

BRITISH MASTITIS CONFERENCE 1998

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INTRODUCTION

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The Eleventh British Mastitis Conference is a measure of continuity of this event but hopefully not complacency in the need to continue seeking to improve mastitis control. The ever changing demands on dairying mean that we need new and revised ideas. The programme today includes two main areas which are always popular - treating mastitis and machine milking. The importance of both is increasingly obvious in an industry experiencing rapid changes in the demands for milk quality and animal welfare. We hope to broaden our knowledge today by inclusion of international speakers and opinion. All our speakers are authorities in their subject and will be extremely informative.

The session on mastitis therapy is wide ranging, from treatment of the whole animal to alternatives to antibiotics. The milking machine session reports on changing ideas on milking higher yielding cows and how the machine is used currently on farms. As usual there is the popular poster session, becoming increasingly more detailed and scientific, and the session updating us on recent research.

The organisers hope that this wide ranging and very topical programme, based on the suggestions of last year's delegates, will satisfy many of the demands of today's audience. Those old friends of the BMC and those new are welcome to this unique forum, both to listen and to discuss mastitis and its control.

MASTITIS THERAPY

MASTITIS TREATMENT - A WELFARE ISSUE

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SUMMARY

The overall problems surrounding the needs for treatment, the aims of treatment and the business and political objectives in dealing with clinical mastitis in the dairy cow are discussed. This paper describes the current and prospective situation as a general introduction to a series of papers reporting on current approaches and different means of treating clinical mastitis. It is argued that the clear objective and obligation of all involved is to produce the best quality raw milk possible but that in doing so the priority for all involved, from producers to consumers, must be the well-being of the dairy cow. This is enshrined in the, now accepted, International Dairy Federation objectives for treating clinical mastitis.

INTRODUCTION

The incidence of clinical mastitis may be minimised by application of considerable effort but cases will still occur in any herd. These have to be dealt with for their own sake, the effect of the infection on milk quality and the consequences of increasing the prevalence of infection in the herd by failing to cure infections. In the short term, priorities for dealing with clinical cases may vary according to their impact but the bottom line must be how any case or episode of cases affects the individual milk production business. Increasingly this is about image and value where value is financial and political.

There is a growing pressure on the quality of means of milk production shown by tightening market requirements for milk quality, quality assurance schemes and, more lately, opposition to various technical developments not least application of antimicrobials in food production. The impact of changing social and political opinion on dairy production, especially udder health, has to be considered. This must include consideration of alternative methods but mostly should involve reconsideration of aims, methods and achievements to achieve practical and economic effectiveness.

TREATMENT OF CLINICAL CASES OF MASTITIS

The intentions in responding to the clinical case of mastitis can be varied and often will vary only with practical matters such as the severity of the problem, simply a few clots or a very sick animal? Strategically this requires more thought to achieve a consistent and effective response. Treatment may be applied to be effective at three levels

- treating disease, often a minimal approach to restoring milk quality - a 'traditional' approach
- treating the infection with the aim of curing the infection,
- treating the signs of disease and their subsequent effects on animal well-being.

Treating disease only may extend from simply ignoring a mild case which resolves rapidly but at a cost of another sub clinically infected quarter. More likely the same effect will be

achieved by use of an udder rub or a minimal course of antibiotics, either likely to resolve 90% cases but cure the infection in many fewer. Treating infections to achieve a bacteriological cure is much more difficult but can be achieved (1). It often requires treatment beyond the label recommendations of use of intra mammary antibiotics. Sales of intra mammary tubes in the UK are approximately double what would be required to treat every case of clinical mastitis with the average recommended dose of three tubes. This only serves to show how outdated and unrealistic are the existing recommendations. There is a growing realisation that effective treatment may also involve medication or physiological applications to ameliorate pain, inflammation, dehydration etc. (2,3,4). Such approaches are often varied in need and specific in requirement, some may only be effective in treating self limiting infections such as some coliform mastitis.

CLINICAL MASTITIS IN CONTEXT

There is fairly broad agreement that the incidence of clinical mastitis varies greatly but still averages 35-40 cases per 100 cows per year (5). It is uncertain if anyone includes summer mastitis or other dry cow cases in this total. However, given 40 cases per 100 cows and a national herd of 2.5M some 1,000,000 clinical cases occur each year. With each case costing £182-218 (6) the bill is approximately £200M annually. Considering the problem in context also involves the consequences of approaches to treatment.

