUSION

This case study demonstrates that with good facilities, following standard operating procedures, the highest levels of stockmanship and attention to detail, udder health can be maintained to the highest level even under intensive conditions in the UK.

ACKNOWLEDGEMENTS

Quality Milk Management Services (QMMS)

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USE OF FARMER RECORDED MASTITIS DATA FOR GENETIC EVALUATIONS OF UDDER HEALTH IN DAIRY CATTLE

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The udder health sub-index in the UK national profit index (£PLI) utilises test day records for somatic cell count (SCC) as well as an udder composite type trait, but does not, as yet, include a direct measure of mastitis. Inclusion of mastitis is expected to enhance the prediction of udder health in genetic evaluations. Some UK dairy herds have been recording mastitis events of their cows through milk recording organisations (MROs) and preliminary analyses on the level of recording suggest that mastitis could be used in addition to present udder health traits (1).

Mastitis events were recorded by farmers voluntarily as part of routine milk recording. Mastitis events that occurred 0 to 305 days after calving in the first three lactations were considered. Mastitis was analysed as a binary trait (0/1), so analyses comprised affected animals and their contemporaries of the same herd, year and season of calving. Univariate analysis of mastitis and bivariate analyses with production, lameness, fertility, SCC, lifespan, and type data were carried out. Genetic evaluations were also carried out on SCC and mastitis data, and proofs were obtained.

The heritability estimate of mastitis was $\sim 0.04 \pm 0.01$ and was strongly genetically correlated with SCC (0.65 ± 0.07) and moderately correlated with udder composite (0.28 ± 0.10), which are traits currently included in the udder health sub-index. Genetic correlations of mastitis with other traits, using a repeatability model, are shown in Table 1.

Table 1. Genetic correlations of mastitis with other traits

Trait type	Trait	Genetic correlation
Production	Milk yield	0.30 (0.07)
	Protein yield	0.31 (0.04)
	Fat yield	0.19 (0.05)
Health	Lactation average somatic cell count	0.65 (0.07)
	Lameness	0.26 (0.09)
Fertility	Calving Interval	0.28 (0.07)
	Days to first service	0.25 (0.06)
Lifespan	Days of productive life	-0.64 (0.06)
Type	Udder composite	-0.28 (0.10)
(in first	Udder depth	-0.50 (0.08)
lactation)	Fore udder attachment	-0.44(0.09)

The following results were obtained from Holstein/Friesian bulls that had a reliability of at least 30 % for mastitis and SCC. PTAs (Predicted Transmitting Ability) ranged from -4.0 to 13.7 % and -18.4 to 71.2% for mastitis and SCC, respectively. Figure 1 shows that the genetic trend of

mastitis closely tracks that of SCC. There has been an increase in PTA's in both traits, in the undesired direction, but recently (since use of SCC in evaluations from 1998) this has stabilised and both PTA's now appear to be decreasing.

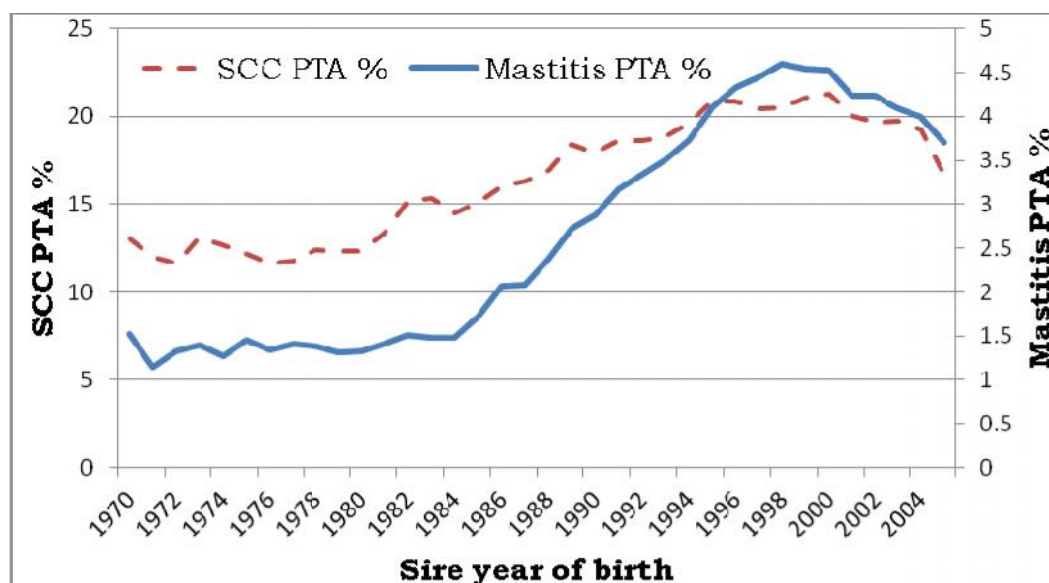


Figure 1 Mean mastitis and SCC PTAs of sires born 1970 to 2005

The heritability of mastitis is low, but sufficient genetic variance and its inclusion together with current udder health traits in an index should help to improve mastitis resistance, as experienced by other countries (2). An antagonistic relationship between mastitis and production traits was found, which suggests animals genetically above average for yield traits are more susceptible to mastitis. Genetic correlations show that increased mastitis resistance would be accompanied by reductions in lameness, increased fertility, and increased length of productive life. Future work should involve encouraging and increasing farmer awareness of the benefits of disease recording for their own herd management, in addition to the use of data in genetic evaluations.

