

# 2009

# BRITISH MASTITIS CONFERENCE

Organised by

*The Dairy Group*

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Topics are:

- Controlling Somatic Cell Counts
- Research updates
- Milking management

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**Wednesday 14<sup>th</sup> October 2009**

**Warwick Complex, Wolfson Theatre  
Stoneleigh Park Exhibition and Conference Centre  
Near Coventry, Warwickshire, CV8 2LZ**

# BMC 2009

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# GENERAL INFORMATION

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## TIMETABLE of EVENTS

09:00	<b>ARRIVE / REGISTRATION / COFFEE and POSTER DISPLAY</b>	
09:45	<b>CHAIRMAN'S INTRODUCTION</b>	<b>Ian Ohnstad,</b> <i>The Dairy Group, UK</i>
	<b>Controlling Somatic Cell Counts</b>	
09:55	Why do we want to control SCC? A purchaser's viewpoint	<b>Jim Begg,</b> Dairy UK
10:15	Nutritional control of SCC	<b>Richard Vecqueray ,</b> EBVC, UK
10:30	Optimising the use of individual SCC information	<b>Andy Biggs,</b> The Vale Veterinary Group, UK
10:45	Questions and Discussion	
11:00	<b>COFFEE and POSTERS</b>	
	<b>Research updates</b>	<b>Brian Pocknee,</b> DHC, UK
11:30	The cow's response to pathogens	<b>Tracey Coffey,</b> IAH, UK
11:45	Recent advances in mastitis detection with AMS	<b>John Baines,</b> Fullwood Ltd, UK
12:00	Is there a role for backflush systems?	<b>Richard Olde Riekerink</b>
12:15	Questions and Discussion	
12:30	<b>LUNCH and POSTERS</b>	
14:00	<b>CHAIRMAN'S INTRODUCTION and VOTING ON POSTERS</b>	<b>Elizabeth Berry</b> DairyCo, UK
	<b>Milking management</b>	
14:05	Teat disinfection – what's new?	<b>Alison Cox,</b> JohnsonDiversey Ltd, UK
14:35	The true costs of mastitis	<b>Martin Green,</b> University of Nottingham, UK
15:05	Environmental challenge in a pasture system	<b>Katrina Roberts,</b> Animal Health Centre, NZ
15:45	Questions and Discussion	
16:00	<b>POSTER AWARD and CLOSE</b>	
16:10	<b>TEA and DEPART</b>	

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J.D.Hanks<sup>1</sup> and A.M.Biggs<sup>2</sup>, <sup>1</sup>Veterinary Epidemiology & Economics Research Unit (VEERU), University of Reading, P.O.Box 237, Reading, UK; <sup>2</sup>Vale Veterinary Centre, The Laurels, Station Road, Tiverton, Devon, UK
- Use of novel teat dip cup** 87-88  
E Berry<sup>1</sup>, C Kingston<sup>2</sup>, J Clarke<sup>1</sup>, R Hiley<sup>2</sup> and R May<sup>2</sup>  
<sup>1</sup>Institute for Animal Health, Compton, Newbury, Berkshire, UK; <sup>2</sup>Ambic, Witney, Oxfordshire, UK
- Suitability of data on clinical mastitis from UK dairy herds for use in genetic evaluations** 89-90  
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DairyCo, Stoneleigh Park, Kenilworth, Warwickshire, UK
- Understanding Mastitis Epidemiology on Individual Dairy Units** 93-94  
A J Bradley<sup>1, 2</sup>, J E Breen<sup>1, 2</sup> and M J Green<sup>2</sup>,  
<sup>1</sup>Quality Milk Management Services Ltd., Unit 1, Lodge Hill Industrial Park, Station Road, Westbury-sub-Mendip, Wells, Somerset, UK; <sup>2</sup>School of Veterinary Medicine and Science, The University of Nottingham, Sutton Bonington Campus, Sutton Bonington, Leicestershire, UK
- Role of Biotin Carboxyl Carrier Protein (BCCP) in the interaction of *Streptococcus uberis* with Bovine Plasminogen** 95-96  
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- Bactoproof: A revolutionary new testing method for mastitis** 105-106  
P D Burr and S Duthie, Biobest Laboratories Ltd, 6 Charles Darwin House, The Edinburgh Technopole, Penicuik, Midlothian, Scotland
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I Janik<sup>1</sup>, H Martin<sup>1</sup>, S Chadd<sup>1</sup> and B Hill<sup>2</sup>,  
<sup>1</sup>Royal Agricultural College, Cirencester Gloucestershire, UK; <sup>2</sup>Micro Research Limited, Ledbury, Herefordshire, UK
- Staphylococcus aureus* isolates from a dairy farm in Beijing: Molecular typing, antibiotic resistance and toxicity test** 109-110  
J Gao, M Ferreri, L B Chen, X Q Liu, J L Su and B Han, China Agricultural University, College of Veterinary Medicine, 100193 Beijing, P.R.China
- National Mastitis Survey** 111-112  
A M Biggs and R Ankcorn, Intervet Schering-Plough Animal Health, Walton Manor, Walton, Milton Keynes, UK

## CHAIRMAN'S INTRODUCTION

Welcome to the 21st British Mastitis Conference. This conference heralds a new phase for BMC as we welcome on to the organising committee DairyCo and the University of Nottingham. We are delighted that both organisations have agreed to become part of BMC and we are confident that this partnership will ensure we continue to provide a first class conference for many years to come.

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.

The first session examines the complex and challenging question of cell count control, focusing on the market demands for high quality milk, the nutritional control of SCC as well as optimising the use of individual cow SCC data.

The second session provides an excellent opportunity to be updated on the latest research being undertaken in the field of mastitis and SCC control. A series of three short summary papers will consider how a cow responds to a mastitis challenge, technical developments in mastitis detection with automatic milking systems and the role of backflush systems on a modern dairy farm.

After lunch, we will turn our attention to milking management, with papers updating our thoughts on the role of teat disinfection, environmental challenges in a pastoral system and the true cost of mastitis.

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. They are: DairyCo, Pfizer, DeLaval, Intervet/Schering Plough, BouMatic, Kilco, Norbrook Laboratories, Boehringer-Ingelheim, Marks and Spencer, Fullwood, Ambic and NMR. As usual the event could not happen without able administration, now provided by Barbara Hepworth at Nottingham University.

Finally, as always, thank you for attending and supporting the conference. I hope you 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**



### Organising Committee

Chairman: Ian Ohnstad  
Conference Secretariat: Barbara Hepworth  
Editor: Brian Pocknee

### Scientific Committee

Ian Ohnstad, *The Dairy Group*  
Elizabeth Berry, DairyCo  
Brian Pocknee, *The Dairy Group*  
Martin Green, University of Nottingham

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A global organization for mastitis control and milk quality

The NMC is a professional organization that promotes research and provides information to the dairy industry to help reduce mastitis & enhance milk quality. For nearly 50 years, the NMC has distinguished itself internationally as a leader in meeting those objectives.

#### What does NMC do?

- Provides a forum for the global exchange of information on mastitis and milk quality
- Publishes educational materials including books, brochures and CDs
- Establishes guidelines for mastitis control and milking management practices
- Monitors technological and regulatory developments relating to udder health, milk quality and milk safety
- Conducts meetings & workshops, providing educational opportunities for all segments of the dairy industry
- Helps fund the National Mastitis Research Foundation

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*A commitment to  
reducing mastitis and  
enhancing milk quality*

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#### Who are the members of NMC?

NMC membership is comprised of people from more than 40 countries, representing a wide range of dairy professionals who share an interest in milk quality and mastitis control. These people include dairy producers, veterinarians, university researchers and extension specialists, milk procurement field staff, equipment and supply representatives, government officials, and students.

#### What can NMC do for you?

The continued pressure to ensure milk safety and improve milk quality, as well as the need to increase production efficiency, requires greater team effort between producers, veterinarians and other dairy professionals. Each team member plays a key role in developing successful mastitis control programs. NMC can serve as your resource for information related to udder health, milking management, milk quality, and milk safety.

#### Why join NMC?

- To receive the latest technical and applied information on udder health, milking management, and milk quality
- To provide leadership on milk quality issues within the industry
- To participate and learn about mastitis and milk quality developments at NMC meetings
- To establish valuable industry contacts
- To support education and research efforts that help raise awareness and understanding of milk quality issues

#### NMC membership benefits

- NMC annual meeting and regional meeting proceedings, containing all of the papers and posters presented at the meetings
- The NMC printed and electronic newsletters, addressing the latest information on udder health, milking management and milk quality
- Access to the "members-only" section of the NMC website, which includes the NMC Proceedings Library, NMC newsletter archives, and NMC membership directory
- Opportunities to network with other dairy professionals concerned with milk quality

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*No other professional dairy  
organization enjoys the wide  
range of expertise found within  
the NMC membership.*

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#### Working together

Since 1961, NMC has coordinated research and education efforts to help control the losses associated with mastitis. By bringing together all segments of the industry, a strong and successful organization has been created to enhance the quality of milk and dairy products. NMC welcomes your active participation and support. Please visit the NMC website for additional information and resources.

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# PAPERS

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## **WHY DO WE WANT TO CONTROL SCC? A PURCHASER'S VIEWPOINT**

**Jim Begg**

DairyUK, 91-93 Baker Street, London W1U 6QQ. e-mail: [jbegg@dairyUK.org](mailto:jbegg@dairyUK.org)

No written paper has been submitted.



## **NUTRITIONAL CONTROL OF SCC**

**Richard J. Vecqueray<sup>1</sup>, Richard Cooper<sup>1</sup>, Alastair J. Hayton<sup>2</sup> and James Husband<sup>1</sup>**

<sup>1</sup>The Evidence Based Veterinary Consultancy, EBVC Ltd. 68 Arthur Street, Penrith, Cumbria CA11 7TX. Email: [richard@ebvc.eu](mailto:richard@ebvc.eu). <sup>2</sup>Kingfisher Veterinary Group, Crewkerne, Somerset, UK

### **SUMMARY**

The impact of nutrition on mastitis and somatic cell counts has been recently extensively reviewed (1, 2). This paper concentrates on how some of these principles may be practically applied in typical UK feeding and production situations to control Somatic Cell Counts (SCC).

### **INTRODUCTION**

Our sphere of practical intervention is fourfold since nutrition may affect SCC by either:

1. Increasing the challenge from the environment due to the production of loose faeces.
2. Inducing Milk Fever that makes a cow more likely to suffer clinical and subclinical intramammary infections.
3. Influencing the volume of milk in the udder prior to drying off.
4. Reducing the cow's ability to respond to a challenge by inhibiting her immune capacity.

Each of these interventions will be reviewed in turn.

It should be noted that whilst associations have been found with ketosis and SCC for example, there is little or no evidence that the ketosis prevention strategies suggested below, such as minimizing social group changes or using choline, influence SCC. Indeed the degree of influence of nutrition on SCC and mastitis although well known is extremely difficult to quantify in significance.

### **CONTROLLING FAECAL CONSISTENCY**

Associations have been made between udder hygiene score, leg hygiene score and the incidence of intramammary infection with environmental pathogens (3). Cows with dirty udders were 1.5 times more likely to have major pathogens isolated from milk samples compared with cleaner cows (4). An observational

study found that groups of cleaner cows had firmer faeces (5). Higher producing cows excrete more liquid faeces (6).

Given the weight given to faecal consistency by modern dairy nutritionists there is a dearth of science on the subject.

Consistency is hard to predict accurately. It is affected by dietary acid detergent fibre (ADF) content which is a marker for structural fibre. The more ADF fed, the stiffer the faeces. Increasing dietary starch intake produces more liquid faeces but not necessarily faeces of lower dry matter content since fibre in faeces retains water. As dietary starch increases, faecal starch increases and pH of the faeces falls. Higher crude protein diets tend to produce more liquid faeces but protein source has an influence (6).

### **Nutritional strategies to create firmer faeces**

1. Increase dietary ADF
  - Feed more chopped straw, hay or lucerne
2. Increase physically effective neutral detergent fibre (peNDF) sources
  - Feed a coarse blend rather than a compound nut or dye ground meal. (peNDF defined by proportion of dry matter retained on a 1.18 mm sieve.)
3. Increase forage NDF (also increases peNDF). UK dietary forage NDFs typically vary from 18% - 26% NDF. The absolute requirement to prevent sub acute rumenal acidosis (SARA) depends on the digestibility of the NDF, the fermentability of the Non-Fibrous Carbohydrate portion of the diet and the diets presentation and processing.
  - Keep forage:conc ratio the same but alter forage type to a higher NDF.
  - Feed less concentrate, but a higher quality concentrate to maintain energy density.
4. Increase the Rumen Undegraded Protein (RUP) % of the overall Crude Protein. Crude protein % remaining constant or reduce overall crude protein %.
5. Improve cow comfort, improving lying times, cudding times and therefore improving salivary buffering of the rumen

### **CONTROLLING MILK YIELD PRIOR TO DRY OFF**

Quarters that form keratin plugs early in the dry period have lower odds of developing dry period intra-mammary infections than those that do not close. The hazard of quarters closing if milk production on the day prior to drying-off was greater than 21 kg was 1.8-times less (7).

### **Nutritional strategies to reduce milk yield prior to drying off**

1. Undertake social group changes.
2. Isolate cows socially.
3. Decreasing dietary energy density. It is important to maintain dietary macromineral (e.g. magnesium), micronutrient supply and sufficient protein to maintain rumen function if utilizing this strategy. It typically involves penning cows and feeding a higher proportion of straw in the diet.

### **CONTROLLING MILK FEVER**

Cows with clinical hypocalcaemia (milk fever) are 8.1 times more likely to suffer from mastitis and 9 times more likely to be affected by E. coli mastitis (8). This may be due to a direct effect on the immune response or due to recumbent cows having a higher bacterial challenge at the teat end and reduced teat sphincter tone.

#### **Milk Fever Prevention Strategies**

Milk fever prevention is extensively studied and relies on supplementing magnesium and reducing the supply of dietary potassium. (9)

The following dietary macromineral concentrations can be targeted:

Magnesium	0.4%
Sodium	0.18%
Potassium	<1.5%
Calcium	0.4-1% requirement depends upon final metabolic acidification, the more acid, the more calcium is required
Phosphorus	0.35%
Sulphur	0.35-0.4%
Chlorine	Level depends on cation loading (Potassium and Sodium). Supplementation requires caution since many sources are unpalatable

### **CONTROL OF COW IMMUNE STATUS**

We have nutritional influence over a dairy cow's immunity through our potential to reduce the severity of negative energy balance and fatty liver; supplement micronutrients and alter dietary fatty acid type.

## **Negative energy balance (NEB) & fatty liver syndrome**

The development of clinical and subclinical ketosis is known to have an adverse effect on immune function, and effects both the humoral and cellular arms of the immune response. In a review paper by Suriyasathaporn *et al.* (10), the effects of hyperketonaemia on the specific immune responses to intramammary infection were that:

- Leukocytes show a lower capacity of the phagocytic defence mechanism;
- Neutrophil phagocytic and bactericidal capacities are reduced;
- Lower amounts of cytokine production after bacterial infection observed;
- The chemotactic capacity of blood leukocytes is impaired.

Fatty liver occurs when blood NEFA levels rise and exceed the liver's capacity to completely oxidize them to CO<sub>2</sub>. In this situation ketones are produced and also triglyceride. Triglyceride is exported as very low density lipoproteins (VLDLs). The export of VLDLs is slow and its capacity is easily exceeded leading to the accumulation of fat in the liver.

Therefore prevention of fatty liver relies on either preventing excessive lipolysis; improving the liver's capacity to oxidize NEFA or its capacity to export VLDLs more rapidly.

Concentrations of blood NEFA increased after calving, as is generally reported (11). Bobe *et al.* (12) stated that Plasma NEFA concentration is a reliable index of the magnitude of adipose fat mobilization and therefore provides an easy and convenient objective measure of disease risk.

Body weight loss in early lactation, the end result of negative energy balance and potential fatty liver disease, is associated with elevated somatic cell count events (13).

## **Nutritional strategies to prevent fatty liver and NEB**

### *Controlling body condition score*

To be able to control body condition is the nirvana of dairy cow nutrition.

Obesity is a significant risk factor for fatty liver development (12) additionally overfeeding in late lactation or in the early dry period may lead to the development of insulin resistance (14).

- Group and feed later lactation cows by body condition rather than production;
- If a cow is fattening dry her off if using a low energy density dry cow ration;

- Feed a low energy density early period dry cow ration;
- Maintain a low herd calving interval;
- Calve Holstein heifers at an average of two years old after rapid average daily growth rates.

#### *Eliminating feed restriction*

A *stable* dry matter intake pre-calving is the prerequisite of good liver health. Precipitous drops in dry matter intake whatever the original level are dangerous. It has been postulated that a high original level leaves the cow more at risk of a large fall than if the original level was low or restricted as the cow approaches calving (15).

Therefore, it maybe *how* cows are managed before calving is more significant to NEB than *what* the cows are fed in terms of formulation (16). With this in mind:

- Provide adequate trough space;
- Limit stocking density in pre and post calving yards;
- Avoid social group changes within one week of calving.

#### *Shortening the dry period*

Whilst the data on shortening the dry period from 60 days remains equivocal in terms of the ultimate consequences for udder health (17, 18 and 19), cows with shortened periods have less body fat mobilisation and decreased liver triglycerides post calving (20). Furthermore they will have a lower yield at drying off which has been shown to be a risk factor for the acquisition of new IMI during the dry period, see below (7).

- A suggestion in the context of controlling SCC would therefore be to adopt a cow specific approach to shortening the dry period:
  - Not 1<sup>st</sup> lactation animals;
  - Not animals with a high cell count;
  - Yes to animals yielding over 21 litres at 60 days prior to calving.

#### *Manipulating dietary oil type*

It is well known that fat, specifically fatty acids, are more than an energy source and are potent modifiers of metabolism (15).

Various studies (21, 22 and 23) have found that challenging cows with saturated fat pre-calving raises blood NEFA concentrations and increases liver triglyceride concentrations post-calving.

In contrast feeding or infusing unsaturated oil as linolenic reduced liver TG and increased liver glycogen whereas longer chain omega 3 fish oils did not. More study is needed in this area however since not all studies have shown consistent results (24).

Dietary PUFA are well known to reduce lipid accumulation in the liver, up-regulating FA oxidation in liver to carbon dioxide and increasing total body glycogen storage (25).

This is an evolving field of research in dairy cow nutrition. On current published evidence a recommendation would be:

- Don't feed saturated oil sources to dry cows;
- Don't feed fish oil to dry cows;
- Consider using a source of linolenic acid.

### *Choline*

Choline has been shown in experimental models involving feed restriction to reduce liver triglyceride by the probable mechanism of a direct effect on the liver (26). This effect has also been found when it has been fed in a protected form before and after calving (27). It is likely to be an effective tool to prevent fatty liver (15).

- Feed Protected Choline before and after calving

### *Propylene Glycol*

Administering propylene glycol has the potential to cause an insulin response and reduce fatty acid mobilization from adipose tissue (28).

It does this consistently in experiments but only when 'pulse fed' as a drench or in concentrate feed (29, 30 and 31)

- Drench or feed in concentrate (parlour or top dressed, not TMR) 300 – 400 mls of propylene glycol daily in the week pre and post calving

### **Nutritional strategies to prevent micronutrient deficiency**

The pathophysiology of micronutrient deficiency has been well covered in recent reviews (1).

### *Vitamin A (retinol) & Beta-carotene*

Vitamin A's role is not fully elucidated as an anti-oxidant but is involved in resistance to infection, particularly mastitis (32). Beta-carotene is also thought to have a role as an antioxidant separate to its role as the precursor to Vitamin A (33).

The NRC recommendation for supplementation of vitamin A (33) for both dry and lactating cows is 110iu/kg bodyweight. For a 700kg cow this is equivalent to 77000IU/day. No requirement for beta-carotene has been set and this is a parameter not normally measured in the field. We have recently done some field testing and found deficient situations where cows have been fed solely conserved forage for some time.

Supplementation of vitamin A should be specifically considered in diets containing high levels of concentrates, low levels of green forages, high levels of poor quality forages and during the periparturient period and periods where the immune system is under "stress" due to reduced competency or of high levels of exposure to infectious pathogens.

### *Vitamin E (alpha-tocopherol)*

This is a fat soluble membrane antioxidant that enhances the functional efficiency of neutrophils by protecting them from oxidative damage following intracellular killing of ingested bacteria and also has a role in maintaining cellular membrane fluidity (34).

Variation in the results from the numerous trials looking at Vitamin E and mastitis are fascinating and the conclusion maybe drawn that there is a good response to supplementation in deficient situations and where there is adequate selenium supplied (35, 36, 37, 38 and 39)

As the basal diet will be highly variable in the level of vitamin E it contains, recommendations for vitamin E are based on the requirements for supplementation. In non-grazed cattle the current NRC recommendations for Holstein cattle are 1000IU/day for dry cows and 500IU/day for lactating cows. In grazed cattle these requirements are reduced to 330IU/day for dry and 160IU/day for lactating cows. (33)

### *Selenium*

This mineral is required for the activity of the anti-oxidant enzyme glutathione peroxidase (34).

Trials have demonstrated a reduction in somatic cell count when they supplemented selenium (40 and 41).

With reference to supplementation level Weiss (42) concluded that there was no clinical evidence for an improvement in udder health beyond a supplemental level of 0.3ppm selenium.

### *Zinc*

This mineral, like copper is essential for the activity of the superoxide dismutase (34). As with copper there is a paucity of evidence to suggest an effect of supplementing zinc on udder health and the author is unaware of any peer reviewed work other than Whittaker *et al.* (43) on the effect of zinc supplementation on udder health, There are a number of non-peer reviewed articles suggesting an effect on a reduction in somatic cell count when zinc was supplemented in the region of 360mg/day, however due to trial design this cannot be definitively attributed to the effect of supplementation (42).

NRC recommendations for requirements for zinc are 300mg/day for dry cows to 1400mg/day for a cow producing 45 litres. Zinc can interfere with copper uptake and it is recommended that the dietary zinc intake should not exceed the dietary copper intake by more than fivefold.

### *Fatty Acid Profile*

Dietary fatty acids can influence immunity beyond any effect on liver fatty acid oxidation through the production of cytokines and molecules involved in the regulation of immune responses. Omega-3 and omega-6 polyunsaturated FA are important immunomodulators of immune reactions (44).

One possible explanation of the mechanism is related to the synthesis of eicosanoids such as prostaglandins and leukotrienes.

Omega-6 FA such as linoleic acid (C18:2n6) and omega-3 FA such as  $\alpha$ -linolenic acid (C18:3n3) lead to the formation of arachidonic acid and eicosapentaenoic acid, respectively. Both arachidonic acid and eicosapentaenoic acid are precursors of eicosanoids, but those that are synthesized from eicosapentaenoic acid do not have as strong biological activity as do those produced from arachidonic acid (45). As a result, feeding plant or fish oil rich in omega-3 PUFA generally reduces inflammatory reactions and production of interleukin-1, interleukin-6, and tumour necrosis factor- $\alpha$  in different animal species, including human. However, many contradictory observations have been reported (46).