Resolving the disease but not the infection leads to recurrence of clinical signs and/or contagious spread, increased prevalence of infection hence poor milk quality from higher Bactoscan and SCC, and increased risk of antibiotic contamination. It is necessary to consider that the 1M cases each year not only induce sales of 6.5M intra mammary tubes but also 6.5M tubes of dry cow products. Dry cow products were developed for two reasons, to prevent new infection in the dry period and to reduce the duration of existing infections. In the initial trials both aims were achieved with about 80% success (7). There is considerable international pressure to limit use of dry cow intra mammary antibiotics, they are seen as contributing to antibiotic resistance. The fairness of this needs to be considered separately. It is clear, however, that the need for such products and their ability to cure 80% existing infections is a measure of failure of treatment (and prevention) of disease during lactation.

The practicality of treating clinical mastitis as a disease has been that there may be 90% resolution of the disease but only 20-80% bacteriological cure (8,9) probably with half of the effect being due to a self cure. In all, a problem that takes as long to resolve if treated than if untreated (10). This return of milk to the bulk tank approach neglects much of any consideration of milk quality, a case of 'if it's white and runs downhill then its milk'.

REQUIRED STRATEGY

Mastitis control methods must now be directed to achieving the best milk quality defined by a low Bactoscan and SCC, and no antibiotic residues. This translates to a lower prevalence of sub clinical mastitis, fewer cases of contagious mastitis, and more diligence against possible increased milk withholding times after treatment. Alternatives include better prevention or other means of treatment. It is important to note the limitations of residue detection. Methods in use only measure inhibition of microbial growth. More refined means of detecting actual antibiotics are increasingly available and likely to be applied with obvious effects. There is

much current debate on ensuring minimising concentrations or complete absence of a whole variety of chemicals and bacteria from raw milk.

The implications of the drive for increasing milk quality are total bacteriological cure, 'SCC' cure and a low risk for milk contamination, actual or perceived. Low risk may mean less or better use of antibiotics or alternative treatments depending on the tests for residues in use. There is increasingly likely to be a conflict in achieving bacteriological and SCC cure by use of antibiotic treatment with residue limitation. It is unclear yet what the economic balance might be and this could be very important. Further pressure will come from political pressure on attempting to limit antibiotic use supposedly to prevent development of resistance.

RESISTANCE TO ANTIBIOTICS AND MASTITIS

It is highly probable that confusion and misapportioning of blame exists by including use of intra mammary antibiotics as contributing in any way to an increase in resistance to antibiotics in potential human pathogens when antibiotics are used for therapeutic or preventive medicine in dairy cows. There is no substantiation that typical strains of bacteria causing mastitis in the dairy cow are particularly pathogenic for man. Such bacterial species have few, if any, mechanisms for transfer of resistance to other bacteria, as occurs with gut pathogens (11). The limited evidence on resistance patterns in mastitis pathogens indicates a decrease in antibacterial resistance over recent years (12).

If there is any risk then it may come from inappropriate management and undefined use of combination treatments. This might include feeding of milk containing antibiotics to calves which gives direct exposure, to relatively low levels of antibiotic, of those enterococci and *Salmonella* species known to have developed antimicrobial resistance, and to be capable of transferring such resistance. It could be envisaged that parenteral treatment could also contribute to developing resistance under certain pharmacokinetic conditions. Intra mammary antibiotic stays in the udder and diffuses poorly to other tissues, it is excreted predominantly in milk. Parenteral antibiotics are formulated to disperse into and accumulate in diverse target tissues but they cannot distinguish infected tissues from non infected sites. Hence, these are as likely to accumulate in gut as in the mammary gland. This leads to unnecessary exposure of those same gut enterococci and *Salmonella* species to antibiotic. Risk can also be minimised by appropriate treatment. Coliform mastitis rarely requires antibiotic treatment to deal with the infection whereas *Streptococcus uberis*, which is very sensitive to appropriate intra mammary antibiotics, is extremely poor at transferring any genetic material.

WHY AND WHEN TO TREAT

The whole issue of treating mastitis raises for the milker, and the vet, a number of questions and concerns, some of which have been considered above. These are also a number of obligations to be taken into account.