ACKNOWLEDGEMENTS

Many thanks to the farmers for recording the data as well as funding from the Defra Sustainable Livestock Production LINK Programme, the Scottish Government, CIS, Cogent, DairyCo, Genus, Holstein UK and NMR.

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APPARENT DRY PERIOD CURE RATES AND DURATION OF INFECTION

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A retrospective performance analysis was carried out for 309 farms who received a Clover Cell Check subclinical mastitis report in August 2010. These farms are served by 25 practices across the UK who make use of the Clover Cell Check web tool. Most practices are members of XLVets.

Clover Cell Check automatically downloads and processes farm milk recording data for veterinary practitioners who have sought permission from their farm clients. Much of the analysis reflects the principles established by NMR's Herd Companion but the Cell Check software also allows the user to monitor performance against a benchmark group of farms served by the same practice or group of veterinary practices. Targets are developed by measuring the upper quartile of farms whose data is processed.

Dry period performance is an important component of herd level cell count control. Clover Cell Check reports the 'apparent dry period protection rate' and 'apparent dry period cure rate' through comparing the last recording of any lactation with the first recording of the subsequent lactation. There is an interaction between these two criteria in that poor apparent protection can lead to a higher proportion of cows that apparently fail to 'cure'. There are also several other weaknesses to these measures when viewed in close detail at herd level. However, comparison of these figures between farms can prove revealing.

Across the 309 herds studied the upper quartile of farms show apparent dry period cure rates of more than 78% and the lower quartile of herds show apparent cure rates of 64% or lower. Apparent dry period prevention rates of 88% are achieved by the upper quartile of farmers while for the same parameter only 78% is achieved by the lower quartile.

The significant difference between apparent dry period cure rates between farms was investigated as part of this report. Barkema *et al.* (2006) have previously shown the relationship between duration of intramammary infection and poor dry period cure rate when dealing with *Staph. aureus* infected animals. The somatic cell count (SCC) recording data from the 47426 cows which from which this dataset arises was analysed to further investigate the relationship between duration of intramammary infection and apparent dry period cure rate.

The number of cows infected for over six months was analysed for each herd and represented as a proportion of the total herd size. The upper quartile of herds had 3.57% of the herd or less infected for over six months, while the lower quartile of the herds had 9.39% or more of the herd infected for over

six months. The average apparent dry period cure rates for each quartile was then calculated to allow comparison and is displayed below.

	Percentage of herd infected over 6months	Average apparent dry period cure rate
Upper Quartile	3.57%	76.26%
Lower Quartile	9.39%	64.95%

The results show a significant decrease in average apparent dry period cure rates as the proportion of the herd infected for over six months increases.

The analysis of data in this way not only allows realistic targets to be set when delivering cell count monitoring services but also has clinical relevance when dealing with herds with a high proportion of the herd infected for over 6 months. The significant difference in apparent dry period cure rates between the upper and lower quartiles of these farms could have considerable financial implications; an increased proportion of the herd starting their lactation with a high cell count could lead to increased use of lactation therapy to try and control SCC and potential bulk milk SCC penalties if thresholds are exceeded.

This data suggests that early intervention for cows' whose recorded cell count may indicate infection, may enhance herd performance in terms of quality milk production.

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IDENTIFYING MARKERS FOR MASTITIS SUSCEPTIBILITY IN BOVINE IMMUNE RECEPTORS

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The genetic selection of cattle less prone to mastitis and other diseases, with little or no impact on milk productivity, would prove beneficial to dairy breeders. Mastitis in the UK continues to have huge economical impact, costing the dairy industry many millions of pounds each year. The ability to resolve mammary infection either asymptotically or with minor clinical symptoms would reduce production losses and costs, as well as improving animal welfare. Appropriate early immune responses to mastitic pathogens are therefore vital if infection is to be controlled. The detection of pathogens within the udder is carried out by specific receptors on resident somatic cells which trigger the onset of the immune response. Changes in these receptors may affect their ability to recognise pathogens. These changes may occur as mutations in the DNA, and inherited in the form of single nucleotide polymorphisms (SNPs). Therefore their identification may provide suitable genetic markers for selection. By choosing animals with favourable mutations, herd mastitis rates could be reduced.

The aim of this study is to identify SNPs in bovine receptors and look at their potential correlation with an animal's susceptibility to disease, including mastitis, as well as their influence upon milk quality and quantity. A Holstein Friesian herd (n= >400) situated in Berkshire was used to obtain genomic DNA for sequencing of target genes. Identified SNPs were then matched to any health or milk productivity traits available through the local InterHerd database and submitted National Milk Records (NMR) data. These included clinical mastitis case histories, lameness, SCC, 305d milk yields and milk protein/fat percentages.