In dairy cattle, linseed a good source of omega-3, has modified prostaglandin secretion and reproduction in dairy cows (47). Little is known so far on the effects of altering fatty acid composition of the diet of dairy cows on cellular and humoral responses which are obviously very important in SCC control.

However, it has been shown that lymphocytic proliferative responses to mitogenic challenge are influenced by dietary manipulation of these n-3 to n-6 ratios in cows (48) and severity and duration of the suppression of the proliferative response has been linked to the incidence of mastitis (49 and 50)

It is too early to give specific recommendations as to how these fatty acids may be manipulated to help dairy cows but it is important to recognise their influence when looking at problems involving immunity or planning diets.

## CONCLUSIONS

The associations between SCC and nutrition are weak compared with those associated with hygiene and environmental challenge.

With this consideration in mind however, there are many established relationships between nutrition and SCC. Therefore we should make every effort to integrate as much of what is known into our daily rationing and advice on *how* cows are managed and fed. We should also encourage research in the areas such as fresh cow nutrition and dietary fatty acid manipulation that remain lacking.

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## **OPTIMISING THE USE OF SCC INFORMATION**

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### **SUMMARY**

The use of Somatic Cell Count (SCC) as a proxy measure of udder health has evolved from its early use at a herd level (Bulk Milk SCC - BMSCC) where the sensitivity, of particularly changes in infection status within a herd, is poor. The adoption of regular individual cow monthly SCC recording by a significant proportion of dairy producers and the advent of interpretive software to analyse the dynamics of individual cow SCC using threshold values to indicate the likelihood of the presence of an intramammary infection, has allowed the performance of cows and herds during both lactation and the dry period to be monitored and benchmarked.

### **INTRODUCTION**

#### **What do SCCs mean?**

The Somatic Cell Count (SCC) measure, expressed as thousands of cells per millilitre of milk, has been used worldwide for several decades as a proxy indicator of udder health. The word Somatic is derived from the Greek word somatikos and means “of the body”. Although a proportion of the somatic cells are mammary epithelial cells shed into the milk, generally in excess of 95% are leucocytes consisting of variable proportions of lymphocytes, neutrophils and macrophages which together function in the generation of an effective immune response. Both the overall absolute number and proportion of the various types of leukocytes in milk will vary with a number of factors including infection status and mastitis pathogen, parity, stage of lactation, seasonal and diurnal variation, milking frequency and interval, milking routine such as post milking teat dipping, day to day variation seen in any biological system and stress. However the most significant changes are seen in response to infection.

Thus SCC is used as a proxy measure for udder health as an increased number of leucocyte in milk generally indicates the presence of an immune response most often as a result of an intramammary infection. In the healthy udder macrophages predominate and act as sentinels for infection which once recognised release chemoattractants resulting in the recruitment and migration of large numbers of neutrophils and lymphocytes to the site of infection. Lymphocytes tend to be involved in “immune memory” whilst neutrophils, which are phagocytosing and destroying the invading pathogens, release

chemicals which induce what can be viewed as paradoxical detrimental effects such as swelling of secretory epithelium cytoplasm, sloughing of secretory cells, and decreased secretory activity (milk production). To mitigate this, resident and newly migrated macrophages help reduce the damage to the epithelium by phagocytosing neutrophils that undergo programmed cell death through apoptosis.

Despite the large numbers of immune cells present in milk they are often considered to have compromised activity when compared to the rest of the body which in the case of neutrophils may in part be due to the ingestion of fat globules thereby reducing their ability to engulf bacteria.

### **What can be measured?**

The use of SCC to determine udder health is most commonly at a cow level and is measured using a commingled composite sample of milk from all four quarters. However the measurement of SCC at an individual quarter level is more appropriate for identifying infected quarter(s) with a view to action such as the treatment of sub-clinical infection where appropriate or for research into the effects of infection on milk SCC and determining a threshold level above which infection is likely. It is widely accepted that milk from an uninfected gland will almost always have a SCC <100,000 cells/ml whereas milk with a SCC >200,000 cells/ml may well indicate the likely presence of an intramammary infection. In addition there is some evidence that a very low SCC may increase the risk of intramammary infection and so somatic cells may offer some protection to intramammary infection as well as being an indicator that it is present.

### ***Herd or Bulk Milk Somatic Cell Counts (BMSCC)***

A herd or bulk milk tank SCC is effectively made up of the SCCs of the individual cows contributing milk to the bulk tank at that time and gives an estimation of how widespread infection is (prevalence) within those cows. Although the impact an individual cow has on BMSCC will depend on a combination of yield and SCC it has been estimated that there is an increase in infection prevalence of 10% for every 100,000 cells per ml increase in BMSCC.

It is worth noting that the BMSCC only gives a measure for the cows currently having their milk added to the bulk milk tank. Clearly this will not include any cows that are currently dry nor will it include cows whose milk is being withheld from the bulk tank. These might include cows under treatment, cows freshly calved and cows with high SCC. This means that the BMSCC of milk being sold from a dairy herd (from the cows that have their milk allowed in the bulk tank) is a “manipulated figure” and will often underestimate the herd SCC and so will not reflect true mammary health status of the herd. For those herds that perform regular monthly individual cow milk recording a better

figure to use is the calculated BMSCC as usually all cows currently in milk are milk recorded even if their milk is being withheld from the bulk tank. By calculating the theoretical BMSCC using the yields and SCC of each cow a more realistic BMSCC is obtained giving a truer picture of udder health status for the herd.

### **Individual Cow Somatic Cell Counts (ICSCC)**

Regular monthly individual cow SCC recording is probably the most common use of somatic cell counting and is almost exclusively performed on a composite commingled milk sample from all four quarters and represents the average SCC of all four quarters (assuming all quarters are of equal yield). As a consequence there is a danger that cows with infected quarters can go undetected particularly if they have only one infected quarter with the other 3 having low SCCs. For example a cow with a SCC below the 200,000 cells/ml threshold, say 150,000 cell/ml (Table 1), could have 3 quarters with a SCC of 50,000 cells/ml and one of 450,000 cells/ml  $[(50 \times 3) + (450) = 600$  which divided by 4 is 150].

**Table 1.**

#### **An illustration of the dangers of interpreting composite cow SCC**

<b>Comparison of quarter SCC for two cows with the same composite (comingled) cow SCC</b>					
<b>Likely infection status</b>	<i>Quarter cell counts</i>				Composite Somatic Cell Count (cow)
	FL	FR	BL	BR	
<b>Infected Cow</b>	50	50	50	450	150
<b>Uninfected Cow</b>	150	150	150	150	150

This should not be a surprise or a worry but merely serve to highlight the limitations of overly interpreting individual cow SCCs. It is generally accepted that when using a 200,000 cell/ml threshold the sensitivity and specificity (i.e. correctly identifying all the infected cows and correctly identifying all the uninfected cows) is about 75%. Or put another way 75% of cows with an infection have a SCC >200,000 cells/ml and 75% of uninfected cows have a SCC <200,000 cells/ml.

The increased cost and practical issues of collecting four times as many samples make regular monthly individual quarter SCC testing really only viable for research projects. However composite samples can be used as an indicator

for further testing at a quarter level using cow-side methods such as the California Milk Test (CMT).

### **Interpretation of SCCs**

BMSCC tends to be less variable over time than individual cow SCCs with a 12 month rolling mean giving the most smoothed data. In the UK BMSCC is measured at least 4 or 5 times each month to give an aggregated monthly figure and a geometric mean of the 3 most recent monthly figures (antilogarithm of an average of the logarithm of 3 months data) being used for payment purposes.

Individual cow SCC are much more variable over time and, after taking the many factors which influence SCC into account as well as sensitivity and specificity issues with various thresholds, it can be seen that they are only a guide to infection status. Any decisions relating to infection status using SCC should be based on multiple results. As is the case with many tests used to assess infection status repeat result and trends will often increase certainty.

Lactation averages which are often displayed on lactation certificates when cows are sold can also be very misleading unless they are low. A much more useful indicator of infection status is the last three SCC results as it is the current infection status we are interested in, not a previous cleared infection increasing the lactation average or worse still a cow dried off with a recently acquired infection but with a sufficient number of low SCC recordings earlier in the lactation to keep the lactation average low.

### **Changing the SCC threshold**

The figure of 200,000 cells/ml is a good general working threshold however the sensitivity and specificity of 75% can be changed by manipulating this threshold. Different populations, for example younger cows or herds with a low or high BMSCC, may justify different thresholds. In common with many tests changing the threshold in a given situation to improve sensitivity will be at the expense of specificity and vice versa. Consequently if it is important to be more certain cows are infected, for instance if they are to be selected for sampling or treatment, the threshold could be raised to 300,00 cells/ml. This would reduce the chance of a cow being selected as infected when it was not thus helping improve the cost benefit of sampling or therapy. If the converse is a high priority and there is a need to be sure cows are not infected, say for running a "clean" low SCC group or if a selective antibiotic dry cow policy (with or without teat seal) was used, with antibiotic dry cow therapy for infected cows, the threshold could be lowered to 150,000 cells/ml or even 100,000 cells/ml. This would ensure that if a misidentification occurred it would be more likely an uninfected cow was labelled as infected. In the "clean" group scenario it might mean an uninfected cow being kept in the dirty group (an error for her) but this

is preferable to an infected cow going into the “clean” group where the error will be compounded by spread of infection within the “clean” group. In the selective antibiotic dry cow scenario an uninfected cow might receive antibiotic, much as would happen in a non selective antibiotic dry cow therapy policy, but again this is preferable to an infected cow missing out her best chance to be cured by not being given antibiotic dry cow therapy.

### **INTERPRETATIVE SOFTWARE USED FOR DYNAMIC SOMATIC CELL COUNT ANALYSIS.**

For many years both BMSCC and monthly individual cow SCC from milk recording have effectively provided a static snapshot of somatic cell counts in a herd. More recently interpretive software has been used to interrogate the large databases of milk recording companies and correlate the data over time - emphasising a dynamic rather than snapshot approach to individual cow SCCs within herds. This has facilitated detailed SCC monitoring both during lactation, highlighting cows that have changed infection status between monthly milk recordings, as well as between drying off and subsequent calving effectively monitoring the dry period performance. Although the software can be setup to use any SCC threshold generally a 200,000 cells/ml threshold is used to indicate change of infection status. The first such program to be used widely in the UK was Herd Companion which was initially developed by the author and Dr James Hanks of the Veterinary Epidemiology & Economics Research Unit (VEERU) at The University of Reading in a development version in MS Access as part of a Milk Development Council (MDC) now DairyCo initiative. The concept has since progressed to utilise other milk recording data such as milk production, fertility and milk quality including milk protein and butterfat. At an early stage Herd Companion was migrated to the milk recording webpage with the help of Dr Andrew James also from VEERU and now exists as a purely web based application available to all National Milk recording (NMR) customers and their advisors which in the main are veterinary surgeons. Herd Companion can be found at <http://www.nmr.co.uk/> by following the link to [NMR Herd Companion](#). These developments have continued in the UK and web based data for dairy herds milk recording with The Cattle Information Service (CIS) including SCC data are available at <http://www.thecis.co.uk/>. PC computer based software is also available for herd data analysis notably Interherd (which the development version of Herd Companion interrogated), Interherd Plus and TotalVet all of which import SCC and milk quality data from milk recording companies.

## SOMATIC CELL COUNT ANALYSIS – BASIC PRINCIPLES USING HERD COMPANION AS AN EXAMPLE

Within lactation the SCC measure of a cow at any milk recording falls into one of seven status groups:

### Low SCC levels (below 200,000 cells/ml)

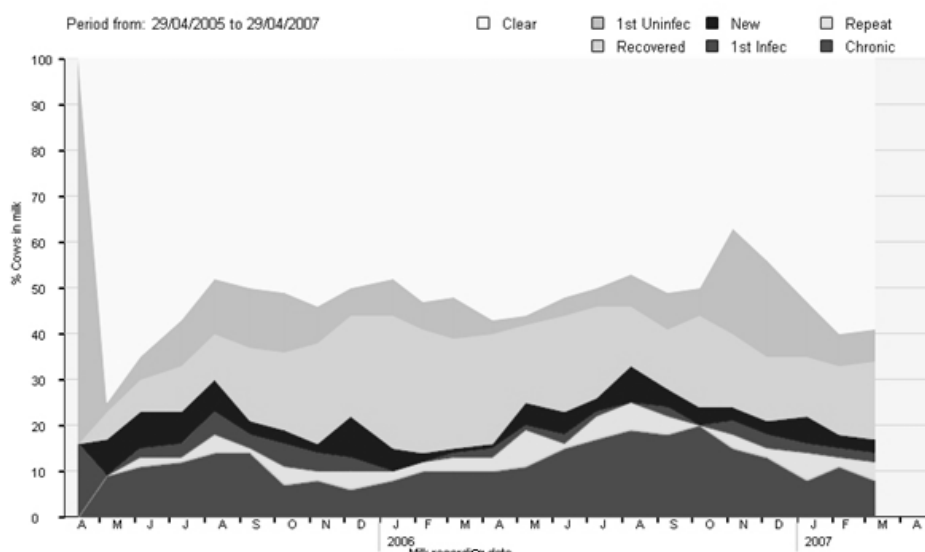
- UNINFECTED: A low SCC at this and the previous milk recordings.
- FIRST LOW: Both a low SCC AND the first milk recording in a lactation.
- RECOVERED: A low SCC level following a high SCC.

### High SCC levels (above 200,000 cells/ml)

*(Target overall < 10% of cows in milk > 200,000 cells/ml)*

- NEW: Not the first milk recording in the lactation but the first high SCC  
*(Target <5% new infection rate. Interference 10% cows in milk)*
- FIRST HIGH: Both a high SCC AND the first milk recording in a lactation  
*(Target <15% calving in > 200,000 cell/ml Interference 20%)*
- REPEAT: A high SCC for at least the second time in the lactation although following a low SCC at the previous milk recording.
- CHRONIC: A high SCC at both this and the previous milk recording  
*(Target < 5% chronic cow in milk. Interference 10%)*

**Fig 1.**



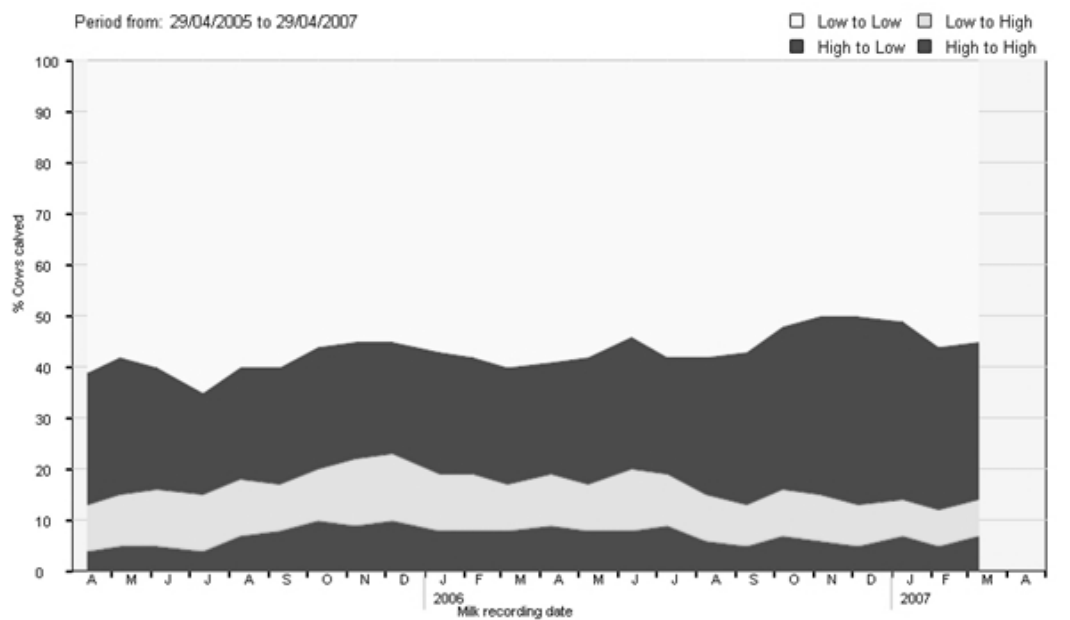
These 7 groups are graphed over time in a colour stacked area graph giving an indication of lactation performance. Effectively the proportion of the herd with new infections, recovering and remaining chronic can be followed over time. The dry period performance can be inferred by the proportions of first infected and first uninfected.

This greyscale version of the graph is not as clear as the colour version however the data is layered in the order above.

During the dry period cows can change their status in four ways:  
(Using 200,000 cells/ml as a threshold)

- LOW LOW: Uninfected cows remaining uninfected during the dry period.  
(Target > 80% of cows drying off low ie dry period protection rate)
- HIGH LOW: Cows eliminating an infection during the dry period.  
(Target > 80% of cows drying off high ie dry period cure rate)
- LOW HIGH: Cows acquiring an infection during the dry period.
- HIGH HIGH: Cows remaining infected through the dry period.

**Fig 2.**



These 4 groups are graphed over time in a colour stacked area graph giving an indication of dry period performance over time. Effectively the proportion of

cows calving in protected, curing, acquiring an infection or apparently failing to cure can be followed over time.

This greyscale version of the graph is not as clear as the colour version however the data is layered in the order above.

There are a number of detailed reports and graphs highlighting trends in the herd SCC profile and listing potential problem animals. The number of animals in each of the 7 lactation groups is listed for each month's milk recording allowing the new infection rate, the number of chronic cows, cows recovering or having repeat infections and cows infected at their first recording after calving to be monitored. The summary report looks at the age profile for the 7 lactation groups, the dry period performance for low and high SCC cows at drying off and trends in recovery rates. The individual animals making up the data groups for the lactation graph (cows >200) and dry period performance (Fig 1 & 2) are listed effectively giving potential attention or action lists.

**Table 2** Options for cows selected on SCC action lists

<b>Action lists from cows with high individual cow SCC (after interpretation and not just from one result)</b>	
<b>Action for selected problem cows</b>	<b>Effect on cow and or herd</b>
<b>No action</b>	
<b>Sample and culture</b>	Cow unaffected. Herd still at risk as infected quarter(s) still being milked through parlour
<b>Withhold milk (or feed to male calves) *</b>	
<b>Treatment</b>	Cow hopefully cured. Herd risk reduced/eliminated
<b>Early dry off</b>	Cow may cure.
<b>Quarter dry off / quarter culling</b>	Herd risk reduced/eliminated #
<b>Cull cow</b>	Cow removed and herd risk eliminated #

\* BMSCC will be reduced by "manipulation" as the high SCC milk is not entering the tank. However the risk of spread is still present although some form of cluster disinfection or milking last would help.

# Often by the time chronically infected cows are identified, and eventually culled, spread to other cows within the herd will have already occurred. So even though, as far as the herd is concerned, culling will effectively remove the infection in that cow infection will often be established in other cows which in time may well become chronic high cell count cows themselves.

The full detail of all the reports and graphs in Herd Companion is out with the scope of this paper but can be viewed by accessing the demonstration farm on the Herd Companion webpage.

A further parameter which although not currently widely used was used in the MS Access development version of Herd Companion but never made it to the web version. It can give a useful single figure assessing the infection transmission dynamics in either the dry period or between milk recordings. The concept is to look at the number of cows going up through the 200,000 cell/ml threshold (apparent new sub-clinical infections - ANSI) (1) compared to the number going down through the 200,000 cell/ml threshold (apparent cures). It has been called the "Infection Ratio" (IR) (2) or Net transmission index" (NTI). (4) However care must be used when viewing the figure in isolation because neither the magnitude of the SCCs greater than 200,000 cells/ml nor the yield are taken in to account and so it is possible for the BMSCC to go down despite the infection ratio being greater than one. This would occur if the fewer in number recovering cows had previous very high SCCs (and possibly higher yields) whilst the more numerate apparent new infections had less significantly elevated SCCs (and possibly lower yielding cows).

## **WHAT CAN BE LEARNT BY LOOKING AT THE WHOLE DATASETS OF MILK RECORDING COMPANIES?**

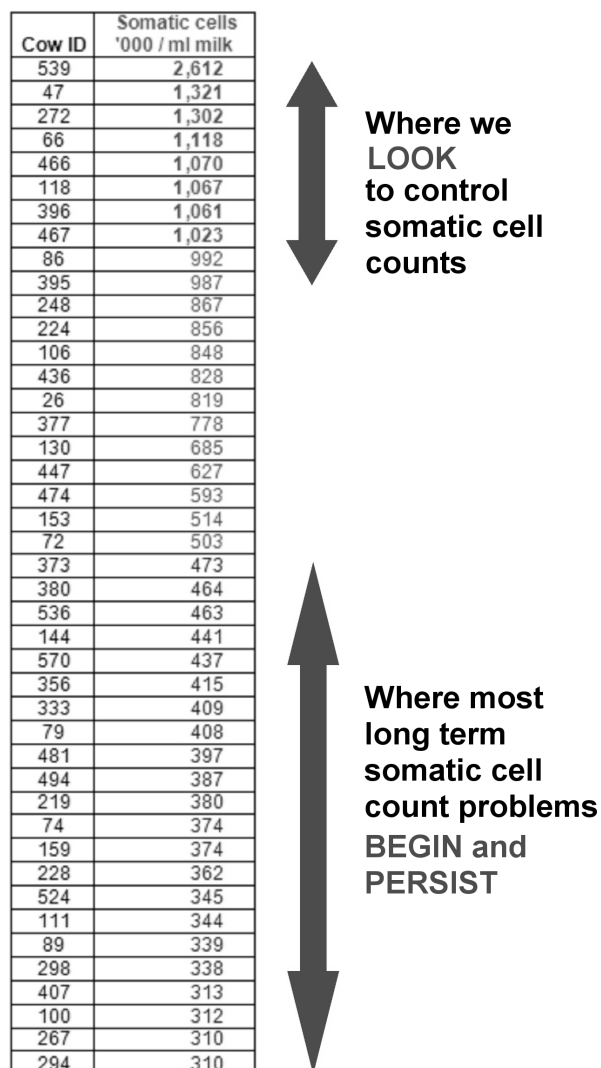
When very large datasets are interrogated without reference to individual herds the influence of individual herd variation is mitigated and trends can be investigated which give some insight into the dynamics of SCC in the UK dairy industry. Approximately 50% of GB dairy herds milk record through National Milk Records (NMR) so their database gives a good representation of herds from England, Wales and Scotland and an increasing number of herds from Northern Ireland.