It is necessary to consider obligations to

- the cow, to maintain its health and well-being
- the consumer via the buyer, to provide milk of the highest quality
- society, to work to the highest standards, sustaining longevity of techniques and conserving resources.

These matters are issues of debate within the International Dairy Federation (IDF) in an attempt to achieve consensus and understanding. The whole area of treating mastitis is being reviewed. There are widely different views and approaches eg in Denmark it is not recommended to use dry cow treatment. In many cases part of the problem can be misunderstanding. Last year it was made clear that the intention in Sweden is to use aggressive treatment, where treatment is necessary (13). Discussion and debate are to achieve mutual understanding and consensus on achieving healthy cows. The outcome has to be good quality raw milk having a minimal level of bacteria (Bactoscan as low as possible), low SCC, no residues and produced by a herd with as low a prevalence (number of infected quarters) as possible. Generally the UK is good at this as comparative international data show (Table 1). Indeed the UK appears to provide the cleanest milk around.

There are a huge variety of ways of maintaining milk quality and all have adherents. What is important is to reach the quality standard necessary and meet all obligations. This may include effective and resilient use of antibiotic treatment by early, appropriate and aggressive treatment (1,14). There may be other preferred methods (15, 16) and there may be additional targets including pain relief (4).

Table 1. Milk quality for selected countries

	Cell count (‘000 cells/ml)	Limit TBC (cells/ml)	Bacterial count Level	
			TBC (cells/ml)	Bactoscan (impulses/ml)
UK	~165	100,000		~15,000
Denmark	247	100,000	8,400	
Holland	190	100,000	1.4% > 100,000	
New Zealand	177		?	
Norway	124	80,000 1 st quality	1.5% > 80,000	
Switzerland	101	80,000 (200,000 Bactoscan)		4.9% > 200,000
USA	350	100,000	?	

In the international marketplace there is a rapid need to develop strategies to be effective, meet political concerns and consumer attitudes, and to be sustainable. The objectives for therapy we have in doing so have been summarised and agreed unanimously by the International Dairy Federation and its 35 member countries (17). They are

Objectives for therapy, applied to cases of clinical mastitis, in order of priority.

1. To cure clinical mastitis and ensure the most rapid limitation of pain and discomfort to the infected animal.
2. To eliminate infection, prevent recurrence of disease and limit spread of infection to uninfected quarters of the same animal and the rest of the herd.
3. To restore milk quality, making it free from bacteria and reducing the leukocyte content, as rapidly and completely as possible.
4. To minimise the impact of the disease, and curative methods, on food production methods and consumers.
5. To maximise the durability and maintain the effectiveness of therapeutic methods.

The basic tenet of all of this is that we have to treat cows rapidly and effectively to minimise suffering. Whether as owner or keeper of the animals, contracted to care for the animals or a consumer of their output, the well-being of the animal is the priority. This has to expand and will expand as an economic and moral need for the farmer too. Some mastitis is inevitable and where it occurs it must be treated with the most effective means possible. The obligations of all involved in caring for animals, producing milk and consuming products must include the acceptance that treating mastitis is primarily a welfare issue, and that other matters must be secondary.

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MAKING MASTITIS TREATMENT DECISIONS

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SUMMARY

Therapeutic efficacy of mastitis can result in frustrating clinical outcomes, depending on the causative pathogen, the severity and duration of infection, and expectations of the dairy producer. In herds that have controlled contagious mastitis, costs incurred from clinical mastitis likely account for the largest proportion of total herd mastitis costs, and in some instances, a majority of these losses results from therapeutic costs, especially discarded milk following treatment. Additionally, mastitis remains the primary cause of antibiotic use on dairy farms and violative drug residues in marketed milk in the U.S. Therefore, an approach to therapeutic decisions for mastitis must address the three fundamental "e"s : efficacy, economics, and evasion of drug residues. This paper will focus on mastitis treatment decision-making and promotes a perspective of treatment as an adjunct to immune function.