Numerous SNPs have been detected within the candidate genes. To date SNPs within one immune receptor gene have shown promise as markers for mastitis susceptibility. Screening for these markers may reduce the number of clinical cases by up to 39%.

Anecdotal evidence suggests that animals less prone to mastitis are less productive, in terms of milk quality and quantity. However initial data indicates that the SNPs identified in this study have little, if any detrimental effect on milk productivity. These preliminary findings warrant further investigation and possible functional studies to determine their effect upon the coding receptor in terms of its production and/or activity.

ACKNOWLEDGEMENTS

This study is contributing as part of a PhD project funded by the BBSRC.

GOOD HYGIENE PRACTICE ON DAIRY FARMS

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Good hygiene practice on dairy farms is essential in order to obtain good quality milk with low bactoscans and somatic cell counts (SCC).

The FAO (Food and Agriculture Organisation) has published a guideline on: Milking, milk production hygiene and udder health. The aim of our poster is to make a critical evaluation of the FAO's guideline and to place the emphasis on the best way to apply it in order to be effective at economically.

General prevention measures should be established for feeding, housing, milking and managing cattle. The farmer must be aware of the importance of management and hygiene control and prevention.

The milking machine equipment must be correctly tested, maintained, cleaned and renewed if necessary, in order to avoid milk and teats contamination.

The housing is a reservoir of pathogens potentially able to cause mastitis. The bedding area must be kept clean and at least 5 m³ area/cow be allowed. An insecticide should be used because flies can transmit the pathogens responsible for summer mastitis.

The teats must be in good condition. Several teat dips with different qualities are available commercially. Choose teat dips with data based on field trials carried out using a recognized protocol.

Several parameters should be used and monitored: teat condition improvement as it is directly related to the milk out time and milk production, bulk somatic cell count which is a milk price major parameter and of course the number of new infection cases.

A method of detection of clinical mastitis must be established: every quarter should be examined and palpated every day. For subclinical mastitis, use a CMT test can be used when SCC are high or after a clinical mastitis.

The treatment must be done in a rational way: treat with antibiotic preparation as prescribed by a veterinarian. Clean hands and the teat before inserting antibiotic treatments otherwise secondary infection can be introduced.

Use dry cow antibiotic or teat sealant at drying off to either prevent or cure infections (antibiotic treatment is necessary if SCCs are high).

The poster gives figures, normal values and limits to choose the best scheme of prevention of mastitis and all the steps are detailed to be effective and to reap the benefits of their application.

ASSESSING MILKING CLUSTER HYGIENE VALUE

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The disinfection of milking clusters following the milking of cows with clinical mastitis or those with high Somatic Cell Counts (SCC) to minimise the transmission of contagious mastitis caused by *Staphylococcus aureus* to herd mates has long been recommended as part of good mastitis control practice. More recently it has been appreciated that environmental mastitis caused by *Streptococcus uberis* can also be spread from cow to cow at milking.

STUDY BASIS

The Sorgene National Cluster Hygiene Study was undertaken by *The Dairy Group* in the summer of 2010 to establish the attitudes and approaches of UK dairy farmers to routine milking cluster disinfection and to identify areas for improvement. It involved consultant interviews with 100 UK dairymen representing a good cross-section of current dairy management practice.

The participating herds range from fewer than 60 to more than 960 cows averaging from under 4500 to over 12,000 litres/cow with rolling average SCCs ranging from 110,000 to 320,000 cells/ml. Reflecting current commercial practice, the vast majority milk in herringbone parlours, although a number have rotary, tandem as well as the most modern automated milking systems.

KEY FINDINGS

For the first time ever, the study presents a clear picture of milking cluster hygiene practices on UK dairy farms.

- Encouragingly, the overwhelming majority of producers (96%) see high standards of cluster hygiene as an essential part of modern mastitis control and vast majority (83%) are currently employing some form of cluster disinfection – as against 53% in the past.
- The extent to which they are routinely disinfecting clusters, however, varies widely from farm to farm, with most (73%) not generally doing so after milking each cow and (12%) never doing so even after milking mastitic cows.
- While more than three quarters of dairymen (76%) see routine cluster disinfection as important in controlling contagious mastitis, noticeably fewer (68%) appreciate its potential role in controlling environmental mastitis.
- Also concerning is the greater apparent rigour in treating clusters following the milking of high SCC cows, only just over half the producers (51%) always or generally doing so compared to the 86% routinely disinfecting clusters after milking cows with clinical mastitis.
- Cluster dipping remains the most popular form of routine disinfection, being undertaken by just under half (48%) of dairymen – against 40% in the past.

Automatic back-flushing systems have, however, seen the most rapid increase in usage, from 2% of herds in the past to a current 22%.