### **1. Persistent high somatic cell count cows: where do they start, where do they get to? (2007) (5)**

From a dataset of 585,277 cows in 5,174 NMR recorded herds during December 2006/January 2007, a total of 56,082 cows were identified with 2 or more consecutive somatic cell count readings over 200,000 cells/ml milk. These "chronic high" cell count cows had all recorded one or more SCC below 200,000 cells/ml milk at the start of the current lactation suggesting the population had acquired infection during the lactation rather than the previous dry period or earlier. It was found that 70% of cows when they first passed up through the 200,000 cells/ml threshold were only between 200,000 & 500,000 cells/ml. In their most recent recording when this dataset was captured some of these chronic cows will have had several months with SCC greater than 200,000 cells/ml, yet 60% were below 500,000 cells/ml.

This indicates that most high somatic cell counts begin and persist below 500,000 cells/ml milk. This could equate to one infected quarter in excess of 1.7 million cells/ml, if diluted by clean milk (<100,000 cells/ml milk) from three uninfected quarters and so just concentrating on the highest cell count cows in a herd enables many new infections to establish and persist undetected (Fig. 3).

**Fig 3.**



**2. Percentage somatic cell count contribution: highlighting the wrong cows (2007) (3)**

From a dataset of 5,104 NMR recorded herds during January 2007, individual SCC contributions (yield X SCC) were calculated for 570,987 cows. Aggregating individual contributions enabled cows to be categorised as either “in” or “out”

of the overall top 10% of cows by SCC contribution. The number of consecutive high (>200,000 cells/ml milk) SCC recordings before the January recording was used to determine the duration of prior infection. The SCC at the following recording was taken to indicate the level of recovery (<200,000 cells/ml). The cows in the top 10% percentage SCC contribution contained a lower proportion (44%) of newly infected cows (those with a 1<sup>st</sup> high SCC) than existed in the overall population (50%) indicating that high percentage contribution cows tend to be more well established infections which experience shows are less likely to respond to therapy. Nearly one quarter (23%) of cows having a second high SCC were not in the top 10% percentage SCC contribution group and so would not have been identified by concentrating on the cows with a high percentage contribution.

### 3. The correlation between somatic cell counts around calving and cow longevity (2009) (6)

Using the NMR milk recording database 59,198 cows from 2,586 randomly selected herds were studied. These cows all calved between October and December 2006 to start their second or higher lactation thus allowing classification of their previous dry period performance using the 4 dry period Herd Companion parameters described above. The data allowed follow-up of cows for up to three years giving sufficient time to either re-calve (survive) or leave the herd. The precise reasons for leaving the herd are not recorded although the great majority will be culls. Table 3 shows a marked variation in the re-calving percentages of cows in each dry period category.

**Table 3.**

Dry period category	Calvings	Failed to re-calve	% Failed to re-calve	Chi <sup>2</sup> p-value for comparison with Low : Low
<b>High : High</b>	7,137	3,063	<b>43%</b>	<0.0001
<b>High : Low</b>	19,998	6,307	32%	<0.0001
<b>Low : High</b>	4,880	1,511	31%	<0.0001
<b>Low : Low</b>	27,183	6,379	<b>23%</b>	-
<b>Total</b>	59,198	17,260	29%	

Cows with raised SCC before and/or after the dry period have a significantly increased probability of failure to re-calve ( $p < 0.0001$ ) when compared to Low:Low category cows. High:High cows are 20% less likely to re-calve than Low:Low cows. The re-calving rates of cows in the Low:High and High:Low categories were not statistically significantly different from each other (Chi<sup>2</sup> p-value: 0.4378).

## **CONCLUSIONS**

The advances in the use of web based interpretive software to regularly and routinely interrogate the large milk recording company databases has allowed veterinarians and advisors to manage, monitor and benchmark many more herds than was possible when paper or computer records were lists of SCC numbers in descending order. As happened with fertility monitoring targets and interference levels for various parameters are now commonly accepted.

Clearly SCCs are only part of a herd mastitis picture and although efforts are always being made to improve the quality of clinical mastitis data it is generally poorly recorded as far as centralised milk recording company computer databases are concerned. The PC computer based clinical mastitis records (often kept on the farm computer or in the veterinary practice) are generally more accurate and recent developments in both Interherd Plus and TotalVet are at last making similar strides seen in the dynamic and interpretive SCC analysis in recent years. Perhaps veterinary surgeons and milk recording companies can in part be blamed for the poor record keeping by farmers for clinical mastitis. Unless the data can be shown to be useful in terms of management and control then the efforts of recording may well be seen to outweigh any benefits offered by full and meticulous recording. A holistic approach incorporating both SCC and clinical mastitis data is clearly the best approach and encouragement to record clinical mastitis by milk recording companies as well as a recent national initiative the DairyCo Mastitis Control Plan is hoped to do just that.

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## THE COW'S RESPONSE TO PATHOGENS

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### SUMMARY

Mastitis, an inflammation of the udder caused by invading pathogens, is one of the most important infectious diseases in dairy production. It is recognised as one of the three most significant health problems of the UK dairy herds, together with lameness and fertility problems (6), raising concerns for animal welfare and economic losses in milk production. This paper discusses new data that aims to provide a better understanding of the pathogenesis of the disease caused by a leading pathogen that may open new avenues for the prevention of this disease and investigates the potential for additional markers for genomic selection for improved milk traits and disease resistance.

### INTRODUCTION

The signs of mastitis vary according to factors in the host and the specific pathogen causing the infection, resulting in sub-clinical or clinical mastitis. While sub-clinical mastitis shows no obvious signs of disease, clinical mastitis can be associated with visible abnormalities in the milk accompanied by pain and swelling in the affected gland, as well production of a secretion which is composed solely of aggregated protein in a serous fluid. In severe cases, the animal can display further signs such as elevated temperature and loss of appetite, which can develop into bacteraemia, septicaemia and death of the animal.

Milk from the uninfected gland contains resident somatic cells, including macrophages and neutrophils, typically <150,000 cells/ml. The somatic cell count (SCC) is an international measure of milk quality and udder health, with only milk with a cell count below 400,000 cells/ml permitted to be sold for human consumption within the European Union. Milk from sub-clinically infected quarters usually has a cell count <250,000 cells/ml but can vary, while clinically infected quarters contain <2,000,000 cells/ml. This increase in cell count is associated with the influx of neutrophils, and the inflammatory reaction associated with mastitis results in a lower rate of milk production and a deterioration of the quality of the secretion.

The vast majority of cases worldwide are due to infection with one of five bacterial species; *Streptococcus uberis*, *Escherichia coli*, *Staphylococcus aureus*, *Streptococcus dysgalactiae* and *Streptococcus agalactiae*. Changes to

the husbandry of dairy cattle and implementation of hygienic milking procedures, which reduced the duration and transmission of infection from cow to cow, have resulted in a decrease in the incidence of bovine mastitis worldwide. In herds where the recommendations are followed rigorously, mastitis is now due exclusively from infections acquired from environmental sources (2), with a greater proportion of infections now due to *S. uberis* and *E. coli*. Subsequent reductions in disease may come about from the targeting of these individual pathogens.

The development of highly effective vaccines and other preventative interventions against pathogens of the bovine mammary is hampered by a lack of information on the components of pathogen and host, and their subsequent interactions, which promote infection and disease. Current work aims to address this by analysing the host response to infection with *S. uberis*. *S. uberis* gains access to the mammary gland through the teat canal and once present within the parenchyma the organism is able to replicate, resist the bactericidal action of neutrophils and induce an inflammatory response (8). However, the precise interactions between host and pathogen that promote infection and disease have not been described. A limited number of investigations of the immune responses to intramammary infection with *S. uberis* have been undertaken (1, 4, 9, 12). These showed the changes associated with mastitis, and identified differences in these changes that were correlated with distinct pathogens. Following challenge with *S. uberis*, elevation of various cytokines (IL-1 $\beta$ , IL-8, IL-10, IL-12, IFN- $\gamma$ , TNF- $\alpha$ ) was detected along with elevated levels of soluble CD14, lipopolysaccharide binding protein (LBP) and the complement component C5a (1). These responses differed from those reported for *S. aureus* (10); indicating that such responses may account for the more severe nature of disease following infection with *S. uberis*. This highlights the variability of the innate immune response to distinct mastitis pathogens, and emphasises the potential correlation with the outcome of infection.

This study aimed to investigate the host response to infection with a virulent field strain by comparison to infection with an avirulent field isolate using an experimental challenge model. A comparative study of *S. uberis* strains of defined and differing virulence provides a unique opportunity to assess the differences in the responses to challenge and correlate these to specific responses of the host, with the potential to determine which combinations of responses are associated with particular aspects of pathogenesis. Such data would significantly enhance our understanding of the specific disease processes relating to this pathogen and our understanding of the immune responses of the bovine mammary gland.

## **EXPERIMENTAL OUTLINE**

***Streptococcus uberis*** - Strains of *S. uberis* differ in virulence for the lactating udder (5). Strain 0140J, a virulent strain, is well used around the world in models of infection in dairy cattle (1, 3, 5, 7, 11). In contrast, strain

EF20 is of considerably reduced virulence. In a comparative study 0140J caused infection and disease in 89% of challenged quarters whereas EF20 colonised all animals transiently at a low level, causing mild signs of disease in only 11% of challenged quarters (5). These strains of *S. uberis*, 0140J and EF20, were used in an experimental challenge model.

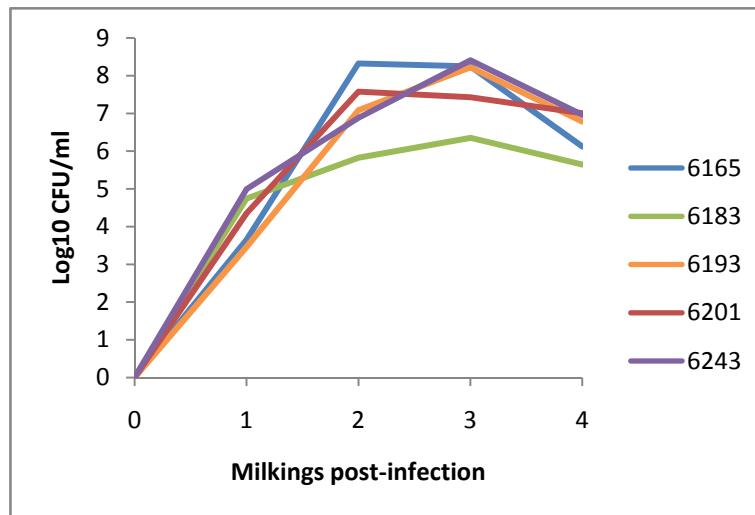
**Challenge studies** - Two groups of five heifers, four to ten weeks post-calving, were challenged ( $10^3$  colony forming units or CFU) in two quarters with either 0140J or EF20 after morning milking. Milk samples were then taken for the next four milkings, in addition to samples taken pre-challenge. From these samples, SCC and bacteriology (CFU) were determined. In addition, somatic cells were harvested for RNA extraction and milk samples were retained for additional experiments. At post-mortem the gross pathology of both infected quarters was determined, and tissues from multiple sites within each infected quarter were harvested as well as samples of the supramammary lymph nodes and blood.

**Immune responses during intramammary infection** - Once the initial host barrier systems have been breached, the innate immune system provides the next level of defence against mastitic pathogens. Description of the innate immune response of the bovine mammary gland is instrumental in understanding the pathogenesis of disease and the subsequent development of control measures. To address this we will be using RNA extracted to perform a temporal study of the transcriptional changes within the somatic cells for the duration of the challenge experiment. Furthermore we will compare the transcriptional profile following infection with the virulent strain 0140J to that from EF20. In addition, the production of immunological markers in the milk will also be determined by ELISA.

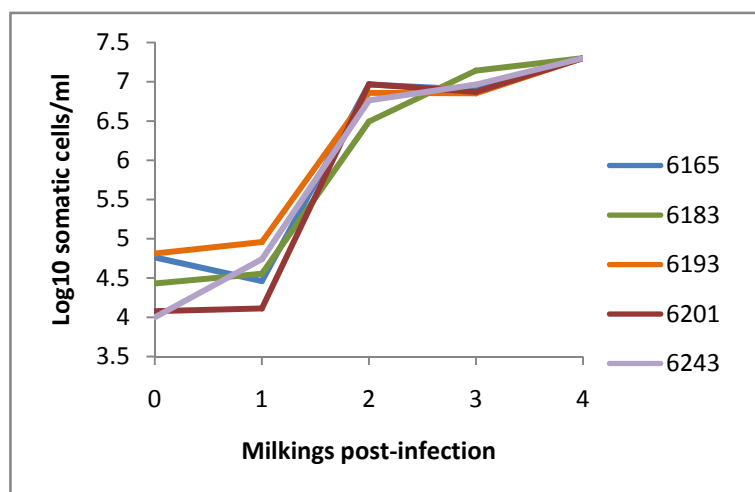
## **PRELIMINARY RESULTS AND DISCUSSION**

Data analysis is very much ongoing as this paper is being written.

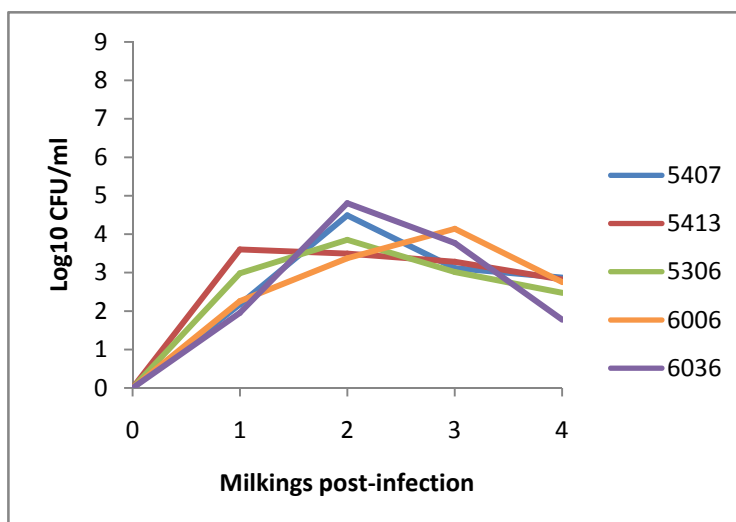
Analysis of CFU following infection with 0140J indicates efficient colonisation of the gland as indicated by rapid replication of the pathogen (see Fig 1). This is concomitant with a rapid increase in SCC by 24hr post-infection (see Fig. 2). In contrast, analysis of CFU following infection with EF20 (see Fig. 3) shows it to be less efficient in colonisation, resulting in a failure to establish itself within the gland and therefore in fewer numbers of bacteria throughout the experiment. Interestingly infection with this strain is also associated with a rapid and sustained raise in SCC (see Fig. 4). This data confirms that both EF20 and 0140J survive equally in the presence of neutrophils and must therefore both be able to inhibit neutrophil function. The failure of EF20 to establish within the gland suggests that the difference in response to these strains relates to processes during the early colonization of the gland.



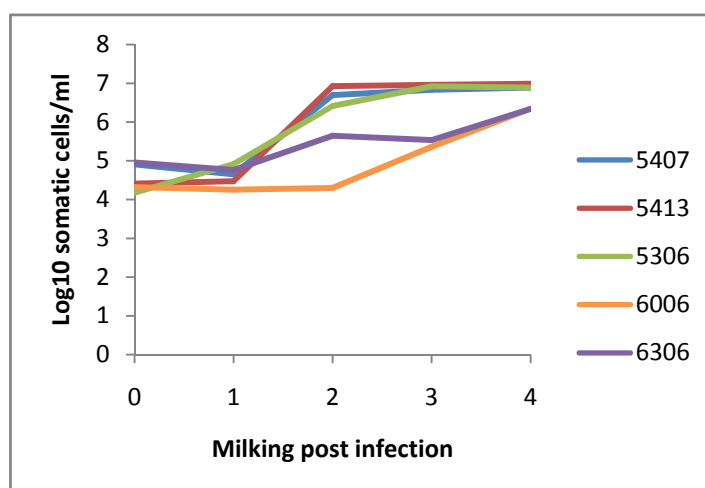
**Figure 1** - Bacteriology data (log<sub>10</sub>) showing CFU of strain 0140J throughout the course of the study. Data is shown as a mean of the two infected quarters.



**Figure 2** - Somatic cell count data (log<sub>10</sub>) following infection with strain 0140J throughout the course of the study. Data is shown as a mean of the two infected quarters.



**Figure 3** – Bacteriology data (log<sub>10</sub>) showing CFU of strain EF20 throughout the course of the study. Data is shown as a mean of the two infected quarters.



**Figure 4** – Somatic cell count data (log<sub>10</sub>) following infection with strain EF20 throughout the course of the study. Data is shown as a mean of the two infected quarters.

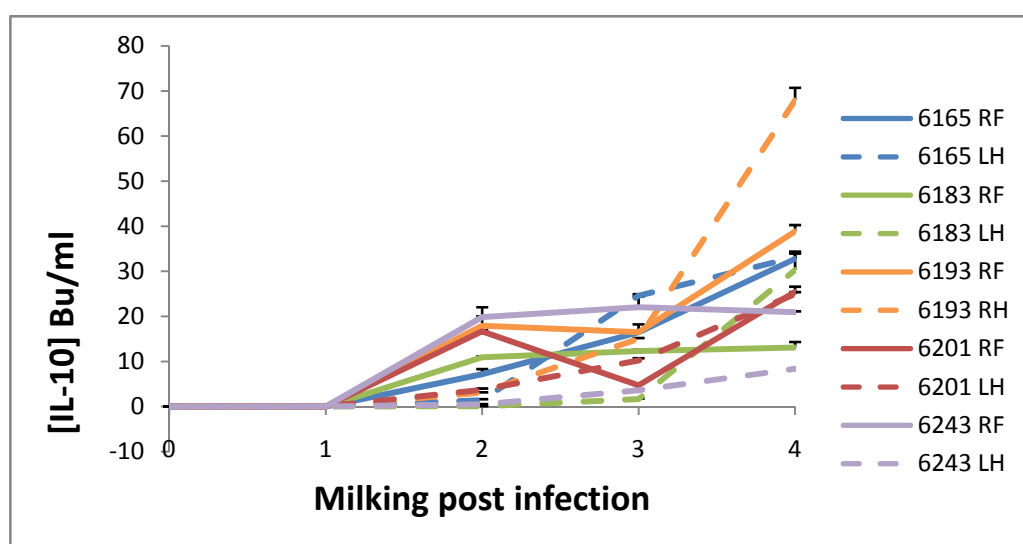
Those animals infected with 0140J displayed typical signs of clinical mastitis, including deterioration in the quality of the secretion, elevated temperature, tenderness and firmness within the infected quarters, contrasting with those animals infected with EF20 which showed much reduced signs of disease.

At post-mortem, examination of the tissues from animals infected with 0140J showed extensive gross pathology in the form of oedema within the mammary tissue. In contrast little or no gross pathology was observed in those animals infected with EF20. Examination of the supramammary

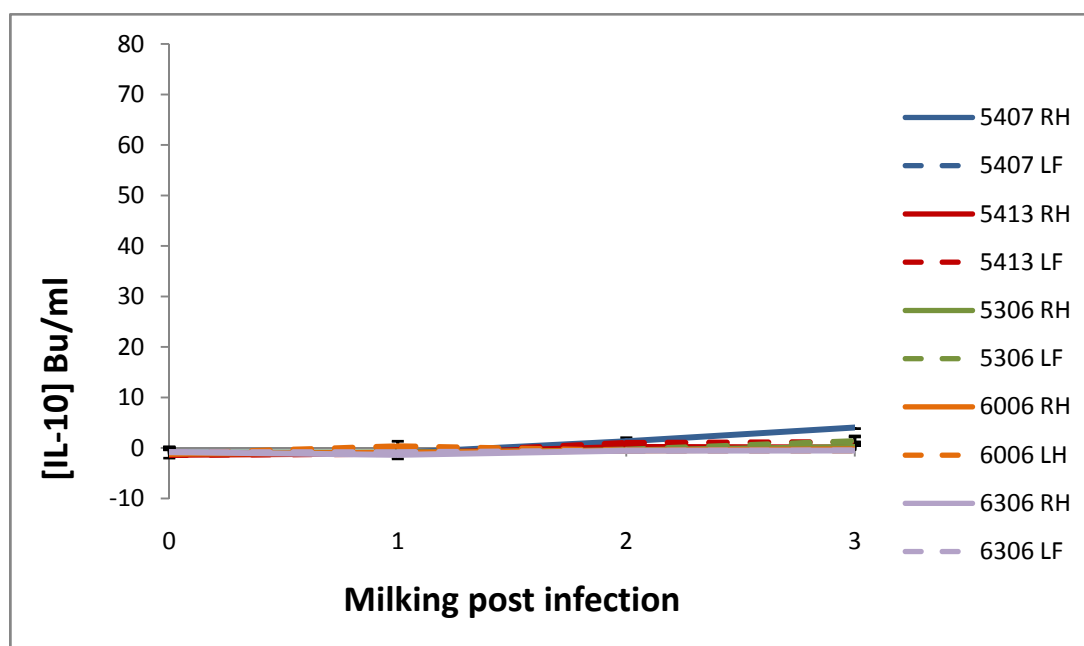
lymph nodes from both groups of animals showed no differences, with neither showing any abnormalities.

### Identification of immunological markers

Preliminary data is available from ELISAs which analysed production of immunological markers in the milk of both groups of animals. Analysis of IL-10, an anti-inflammatory cytokine, showed production in those animals infected with the virulent isolate to increase over the course of the study (see Fig. 5). In contrast, little or no IL-10 was produced by those animals infected with EF20 (see Fig. 6). The same trend was seen for the production of the pro-inflammatory cytokine IL-12 (data not shown). This work will be extended to cover other molecules of interest, including the potent chemoattractant CXCL8. CXCL8 and its receptors CXCR1 and CXCR2 (present on the surface of neutrophils) are also being studied as potential markers for genetic selection, and this will be discussed further at the conference.



**Figure 5** – Production of IL-10 (bovine units per ml) following infection with strain 0140J throughout the course of the study. Data is shown on a per infected quarter basis.



**Figure 6** – Production of IL-10 (bovine units per ml) following infection with strain EF20 throughout the course of the study. Data is shown on a per infected quarter basis.

### Further work

A more detailed presentation and discussion of the preliminary findings of this research and the next steps in this work will be made at the conference.

### ACKNOWLEDGEMENTS

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## **RECENT ADVANCES IN MASTITIS DETECTION WITH AUTOMATIC MILKING SYSTEMS**

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### **INTRODUCTION**

Mastitis, together with lameness and failure to reproduce, is one of the main production diseases of dairy cows. Presence of high levels of mastitis pathogens, in raw milk at least, can constitute a risk to human health and is regarded as an indication of less than adequate milking methods. Recent survey results indicate that annual incidence remains, stubbornly, at an average of 47 – 65 clinical cases per 100 cows per year (2).