INTRODUCTION

Throughout the 1960s and 1970s a wide variety of antimicrobials became available as intramammary infusions for treatment of mastitis. Initial successes suggested 75% efficacy (cures) in both lactating and dry cow formulations. However, there has been growing skepticism that in some cases, we achieve far less than originally reported. Chronic intramammary infections caused by pathogens such as *Staphylococcus aureus* pose difficult therapeutic problems, and typical labelled dose regimens that provide antibacterial concentrations for 24 to 48 hours will not provide cures in many cases. Additionally, the major thrust of development of antimicrobials for treatment of mastitis has been directed against Gram-positive organisms, particularly staphylococci and streptococci. These continue to be important pathogens, however, many herds have seen the emergence of Gram-negative organisms as a substantial cause of mastitis losses, which are resistant to many current products.

Traditionally, the most significant economic losses of mastitis have been attributable to lost production resulting from inflammation. However, application of management practices that decrease the prevalence of contagious pathogens, has also altered the focus of mastitis control to environmental pathogens and economic losses that arise from clinical mastitis. Estimates on the cost of clinical mastitis are \$110/case and \$40 to \$50 per cow in herd per year (1,2). In a large Michigan herd using daily milk weight technology, discarded milk following treatment accounted for 70% of lost marketable milk (2), and in herds that do not have a judicious treatment program losses from discarded milk alone can exceed \$70 per cow in herd per year. Thus, there is increased awareness among producers of treatment-related costs, and the economic value of extensive antimicrobial therapy for mastitis.

In Michigan, mastitis remains the most frequent cause of antimicrobial use in dairy cows and accounts for 90% of occurrences when violative residues are detected in marketed milk. Considerable debate among the public and regulatory sector exists as to the dangers that

antimicrobials and other drugs in milk pose as a health risk. Nonetheless, whether the dangers are real or perceived, frequency of milk testing and regulatory control of drug use on dairy farms is increasing and likely to increase in complexity as time continues. Thus, because of accountability to dairy producers and the consuming public, we must address two other key issues other than efficacy. Therapy must be economically viable and must not increase risk of drug residues in marketed milk.

Although these guidelines serve as a basis for therapeutic decisions, the practical realities faced by farmers and health advisors such as veterinarians still require that cows with mastitis will be treated. Although simplified, a mastitis therapy protocol can be designed from two "on-farm" perspectives; what to do with acute, systemic mastitis cases, and the other is the problems associated with chronic or relatively mild clinical cases. The key variables that influence the approach to formulating a treatment protocol are drug selection, causative agent, and cow immune status.

PHARMACOKINETIC AND PHARMACODYNAMIC CONSIDERATIONS FOR MASTITIS TREATMENT DECISIONS

Antimicrobials have been the class of drugs most often advocated in clinical practice and research as a therapy for mastitis (3). This direction has seemingly offered the most potential because the purpose of these drugs is to inhibit the growth of, or kill infectious, pathogens. However, because of the limited therapeutic dosing of these drugs that is usually employed due to economic and residue avoidance concerns, we are often unable to maintain effective inhibitory concentrations. Our goal in selecting the best antimicrobial treatment regimen for an intramammary infection is simple, administer the drug at a dose and site that will allow accumulation in the mammary gland (pharmacokinetics), and identify the pathogen and minimal inhibitory concentration (susceptibility) so that we can maintain effective drug concentrations (pharmacodynamics). Although adequate for pathogens such as *Streptococcus agalactiae* and *dysgalactiae*, the minimal dose amount and frequency as suggested for many commercial intramammary preparations is inadequate from a pharmacokinetic and pharmacodynamic standpoint for more invasive pathogens such as *Streptococcus uberis* and *S. aureus*. Number of doses with intramammary preparations are usually maintained according to milking times of the cow. However, a more novel approach has been to apply parenteral (systemic) administration as an adjunct to intramammary therapy. Systemic use of antimicrobials has been successful for increasing cure rates for chronic *S. aureus* intramammary infections in dry cows and lactating cows in studies from Israel, Netherlands, and Louisiana, but not in dry cows in studies from Michigan and Louisiana (4,5,6,7). In these studies, antimicrobials such as fluorquinolones, macrolides, and tetracyclines were selected as good pharmacokinetic candidates because of good volume of distribution (lipophilic), relatively long half-life, and high bioavailability (low serum protein binding). Because of a high degree of resistance to antimicrobials in commercial intramammary products, systemic antimicrobial therapy for the treatment of acute Gram-negative mastitis has been attempted. Research from Alabama (8, intravenous ceftiofur, low bioavailability), Minnesota (9, intramuscular erythromycin, poor susceptibility; gentamicin, poor tissue distribution) and Finland (10, sulfatrimethoprim) have all realized poor therapeutic results with natural and experimental coliform infections. A recent Israeli study (11) with a fourth generation cephalosporin (cefquinome) with good tissue distribution and antibacterial

activity reported better clinical outcomes. Thus, it is important to identify the pathogen and apply sound pharmacologic principles to promote the best probability for therapeutic efficacy.