- Although a few producers still employ water alone, there is widespread recognition of the need to use a good disinfectant in cluster hygiene, with peracetic acid/hydrogen peroxide combinations being preferred by most (70%).
- The way disinfection is carried out on farm may not be making the most of its value, however. The fact that a good third (35%) consider cluster spraying to be as effective as dipping or back-flushing and nearly a quarter reckon (24%) that good pre- and post-milking teat disinfection makes disinfecting cluster unnecessary clearly underlines this.
- As in so many things these days, time appears to be a far more important constraint to improved cluster hygiene practice than understanding.
- The vast majority of producers (82%) recognise the value of routine cluster disinfection in reducing mastitis cross-contamination. Balanced against this, however, around two thirds (67%) regard its time-consuming nature as a major drawback.
- Interestingly, in practice most producers employing the technique (63%) – including many using the most time-consuming dipping regimes – actually find it adds less than five minutes to their milking time.

CONCLUSIONS

Most UK dairymen clearly understand the benefits of good cluster hygiene quite well. However many are failing to make the most of routine cluster disinfection in their day-to-day management, either through less than ideal practice, concerns over extra time-consumption or both.

To address this position, milk producers need to appreciate that:

- Environmental mastitis caused by *Streptococcus uberis* as well as contagious infections can readily be spread from cow to cow at milking.
- The risk of mastitis spread from high SCC cows at milking is at least as high as that from animals showing clinical disease.
- Good quality disinfectants need to be used at the concentrations and with the contact times and programmes recommended by their manufacturers.
- Time need not be such a barrier to effective routine cluster disinfection as it is often perceived to be, even with manual systems.

Although automatic back-flushing systems can be valuable in reducing the time requirement of routine cluster disinfection, they need careful cost-benefit appraisal prior to installation.

ACKNOWLEDGEMENT

The Sorgene study was funded by UK rural hygiene leaders, BASF Pest Control Solutions.

Further information and copies of the full report are available from Paula Chatterton on 0151 422 4810 or paula.chatterton@basf.com.

THE ROLE OF COMPUTER SOFTWARE IN THE ANALYSIS OF INFECTED QUARTERS TO DESCRIBE AND CHARACTERISE THE MASTITIS CHALLENGE IN A HERD

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INTRODUCTION

Where farmers are recording the quarter(s) affected by mastitis there are opportunities to analyse mastitis in much greater detail. Characterising the disease at a quarter level in this way can provide valuable indications of the type of infection in the herd.

Routine milk recording generally only provides detail on cow cases, indicating that the cow has had one or more cases of mastitis on a specific date. It is not possible to determine if the infection was in one or more quarters. Also, when a cow has a subsequent mastitis event, there is no means of telling if the infection is in the same quarter (a repeat case) or in a different quarter (a new case).

In contrast, with quarter data, a mastitis case equates to an infection in a single quarter. Hence a cow can have up to four cases (one in each quarter) on a specific date. This also opens the opportunity to distinguish between quarters affected for the first time in a lactation (a **new** case) or a re-infection (a **repeat** case). It is these characteristics that can guide the veterinarian towards the underlying cause of the disease.

DEFINITIONS

When the quarter is recorded a greater range of parameters is available:

Cows affected: the number of cows in a cohort that have had one or more mastitis cases since calving. This can be calculated for different time periods after calving, e.g. % cows affected by 7, 30, 100, 150 days after calving

Mastitis case: a case is a single QUARTER affected once. A cow with mastitis in 2 quarters on the same date is 1 cow affected, 1 cow case and 2 (quarter) cases.

New cases are quarters entering the mastitis cycle for the first time in the cow's current lactation.

Repeat cases are subsequent cases in a quarter (in the same lactation).

Total quarter cases: The sum of new and repeat cases.

Recurrent quarters are quarters that have had one or more repeat cases.

INTERPRETATION

Cows affected: % **cows affected** (mastitis rate) provides the proportion of

cows in the cohort affected in one or more quarters.

Quarter cases per 100 cows indicates the mastitis incidence in the herd.

New infection rate (% new cases): A high new infection rate indicates a high proportion of mastitis-free quarters entering the mastitis cycle.

Repeat infection rate (% repeat cases): proportion of total cases that are repeat cases. This parameter is highly influenced by culling policy (how many repeats are allowed before the cow is culled?)

{HIGH level suggests - Either persistent recurrent infections with poor response to treatment and potential for CONTAGIOUS spread (e.g. *Staph aureus*/cow adapted *Strep uberis*), or (less commonly) a very high challenge of potentially short lived or easily cured infections (e.g. *E.coli*) where repeat cases are often fresh infections.

LOW level suggests - Short-lived/easily cured infections (e.g. *E.coli*) with a lower challenge but likely ENVIRONMENTAL source.}

Recurrence rate (% recurrent cases) is less influenced by culling policy and indicates the proportion of affected quarters requiring one or more repeat treatments. This approximates to the proportion of chronically infected quarters which is most often contagious mastitis (particularly *Staph. aureus* or *Strep. uberis*) but can, like repeat rate, also be influenced by a high infection pressure.

{HIGH % suggests - Persistent infections such as *Staph. aureus* or cow-adapted *Strep. uberis* with a potential for recurring cases and CONTAGIOUS spread.