Dairy Hygiene Legislation requires that “that milk from each animal is checked for organoleptic (taste, colour, feel, smell) or physico-chemical abnormalities by the milker or a method achieving similar results and that milk presenting such abnormalities is not used for human consumption” (11). The clear implication is that, with automatic milking systems where a human milker is not present, sensors are provided, used as a basis to divert abnormal milk and that the “gold standard” is that which can be achieved by a human.

A patent search on the words “mastitis sensor automatic milk” reveals that there are at least 641 patents already applied for or granted (5). There are also many published scientific papers discussing a myriad of possibilities for automated sensors for mastitis. In spite of this, the range of technologies being used in practice is relatively limited. This paper sets out to discuss the application of sensor technology as a tool for compliance with legislation and to manage udder health.

### **MASTITIS - THE DISEASE AND SYMPTOMS**

Mastitis is not one disease but can be caused by a large number of pathogens.

Clinical mastitis signs vary from seeing a few clots in the milk with perhaps some swelling of the infected quarter to severe signs which include a swollen quarter or whole udder. The cow may have a high temperature, fever, loss of appetite, and be dehydrated (2). In severe cases, the disease can result in the death of the animal.

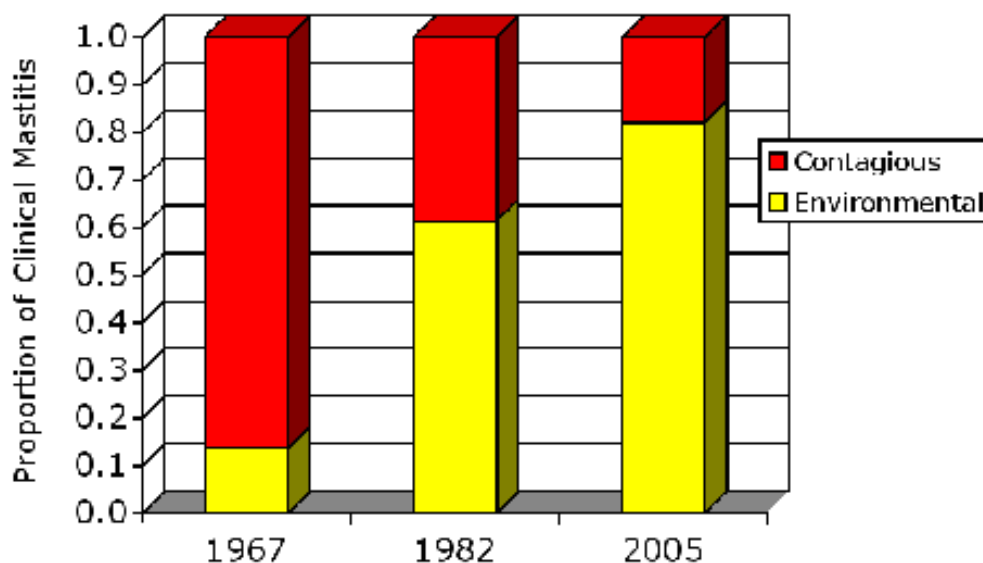
Sub clinical mastitis is when there is bacterial infection present with no clinical signs. The cow could have a raised somatic cell count (number of

white blood cells/ml of milk) and there may be bacteria in milk cultures. However, legislation does not demand that such milk is rejected.

It is generally accepted in the UK that a somatic cell count of >200,000/ml of milk is an indication that a cow or quarter is infected. Care has to be taken when using this figure as a cow could have a cell count just below 200,000 and have  $\frac{3}{4}$  with a cell count of less than 50,000 and  $\frac{1}{4}$  with a cell count well over 200,000/ml. Where a herd risks being penalised on the basis of somatic cell counts, rejection of milk from cows known to have a high somatic cell count can be a useful milk quality management tool. Of course, the cost of that milk must be weighed against the likely penalty.

One significant trend which should not be overlooked is the change in the relative proportions of contagious and environmental mastitis over the last 40 years. As a result of the successful implementation of mastitis control strategies, the proportion of contagious infection has declined to less than 20% of the total (2).

**Table 1 Contagious and Environmental Clinical Infections 1967 – 2005 (2)**



## **SENSOR TECHNOLOGY FOR DETECTING MASTITIS**

The extremely wide range of types of pathogen associated with mastitis makes it difficult to see how any single sensing technology on its own can be successful.

As already indicated, there are a large number of patents relating to the use of mastitis sensors on automatic milking units. In the wider context, a patent search on “mastitis sensor” indicates that there are at least 1357



those effects occurring earlier in the cause effect spectrum are much more likely to be associated with “self cure” where therapeutic intervention may not be justified.

In the context of automatic milking, the last thing an operator wants or needs is to be burdened by mastitis alerts where there are no visible symptoms, where intervention may not to be justified or has a negative cost benefit. However, there is a contrasting argument, from the point of view of udder health management, that early warning of impending problems can be useful. This highlights the fact that it is essential to educate users in respect of the correct use of sensor technologies.

## **SENSORS IN PRACTICAL USE FOR MASTITIS DETECTION**

In 2004, in a paper with a similar brief, Reinemann (12) reported on the basis of European and American studies that electrical conductivity and milk colour were the most widely used on-line milk sensing methods. He also noted that, deviation in milk yield and milking interval are widely used supporting diagnostic techniques. He also predicted that cow side measurements of somatic cell count were likely to be applied to automatic milking in the future.

Whilst, superficially, the situation would seem to be largely similar today, a number of significant observations can be made.

Most sensor technology implemented today has been driven by the need to detect, or at least predict, onset of clinical mastitis symptoms.

An ideal sensor should be non-invasive, cleanable, avoid damage to the milk and not require the use of reagents, particularly where those reagents may be dangerous to the environment or difficult to dispose. This point has ensured that sensor technologies such as electrical conductivity and optical sensing methods, including colour, continue to be attractive. The use of such sensors also strikes a balance between effectiveness and low running cost.

Although the use of electrical conductivity has been discussed, supported and condemned for many years, the main point to note is that there have been significant developments in the refinement of its use.

Significantly, Hamman & Zeconi (6) concluded that “within cow comparison between quarters seems to be the best way to use conductivity measurements” and “measurement of electrical conductivity in milk requires a high degree of standardisation”. These conclusions indicate that the main challenge with electrical conductivity measurement is in standardisation of the measurement technique and interpretation of the physical data.

Mein *et al.* (8), using temperature correction and more sophisticated algorithms to take account of milking interval and other factors, have shown a sensitivity of 92% and specificity of 95% where a true positive was defined as the presence of a major pathogen. A further study was carried out by the same authors on 4 herds and reported sensitivity of 80% and specificity of 93%. In that study a true positive was defined as a cow with visible clots in the foremilk of one or more quarters at a monthly herd visit. True negatives had no visible signs of clots in their milk and low CMT (scores of 1 or 2). The sensitivity and specificity data achieved are favourably comparable with that reported for human observation by Hillerton (7).

The predicted (12) use of cow-side somatic cell counting or estimating is not regarded as an indicator of abnormal milk (10) and has not, by any means, been universally adopted.

The use of optical technology to measure various milk components, including blood, is already showing promise whilst still satisfying the ideal of being non-invasive and not requiring reagents.

The final comment from Reinemann (12), that inputs from a number of sensors, including milk composition, animal behaviours and milking characteristics need to be co-ordinated via a “smart” system, is as true now as it was then. This is particularly relevant when considering the predominance of environmental pathogen related mastitis. Often the first symptoms can be failure to attend the automatic milking machine and/or loss of milk production. For many farmers, one of the most important sensors is the list of cows not attending the machine.

## **CONCLUSION**

An enormous array of technologies is possible for detecting mastitis related characteristics within milk.

The application of such sensing technology is driven by legal requirements to detect and divert abnormal milk.

Other technologies must provide a very clear benefit at a cost which is recoverable.

Sensors should, ideally, not damage milk, nor require discard of milk and not require reagents.

The most important factor in sensor technology for mastitis is an intelligent management system to combine the output of various sensors and permit “management by exception”.

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## **IS THERE A ROLE FOR BACKFLUSH SYSTEMS?**

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### **SUMMARY**

Automated dipping and flushing (ADF) systems have existed for some years, but their effect on udder health was never examined in a field study on commercial dairy farms. The objectives of this study were therefore to evaluate the effect of introducing an ADF system in a herd on bulk milk somatic cell count (SCC) and individual cow SCC. Dairy herd improvement data were collected over a 30 mo period of 25 sets each of 3 farms. Each set of three farms contained a farm that installed an ADF system, one that disinfected teats using dip cups after milking and one that sprayed teats after milking. Data were analyzed using linear mixed models. Bulk milk SCC on farms that sprayed or dipped before installing an ADF system was 14,000 and 31,000 cells/ml lower in the 12 mo period starting 6 mo after installation, respectively, than on farms that continued spraying or dipping the teats after milking. Proportion of cows with an elevated SCC were in the same periods compared with farms that sprayed or dipped 4.8 and 1.4% lower, respectively, after installation of an ADF system.

Installing an ADF system had a beneficial effect on bulk milk SCC and individual cow SCC. The effect was most prominent after approximately 6 mo after installation.

### **INTRODUCTION**

Proper post-milking teat disinfection (PMTD) is an effective management practice to prevent transmission of contagious mastitis pathogens from spreading from cow to cow (8, 9, 11). Post-milking teat dipping can use up to 10% of the milker's routine in the milking parlour (2). With farms increasing in size and increasing number of rotary milking sheds, the need for automation of PMTD is mounting.

Disinfecting and flushing milk liners has proven to reduce bacteria counts and the incidence of IMI in experimentally challenged cows (5). Several systems are commercially available, such as backflushing, steam cleaning, and an air wash system, each of them with their own advantages and disadvantages. Backflushing reduces the significance of the milking liner unit as a fomite for *Staph. aureus* transfer between cows (4, 7).

The system described in this manuscript automatically covers the teat with a disinfection fluid after milking and flushes the liner (Hogewerf *et al.*, submitted). A similar system has been described earlier by Galton (5). In this system the disinfectant is injected through the short milking tube instead of the head of the liner. An older study by Grindal and Priest (6) describes a system in which the disinfectant delivery tube is conveyed within the long and short pulse tubes and injects the disinfection fluid into the mouthpiece chamber. Both studies were carried out in an experimental set up. The current system is the first commercially available system in several countries. However, no long-term field studies on multiple commercial dairy farms have been conducted which study the effect of installing an ADF system on SCC and BMSCC. The objectives of this study were therefore to evaluate the effect of introducing an ADF system in a herd on BMSCC and ICSCC.

## **MATERIALS & METHODS**

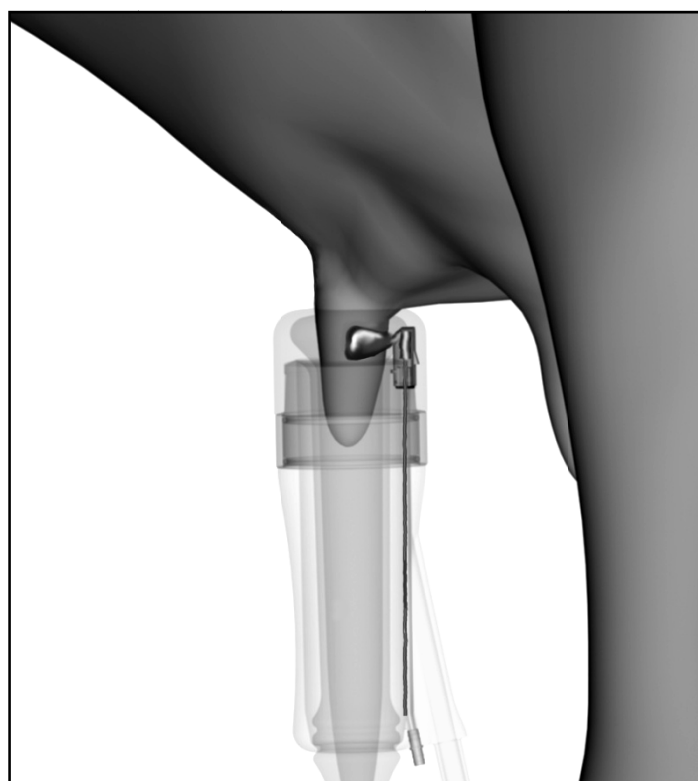
Twenty-five farms were recruited in the United Kingdom that had an ADF system installed between December 2004 and June 2007. Each farm that had installed the ADF system was matched with 2 farms: one that applied PMTD using a sprayer, and one that applied PMTD using a dip cup. Method of applying PMTD has a significant impact on BMSCC (3). Farms were matched on herd size, average BMSCC, and geographic location. In total, 75 farms participated in this study, 25 sets of 3 farms.

Monthly average BMSCC and ICSCC for each lactating cow in lactation were obtained from the British Dairy Herd Improvement program.

The system used in this study was the Automated Dipping and Flushing system (Research Development & Innovations bv, Gorssel, the Netherlands). After a cow has finished milking, a signal to the individual control system is given, which closes the milk line and an iodine based dip is sprayed in the mouthpiece chamber of the teat liner (Fig. 1).

The unit sprays teat dip followed by air. Upon detachment, the system allows approximately 5 to 20 s for the disinfection fluid to disinfect the liner, after which the liners are rinsed with, alternating cold water followed by compressed air to remove any iodine residues and clean each liner.

**Figure 1 Schematic delivery of teat disinfection fluid to milking liner mouthpiece chamber.**



Comparison of BMSCC between farms in the matched sets of farms was performed using linear mixed models with herds as random effects. To approximate the normal distribution, a natural logarithmic transformation of SCC values (1,000 cells/ml) was used (1). The model for the effect of installing an ADF system on the BMSCC at the  $i^{\text{th}}$  measurement in herd  $j$  in herd set  $k$  was as follows:

$$\begin{aligned} \ln\text{BMSCC}_{ijk} = & \beta_0 + \beta_1\text{installed}_{ijk} + \beta_2\text{dip}_{jk} + \beta_3\text{dipaft}_{jk} + \beta_4\text{adf}_{jk} \\ & + \beta_5(\text{dip}*\text{installed})_{ijk} + \beta_6(\text{dipaft}*\text{installed})_{ijk} \\ & + \beta_7(\text{adf}*\text{installed})_{ijk} + \nu_k + \omega_{jk} + \varepsilon_{ijk} \end{aligned}$$

where  $\beta_0$  is the intercept;  $\beta_1$  is the regression coefficient for the installation of the ADF system, i.e. after the installation date this variable changed from 0 to 1;  $\beta_2$  to  $\beta_4$  are regression coefficients for design variables;  $\beta_5$  to  $\beta_7$  the regression coefficients interaction between the design variables and the installation of the ADF system;  $\nu_k$  is the herd set random effect;  $\omega_{jk}$  is the herd random effect, and  $\varepsilon_{ijk}$  is the error term.

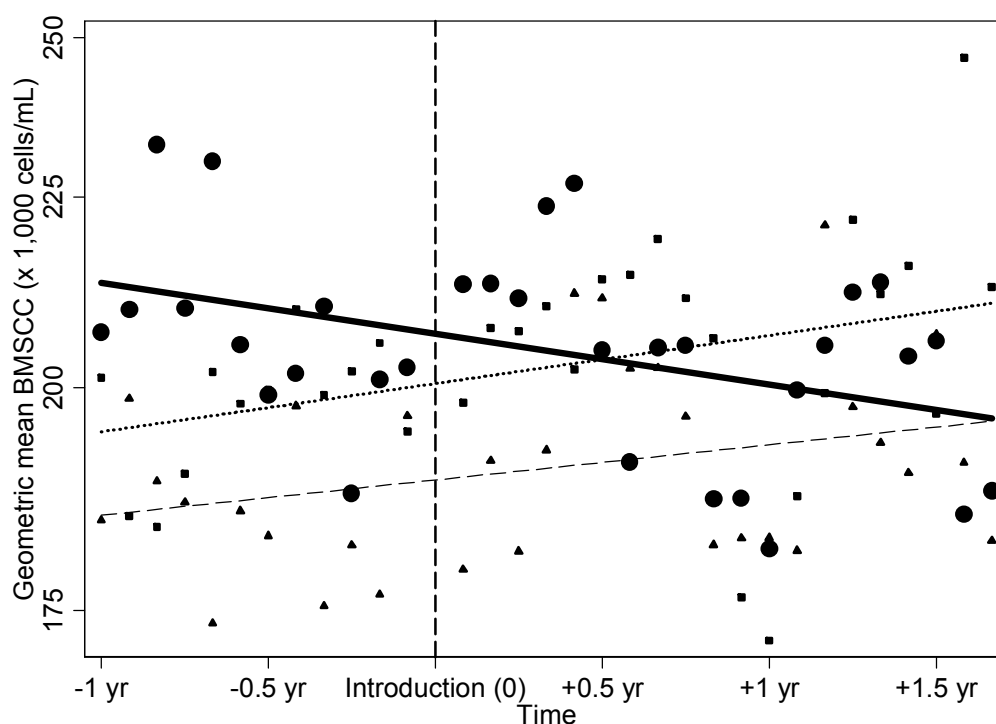
BMSCC was analyzed using the 12 mo period before installation of the ADF and a 12 mo period adjacent to installation, 3 mo after installation, and 6 mo after installation, because we expected that the effect of installing a different PMTD system would be measurable after some months.

Probability of an elevated ICSCC was compared between the different PMTD situations using a 5-level hierarchical logistic model, while including random effects of herd sets, herds, cows, and lactations.

## RESULTS

Mean and median herd size of participating farms was 270 and 240 cows, respectively, ranging from 94 to 790 cows. Average 305-days milk production for all herds was 8,265 kg, ranging from 6,300 kg to 10,580 kg. Geometric mean BMSCC before installation of an ADF system was 210,000, 184,000, and 198,000 cells/ml and after installation 193,000, 182,000, and 201,000 cells/ml for farms that installed an ADF system, dipped, and sprayed, respectively (Fig. 2).

**Figure 2 Monthly geometric mean bulk milk SCC before and after introduction of an ADF system and for farms that installed an ADF system (solid circles and solid trendline), farms that dipped (triangles and dashed trendline), and farms that sprayed (squares and dotted trendline).**



In the final models, the interaction between installation and some design variables for BMSCC and ICSCC was significantly different from 0, meaning that there was a difference between the systems after installation of the ADF system on the ADF farm. Bulk milk SCC on farms that sprayed or dipped before installing an ADF system was 14,000 and 31,000 cells/ml lower in

the 12 mo period 6 mo after installation, respectively, than on farms that kept spraying or dipping the teats after milking (Table 1).

**Table 1 Modeled mean BMSCC per period and PMTD method**

PMTD method		Mean BMSCC		Difference
Before installation	After installation	Before installation	6 mo after installation	
Spray <sup>1</sup>	spray	199	201	2
Dip <sup>3</sup>	dip	184	183	-1
Spray	ADF <sup>2</sup>	206	192	-14
Dip	ADF	213	182	-31

Proportions of cows that had an elevated ICSCC were in the same periods compared with farms that sprayed or dipped 4.8% and 1.4% lower, respectively, after installation of an ADF system (Table 2).

**Table 2 Modeled proportion of elevated ICSCC (>200,000 cells/ml) per period and PMTD method**

PMTD method		Mean proportion of elevated ICSCC		Difference
Before installation	After installation	Before installation	6 mo after installation	
Spray <sup>1</sup>	spray	21.1	27.3	6.1
Dip <sup>3</sup>	dip	20.6	24.0	3.4
Spray	ADF <sup>2</sup>	23.5	24.7	1.3
Dip	ADF	22.9	24.8	2.0

## DISCUSSION

This study was to our knowledge the first field study measuring the effect of an ADF system on udder health parameters. Previous studies were time- and motion studies or experimental challenging studies within one farm. Bulk milk SCC and ICSCC decreased in herds that installed an ADF system compared with herds that did not.

One of the disadvantages of an ADF system seemed to be the poorer coverage of teats with disinfecting fluid. However, Hogewerf *et al.* (submitted) showed that coverage is not as good as manual dipping but still reasonable and that manually dipping is sometimes skipped in some cows by accident. One of the big advantages of implementing an ADF system is the increased efficiency of milking routine. As stated before, PMTD can take up to 10% of milking time (2). Another study, carried out in the UK showed a reduction in milking time following the installation of the ADF system (10).

The effect on SCC parameters of switching from spraying to ADF was larger than the effect of switching from dipping to ADF, and overall farms that dipped had lower BMSCC than farms that sprayed. The latter was more or less expected, because in order to apply effective teat coverage, PMTD using a sprayer is in general less effective than using a dip-cup (3).

In general, most farms that installed an ADF system had higher BMSCC before installation than the farms that did not install one. A reason for this could be that farmers that decided to install an ADF system were more motivated to change management to decrease the SCC on their farm and were therefore more willing to invest in an ADF system.

Matching farms in sets of 3 enabled the exclusion of seasonal effects, because the effect of installing an ADF system was measured relatively to 2 farms that did not install an ADF system (one farm used PMTD using a sprayer, and one farm using a dip-cup).

The effect of installing an ADF was not significant in the 12 mo period immediately after installation. In the 12 mo period starting 6 mo after installation, the effect of decreasing BMSCC of installing an ADF system was clearly there. This difference is likely to be due to 2 reasons: 1) the effect of a new system in a milking parlour will take some time for the milkers and cows to get used to, and the milking cluster is different than the cluster that was previously used; and 2) the ADF system reduces the new IMI rate and this will have only an effect after a period of time, because chronically infected (elevated SCC) cows will need to be cured, culled or dried off. Therefore, if the first few months after installation were omitted in the analyses, the effect was visible.

## CONCLUSION

Installing an ADF system decreased BMSCC, ICSCC and the proportion of newly elevated ICSCC. The effect was most prominent after approximately 6 mo after installation.

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## **HYGIENE AND MASTITIS CONTROL**

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### **SUMMARY**

The control of Somatic Cell Counts (SCCs), mastitis and Bactoscan results are seldom considered as a single concern.

On many farms where the control of mastitis is poor, hygiene is often given inadequate or varying degrees of priority. This is often down to the lack of knowledge, the perception that there is insufficient time or that prevention is too costly.

Giving priority to hygiene for all aspects of mastitis control will ultimately have a positive impact on reducing the challenge of bacteria gaining access to both the udder (thus causing mastitis) and the bulk milk supply.

### **INTRODUCTION**

This paper is the collation of data documenting JohnsonDiversey experience of farm hygiene across the country. The information has been gathered from the many hundreds of farm visits carried out by JohnsonDiversey each year and also includes information gleaned from the JohnsonDiversey State of the Nation Report 2006.

It has been produced as an overview of farmer's perceptions of hygienic milk production and its relationship to mastitis control and as an educational tool for effectively installing a strong hygiene routine on the unit in a timely and cost effective manner.

It is important to impress that this is not aimed at criticising the people involved with milking cows nor is it to preach, but as a means of raising questions and debate which will ultimately help and inform.