MASTITIS THERAPY DECISIONS: COW'S POINT OF VIEW

It is well established that cows that have concurrent metabolic disease, inadequate nutrition, or that are subjugated to stress, including calving, are more likely to be affected by infectious agents. Mastitis is no exception to this principle, and numerous studies have demonstrated the direct correlation between *in vitro* phagocyte function and clinical outcome of experimentally challenged cows. Anti-oxidant supplementation of dietary rations improves *in vitro* anti-bacterial function of neutrophils collected from supplemented cows and decreases incidence and severity of clinical mastitis (12,13). Conversely, alteration of neutrophil migration into the gland occurs near calving and results in more severe coliform infections (14). Neutrophil function in clearance of pathogens from the mammary gland is critical, however the understanding of immunology has increased so that we now know that the neutrophil is part of a larger symphony of immune effectors that includes macrophages, lymphocytes, immune modulators such as cytokines, inflammatory mediators, and acute phase reactants (3). As a primer of humoral immunity, use of core-antigen vaccines improves the clinical outcome of cows with Gram-negative mastitis. Thus, preventive measures to establish optimum cow immunity are desirable. However, cow immune response to even the best of preventive programs is highly variable. At the time of clinical mastitis treatment, our ability to manipulate immune function is limited, and learning to assess immune function, even on a crude scale may offer some insight to therapeutic success. This is based on the premise that no antibiotic can clear an infection without a functional immune system and learning to read what a cow's defences are telling us may help in deciding our options or at least expectations. We need to support basic research that will allow us to gain recognition of cows with impaired immunity that pose a higher risk of unsuccessful therapy. Mammary quarters with infections of longer duration, that more consistently shed pathogens over time, and from cows that have multiple quarters with infections are a poorer therapeutic risk (5,15). These are somewhat crude predictors of therapeutic success, but suggest that we should explore genetic markers in dairy cattle that may allow us in the future to identify potentially immune impaired breeding lines as well as target our therapeutic efforts and expectations of treated animals.

A particularly attractive alternative would be to develop new strategies of dry cow therapeutics. This is an opportunity that could best realize the three fundamental criteria of therapy. Cost to the producer, particularly from discarded milk should be minimal, risks of residues in milk are certainly reduced, and as the involuted gland is a more hostile environment to bacterial survival than a lactating gland, improved efficacy gained through synergy with the immune system should be possible. Initial attempts to accelerate involution or potentiate immune activity at the end of lactation, or in lactating cows have not demonstrated practical clinical success (16,17,18,19), but are well targeted considering the high incidence of new intramammary infections during the early dry period.

DECISIONS FOR THERAPY OF ACUTE MASTITIS

Acute, or systemic, mastitis is most often caused by coliform and other Gram-negative organisms (20). However, numerous other pathogens including Gram-positive cocci and mycotic organisms can all result in severe mastitis. The case can be life threatening to the cow, and is often accompanied by marked production loss. If survival occurs, affected cows often perform poorly and may undergo premature culling. From a cowside appraisal, treatment of these cases is a forced decision, i.e. treatment is indicated, if only to relieve the cow of systemic signs. Supportive care is usually indicated, and in the case of coliform mastitis may be the most beneficial component of the therapeutic regimen (3). The obvious basis for antimicrobial therapy is knowledge of the causative pathogen. However, this is not attainable for some hours after initial onset of case recognition, and thus the practical problem remains of basing treatment on best clinical guess. Epidemiological information on the herd such as previous bacteriology, patterns of stage of lactation, season, age, etc suggest our best alternatives. Nonetheless, we remain in the situation of prescribing treatment to best cover all possibilities. Typically, intramammary therapy to inhibit Gram-positive growth in addition to parenteral (systemic) antimicrobials that have broad spectrums of activity are administered. Pharmacokinetic principles as previously discussed apply to severe mastitis cases. Although the drug may be available for distribution in the mammary gland, maintaining effective MIC can be more difficult due to increased resistance of many of these organisms. Caution should be employed in extending therapy for cows that have demonstrated marked clinical improvement, especially for cases of coliform mastitis, as recovering cows affected by these organisms have cleared the infection, and generally do not need antimicrobials to complete recovery. Unnecessary extension of therapy in these instances results in increased discarded milk expense for the dairy producer and risk of antimicrobials in marketed milk.