LOW % suggests - Short lived infections such as *E.coli* with a likely ENVIRONMENTAL source.}

Average quarter cases affected cow: An estimation of the number and/or duration of existing infections within each affected cow. Can be greater than four.

Average Quarter cases/cow case: The average proportion of the cow's udder affected at once with clinical mastitis (maximum ratio is 4). Herds with high case to cow-case ratios combined with high percentage of herd affected are likely to be affected with environmental mastitis and usually suffer substantial losses. The ratio will vary at different stages of lactation. Often with *Staph. aureus* problem herds the ratio will be close to one.

Average quarters affected/cow: Out of a maximum of four this indicates the average number of quarters that have been affected per affected cow.

CONCLUSION

The mastitis parameters discussed are interrelated and need careful studying and analysis. They will vary with not only pathogen and pathogen behaviour but also across parity and stage of lactation. This example of computer analysis and reporting (InterHerd+) can augment experienced veterinary interpretation and pattern recognition of mastitis data. An approach of routine automated analysis also lends itself to the generation of benchmarking data.

BENCHMARKING AND SETTING INTERVENTION TARGETS FOR KEY SOMATIC CELL COUNT PARAMETERS

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Routine milk recording provides reliable measures of herd Somatic Cell Count (SCC) over time. Since 2005 Herd Companion has allowed veterinarians to benchmark herd level SCC against significant numbers of other herds recording with National Milk Records (NMR). Herd Companion uses past milk records to generate 12 month rolling averages that can be used to indicate trends in performance.

A study of 252 herds (representing all client herds from 10 veterinary practices and consultants located from Devon through to Cheshire) enabled the calculation of summary statistics for five important SCC parameters and relationships between them.

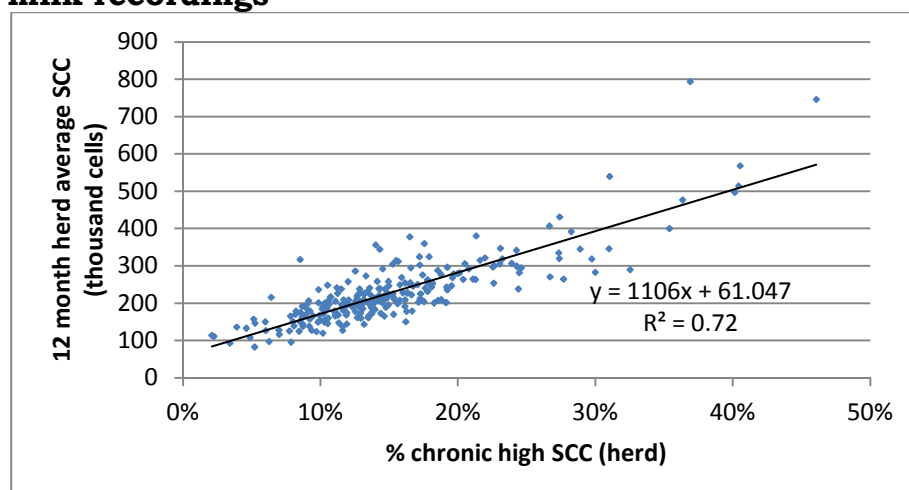
Table 1 Median and quartiles for five SCC key performance indicators for the 252 herds

Parameter (12 month rolling average)	Median	Lower quartile	Upper quartile
Herd SCC: Overall herd SCC ('000 cells/ml)	210	178	263
%High SCC: % milk recordings with SCC >200,000 cells/ml	25%	21%	30%
%SCC>500: % milk recordings with SCC >500,000 cells/ml	9.6%	7.6%	12.2%
%Chronic SCC: % milk recordings >200,000 cells/ml at consecutive recordings	14%	11%	18%
%1stHigh SCC: % lactations starting with a SCC >200,000 cells/ml at the first milk recording in the new lactation	20%	16%	27%

Examining how the Herd SCC correlates with other SCC parameters may provide additional indicators of high Herd SCC and also may shed light on where remedial action could be targeted.

The overall herd SCC was plotted against the other four parameters to establish the level of correlation. As an example, Figure 1 shows the strong correlation between the herd SCC and the % of milk samples that are of the “chronic” Herd Companion category (from cows with high SCC at consecutive milk recordings).

Figure 1. Overall Herd SCC against the percentage of chronic high SCC milk recordings



This allows the allocation of “Low”, “Medium” and “High” bands for the % chronic cows in a herd with regard to the risk that overall Herd SCC is over 200,000. Thus Table 2 shows that a herd where less than 13% of milk records are chronic highs has under 25% risk of having a herd SCC over 200,000 cells. In contrast, a herd with over 14% of milk from chronic cows has over 90% risk of a herd SCC over 200,000 cells.

Table 2. Correlation between the % of chronic high SCC milk records and overall Herd SCC

% milk records Chronic (%Chronic SCC)	Herds %		% herds with overall Herd SCC >200,000 cells (Herd SCC)	
	Count	Percentage	Risk	Risk Band
<13%	103	(41%)	24%	Low risk
13% to <14%	24	(10%)	54%	Medium risk
>=14%	125	(50%)	92%	High risk

Risk bands for all the SCC parameters, derived in a similar fashion, are summarised in Table 3.