The simple fact is that; at farm level the hazards of bacteria in milk are associated typically with the Bactoscan results alone. Where results fall within the non-penalty bracket for milk payment, it is often thought that hygiene measures are satisfactory even when the level of clinical mastitis or cell counts may be at an undesirable level.

Very little has changed to the range of bacteria that cause udder infection on farm (predominately *S. aureus* and *S. uberis*) and despite the vast array of research, knowledge, advice and information available regarding the control

of mastitis, the incidence of clinical cases of mastitis is, reportedly on the increase.

It is suggested that the application of effective preventative measures on farm remains the key to achieving improved mastitis control, revolves around education, motivation and dedication to adopt sound sensible and consistent routines (Bradley *et al.*2007).

The fact remains that on many farms with SCC problems, mastitis control is poor, not because there is a failure to understand the reasons behind the required management regimes, such as the “5 Point Mastitis Control” programme, but simply because hygiene is given varying degrees of priority within these control procedures.

The factors which lead to the varying degrees of dedication to farm hygiene are often rooted in the belief that prevention is more costly than curing a problem which, the farmer believes, may or may not occur. According to research the total cost of clinical mastitis per case in dairy cows can vary widely from £150 to £1710, with the average total cost per case in the region of £177 (*Daisy Report No 5*).

Contagious and Environmental mastitis are terms that are often recognised and understood at farm level and are categories to which farmers can allocate their mastitis issues to in a bid to tackle their problems, but this can also create gaps in their control measures as they focus on controlling one pathogen rather than taking a complete and unified approach.

This paper will outline good hygiene practice regimes which should therefore, be the grounding to hygienic milk production and mastitis control.

## **DISCUSSION**

The control of Bactoscan and SCC results are interlinked, with hygiene being the lynch pin by which to reduce the levels of micro-organisms that can lay challenge to mastitis outbreaks and fluctuating levels in the bulk tank.

Mastitis is a multifaceted disease. Very rarely is an outbreak of mastitis in a herd down to one single event or reason. This said, a high proportion of mastitis today, has causes associated with the environment. Reducing the challenge of infection should be a top priority especially in herds with high levels of environmental mastitis, as the spread of infection at milking time becomes far more likely.

So, if mastitis is the inflammation of the udder and is caused by a wide range of different bacteria surviving in all areas of the cows’ environment then why is hygiene on farm not a higher priority for the farmer?

### **Why is hygiene important?**

- to produce a marketable product
- to maximise bonus revenue
- to prevent losses associated with mastitis

### **Why is Hygiene given varying degrees of priority?**

Some of the most common reasons are:

- Sheer ignorance, lack of understanding or lack of training
- Insufficient time or poor allocation of time
- Lack of staff, particularly during milking
- Perceived cost in time, labour, materials that outweigh the benefits

### **What is the opinion on improving hygiene at farm level?**

For many farmers the 'quick fix' approach to a problem is an easier option than addressing the hygiene issues associated with the problem. Changing teat and parlour cleaning products, post dip formulations, antibiotic treatment, removal of cows from the tank, farm employees, culling cows or even buying new gadgets, powders, lotions or potions are often considered, rather than taking an objective look at operations, particularly during milking.

Although changes to the farm routines may increase milking time initially, it will ultimately have a positive impact on the reduction of bacteria in the milk and on the incidence of mastitis within the herd.

### **Farm level myths about mastitis control and hygiene**

- Higher bulk tank somatic cell count can reduce acute mastitis
- It requires too much effort and this effort far outweighs the financial benefits that could be achieved
- Improving hygiene will extend milking times
- Improving hygiene will cost more money than can be recouped

### **What defines a good routine?**

For a farm hygiene routine to be effective, the most critical piece of information that should be retained is that; above all, the routine should be carried out consistently, with a systematic approach, by well trained staff.

Attention to detail and good housekeeping are necessary to maintain hygiene levels at an acceptable level and reduce the need for blitz operations in order to bring conditions back under control.

Putting measures in place to assess and maintain correct procedures involving hygiene and mastitis control and by keeping comprehensive records to identify achievements and highlight areas requiring further development is critical for any farming unit.

The choice of products used to clean and disinfect on farm should be selected to compliment sound, effective routines and not as the quick fix answer to a long term problem.

## **APPROACHING MASTITIS CONTROL WITH HYGIENE**

The “5 Point Mastitis Control Plan” should always be the basis to any mastitis control programme whether the incidence of mastitis is related to contagious or environmental infection or clinical or sub-clinical cases. It is not without possibility that a herd with a high incidence of *S. uberis*, could spread the infection during milking time even though the source of the infection stems from the housing environment.

### **Personal Hygiene**

Working with soiled gloves or hands and soiled clothes is a sure sign of a poor understanding of the impact that hygiene has on the quality of the raw material being harvested and the transmission of udder disease.

There should be adequate provision for staff who are milking cows, to have access to clean toilet facilities and hand washing stations and for dedicated milking attire and protective gloves to be worn at all times whilst in close contact with the animals.

### **Milking Machine**

It goes without saying that the milking equipment should be serviced and tested regularly to maintain its ability to milk the cows correctly. Liners should be changed at the correct time and pipe work associated with the cluster kept free of splits and holes.

It would be fair to say that a parlour would have to be visually, very heavily soiled for there to be any real possibility of it affecting the incidence of mastitis within a herd. It will however, have a negative impact on the Bactoscan results. It should be noted though, that a parlour that is allowed to become heavily soiled inside or out, would suggest an element of neglect or lack of general hygiene measures especially at milking time and this could pave the way to inadequate mastitis control at milking.

The aim therefore should be clean equipment inside and out, before, during and after milking. The environment in which the parlour resides should also be clean and free of slurry, spilt or excess cake and pools of water in a bid to prevent fly and vermin contamination and bio-film formation.

## **Pre Milking Teat Preparation**

A good milking routine should be designed to produce bacterially clean dry teats and detect mastitis without the spread of infection from one cow to another and encourage speedy and full milk let down.

Pre-preparation should be carried out with clean disinfected hands and gloves, clean teat disinfectant equipment and individual towels, paper or wipes. The choice of preparation is wide and varied, but the use of a pre milking disinfectant followed by dry wiping is known to have the biggest impact on milk quality and will produce healthier, cleaner teat skin with a lower bacterial challenge.

## **Milk Harvesting**

Clusters should be clean before attachment to avoid contaminating the milker's hands and where the cluster becomes detached from the animal during milking the cluster should land, where ever possible, on a clean floor and be rinsed off before re-attachment.

Post milking cluster disinfection has, in the last few years, become an accepted practice either carried out after every cow has been milked (often in herds with a significant problem) or (more commonly) after a clinical case or known high cell count cow.

There are a number of automatic options available, but where manual practices are carried out it is necessary to renew the bucket solution on a regular basis to prevent recontamination and to maintain clean equipment used to deliver the solutions.

## **Post Milking Teat Disinfection**

Post milking teat disinfectant must be applied to the whole length of the teat, immediately after the cluster is removed from the cow so as to gain the maximum benefit from the product, in terms of emolliency and biocidal effect. The product should also be applied through clean equipment (i.e. dip cup) by clean hands.

The recommended window in which to apply a post-dip is within 4 seconds of the cluster being removed. This is often impossible to achieve by manual application, but making a conscious effort to apply the dip as swiftly as possible after cluster removal, is significantly more effective than waiting for the whole row of cows to finish milking before applying post teat disinfectant: by then it is simply too late to be properly effective.

## **Exiting the parlour**

Cows should leave the parlour via well scraped walkways into feeding and loafing areas where fresh food and clean plentiful drinking water is available with easy access. Where cows return to grass the same should apply to farm tracks and gate ways. Encouraging the cows to remain standing for *ca* 20 minutes after milking, in the scraped yards also reduces the challenge to the udder, posed by the environment, whilst the teat end is most vulnerable.

## **Housing**

Milked cows should return to clean, dry, well bedded housing which, in effect, then ensures clean cows returning at milking time. Dry cows require the same housing/bedding management treatment as the milking herd, if not better. The concept that the dry cows are highly susceptible to mastitis infection during the start and end of the dry period is quite often lost, forgotten or ignored at farm level.

The use of disinfectants and guidelines for bedding conditioners can be included in a management routine for all housing, but ultimately ventilation, bedding dryness and the reduction of slurry levels will keep cows cleaner and reduce the challenges from micro-organisms.

## **Parlour cleaning**

At the end of milking the parlour should be thoroughly cleaned both inside and out taking careful note of the water temperature and mechanical action throughout the parlour to ensure it meets the required standards. Correct chemical use is another key factor that can lead to poor parlour cleaning which in turn lead to deposits that can provide nutrients for bacterial growth.

## **Clusters**

For best results, clusters should be thoroughly manually scrubbed in hot foaming detergent water, rinsed and attached to clean jettors, ensuring that for the fixed jettors, there is no stagnant water left in the base. Unfortunately this is not a common practise as it is considered to take too long and be impractical with ACR's.

Normally clusters are attached to jettors prior to being volume washed, but this often leads to liner shells being clean on one side and not on the other thus leaving contamination for the next milking. This process is frequently carried out during circulation cleaning which will cool the circulating water and reduce its efficacy or it is carried out too late and the soiling is baked onto the hot surfaces. In this event, volume washing clusters wastes far more water than manual cleaning and it is not as efficient.

## CONCLUSION

Ultimately hygiene should be the basis to any regime designed to control the challenge of micro-organisms to the udder. Until this becomes a standard practice on all farms, be they large or small, the instances of mastitis will continue along the current, worrying trend.

Focusing on aspects of hygiene relating to the cows environment and to the process of removing milk from the udder at milking will have a positive impact on reducing the challenge of bacteria that can gain access to the udder or the bulk tank.

Introducing simple focussed hygiene measures will improve milk quality and bonus incomes available, reduce the cost of treating mastitis and the losses associated with clinical mastitis, boost yields and produce a healthy teat (and cow) and create a better atmosphere to work for better motivated staff.

## ACKNOWLEDGEMENTS

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## **THE TRUE COSTS OF MASTITIS**

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### **SUMMARY**

The widespread implementation of mastitis control programmes over many years, based on the five point plan, has resulted in a dramatic reduction in classic 'contagious' mastitis. It is no longer possible or appropriate, however, to deliver generic advice to a dairy unit and assume it will be cost effective for that individual farm. It is vital to understand causes of and reasons for mastitis on each farm, and in particular the cost benefit of different mastitis control measures. The purpose of this paper is to assess the current financial losses that are caused by clinical and sub-clinical mastitis in UK dairy herds and the cost effectiveness of mastitis control measures. A practical approach to assessing the cost benefits of mastitis control is described and is available to all mastitis advisors in the UK through the DairyCo Mastitis Control Plan.

### **INTRODUCTION**

Management of disease in dairy herds involves making decisions that improve the health and welfare of the cows on the unit. An important element of this process is to continually assess the cost effectiveness of the interventions available to the unit in question, i.e. the balance of potential benefits and costs have to be weighed. Unfortunately, contrary to the tendency to oversimplify this procedure, estimating both benefits and costs can be difficult for an individual unit. The purpose of this paper is to consider the costs of mastitis in UK dairy herds and to examine the difficulties of assessing whether interventions are likely to be cost effective on individual dairy units.

The paper will review and signpost some recent research on mastitis costs, provide an estimate of the current financial consequences of mastitis in UK dairy herds and discuss the importance of assessing return on investment when undertaking mastitis control.

### **WHY DOES MASTITIS COST?**

The costs of clinical and subclinical mastitis have been estimated on numerous occasions in many countries, for example [1, 2, 5, 10, 11, 13-17],

and an overview of the literature has recently been published as part of a research thesis [9]. The major components that make up financial losses associated with mastitis are:-

- Discarded milk due to treatment or changed secretion (net cost dependent on whether milk is fed to calves)
- Losses in milk production during current and subsequent lactations
- Loss of milk value due to penalty payments, for example because of changes in somatic cell count
- Culling
- Therapeutic costs
- Other veterinary costs
- Farm labour
- Cost of preventive measures
- Cost of diagnostic measures
- Knock-on effects of a case of mastitis (such as mastitis transmission, increased susceptibility to other disease and reduced fertility)

It is clear, however, that the magnitude of these costs vary a great deal between farms, depending on individual circumstances. In general we are interested in the cost of mastitis on an individual farm: the problem is that, all too often, 'average' values for mastitis costs are inappropriately used.

## **MASTITIS COSTS ON UK DAIRY FARMS**

### **Clinical Mastitis**

The problem with estimating and using an average cost of clinical mastitis is that it only applies to some theoretical average farm and this may be considerably different to the farm of interest, on which preventive measures are being considered. An average figure can be calculated from the useful framework previously reported [4] by incorporating current values for milk price, labour etc. However, to present 'average' data in a specific farm situation can be misleading to the point of causing erroneous decision making.

There are different ways to assess individual farm costs for clinical mastitis, but ultimately an estimate needs to be made for the cost components described above, for the individual unit. Some of these are straightforward (such as milk price) and some less so (such as the lost milk production following a case of mastitis). We present two methods to illustrate the probable variation in clinical mastitis costs for UK dairy herds.

### ***A simulation-based model of clinical mastitis costs***

By combining the estimated ranges of each cost (a reasonable assessment of the lowest and highest values), it is possible to approximate the variation in costs for clinical mastitis between farms. Essentially this approach looks at

all possible cost scenarios (i.e. all combinations of costs) on the basis that different farms could have different values for the cost components.

A (stochastic) simulation model was constructed to estimate costs of clinical mastitis on UK dairy farms, using the ranges of cost components shown in Table 1, and uniform distributions (the probability of each cost value between minimum and maximum being the same).

The simulation combined the costs of mild (clinical signs confined to the mammary gland), moderate (some systemic signs such as high temperature, listlessness and loss of appetite) and severe (toxic) cases. The proportion of each type of case will vary between farm and the possible ranges investigated were mild 60-100% of cases, moderate 0-30% cases and severe 0-10% cases (such that total cases = 100%).

Results of the simulation are given in Table 2. The ranges in costs are large, as would be expected between farms with different cost structures and that manage mastitis in different ways. The results can be compared to some real farm data, collected from approximately 150 farms as a part of the National Mastitis Control Plan (<http://mastitiscontrolplan.co.uk>) and shown in Figures 1 and 2. The range of clinical mastitis costs estimated from farm data are slightly larger than the simulation model, but of a similar order of magnitude.

It is probably no surprise to those involved in mastitis control on dairy farms that the range in financial losses caused by clinical mastitis is vast: The range is probably from less than 0.6ppl to greater than 6ppl of milk produced. This gives a massive difference in the room for investment in mastitis control on different farms and clearly demonstrates the importance of knowing farm specific costs when considering preventive strategies. Farm specific costs of clinical mastitis can be estimated using the DMCP full mastitis cost calculator, a tool freely available to trained plan users at (<http://mastitiscontrolplan.co.uk>).

**Table 1. Cost ranges used to explore between herd variation in the costs of clinical mastitis in UK dairy herds.**

<b>Cost Component</b>	<b>Range of Component for Mild Mastitis</b>	<b>Range of Component for Moderate Mastitis</b>	<b>Range of Component for Severe Mastitis</b>
<b>Milk price (ppl)</b>	21 - 29	21 - 29	21 - 29
<b>Feed and fertiliser per litre (ppl)</b>	3-10	3-10	3-10
<b>Net cull loss (£)</b>	1000 - 2000	1000 - 2000	1000 - 2000
<b>Vet hourly fee (£/hr)</b>	60 - 150	60 - 150	60 - 150
<b>Farm labour value (£/hr)</b>	5 - 20	5 - 20	5 - 20
<b>Cost of diagnostics (£ per test)</b>	5 - 10	5 - 10	5 - 10
<b>Quantity of lost future milk (litres)</b>	0 - 800	0 - 3000	2000 - whole lactation
<b>Milk discarded (litres)</b>	0 - 300	0 - 400	0 - 1000
<b>Probability of cull for a case</b>	0 - 0.10	0 - 0.25	0.10 - 0.95
<b>Treatment costs (£)</b>	1- 10	1 - 40	10 - 100
<b>Vet time required (hr)</b>	0 - 0	0.0 - 0.5	0.5 - 1.5
<b>Farm labour time (hr)</b>	0.1 - 0.3	0.1 - 0.5	0.5 - 5.0
<b>Knock on health and fertility costs (£/case)</b>	0 - 100	0 - 500	0 (if dies) - 1000
<b>Herd size</b>	70 - 500	70 - 500	70 - 500
<b>Mastitis rate on farm (cases per 100 cows/yr)</b>	20 - 120	20 - 120	20 - 120
<b>Annual yield (Litres / cow/yr)</b>	6000 - 10000	6000 - 10000	6000 - 10000

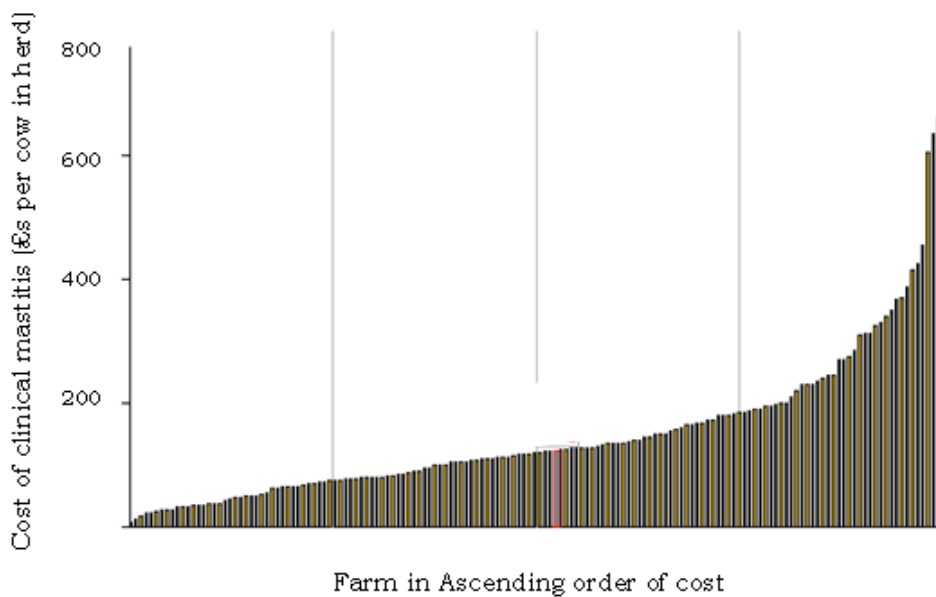
**Table 2. Results of the simulation model providing estimates of the range in costs of clinical mastitis for different farms.**

<b>Cost Element</b>	<b>Estimated range of cost between 'highest and lowest' farms</b>	<b>Central estimate</b>
<b>Cost of case of mild clinical mastitis (£)</b>	64 - 344	194
<b>Cost of case of moderate clinical mastitis (£)</b>	196 - 952	521
<b>Cost of case of severe clinical mastitis (£)</b>	843 - 2569	1603
<b>Average cost per case of clinical mastitis (for all severities) (£)</b>	149-515	313
<b>Cost of clinical mastitis per cow in the herd (£/yr)</b>	57 - 489	204
<b>Cost of clinical mastitis per litre of milk produced (ppl)</b>	0.7 – 6.6	2.6

**Figure 1. Cost of clinical mastitis (pence per litre of total milk production) estimated from real farm data collected as a part of the National Mastitis Control Scheme.**



**Figure 2. Cost of clinical mastitis (£s per cow in the herd per year) estimated from real farm data collected as a part of the National Mastitis Control Scheme.**



## **Subclinical Mastitis**

Estimating costs of subclinical mastitis are more difficult than clinical mastitis because it depends heavily on the bonus/penalty bands applicable to the herd and how close the bulk milk somatic cell count is to the SCC payment threshold. The risk and size of penalties influences the cost components described above, such as the probabilities of culling a high cell count cow, of treating infected cows or removing milk from the bulk tank. It is also difficult to be sure of the future lactation losses on a specific farm due to subclinical mastitis and this has been the subject of a variety of studies and meta-analyses [10, 15]. A value of 0.5 litres (~1.5%) loss per day for each twofold increase above 50,000 cells/ml or 100 litres (~1.5%) loss per lactation for each twofold increase above 50,000 cells/ml (lactation mean) has been reported for individual cows. However, making such calculations from individual cow records to provide a herd estimate is fairly time consuming!

A similar simulation model was constructed as described for clinical mastitis, to estimate likely variation in financial losses for subclinical mastitis for UK dairy farms. The cost components used for this simulation are shown in Table 3 and the results provided in Table 4. Again, as expected, results suggest a huge between herd variation in the costs of subclinical mastitis.

Individual farm data can be used to estimate the costs of subclinical mastitis again using the DMCP full mastitis cost calculator, (<http://mastitiscontrolplan.co.uk>).

**Table 3. Cost ranges used to explore between herd variation in the costs of sub-clinical mastitis in UK dairy herds.**

<b>Cost Component</b>	<b>Range of Component for Sub-clinical Mastitis</b>
<b>Milk price (ppl)</b>	21 – 29
<b>Feed and fertiliser per litre (ppl)</b>	3-10
<b>Net cull loss (£)</b>	1000 - 2000
<b>Farm labour value (£/hr)</b>	5 - 20
<b>Cost of diagnostics (£ per test)</b>	5 – 10
<b>Quantity of lost future milk for cows affected (litres)</b>	0 - 300
<b>Milk discarded from each high SCC cow (litres)</b>	0 - 300
<b>Probability of cull for a case of subclinical mastitis</b>	0 – 0.30
<b>Cost of diagnostics (£ per test)</b>	0 - 10
<b>Knock on health and fertility costs (£/case)</b>	0 - 200
<b>Treatment costs (£)</b>	0- 50
<b>Farm labour time (hr)</b>	0 – 0.5
<b>Herd size</b>	70 - 500
<b>Cows affected with subclinical mastitis (% per year)</b>	10 - 40
<b>Annual yield (Litres / cow/yr)</b>	6000 - 10000

**Table 4. Results of the simulation model providing estimates of the range in costs of sub-clinical mastitis for different farms.**

<b>Cost Element</b>	<b>Estimated range of cost between ‘highest and lowest’ farms</b>	<b>Central estimate</b>
<b>Cost of case of sub- clinical mastitis (£)</b>	66 - 589	290
<b>Cost of sub-clinical mastitis (pence per litre)</b>	0.15 – 2.4	0.80
<b>Cost of sub-clinical mastitis (£s per cow in herd pa)</b>	12 - 184	64

## **RETRIEVABLE LOSSES**

Even when farm specific costs of clinical and subclinical mastitis have been estimated for a dairy unit, an assessment is required of how much of the loss is realistically retrievable. Clearly it will never be possible to reduce mastitis to zero and save all the losses caused by mastitis.