DECISIONS FOR THERAPY OF CHRONIC MASTITIS

Many intramammary infections that are chronic or are observed as mild clinical cases offer a more voluntary approach to therapy. Elimination of infections can result in increased production and, in the case of contagious pathogens, remove the reservoir of infection for non-infected cows. However, many of these infections are of long duration, frequently recur with mild clinical mastitis despite previous therapy, and can add substantial costs and risks associated with treatment. Treatment of subclinical IMI in lactating cows, especially caused by pathogens other than *Str. agalactiae* is usually uneconomical. The foundation for decisions should be based on bacteriology and sound pharmacology. Given the slow onset of infection, identification of the pathogen should be performed before any extensive therapy is instituted. Drug distribution, although theoretically available in the mammary gland, may not be efficacious because of extensive fibrosis and micro-abscess formation in the gland. Finally, chronic cases offer us more leisure to determine the cow's immune status from a perspective of duration of infection, number quarters infected, and other variables as previously discussed.

CONCLUSION

Mastitis therapy has hit a critical impasse, dairy producers are demanding more accountability of economic consequences of mastitis therapy, consumers, and thus regulatory bodies, are elevating their perception of food safety. We must rethink our approach to mastitis therapy so that any new technologies will meet the criteria of efficacy, cost effectiveness, and decreased residue risks. Particularly in the case of more chronic infections, I propose that we maintain our interest in bacteriology and pharmacology, but increase our awareness of the cow's immune status as well. This may result in better realization of our therapeutic expectations.

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MASTITIS THERAPY ON FARM - KEEPING UP WITH THE MOVING GOAL POSTS

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SUMMARY

Attitudes to treating mastitis have changed in response to techniques of therapy and more recently in response to marketing needs for milk. This has led to a need to re-assess the types of treatment available and the aims of using any form of treatment. Different approaches are recommended for different conditions. Aggressive treatment may be used to tackle infections by *Streptococcus uberis* or *Staphylococcus aureus*, if they are likely to be persistent as disease or sub clinical mastitis. In future, the effectiveness of treatment should be improved by better detection and understanding of infection dynamics. However, prevention rather than treatment should remain the priority.

INTRODUCTION

Approaches to therapy on farm have evolved considerably from the pre-antimicrobial era where symptomatic treatments such as massage, use of embrocations and "stripping out" were all that was available. The discovery of antimicrobials heralded a new era and hopes were that mastitis would be eliminated. However the prevalence of infected quarters on any farm is a result of two factors, the *new infection rate* and the *duration of infection*. Both of these factors can be influenced by antimicrobial treatment, either during lactation or at drying off, and by management control strategies such as teat dipping, culling policy, keeping a well maintained milking machine and general levels of hygiene. Increasingly antimicrobial therapy (both milking and dry cow therapy) is matched to the pathogen profile on a farm. The financial penalties based on bulk milk somatic cell count (BMSCC), levied at a producer level, have increased over recent years and the more recent reduction in UK milk price has made these penalties of increasing significance to farm profitability. The ability to finely control individual cows cell counts is becoming of paramount importance. As a result effective and accurate early identification of infection, often by laboratory tests, the selection of specific antimicrobial therapy, again often based on laboratory tests, and the application of a specific treatment regime will become an important part of herd mastitis control. As an adjunct to treatment, appropriate advice on the relevant management control measures to be reviewed can be given in the light of the epidemiological knowledge of the predominant bacteria involved. This will help stop infection spreading to other uninfected cows in the herd. By using this approach both *duration of infection* and *new infection rate* are closely scrutinised.

TYPES OF "TREATMENT" AVAILABLE

Self cure

Self cure is perhaps more common than is often supposed. Despite the fact that the teat orifice is continually being challenged and penetrated by bacteria the cow's normal defence mechanisms make establishment of infection a relatively rare occurrence. It is also possible for infections to become established and then be eliminated by self cure at a later date. It is clear that on occasions things get better despite what we do rather than because of what we do.