Table 3. Correlation between four SCC key performance indicators and the overall Herd SCC

Parameter (12 month rolling average)	R ²	Low risk	High risk
%High SCC: % milk recordings with SCC >200,000 cells/ml	0.78	<25%	>=26%
%SCC>500: % milk recordings with SCC >500,000 cells/ml	0.90	<8%	>=9%
%Chronic SCC: % milk recordings >200,000 cells/ml at consecutive recordings	0.72	<13%	>=14%
%1stHigh SCC: % lactations starting with a SCC >200,000 cells/ml at the first milk recording in the new lactation	0.61	<18%	>=22%

HERD SIZES IN THE UK HAVE INCREASED ARE PARLOUR SIZES KEEPING UP?

Andrew Biggs

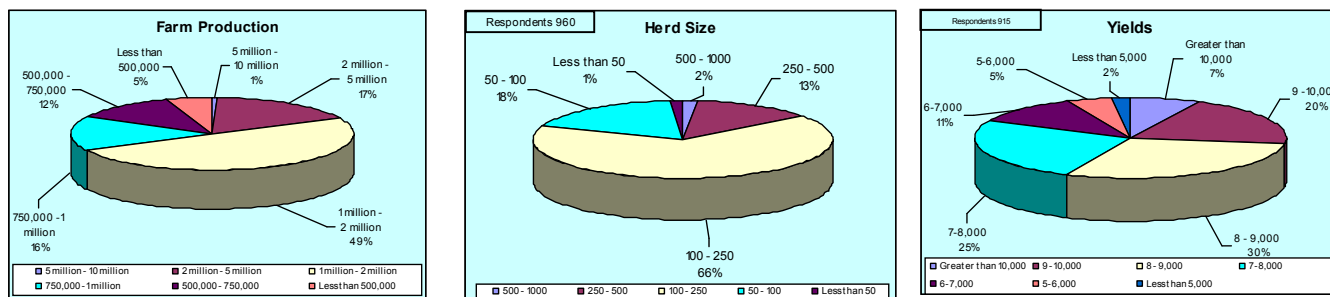
The Vale Veterinary Centre, The Laurels, Station Road, Tiverton, Devon, EX16 4LF; email: ValeLab@btinternet.com

The National Mastitis Survey undertaken by Intervet Schering-Plough Animal Health in 2009 and 2010 has yielded some very useful data about the UK dairy industry.

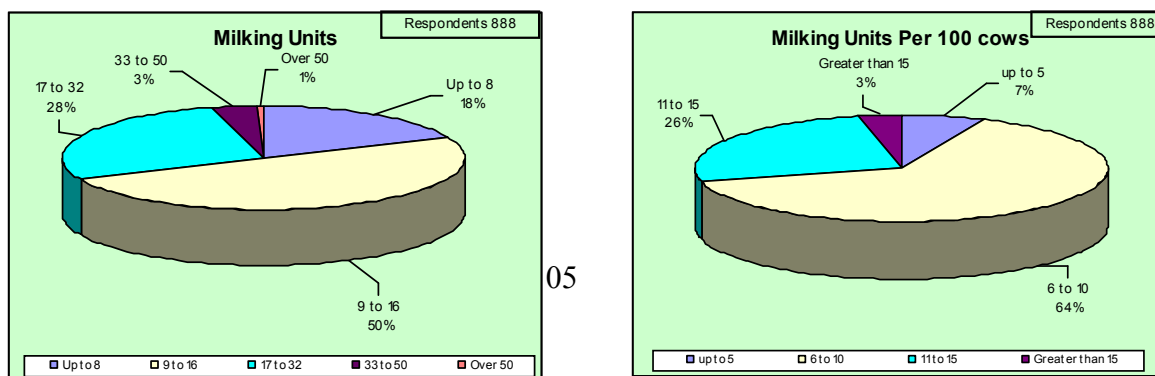
This poster explores the relationship between farm production, yields, herd size and number of milking units or standings in the parlour. It is postulated that the volume of milk harvested per milking unit per annum may be a useful discussion point for both cow and milking personnel health.

Apart from in herds that group cows the milking time (time to milk the herd) and thus standing time (the time cows spend standing before they are milked) will be related to the litres of milk produced per annum and the number of milking units or standings in the parlour. Excessive standing times can influence the health and productivity of cattle in a number of potential ways by for example limiting dry matter intake or increasing lameness. Prolonged milking times can also influence the ability of milking operatives to maintain a high level of attention to detail throughout milking and therefore may be related to intramammary infections. Prolonged milking times could also influence wellbeing of milking operatives as they will spend excessive times in the parlour.