Mastitis literature as a whole reveals a vast deficiency in the area of controlled intervention studies (other than on therapeutic products) to quantitatively assess different management measures for mastitis control. Thus, whilst it is reasonably well established that a few measures are, on average, cost beneficial (e.g. post milking teat disinfection and dry cow therapy [3, 12, 18]), the differences in the cost benefit of most measures, in specific herd circumstances, remain poorly understood. Furthermore, because most economic studies deal with average outcomes over many herds, results can only be applied to an individual herd with great caution.

In particular, interventions that involve management of the environment are rarely the subject of good controlled research and thus have very little evidence of efficacy in specific farm conditions; this is frustrating for the mastitis advisor. One of very few intervention studies was conducted recently in the UK and found that implementation of a holistic control plan resulted in a reduction of clinical mastitis by 20% (and nearer to 30% for herds that fully complied with the plan [7]). Specific management measures associated with the dry period were identified as important protective factors for mastitis in early lactation [6, 8].

Since, in practice, we have to decide when to make management changes to improve mastitis control, an estimate of intervention efficacy and the likely net marginal benefit or minimum return on investment is essential, although difficult.

## **RISK AND MINIMUM RETURN ON INVESTMENT FOR MASTITIS CONTROL**

As indicated above, when estimating return on investments for mastitis control, the costs of clinical and subclinical mastitis can be estimated with reasonable accuracy at the farm level and the costs of the intervention itself should be also be fairly straightforward to evaluate. The greatest area of uncertainty is the impact that specific changes will have in a given situation. This uncertainty doesn't only arise from uncertainty about the effectiveness of the management change itself, but also from many other factors that may change over time (for example, climate, introduction of new pathogens, age structure of the herd, etc). These may not be in the control of the advisor or farmer. Such uncertainties are an inevitable part of practical mastitis control (which needs to be recognised) and handling this is an area of current research.

A minimum return on investment (i.e. the minimum financial benefit that should occur beyond the costs incurred), therefore, has a probability or risk attached to it in each situation (i.e. it is not certain). If an intervention cost is small and the potential benefit in reduced mastitis is large, then the probability of a good return on investment will be high. Clearly, more difficult decisions arise when intervention costs are high (such as capital investments) because in this case larger and more certain improvements in mastitis are needed to guarantee a return on investment.

Making an assessment of possible minimum return on investment is a critical part of mastitis control and yet is often overlooked. A key here is communication. It is tempting to overstate or overestimate the certainty about the efficacy of a mastitis control measure but for many good reasons the results may not live up to expectations. However, even in light of some uncertainty, we need a workable method for practical mastitis control.

### **A PRACTICAL APPROACH TO ADDRESSING MASTITIS CONTROL AND COST BENEFIT**

Despite the uncertainty in estimating farm mastitis costs and, to an even greater extent, the likely minimum return on investment in a specific situation, we still need to find a sound practical basis for mastitis control. This is possible, particularly if an iterative approach is used in which the farm patterns of mastitis and the results of mastitis control measures are closely monitored and scrutinized.

It was largely from such considerations that the National Mastitis Control Scheme (DairyCo Mastitis Control Plan (DMCP)) came to fruition. The plan provides a clear route to examine costs of mastitis and to weigh up the potential cost effectiveness of implementing a holistic plan. The plan is based on scientific literature [7], but is not “mastitis control by numbers” – that is, the plan requires sensible clinical judgements to be made to ensure it meets individual farm needs. Another key feature of the DMCP is that tools are provided for plan users specifically to make estimates on current farm mastitis costs, and also the possible benefits from implementing control measures.

The key steps to evaluate costs and benefits using the DMCP are as follows:-

- Conduct a thorough evaluation of the herd mastitis patterns and highlight the main areas where new infections are occurring
- Undertake a full assessment of the current costs of clinical and subclinical mastitis using the DMCP cost calculator.
- Carry out a farm visit to gather information to assess current mastitis control policies and to identify the key (usually up to 10) areas for improvement.

- Make an estimate of the likely reduction in mastitis from applying the control measures on the unit (this has been evaluated for the plan [7]) and, using the DMCP cost calculator, subsequent return on investment.
- Fully discuss the measures required, the possible cost benefit of implementing the measures and the perceived risks.
- After implementation of the control measures, monitor farm mastitis indices carefully (using both individual cow somatic cell counts and clinical mastitis) and report/discuss the results on a monthly or quarterly basis.
- Reassess and adjust the mastitis control measures in light of the changes identified in mastitis patterns on the farm.

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## **CONTROLLING MASTITIS IN PASTURE-BASED SYSTEMS**

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### **SUMMARY**

The characteristics of mastitis within pasture-based milk production systems and the management practices within those systems require targeted control programs, taking account of their largely seasonal and low cost nature. Control strategies have historically focused on the prevention of new infections and reducing the duration of infection, primarily through the use of antimicrobial therapies. Alternative strategies have been evaluated, including the use of teat sealants, enhanced host resistance by dietary manipulation and vaccination. The involvement of rural professionals in control programs is effective in improving milk quality, however, such involvement needs to take account of the management style and priorities of individual herdowners in order to maximise adoption of new strategies.

### **INTRODUCTION**

Mastitis remains a significant animal health, welfare and economic cost to all dairy industries. In the pasture-based systems used in New Zealand (NZ) it is estimated that mastitis is costing about NZ\$36/cow/annum or NZ\$144 million/annum across the dairy industry (33). The aim of this review is to examine the control strategies for mastitis in pasture-based systems, their effectiveness and the factors influencing their adoption by herd-owners.

### **CONTROL PLANS**

The '5 point plan' mastitis control strategy developed by the National Institute of Research in Dairying at Reading in United Kingdom (UK) during 1950s and 60s (44) has formed the basis of subsequent control plans internationally. The basis of this plan is that the prevalence of infection is related to the number of new infections over time (incidence rate) and the duration of infections. Thus prevalence can be reduced either by reducing the incidence rate of new infections and/or by reducing the duration of infection.

The '5 point plan' was introduced into the pasture-based systems of Australia in the 1970s which resulted in reduced prevalence of infection, incidence of clinical mastitis and the bulk tank somatic cell count (BTSCC; 23). Economic analysis found that such an approach was cost effective in such production systems (4). The programs were effective against *Streptococcus agalactiae* and *Staphylococcus aureus*, but *Streptococcus uberis* was little affected (43).

Regional mastitis control programs were instigated in NZ from the 1950s (7), the 5 point plan was introduced in the 1970s and was revised in the 1990s to form the basis of the Seasonal Approach to Managing Mastitis plan (SAMM plan; 61). This plan recognised the seasonal nature of the calving used in NZ and presented the control measures in a seasonal context. National prevalence and incidence data are missing, but bacteriological data suggest a significant change from predominantly contagious mastitis pathogens in the 1960s (8) to predominantly environmental pathogens more recently (37). However, despite early successes, the BTSCC has increased over the last 5 years in NZ. Hence, a re-examination of the technical content, marketing and approach to on-farm adoption is required to ensure that milk quality again improves under the NZ pasture-based system.

The Australian dairy industry started developing the Countdown Downunder mastitis control program in the late 1990s. This program took an industry-wide approach to mastitis control, and recognised the importance of an industry-wide base of agreed knowledge and a consistent approach to mastitis management by rural professionals. Evaluation of the program found that herdowners produced good initial management plans, but that ongoing monitoring and alteration of the plan with changed circumstances was lacking, so subsequent revision shifted the emphasis to a risk-management approach aimed at ensuring ongoing mastitis control (49).

## **CONTEXT OF MASTITIS CONTROL UNDER PASTURE-BASED SYSTEMS**

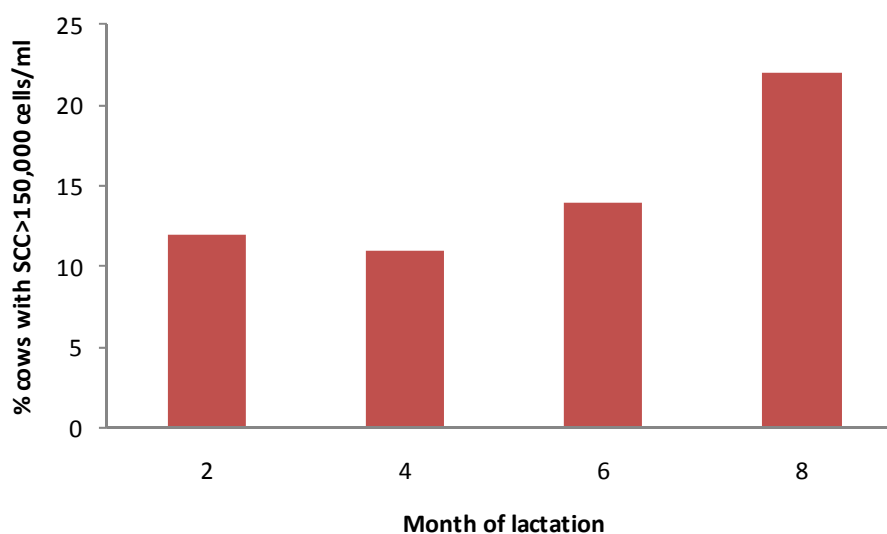
The epidemiology and economics of mastitis control are influenced by production systems. The seasonal pasture-based systems are characterised by relatively low nutritional and infrastructure inputs with limited use of concentrate feeds and no housing of cattle. The result is lower yields and potentially increased exposure to environmental mastitis pathogens as cattle move from pasture to the milking parlour. Additionally labour productivity is relatively high with one milk harvester per 150 cows. Pre-milking udder preparation is generally not done, potentially resulting in reduced sensitivity of detection of clinical mastitis, no stimulation of milk let down before application of the claw and increased risk of new environmental infections. One advantage of the seasonal production system is that all cows cease lactation on one or two calendar dates, allowing dry cow therapy to be applied to all cows simultaneously with subsequent removal from the milking system. This in turn reduces the incidence rate of new intramammary infections (IMI) and maximises cure proportion of existing IMI over the dry period.

New Zealand exports >95% of milk produced and herdowners are paid international market prices for milk. The relatively low returns on investment result in a strong focus on cost effectiveness of mastitis treatment and control programs by herdowners and veterinarians. Systems

that may be effective in one production system may not be biologically effective, cost effective or relevant in another (64). While this does not preclude use of management systems developed under different systems, such systems must be evaluated in the context of the production system to which they are being applied. Additionally, as every farm system is different in terms of financial situation, infrastructure, cow genetics, management systems, and skill of management and labour decision making about mastitis control programs needs to be at herd, not regional or national level (53).

## CURRENT PREVALENCE AND INCIDENCE OF MASTITIS IN NEW ZEALAND

The last national survey of the prevalence of IMI in NZ was performed in 1965/6, when the cow level prevalence of any IMI was 41% (8) and 'haemolytic Staphylococci' and Streptococci spp. were isolated from 41% and 33% of cows, respectively (14). No recent data are available on a national basis, but assuming that an individual somatic cell count (SCC) of >150,000 indicates IMI, the prevalence in the Waikato region is estimated to be between 11 and 22% depending on stage of lactation (Figure 1).



**Figure 1.** Estimated average herd prevalence of intramammary infection based on the percentage of cows within herd with a SCC >150,000 cells/ml at milk production recording (based on 388 dairy herds in the Waikato region of New Zealand in 2008/09 lactation; McDougall unpubl. data).

The lactational incidence of clinical mastitis was 13 cows/100 cows in a study of 28 herds from across NZ. More than 50% of cases occurred within 14 days of calving, the risk of clinical mastitis was higher in heifers and 9+ year old cows, and Friesians were more likely to have clinical mastitis than Jerseys or crossbreds. *Strep. uberis* was the most prevalent isolate from clinical cases of mastitis, but with an increasing proportion of *Staph. aureus*

later in lactation (37). Some herds from the Northland region were reported to have a high incidence of *Staph. aureus* throughout lactation (50).

## **CONTROL STRATEGIES**

A number of the key mastitis control strategies developed under more intense, housed production systems have been evaluated in NZ and/or Australia. The following section outlines the efficacy of some of these control systems within pasture-based production systems.

### **Preventing new infections**

The focus for preventing new infection has been on milking clean dry teats, including use of teat antiseptics and environmental management; effective maintenance of the milking machine, and use of dry cow therapy (or teat sealants) to prevent new infections and cure existing infections over the dry period.

Poor udder hygiene post-calving was associated with increased risk of subclinical mastitis (11), and density of animals on pasture, a proxy for increased risk of poor udder hygiene, was associated with increased risk of clinical mastitis in NZ (48). Anecdotally, use of small areas in which supplementary feeds are fed, or farm tracks on which mud or faeces pool, may increase incidence of clinical mastitis. High numbers of *Strep. uberis* are found on areas of the farm where there is a high density of cow traffic, for example the laneways leading to and from the dairy parlour (31). While not formally examined, it appears logical that improved cow hygiene via improved management of mud and faeces on the farm may reduce the incidence of mastitis in pasture-based systems.

Application of an iodine teat spray on the milking platform three times weekly pre-calving to primiparous cows grazed on pasture tended to reduce the prevalence of *Strep. uberis* on the teat-end one to two days before calving and significantly reduced the prevalence of IMI associated with *Strep. uberis* at the first milking after calving, although the overall incidence of clinical mastitis was not altered (32).

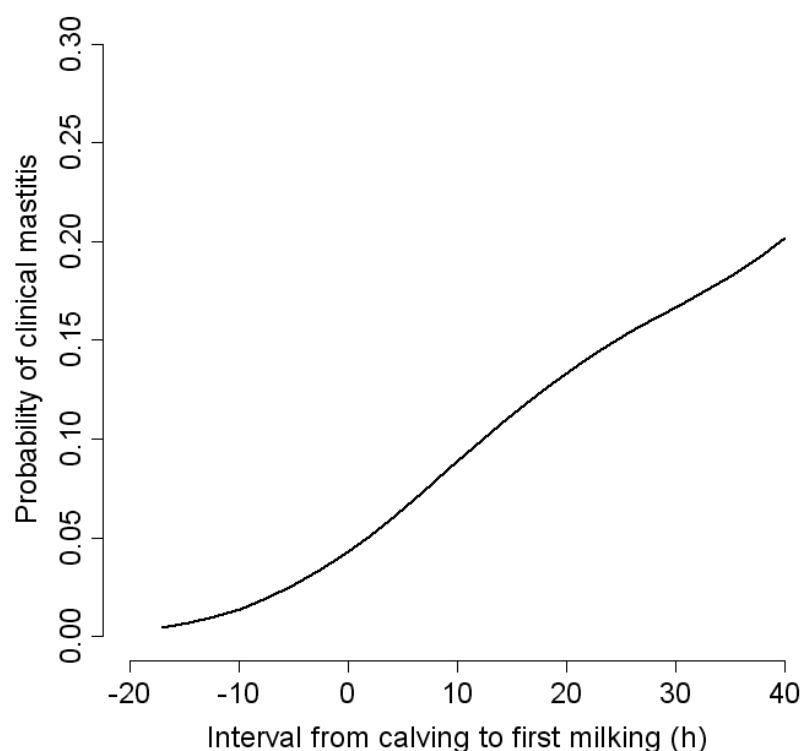
Poor milking function may increase the risk of mastitis via increased prevalence of teat hyperkeratosis or fomite transmission. However, the association between teat-end scores and pathogen specific risk of clinical mastitis are conflicting, with evidence from the UK that severe hyperkeratosis increased the risk of clinical mastitis associated with *E. coli*, while the risk of infection with *Strep. uberis* increased with smooth teat ends (6). One NZ survey suggested that only 9% of milking machines were compliant with ISO standards. This suggests that improvements in milking machine maintenance are required, and this may have beneficial effects in reducing mastitis incidence.

Dry cow therapy or use of teat sealants reduces the incidence of new infections over the dry period, especially those due to *Strep. uberis*. Treatment of bacteriologically negative quarters with dry cow therapy significantly reduced the incidence of clinical mastitis and new IMI rates over the dry period (60). Selective dry cow therapy is commonly used in NZ, but increasing the proportion of cows treated with dry cow therapy is associated with reduced risk of clinical mastitis over the dry period and reduced BTSCC in the subsequent lactation (35).

Teat sealant was found to be as effective as dry cow therapy alone in preventing new infections and there was no apparent benefit of a combination of dry cow therapy and teat sealant in one NZ study (62). However, a more recent study found that addition of an internal teat sealant reduced the risk of clinical mastitis in the next lactation by 0.63 relative to antibiotics alone (29). Additionally, infusion of a teat sealant into primiparous cows around 39 days before calving resulted in a 66% reduction in the incidence of new IMI, a 75% reduction in prevalence of IMI post calving, and a 74% reduction in the incidence of bacteria-positive clinical mastitis compared with no infusion (47).

Prompt milking of heifers after calving and avoidance of over-conditioning animals leaking milk pre-calving have been recommended as means of controlling mastitis (46). Early milking post-calving may reduce the risk of udder oedema and milk leakage which are known risk factors for clinical mastitis (55, 59). Prepartum milking of heifers has been shown to both reduce udder oedema and the incidence of clinical and subclinical mastitis (51). A NZ study demonstrated that reducing the interval between calving and first milking of primiparous cows by 10 hours reduced the risk of clinical mastitis post-calving by 45% and there was a linear increase in the incidence of clinical mastitis with increasing interval from calving to first milking (Figure 2; (10)).

Other strategies to minimise new infection include enhanced host resistance. This may be achieved by dietary manipulation to minimise the impacts of negative energy balance postpartum which has been associated with increased risk of disease, enhancing host immunity via genetic selection or by vaccination against specific pathogens.



**Figure 2.** Smoothed probability of cumulative incidence of clinical mastitis in dairy heifers within 21 d of calving by interval (h) between calving and first milking (from 10).

Dietary restriction at the time of drying off, which 86% of herdowners do in an attempt to reduce milk dripping after drying off, has been shown to increase the risk of clinical mastitis during the dry period and increase the BTSCC in the subsequent lactation (35). Many primiparous cows also lose body condition score (BCS) pre-calving indicating that they are in negative energy balance. Loss of >0.5 units of BCS is associated with an increased risk of udder oedema which is in turn associated with an increased risk of clinical mastitis in primiparous cows (11). Negative energy balance peripartum is associated with elevated concentrations of non-esterified fatty acids and depressed neutrophil function (20). Logically, management to minimise negative energy balance after calving should reduce peripartum disease, including mastitis. Feeding approximately 25% of the pre-calving diet as pasture hay, with the intention of modulating BCS and preventing excessive lactogenesis pre-calving, failed to reduce the prevalence and incidence of subclinical and clinical mastitis in primiparous dairy cows (10). In another study, feeding the ionophore monensin pre-calving to primiparous cows increased BCS at calving, but did not affect the prevalence of subclinical infection or the incidence of clinical mastitis (39). However, use of the ionophore lasalocid was associated with reduced incidence of clinical mastitis (41), a finding supported by a meta-analysis in which the relative risk of clinical mastitis was 0.91 for monensin treated cows compared with controls (13).

Many NZ soils have selenium levels below that required to achieve adequacy for grazing ruminants (17). Selenium supplementation of previously selenium-deficient animals reduced the incidence of clinical mastitis and reduced SCC (24). Selenium supplementation reduces the risk of retained foetal membranes in deficient cows (21) and cows with retained foetal membranes have greater risk of subsequent mastitis (19). However, there was no effect on mastitis prevalence or incidence following pre-calving selenium supplementation of primiparous cows where pre-treatment selenium concentrations were sufficient (10).

The inclusion of resistance to mastitis within breeding objectives is common internationally and currently log base 2 SCC is weighted at 6.7% in the NZ breeding objectives. The paucity of clinical mastitis records has generally meant that selection is based on individual cow SCC data. The correlation between the incidence of clinical mastitis and SCC is relatively high at about 0.7 (22). The heritability of SCC is relatively low (0.08 to 0.19), but sufficient variation and its relatively low cost mean that it can be used for selection (22).

Enhancement of immunological response by vaccination is an attractive proposition for mastitis control (30). This is especially true for environmental pathogens and for prepartum heifers where control strategies designed for contagious pathogens may be less effective. A number of vaccines have been developed and assessed in heifers. Vaccination with killed whole cell, live or subunit *Strep. uberis* vaccines reduced the severity and the duration of mastitis, the number of bacteria and the SCC compared with unvaccinated controls (reviewed by 30). However, field studies demonstrated no effect of killed vaccines on the prevalence of *Streptococcus spp.* (16). In one NZ study using a killed trivalent *Strep. uberis* vaccine, vaccinated cows (n=695) tended to have a lower prevalence of *Strep. uberis* in any gland within seven days of calving than not vaccinated cows (n=711; 0.12 vs. 0.16, p = 0.06). However, there was no difference in the prevalence of *Strep. uberis* at gland level (0.043 vs. 0.055) or the incidence of clinical mastitis associated with *Strep. uberis* at either cow or gland level (McDougall unpubl. data). Recently it has been proposed that the optimal vaccine to control *Strep. uberis* should target the T-cell response, using a sub-unit vaccine with a specific adjuvant that will promote T-cell entry into the udder (12).

### **Reducing the duration of existing infections**

Strategies commonly used to reduce duration of existing infections include antimicrobial treatment of subclinical and clinical mastitis cases either during lactation, or at the end of lactation, and removal (culling) of infected cows.

Parenteral treatment or intramammary infusion of antibiotics during lactation for therapy of clinical mastitis cases in NZ resulted in clinical and bacteriological cure rates of approximately 85% and 80%, respectively (34,

36, 37). The cure proportion was lower for glands from which *Staph. aureus* was isolated relative to other pathogens (26% vs. 85%; (36)).

Therapy of subclinical mastitis remains little studied in NZ. In one preliminary study, the bacteriological cure proportion of glands infected with *Staph. aureus* increased linearly with increasing duration (0, 3 or 6 tubes) of intramammary treatment with 250 mg of cefurozime (54). Similarly the cure proportion of subclinical IMI associated with a variety of pathogens increased with increasing number of daily I/M treatments of 5g of penethamate hydriodide ( $0.16\pm 0.04$ ,  $0.32\pm 0.06$  and  $0.56\pm 0.02$  following 0, 3 or 6 treatments, respectively;  $p < 0.001$ ; Nicole Steele, pers. comm.). However, the economics of treatment remain unclear; some international studies suggest that it is cost-effective to treat sub-clinically infected cows under some circumstances (57). Part of the economic benefit of treatment is the indirect effect of reducing the number of secondary infections by curing existing infections. Modelling suggests that in herds where there is a high rate of secondary infections treatment of subclinical cases may be of limited value (3). Those authors raise the point that the data required to assess whether a specific herd is likely to benefit from subclinical treatment is complex and expensive to obtain. There are no data to provide estimates of the rate of cow to cow transmission in pasture-based systems, hence the cost effectiveness of treatment of subclinical infections in these systems remains unclear.