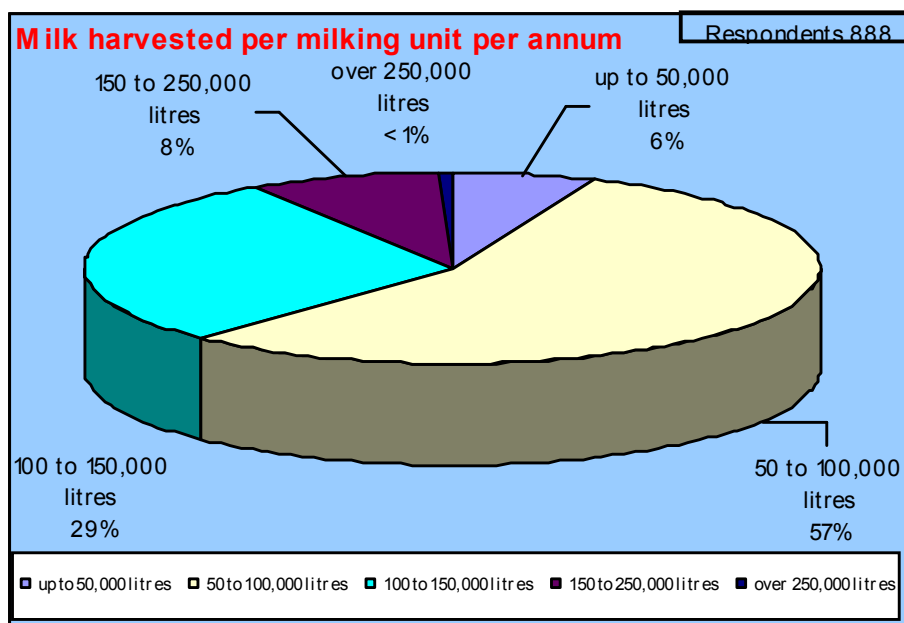
The following graphs indicate the production, herd size and yields of nearly 1,000 respondents in the National Mastitis survey



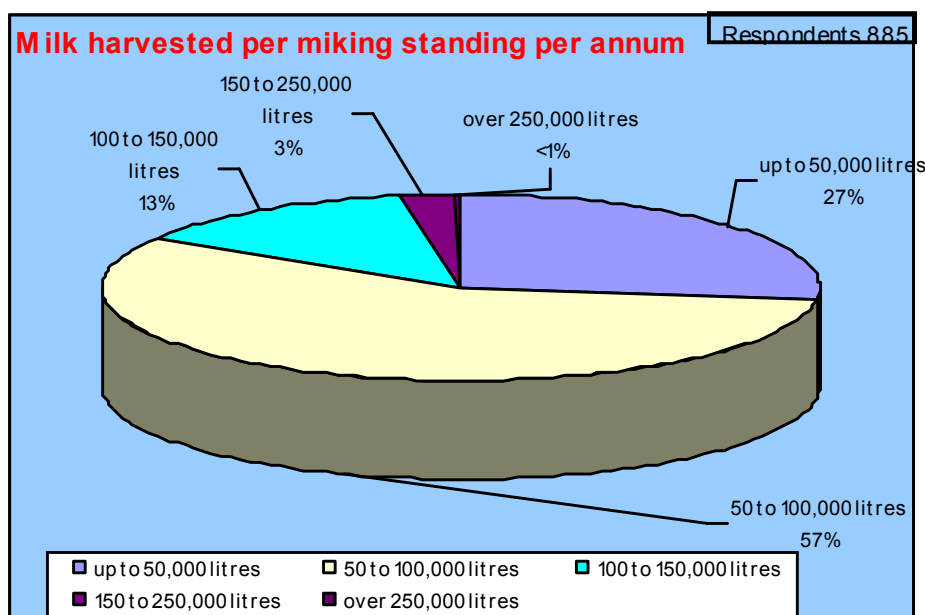
The following graphs indicate the range of the average number of milking units per parlour and the milking units per 100 cows (54% of parlours were swing over parlours)



The figures for milk harvested per milking unit per annum will be influenced by herd size and average cow yield but should still give some indication of likely standing times in herds where the cows are not milked in separate groups. This figure will also be influenced by whether the parlour is doubled up (one milking unit per standing) or a swing over parlour.



The data has been recalculated for milk harvested per standing and unsurprisingly this increases the proportion of herds in the lowest category of up to 50,000 litres per standing compared to the same category by milking unit although the largest group remains the 50 to 100,00 litres per standing or milking unit.



OBSERVATIONS OF DRY PERIOD CURE RATES USING ROUTINE MONTHLY SCC DATA FROM COWS THAT HAVE FAILED TO CURE IN THEIR PREVIOUS DRY PERIOD

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Many cows in the UK are treated with parental antibiotic therapy at drying off in an attempt to improve dry period cure rates. This preliminary analysis of cure rates in the dry period subsequent to a previous dry period failure to cure in a substantial number of cows is intended to be used as a benchmark template in an intervention study where adjunct parental antibiotic therapy is used at drying off to improve cure rates in known infected cows at drying off.

Routine individual cow monthly SCC records from approximately 400 cows with dry periods between 2005 to 2010 were used to identify cows in a variety of SCC patterns indicating a failure to cure an infection in that dry period. The dry period cure rates in the subsequent dry period were then assessed to see if the SCC pattern in the previous two lactations can predict cure rates

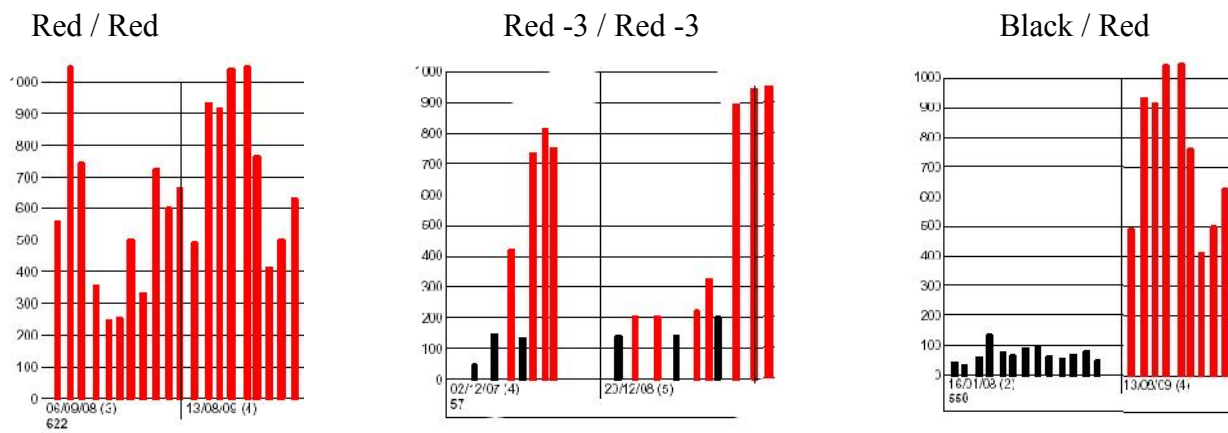
All cows were selected by having a failed dry period cure and had a minimum of three successive SCCs >200 immediately before and after the failed dry period AND three successive SCCs >200 at the end of their subsequent (2nd) lactation. The subsequent dry period performance after the 2nd lactation was then assessed using the first 2 SCCs of the lactation following that second dry period. A dry period cure required both of the first 2 SCCs of the new (3rd) lactation to be < 200.

The cows were also grouped by the number of SCCs >200 and <200 in the two completed lactations either side of the initial failed dry period. The dry period performance of the subsequent dry period of these groups was then analysed.

The dairy cow management program Interherd was used to select the cows for a failed dry period cure and allocate them into various SCC pattern groups. Interherd was used to display the routine monthly cow SCCs for 2 successive lactations in graphical format with SCCs >200 as red and SCCs <200 as black. All cows selected had to have a minimum requirement of at least the three successive “red” SCCs before and after a dry period to be selected as a dry period “fail to cure”. The cows were then allocated to groups as described in the tables below.

The following three graphical displays give examples of how the SCC data was used to select the cows for a failure of a dry period cure and allocate cows to their relevant groups. Lactations described as “red” have all SCCs in

that lactation >200 whilst lactations described as “black” have all SCCs <200. The middle group shows lactations described as “Red minus 3” indicating that despite having the last three successive SCCs >200 (a minimum requirement for selection into the dataset) it was characterised by having three of any of the other SCCs in that lactation <200.



	Red/Red	Red-3/Red	Red/Red-3	Red-3/Red-3	Black/Red	Black/Red-3
	All recordings over 200 both lactations	1st lactation 3 recordings below 200 with the remainder over 200 2nd lactation all recordings over 200	1st lactation all recordings over 200 2nd lactation 3 recordings below 200 the remainder over 200	Both lactations 3 recordings below 200 the remainder over 200	1st lactation all recordings below 200 2nd lactation all recordings over 200	1st lactation all recordings below 200 2nd lactation 3 recordings below 200 the remainder over 200
TOTALS	51	44	6	47	56	27
FAIL	43	35	2	32	32	10
CURE	8	9	4	15	24	17
% FAIL	84%	80%	33%	68%	57%	37%
% CURE	16%	20%	67%	32%	43%	64%

	2/2 lact 2	3/3 lact 3	Heifers -1 to 3
	1st lactation last 2 recordings pre dry over 200 2 nd lactation first 2 recordings over 200 3 rd lactation last 2 recordings pre dry off over 200	1st lactation last 3 recordings pre dry over 200 2 nd lactation first 3 recordings over 200 3 rd lactation last 3 recordings pre dry off over 200	Heifers with up to 3 recordings below 200 the rest over 200
TOTALS	55	56	50
FAIL	28	41	20
CURE	27	15	30
% FAIL	51%	73%	40%
% CURE	49%	27%	60%

Totals		
All data including 2/2 lact 2 & heifers	Data without 2/2 lact 2 cows	Data without 2/2 lact 2 cows and heifers
392	337	287
243	215	195
149	122	92
62%	64%	68%
38%	36%	32%