Dry cow therapy cures between 65% and 100% of major Gram positive pathogens in pasture-based systems (*Staph. aureus*, *Strep uberis*, *Strep dysgalactiae* and *Strep agalactiae*) in Australasia (9, 60).

About 2.5% of cows are removed from dairy herds each lactation for reasons associated with mastitis in NZ (63). Moreover, mastitis increases the risk of cows being removed for fertility failure (38, 51). The economics of culling to control mastitis relative to the cost effectiveness of other strategies has not been examined under pasture-based systems. Modelling of mastitis control methods appears to have ignored the use of culling as control tool despite the fact that culling is an expensive decision.

## **IMPLEMENTATION AND ADOPTION OF CONTROL STRATEGIES**

### **Economic benefit of interventions**

A number of studies have demonstrated a positive cost-benefit of implementing mastitis control (1, 15, 52). The optimal program varies by pathogen with, for example, vaccination being an important component of *E. coli* management, while dry cow therapy and other preventive strategies are important for control of *Staph. aureus* (1).

## **Effect of involvement of rural professionals in mastitis control**

Herds belonging to owners that interacted with veterinarians trained in and having access to tools to improve milk quality subsequently had lower SCC. However, these herds initially had low SCC, suggesting that the herdowners were already interested in improving milk quality (28). Modelling suggests that rural professionals' prior beliefs will influence their expectation of the economic returns from a mastitis control program (18). Rural professionals are likely to differ in their expectations of the outcomes, and the degree of effort that they put into implementation is likely to be influenced by their prior beliefs. Just as herdowners vary in their motivations, beliefs and learning styles, so too do the rural professionals involved in implementing mastitis control plans. The implication is that technical and learning materials for rural professionals needs to meet a variety of learning styles, and it should not be assumed that veterinarians and their businesses are motivated only by profit or technical reasons.

## **Factors affecting adoption of mastitis control programs**

The motivators for a farmer to adopt on-farm change involve both internal (i.e. within farm) and external factors as well as monetary and non-monetary factors. Factors associated with the individual farm performance were more important than industry wide factors (e.g. external recognition), and financial penalties for poor milk quality were more effective than bonus systems (58).

Herdowners underestimate losses associated with mastitis, and if they were more aware of the true cost of mastitis they may be more motivated to improve milk quality (25). Qualitative research in NZ found that few farmers had accurate records of mastitis cases and little understanding of the costs of mastitis (42). The recognition that there was poor data quality, lack of a standardised way of calculating summary measures of mastitis and difficulties in accessing data led to the development of the mastitis focus report by Dairy Australia (49). Thus consistent reporting approaches and cost calculators that can be customised for a specific farm are important tools as part of mastitis control programs.

Farmers' attitudes are more closely correlated to disease incidence and production than to specific-biophysical measures (5). A 'quick and dirty' management style was found to be associated with an elevated BTSCC (2). This implies that unless attitudes and beliefs are changed, or the management style acknowledged, then biophysical changes in farm systems may not result in the expected improvements. A recent study in Denmark found that cow welfare and team work were more important to herdowners than production and profit, yet the veterinarians providing services to these farms were focused on production and profit (26). Paradoxically, although the veterinarians thought the farmers were interested in production and

profit, the farmers did not regard the veterinarians as knowledgeable on the economics of the farm business and would not invite the veterinarians to be on the 'farm board', if it existed. Clearly an understanding of farmer beliefs and needs is essential before any new production medicine/herd health program is instituted.

Understanding by rural professionals of the learning style and beliefs of farmers can improve the uptake and adoption of advice they give (45). While some herdowners need and want to understand the technical basis of advice before implementing it in their farm systems, others wish simply to be given clear guidelines without detailed background information, while others will adopt practices only after trying and modifying the technology to suit their farm system. Yet others may be characterised as 'hard to reach' by rural professionals, but this group are heterogeneous and require alternative ways of communication (56).

## **RESEARCH PRIORITIES**

A distinction can be made between research designed to increase underlying knowledge in some particular area of biology of mastitis and research aimed at increasing uptake and adoption of knowledge on farm.

*Streptococcus uberis* is the most common pathogen of clinical mastitis in pasture-based systems and control remains a challenge under such systems (30). Treatment of clinical and subclinical *Strep. uberis* infection is reasonably successful with >75% cure rate with both lactating and dry cow therapy. However, as the current control programs have limited effect on prevalence and incidence of infection with *Strep. uberis*, novel management strategies are required. Molecular epidemiology techniques are providing new information on the epidemiology of *Strep. uberis* (31, 40, 65). Further studies are required to convert these findings into practical control measures. Vaccination remains an attractive proposition, but there are still technical difficulties present suggesting that a cost effective vaccine is still some time away.

Where research and extension is aimed at optimising complex decision making on farm, it has been argued that quantitative research methods such as treatment *vs.* control research models result in incorrect inferences being drawn. This may be due to complex decision making by data collectors e.g. veterinarians using varying criteria on which to base diagnosis and treatment decisions. There may also be an interaction between rural professionals and herdowners whereby outcomes for the control group may be influenced by increased understanding and awareness by the rural professional, who is also involved with the treatment group. A partial solution is to use both quantitative and qualitative techniques, termed 'mixed model research' (27).

## CONCLUSIONS

Despite considerable research into mastitis epidemiology and treatment under pasture-based systems, further work is required. Current gaps include understanding the epidemiology of environmental pathogens, particularly *Strep. uberis*, so that better control methods can be implemented.

However, many herdowners can achieve good mastitis control as evidenced by high milk quality. Thus the barriers to mastitis control under pastoral (and other production) systems are not entirely technical. Recent research has highlighted that farmer beliefs and motivation are as important as technical knowledge in disease management. Thus understanding herdowner beliefs, goals and farm system is important in achieving on-farm improvements in milk quality. Rural professionals have historically tended to focus on technical and production related outcomes when working with herdowners, but to achieve real change their skill sets, focus and approach needs to evolve. Not all rural professionals are trained or interested in this broader approach to herd health, hence it is likely that such services will be provided by specialist rural professionals with interests and skills in this approach.

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## THE CORRELATION BETWEEN SOMATIC CELL COUNTS AROUND CALVING AND COW LONGEVITY

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### INTRODUCTION

Individual cow Somatic Cell Count (SCC) measures obtained from milk recordings before and after a dry period provide a ready means to assess and benchmark dry period performance. The Herd Companion program compares each cow's SCC measures immediately before and after the dry period against a threshold value (usually 200,000 cells/ml milk) as an indication of the likely infection status of the udder. This is either High (greater or equal to the threshold) or Low (below the threshold) resulting in four possible categories. These Herd Companion parameters are now widely used and understood within the industry:

- **High:High** : The SCC was above the threshold at both milk recordings immediately before and after the dry period suggesting infection at the end of the previous lactation and failure of a dry period cure.
- **High:Low** : The SCC was above the threshold at the last recording of the previous lactation but below at the first milk recording in the new lactation. This suggests infection at the end of the previous lactation followed by a dry period cure.
- **Low:High** : The SCC was below the threshold at the recording before drying off then above at the first recording in the new lactation. This suggests infection acquired in either the dry period or early lactation.
- **Low:Low** : The SCC was below the threshold before and after the dry period indicative of dry period protection.

### THE STUDY OBJECTIVE AND FINDINGS

The study investigated associations between the dry period status of a cow and the probability of re-calving in the subsequent lactation.

Using the milk recording database of National Milk Records (NMR) 59,198 cows from 2,586 randomly selected herds were studied. These cows all calved between October and December 2006 to start their second or higher lactation thus allowing classification of their previous dry period performance using the Herd Companion parameters described above. The data allowed follow-up of cows for up to three years giving sufficient time to either re-calve (survive) or leave the herd. The precise reasons for leaving the herd are not recorded although the great majority will be culls. Table 1 shows a marked variation in the re-calving percentages of cows in each dry period category.

**Table 1. The re-calving performance by Dry Period Category of cows calving in 2,586 herds during October-December 2006**

Dry period category	Calvings	Failed to recalve	% Failed to recalve	Chi <sup>2</sup> p-value for comparison with Low : Low
High : High	7,137	3,063	<b>43%</b>	<0.0001
High : Low	19,998	6,307	32%	<0.0001
Low : High	4,880	1,511	31%	<0.0001
Low : Low	27,183	6,379	<b>23%</b>	-
	59,198	17,260	29%	

Cows with raised SCC before and/or after the dry period have a significantly increased probability of failure to re-calve ( $p < 0.0001$ ) when compared to Low:Low category cows. The re-calving rates of cows in the Low:High and High:Low categories were not statistically significantly different from each other (Chi<sup>2</sup> p-value: 0.4378).

### **Re-calving performance in cows with moderately high SCCs (200,000 – 500,000 cells/ml)**

Many of the high SCCs were relatively modest. Of all cows in the study, 30% had raised SCCs before and/or after the dry period, but never exceeding 500,000 cells/ml. These “moderate” SCC levels should offer the best chance of cure, yet often pass unobserved with farmers and veterinarians distracted by animals with the highest SCC readings. Table 2 shows that these cows still have a significantly higher probability of failure to re-calve than Low:Low category cows. As in the full dataset, differences in re-calving rates of cows in Low:High and High:Low categories were not statistically significant (Chi<sup>2</sup> p-value: 0.6629).

**Table 2. The re-calving performance by Dry Period Category of cows with only moderately high SCCs (200-500,000cells/ml) at milk recordings immediately before and/or after calving in 2,586 herds during October-December 2006**

Dry period category	Calvings	Fail to recalve	% Fail to recalve	Chi <sup>2</sup> p-value for comparison with Low : Low
High : High	1,948	711	<b>36%</b>	<0.0001
Low : High	2,661	780	<b>29%</b>	<0.0001
High : Low	13,103	3,893	<b>30%</b>	<0.0001

### **Effect of lactation number**

While the percentage of cows failing to re-calve was lower when limited to cows from lactations 2 and 3, the same pattern persisted with all high SCC categories significantly less likely to re-calve than Low:Low cows. As in the full dataset, differences in re-calving rates of cows in Low:High and High:Low categories were not significantly significant (Chi<sup>2</sup> p-value: 0.2276).

## USE OF NOVEL TEAT DIP CUP

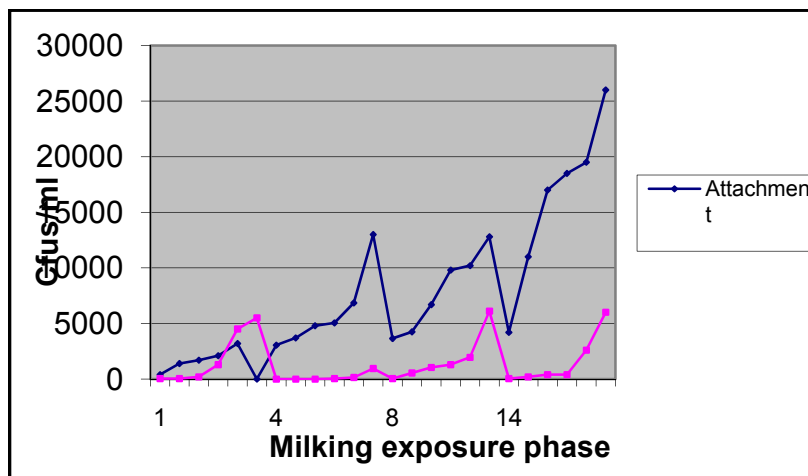
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Teat disinfection is a well-proven control measure, preventing up to 50% of new infections depending on the causal organisms. It is most efficacious against contagious organisms *Staphylococcus aureus* and *Streptococcus agalactiae* but dip cups can become contaminated with a variety of organisms both from the teat skin surface and from milk.

A novel teat dip cup with an attachment consisting of bristles which 'sweep' the teat as the teat dip cup is removed is available. This novel type was compared to a conventional non return teat dip cup type. Teat dip cups and any attachments were dipped in absolute alcohol for 5 minutes prior to each exposure period. Each dip cup type was used for one milking, four milkings (two days), eight milkings (four days) and 14 milkings (seven days). The teat dip cups were not washed in-between milkings, during the consecutive milking studies, unless visibly contaminated. The teat dip cups were used for post milking disinfection only, at routine milking at the IAH Mayfield Dairy unit. During the study period some cows were housed continuously and some cows were allowed access to pasture. A commercial iodine teat dip was used.

**Figure 1 - Total bacterial counts for the different teat dip cup types against milking exposure phase**

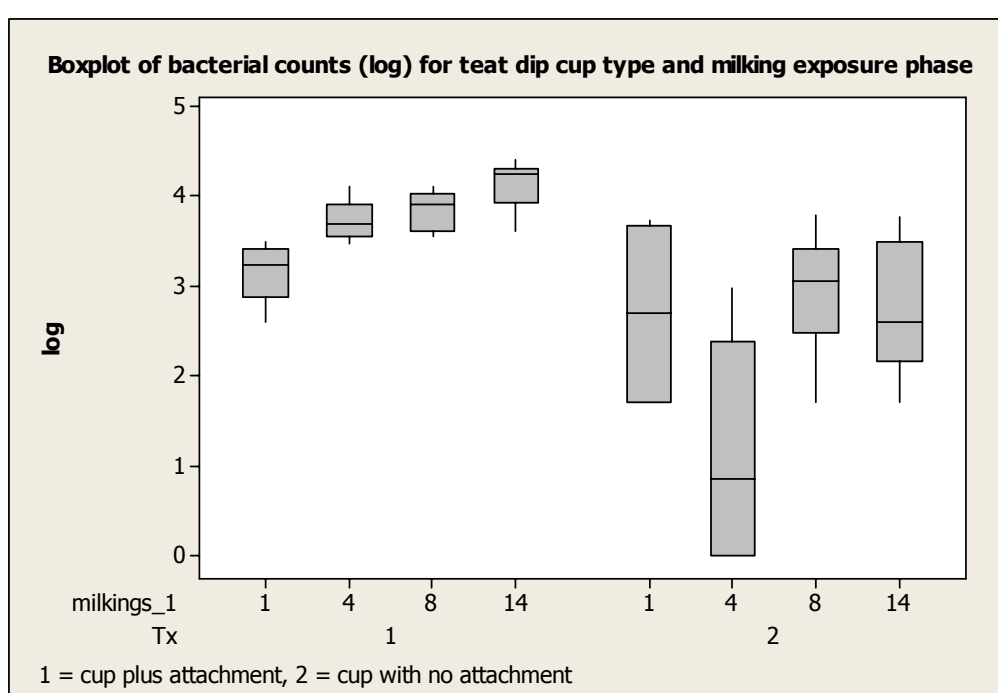


Bacterial numbers were determined after each exposure period (Figure 1). For the teat dip cup with attachment there was a consistent rise in bacterial counts with increasing milking exposure phase but this increase was not observed for the teat dip cup with no attachment. Counts for the teat cup

with attachment were a log count higher for the milking exposure phases 4, 8 and 14 milkings (but not for the one milking exposure phase)(Figure 2).

Treatment and milking exposure phase were both significant factors for bacterial count ( $F_{1,46} 41.94$   $p < 0.0001$  and  $F_{3,46} 5.26$   $p = 0.004$ ) but not for the one milking exposure phase ( $p=0.3$ ). Most of the bacterial counts were made up of *Bacillus* spp. (*Bacillus licheniformis* or *Bacillus subtilis*). These are spore formers and usually come from the environment on to the teats.

The bacteria isolated in this study were not common causes of mastitis, the potential for a build up of pathogens to occur is a risk. This study highlights the importance of being able to easily disinfect the teat dip cup attachment in order to minimise potential bacterial contamination of teats at milking.



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## **SUITABILITY OF DATA ON CLINICAL MASTITIS FROM UK DAIRY HERDS FOR USE IN GENETIC EVALUATIONS**

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Clinical mastitis (CM) and somatic cell count (SCC) are direct and indirect measures of udder health. SCC was first introduced into UK genetic evaluations in 1998. The udder health sub-index in the UK national profit index (£PLI) utilises the test day records for SCC as well as udder composite type trait, but does not, as yet, include a direct measure of mastitis. The level and accuracy of CM recording in the UK is less certain and therefore its usefulness for genetic evaluations is unknown. Inclusion of CM alongside SCC 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 National Milk Records Ltd (NMR) for a number of years. The study aim is to assess the suitability of NMR data on CM for UK dairy genetic evaluations.

The recording of CM events was performed by farmers on a voluntary basis as part of routine milk recording and the data was available from NMR, a milk recording organisation in the UK. Only herds participating with CM recording were included in this study. Animals affected with CM were recorded with the date of incident and these data were linked with calving and yield, including SCC, data. Clinical mastitis events that took place between the day of calving and 305 days after calving in the first five lactations were considered for cows calving between 2000 and 2008. The dataset comprised affected animals and their contemporaries of the same herd, calving year and lactation number. After data editing, there were 147,845 animals from 4,240 herds affected by mastitis at least once during the first five lactations, out of a total of 681,109 animals.

The number of animals affected at least once during lactation increased from 7,002 in 2000 to 33,518 in 2007, which, in part, reflects the increased number of herds actively recording clinical mastitis. The proportion of animals reported to be affected by CM each year increased from 6.7 % (2000) to 20.9 % (2008). There could be 2 reasons for this 1) a greater emphasis on CM recording in more recent years or 2) an increase in the overall level of mastitis. Increased recording of CM is most certainly due to the increased penetration of on-farm software, and 'event data' that includes CM, recorded by the farmer, is generally downloaded directly from the farm software to NMR central database. The percentage of animals affected with CM increased with increasing lactation number, and ranged from 10.72 % (lactation 1) to 22.15 % (lactation 5) (Table 1). Some animals had repeated CM events during lactations and across lactations. Animals with CM tend to have higher SCC as expected. SCC also increased with lactation number and the difference in SCC between affected and non-affected animals also increased with increasing lactation number (Table 2). Higher yielding

animals tend to be at higher risk of CM, with the largest milk yield difference of 337 kg between affected and non-affected animals observed for cows in the third lactation.

**Table 1** Number and percentage of mastitis animals recorded in the first five lactations.

Lactation No.	1	2	3	4	5
No. affected	40636	45292	43613	36017	26928
Total Count	378926	332524	261370	185839	121578
% affected	10.72	13.62	16.69	19.38	22.15

**Table 2:** Mean SCC (1000 cells/ml) and milk yield (kg) for affected (CM) and non-affected (NA) animals for the first five lactations

Trait / Lactation No.		1	2	3	4	5
SCC	CM	252	288	354	411	469
	NA	130	151	193	238	274
Milk Yield	CM	7174	8271	8531	8445	8212
	NA	6949	7934	8194	8113	7909

The numbers of animals affected by mastitis are similar to published estimates of 10.9% (1) and 10.1% (2) in heifers. Many authors have found an increased percentage of animals affected with mastitis with increasing parity (2, 3). Similar to these results, estimates of affected cows were 10%, 12% and 15% in Swedish Holsteins in parities one, two, and three, respectively, for the period 10 days before to 150 days after calving (1).

The trend that shows CM affected animals have higher SCC supports the use of SCC as a measure of CM for management purposes. The number of records available on CM incidence and the trends in the data being comparable with other studies indicate that CM could be used as a trait together with SCC to improve prediction of udder health in UK dairy cattle.

## ACKNOWLEDGEMENTS

Many thanks to the farmers and NMR for recording and supplying the data

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## DAIRYCO MASTITIS CONTROL PLAN

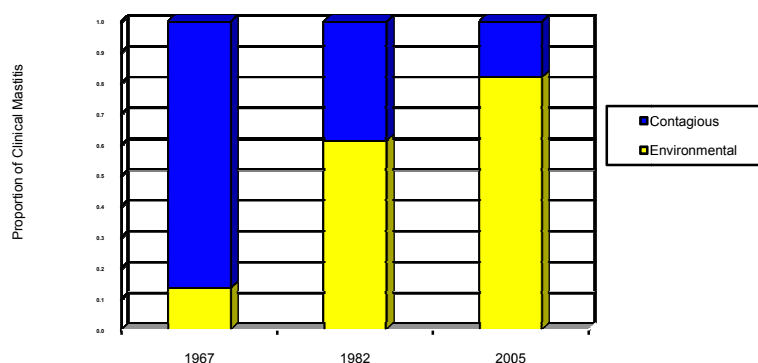
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Implementation of the Five Point Plan in the late 1960s along with other mastitis control strategies has led to a reduction in both the incidence and prevalence of clinical and subclinical mastitis in the UK. In addition, the last two decades have seen a dramatic change in the aetiology of bovine mastitis, with a dramatic shift away from the contagious mastitis pathogens towards those of primarily environmental aetiology.

In an attempt to address the difficulties faced by the modern dairy farmer, DairyCo funded research aimed at improving mastitis control in UK dairy herds. This research led to the development of the DairyCo Mastitis Control Plan.

**Figure 1:** An illustration of the change in the aetiology of bovine mastitis in the UK over the past 40 years.



Implementation of the Plan involves a detailed investigation and analysis of mastitis epidemiology, followed by targeting of control measures (from an extensive list) based on a hierarchical approach influenced by the patterns of disease. Importantly, **every** plan is farm specific with the aim of ensuring the best return on any investment in mastitis control.

Following this research and a successful pilot study, DairyCo have decided to roll out the mastitis control plan as part of a nationwide initiative to reduce mastitis. The aim of this initiative is to train Plan Users with a view to facilitating implementation of the plan on 750 farms over the next three years

and becoming self funding. Plan users attend two days of CPD. There are course notes along with an electronic version of the plan where plans can be archived, along with electronic mastitis cost calculators, mastitis treatment decision support tool, and access to the Quality Milk Manager software.

It is anticipated that with the enrolment of more farms, detailed national statistics will become available.

## **UNDERSTANDING MASTITIS EPIDEMIOLOGY ON INDIVIDUAL DAIRY UNITS**

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Over 50 years of implementation of mastitis control programs, in the developed world, has resulted in a dramatic reduction in sub-clinical and contagious mastitis and an increase in the diversity of mastitis aetiology and epidemiology. It is therefore no longer possible or appropriate to deliver 'generic' advice to dairy units and it is necessary to tailor the approach to mastitis control to the specific issues facing an individual unit. The first steps in tailoring advice are to accurately define mastitis patterns on a unit and to institute a system that allows detailed monitoring of disease. Key indices and intervention levels that are easily measured on farm or collated from existing records enable the practitioner to determine the relative importance of the dry period and lactation, and monitor the behaviour of pathogens in terms of persistence of infection.

A number of somatic cell count and clinical mastitis measures and indices have been developed by the authors (1 - 4) and incorporated into software (TotalVet, QMMS and SUM-IT). This software facilitates the rapid analysis, evaluation and monitoring of both the relative importance of the dry period and lactation on an individual unit, as well as characterising the apparent behaviour of the pathogens involved.

Individual cow somatic cell count data is analysed to allow the calculation of the rate of new infection in lactation, the rate of new infection during the dry period, dry period cure rates, clinical and sub-clinical mastitis cure rates in lactation, the interaction between the proportion of cows infected and chronically infected in the herd, the relative contribution of the dry period and the relative contribution from 'legacy' cows carrying infection from earlier lactations.

Clinical mastitis data is analysed to allow the calculation of both putative dry and lactating period clinical mastitis rates, based on a probabilistic assessment of the likely origin of infection; this also allows an estimation of the relative contribution of the dry period and lactation. In addition, rates of clinical mastitis recurrence, distribution of cases in lactation, by age as well as seasonal distribution can be rapidly assessed.

Within all of the calculated indices it is also possible to assess the impact of stage of lactation, parity, management group as well as allowing cohort

analysis of groups of problem cows.

To date several hundred herds both in the UK and overseas have been assessed using the methodology described. These analyses have demonstrated significant variation in the measured indices both between farms and within farms over time. For example, putative dry period origin clinical mastitis has been shown to account from <10% to >80% of all the clinical mastitis on an individual unit. Using SCC indices, lactation and dry period new infection rates have been shown to vary from <5% to >30% and <5% to >60% respectively. Clinical mastitis cure rates, as assessed by SCC response and lack of recurrence of clinical disease vary from <10% to >70%. As a general rule of thumb, if the relative contribution of the dry period is >33% more benefit is likely to be gained from intervening in the dry period than in lactation on the specific unit under consideration.

Importantly, investigation of correlations between SCC and clinical mastitis indices across a large number of herds has demonstrated that there is little or no correlation between clinical and sub-clinical disease.

Use of monthly cell count changes and strategic rates of clinical mastitis occurrence can improve mastitis definition on dairy farms. It is crucial to analyse and record both clinical and sub-clinical mastitis on a unit in order to get a true picture of the patterns of IMI in a herd; over-reliance on SCC or clinical mastitis alone for monitoring performance and targeting interventions is likely to lead to erroneous decisions in some cases. The approach outlined in this abstract facilitates implementation and allows targeting and monitoring of control strategies in evidence based approaches to mastitis control.

## **ACKNOWLEDGEMENTS**

The authors would like to acknowledge the expertise of the programmers at SUM-IT who developed the software to enable the analyses outlined in this abstract.

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## **THE ROLE OF BIOTIN CARBOXYL CARRIER PROTEIN (BCCP) IN THE INTERACTION OF *STREPTOCOCCUS UBERIS* WITH BOVINE PLASMINOGEN.**

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Bovine mastitis or inflammation of the bovine mammary gland is not only a significant welfare issue, but also one of the most economically important diseases in the dairy industry (1). *Streptococcus uberis*, one of the main causative organisms, is responsible for approximately 20% of all clinical cases in the UK (2). The ability of *S. uberis* to utilise host proteins as source of amino acids for growth has been postulated as a means by which *S. uberis* facilitates colonisation. *S. uberis* is able to convert bovine and ovine plasminogen to the active form, plasmin (3). The presence of plasmin can lead to the hydrolysis of casein, releasing amino acids and peptides required for growth (4).

Previous research on the interaction of *S. uberis* with bovine plasmin(ogen) putatively identified a novel candidate as receptor for bovine plasmin, the biotin carboxyl carrier protein (BCCP) (unpublished data). Preliminary data has characterised the receptor as a 25kDa protein with a preference for the active serine protease, plasmin. Since plasmin binding is postulated as a central virulence mechanism employed by *S. uberis*, evidence supporting BCCP as the plasmin receptor would implicate this in pathogenesis and as a potential target in the control of environmental mastitis.

The interaction between bovine plasmin and BCCP was investigated. BCCP was shown to be present in extracts from the capsule, cell wall & growth medium of *S. uberis* 0140J (wild type) and most prominently in the cell wall and media components of a mutant strain (hasA mutant) that lacked the ability to produce a hyaluronic acid capsule. However, purified anti-BCCP IgG was unable to block binding of plasmin to either strain.

This investigation confirmed that the BCCP is present on the outer surface of the bacterial cell but did not support the hypothesis that BCCP acts as a plasmin receptor in *S. uberis*.

### **ACKNOWLEDGMENTS**

This project was undertaken as part of the BVM BVS BVMedSci. I would like to thank SVMS University of Nottingham for financial support and the Institute of Animal Health for the use of their facilities during this study. I would also like to thank Professor J. Leigh and his research team for supervision of this project.

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## **INVESTIGATION OF THE RESISTANCE/SUSCEPTIBILITY OF *STREPTOCOCCUS UBERIS* TO LACTOFERRIN/LACTOFERRICIN.**

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*Streptococcus uberis* is an important mastitis pathogen; one of the most prevalent in today's dairy cow. A well-characterised protein, lactoferrin (Lf) and a peptide derivative of lactoferrin, lactoferricin B, both possess a range of antibacterial actions. The aim of this study was to investigate the antibacterial effects of lactoferrin and lactoferricin B on the growth of *S. uberis*. Overnight bacterial growth assays were carried out with a range of concentrations of both proteins, to assess their effects on bacterial growth.

No change in the growth of *S. uberis* was observed in the presence of lactoferrin at concentrations up to 20nM, demonstrating the resistance of *S. uberis* to the antibacterial action of Lf. However, similar molar concentrations of lactoferrin were able to completely inhibit growth of a *S. uberis* mutant strain lacking the protein MtuA. MtuA is a protein essential for high affinity assimilation of manganese from the surrounding medium. The data suggests that lactoferrin exerts an inhibitory effect by sequestration of manganese from the surrounding medium. This is not sufficient to prevent growth of *S. uberis* in the presence of the high affinity uptake system mediated through the actions of the ABC transporter (MtuABC).

In contrast, lactoferricin B caused complete growth inhibition of *S. uberis*. The data clearly reflects the difference in the mode of antibacterial action of lactoferrin and lactoferricin B.

### **ACKNOWLEDGEMENTS**

This project was undertaken as part of the BVM BVS BVMedSci degree. I would like to thank SVMS University of Nottingham for financial support and the Institute of Animal Health for the use of their facilities during this study. I would also like to thank Professor J. Leigh and his research team for supervision of this project.



## **USE OF TILMICOSIN (MICOTIL) AS AN ADJUNCT TO INTRAMAMMARY ANTIBIOTIC DRY COW THERAPY FOR THE TREATMENT OF INTRAMAMMARY INFECTIONS IN CATTLE**

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A retrospective survey of somatic cell count dynamics was carried out to investigate the benefits of using tilmicosin as an adjunct to intramammary dry cow therapy at, or shortly after, the time of dry off in cows with persistent or recurrent intramammary infections.

Tilmicosin (Micotil; Elanco) is a macrolide antibiotic, licensed for the treatment of pneumonia and lameness in sheep and cattle. Its ability to concentrate in polymorphoneutrophils has led to the suggestion that it may be of use in the treatment of intramammary *Staphylococcus aureus* infections. Parenteral administration of tilmicosin at the time of dry-off in herds with milk quality problems has become increasingly widespread since 1998, when a report demonstrated an 85% success rate in curing chronic *Staphylococcus aureus* infections alongside conventional intramammary dry cow antibiotic therapy (1). However, the product is currently not licensed for this use and there is very little data available on which to base prescribing advice. At the end of July 2006, Micotil became limited to vet use only, allowing the accurate identification of cows treated with Micotil to be possible.

Since July 2006 a total of 102 cows on 6 farms where full somatic cell count histories were available, had their identity recorded when the vet administered Micotil at, or shortly after, dry-off. The typical dose size used was 20cc (10mg/kg) subcutaneously.

Cows were defined as infected if their cell count was above 200,000 cells per ml at the last milk recording prior to dry off. In all herds milk recording was carried out on a monthly basis. The duration of infection was the number of months since the cow had two consecutive milk recordings with a cell count below 200,000 cells per ml.

Cows were defined as cured if the first two consecutive recordings were below 200,000 and if the first recording was above 200,000 and the two subsequent recordings were below 200,000.

Using these criteria, 89 of the 102 cows were defined as infected at dry-off. The remaining cows, while recording below 200,000 cells/ml at the last recording frequently had a long previous history of cell count milk recordings greater

than 200,000 cells/ml. No clinical records were examined but it may be presumed that others may have had a history of clinical mastitis.

21.3% of treated cows had a composite somatic cell count at last milk recording of over 1 million cells/ml. The average duration of infection was 3.8 months and the average parity at dry-off was 3.0 lactations.

63 treated cows were defined as being cured after the dry period. This figure represents 70.8% of cows infected at dry off and 60.2% of all cows treated. There was a wide farm to farm variation although no farm saw lower than 50% of all cows being cured.

The cure rate of all cows treated by parity ranged from 58% to 80%. There was no apparent relationship between success rate and parity at dry-off. Even the 13 cows ending their 6<sup>th</sup> lactation or older showed a 62% cure rate.

The cure rate of all cows treated by duration of infection ranged from 50% to 84%. There was no apparent relationship between success rate and duration of infection at dry-off. The 26 cows ending their lactation having been infected for 6 months or more showed a 62% cure rate.

Whilst it is recognised that there is no information regarding which pathogens may be involved and that monthly composite cell count data is a relatively blunt means of assessing infection status at the quarter level, the results indicate that there may be a benefit in targeting individual cows for Micotil treatment at the time of dry off.

It is possible that because the herds involved generally had poor milk quality performance, the infection pressure on cows that were cured may have been higher than in herds with better milk quality performance, leading to an underestimate of cure rates.

Further controlled studies on the use of Micotil as an adjunct to conventional intramammary antibiotic dry cow therapy are required to validate any advantage of using this combination over intramammary antibiotics alone. The relative cure rates and the possible cost-benefits of this use need to be established across a range of recognised udder pathogens.

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## **USE OF A FLOW DIAGRAM TO AID CONTROL OF STAPHYLOCOCCUS AUREUS MASTITIS- A CASE STUDY**

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An organic dairy herd consisting of 120 Holstein Friesian cows had a longstanding subclinical mastitis problem. *Staphylococcus aureus* had been identified as the main causative pathogen. Bulk Milk Somatic Cell Counts (BMSCC) had exceeded 550,000 cells per ml during the three month preceding the investigation and the rolling 12 month average was 480,000 cells per ml. Previous recommendations regarding milking routine had been implemented and no major flaws were noted in the current milking routine. The clusters are fitted with a back flush system. Annual production was around 7000 litres per cow. Annual costs associated with the subclinical mastitis problem were estimated to be between £30,000 and £40,000 a year.

The dairy herd is part of a large estate. During the investigation it became clear that differences between the various management layers and the herdsman plus relief milker were a major obstacle in managing the mastitis problem, in particular in reaching decisions relating to treatment and culling.

A flow diagram based on the literature (1-4) was constructed to provide guidelines to manage and control *Staphylococcus aureus* in the herd. Cows were grouped and milked according to perceived mastitis risk.

### **RESULTS**

- Rolling BMSCC has been reduced from almost 500,000 cells/ml to around 300,000 cell/ml.
- Culling rate increased short term from 22% to 32%; average parity has been reduced from 3.5 to 3.
- Number of chronically infected cows reduced from over 40% to under 15%.
- BMSCC of newly added Gloucester cattle bought in as heifers (now milked first) has remained around 50,000 cells/ml (assumed uninfected).

It is hoped that continued implementation of the control plan will reduce level of infection and BMSCC further in future.

The use of a flow diagram aided the management of BMSCC and level of *S.aureus* mastitis in this organic herd. Different economic and herd parameters would influence decisions in other herds.

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## **THE RELATIONSHIP BETWEEN HERD SOMATIC CELL COUNT AND HERD COMPANION INFECTION RATES**

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Somatic Cell Count (SCC) levels in milk have for many years been used as an indirect guide to the level of infection present in either the bulk milk sample (the whole herd indicator) the cow or even the individual quarters. The problem has often been one of how to use the huge variations in individual cow SCC recordings to best describe the epidemiology of mastitis infection in the herd.

The NMR Herd Companion Program analyses dynamic changes in SCC records from monthly milk recording data. Cows with SCCs above a threshold level of 200,000 cells/ml milk are allocated into four infection categories based on the trend between results at consecutive milk recordings. The word “infection” is used as a point of emphasis rather than confirming actual clinical infection in the cow. The four Herd Companion categories are:

- New infection – A SCC above the threshold for the first time in the current lactation, having had earlier milk recordings that were all below the threshold.
- First infection – The first milk recording in the current lactation AND with an SCC above the threshold
- Repeat infection – A cow that was below the threshold at the previous recording but has now returned to a SCC reading above the threshold, having had high SCC recordings at some point earlier in the current lactation.
- Chronic Infection – an SCC over the threshold at both the current and previous milk recordings in the current lactation

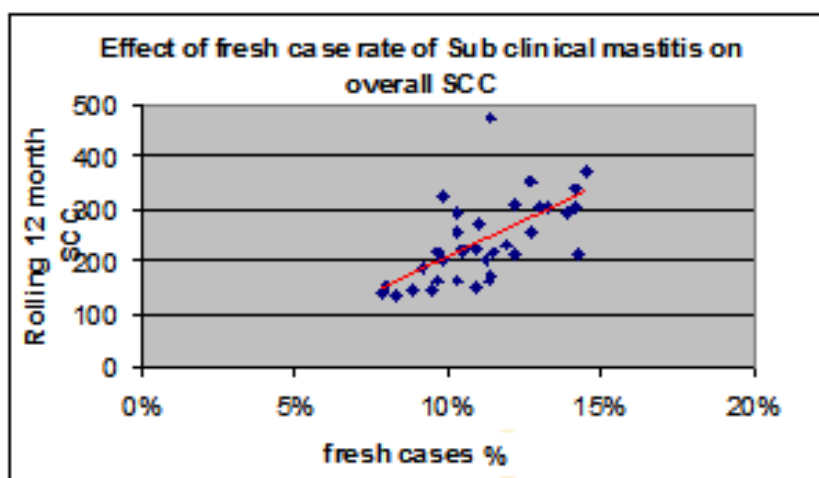
Using data from 36 client herds, these Herd Companion parameters were examined to look at the variation within each infection category and the level of correlation to overall herd indicators for infection, such as recorded herd SCC and percentage cows over 200,000 SCC. Annual rolling averages (of 12 monthly milk recordings) were used throughout to dampen down any monthly variation and to produce a representative herd value.

The parameters most strongly related to overall herd SCC were the level of Chronic Infections (Figure 1) and the percentage of cows over 200,000 SCC.

### Figure 1 Herd SCC against the level of Chronic infections

The generally narrow range in values for “New”, “First” and “Repeat” infections resulted in no clear correlation with overall Herd SCC. However, aggregating these in a new definition, “Fresh Infections” (New + First + Repeat), resulted in a clear correlation (Figure 2).

### Figure 2 Herd SCC against the level of Fresh infections (New + First + Repeat)



These findings are being extended to a much larger number of herds within the National Milk Records (NMR) database.

### CONCLUSION

Hugely variable individual SCC results require cows to be aggregated in to “epidemiological” groups that reflect possible infection trends. These broader definition groups based on the Herd Companion program SCC data have been used in our farm practice as part of a new initiative to make SCCs more use in understanding herd infection trends.

## **BACTOPROOF™: A REVOLUTIONARY NEW TESTING METHOD FOR MASTITIS**

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Mastitis is one of the most significant health and welfare problems in UK dairy herds. The full economic cost of mastitis is difficult to assess on either an individual cow or herd basis due to the multiple and complex losses involved but costs include lost milk, increased labour on farm, treatment cost, decreased fertility and increased culling. Mastitis also has a significant effect on milk quality and an EU limit of 400,000 cells/ml is imposed for milk destined for the liquid market. Purchasers often apply price penalties for somatic cell counts significantly below this level.

Although described as a single disease it is caused by an array of different pathogenic bacteria. While these have in the past been conveniently categorised as contagious or environmental pathogens some authors have begun to question whether the division is clear cut. There is no doubt that management of the herd has a substantial influence on the rates of mastitis and it is likely that genetic factors also influence the susceptibility to the disease.

For many years mastitis control has revolved around the 'five point plan'. This plan relied heavily on the use of antibiotics to treat cases and for the dry cow period. Without a clear bacteriological diagnosis this approach is not allowed in organic herds and is also questioned by those concerned over the development of resistance to antibiotics. More recent control plans have placed greater emphasis on obtaining evidence of the bacteriological cause of the mastitis as this is essential in identifying the critical management factors for the long term control of disease and also ensuring antibiotic treatment is both justified and targeted correctly to provide the most chance of a bacteriological cure.

Bactoproof™ is a new laboratory method to rapidly and accurately identify the cause of mastitis. It uses quantitative real time PCR to identify the bacteria involved. It has significant advantages over the existing method of identifying the cause of mastitis by culture.

- Speed:
  - 24-hour cycle from collection to reporting via text or web compared to a typical 4 days for conventional culture.
- Accuracy:
  - Precise identification of over 99% of mastitis pathogens and the only resistance gene of significance in antibiotic treatment selection.

- Yields results in many samples that would show ‘no growth’ in conventional culture.
- Test results reflect situation at sampling not after minor contaminants have grown in transit.
- Quantitative detection of pathogens including mixed infections, more information from one test.

In addition to the technical advantages described Bactoproof™ is available as a convenient kit which allows the farmer to take control of mastitis diagnosis.

- Ready to use on dairy shelf when mastitis identified.
- Simple collection and dispatch, no need to keep sample cold.
- Result available at a convenient time to farmer via internet or SMS.
- Farmer makes decision to test and receives result in 24 hours: can then discuss treatment and control with vet.
- Easy to understand results, no confusing jargon.

Bactoproof™ is a significant advance in mastitis diagnosis. Accurate and speedy diagnosis is the 1<sup>st</sup> step to effective management and treatment of any infectious disease. Use of this new test will reduce the time to effective evidence-based treatment, and hence damage to udder as well as reducing wasted milk. This test should be of significant benefit in any mastitis control programme and hence to dairy farming in the UK.

**Summary Table: Bacterial culture vs PCR in mastitis diagnostics**

Bacterial culture	Bactoproof Mastitis PCR Assay
72 hours in lab	8 hours in lab
No-growth in 25-40% of cases	Significantly less (>40% less) ‘no growth’ results
User experience may affect reliability	Objective results for all bacteria
Mainly qualitative	Qualitative and quantitative -can be used for hygiene monitoring and bulk tank milk testing
Unreliable during antibiotic treatment	Not affected by antimicrobials so can be used for following and studying treatment efficacy, reliable for repeated testing even if antibiotics have been/are being used
Prone to error due to bacterial growth during transportation	Bronopol-preserved milk eliminates major source of error.

## MILK PARTICULATES AND MASTITIS IN DAIRY COWS

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We have investigated the hypothesis that the particulate content of milk, as detected by a liquid particle counter, is related to health status of a lactating cow. Twenty one Holstein dairy cows were monitored from the first day of a clinical mastitis outbreak until complete recovery (monitoring for a minimum of twenty days). The controls for this experiment were two heifers monitored over the period of their first lactation. During the experiment, changes in all four quarters and the mix of milk from each of them were measured. For each sample, the following parameters were collected: somatic cell count (SCC), fat content, lactose, protein, and the number and size distribution of milk particles and milk fat globules (MFG), as described by Janik *et al.* (1).

This project began with the intention of using the raised SCC during infection as a marker for physiological condition and infection status. However it was quickly concluded that MFG constituted the most numerous population of particles in raw milk. Moreover it also became evident that the non-cellular particulate content of milk (including MFG) is a good indicator of mammary gland condition and their relative abundance made detection and measurement far more reliable and effective than SCC. This finding agreed with research work on behaviour of MFG during mastitis first published in the 1960s. It was found by King (2 and 3) that mastitis is associated with a decrease in the number and an increase in the average diameter of MFG. However this work did not progress and no further work was done on the physiological relationship between MFG populations and mastitis.

During our experimental work more than 2000 samples of foremilk were analysed from animals at varying stages of lactation and age, over different times of the year. We collected data from over thirty five outbreaks of clinical and sub-clinical mastitis.

The average total particle count in milk from healthy animals was found to be in the order of  $10^{11}$  to  $10^{13}$  particles per ml over the size range  $2\mu\text{m}$  to  $30\mu\text{m}$ . Our results suggested that the size distribution of particles changed significantly in all four quarters, even if only one was infected, during clinical as well as sub-clinical mastitis. Changes in the number of particles during mastitis were so great that they would not be expected to be seen in milk from healthy animals. The SCC, used to confirm presence of infection, increased only in milk from the infected quarters.

Changes in the particulate content of milk as well as in size distributions of MFG were seen up to three days before clinical signs of infection were present in 75% of cases.

The use of particle counting technology for the examination of MFG particles may allow a rapid analysis of the inflammatory condition of the udder and may also open an opportunity for diagnosis prior to clinical signs becoming evident. Moreover particle counting technology may be used to detect sub-clinical mastitis as there are currently no simple tools for detecting this condition.

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## **STAPHYLOCOCCUS AUREUS ISOLATES FROM A DAIRY FARM IN BEIJING: MOLECULAR TYPING, ANTIBIOTIC RESISTANCE AND TOXICITY TEST**

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Occurrence of antibiotic resistance in *Staphylococcus aureus* and also various molecular types of *Staphylococcus aureus* have been described [1]. The aim of the project was (a) to evaluate *S. aureus* molecular type diversity from one dairy farm, and (b) assess the variation in antibiotic resistance and toxicity among clinical mastitis *Staph. aureus* isolates.

This poster surveys clinical and subclinical mastitis using a combination of molecular- and microbiological methods were applied. Samples were obtained from clinical (n=15) and subclinical (n=19) mastitis cases as well as extra mammary sites (n=13) from one dairy farm in Beijing. Somatic Cell Counts (SCC) were conducted with a micro somatic cell counter [2] (Digital Bio Technology©, South Korea). Samples from affected quarters were plated for initial identification by colony morphology, selective medium growing and gram staining followed by an assessment of biochemical traits and 16SrRNA gene sequencing [3]. Multiple-locus variable-number tandem-repeat analysis (MLVA) for six loci were applied for molecular types discrimination [4]. Antibiotic resistance of *S. aureus* from clinical mastitis was assessed by detection of minimal inhibitory concentration (MIC). Toxicity was assessed by challenging mice (n=30) with  $10^8$  CFU/ml of defined *S. aureus* molecular type.

MLVA showed that the isolates from this dairy farm belong to a single molecular type. Challenged mice with *S. aureus* isolates differed, with lethality rates ranging from 0-80%. Additionally, antibiotic resistance varied in resistance from penicillin (86.7%), erythromycin (50.0%), clindamycin (6.7%) to tobramycine (6.7%).

We discuss these findings at one time point for the herd and then following pathogenic transmission within the herd. Changes in antibiotic resistance could be interpreted with an 'adaptive radiation' caused by previous treatment of individual cows. Based on these findings we propose a further study on virulence genes.

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## **NATIONAL MASTITIS SURVEY**

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No written paper has been submitted.