Treatment during lactation

Historically, treatment during lactation involved regular "stripping out" of the infected quarter, application of various topical treatments and massage to encourage the "circulation". The advent of antimicrobials brought a "magic bullet" approach and drugs were developed with varying degrees of efficacy against the common mastitis causing pathogens.

It appears that the "normal" three tubes used for a treatment course has become part of mastitis history but it is difficult to find any reference to the logic by which this three tube approach was chosen. Initially one tube was infused and only repeated if necessary depending on the clinical response. Gradually it became common place for "data sheets" to suggest three tubes were infused at 24 hourly intervals (every other milking). Latterly the "data sheet treatment regimes" have tended to move towards three tubes infused at 12 hourly intervals (every milking). It is felt that more "aggressive" therapy in the early stages of infection (12 hourly tubing) gives a better chance of a bacteriological cure. This approach also benefits the producer by shortening the duration of treatment and thus reduces the amount of milk discarded during treatment and milk withhold period.

In an attempt to achieve clinical, bacteriological and cell count "cure" various treatment regimes have been employed. These types of treatment regimes are used where infections are known to be refractory to treatment and may have resulted in previous treatment failures, recurrent clinical cases or persistently elevated somatic cell counts. Any variation from the "data sheet recommendations" will result in voiding the validity of the drug companies published withhold period. It is important to note that even increasing the number of tubes infused during a course of treatment will mean that individual cows will have to either have a "standard" milk withhold period applied, currently one week, a withhold period calculated or their milk tested by a suitable "inhibitory substance test" prior to returning their milk to the bulk tank.

Self cure and prompt treatment during lactation predominantly affect the *duration of infection*, by eliminating the infection from the affected quarter. However by eliminating infections from mastitic quarters a *reduction in new infection rate* is also likely because, as there are fewer infected quarters present in the herd, the chance of spread of infection is reduced.

Treatment at drying off

Infusion of antibiotic in all quarters at drying off (Dry Cow Therapy) is one of the key points in the NIRD 5 point mastitis control plan and has been used successfully for almost 30 years. This treatment fulfils both of the most important criteria for disease control namely *reduction of the duration of existing infections* and *reduction of the new infection rate*. Antibiotic therapy at drying off is more likely to be successful at eliminating intra-mammary infections than antibiotic therapy during lactation.

Eliminating existing infections (reducing duration of infections) can be of importance with contagious infections such as staphylococci and streptococci. Many cows infected during lactation do not show signs immediately and in some instances may not have a clinical episode for the whole lactation. The infection remains sub-clinical. Some infections do not result in consistently elevated cell counts. Protection of the dry udder (reducing new infection rate) can be of importance with infections such as *Streptococcus uberis* or those that cause summer mastitis. *Staphylococcus aureus* treatment efficacy declines as the cow increases in age so it is imperative to try and keep cows free from *S. aureus* for as long as possible. This coupled with the fact that cows are very susceptible to new infections during the first two weeks of the dry period, especially to *Str. uberis*, means that it is advisable that all cows in the herd receive dry cow therapy. Unfortunately the cow is also very susceptible to infections during the last two weeks of the dry period but to ensure milk produced after calving is free from antibiotic residues protection can not be afforded right up to the point of calving.

Culling

Removal of chronically infected cows is also one of the points in the NIRD 5 point mastitis control plan. Culling predominantly affects the *duration of infection* by eliminating the cow and thus the infection from the herd. However, by removing infected cows from the herd, the new infection rate is likely to be reduced because the chance of spread of infection is also reduced.

THE AIMS OF TREATMENT

This is the area where the goal posts have moved most. Take the statement "Early treatment is crucial to get the best chance of success". What meant by early and success can be considered to give some idea how things have changed.

Early can be taken as "at the first signs of abnormality". For clinical mastitis this would be visible changes (e.g. clots in the milk or a swollen quarter). For sub-clinical mastitis this would require laboratory tests. (e.g. elevation of somatic cell count SCC and or the isolation of mastitis causing bacteria)

The aims of successful treatment have evolved as the identification of mastitic cows has become more discriminate. Cows with a slightly elevated SCC, which only a few years ago would have been deemed to be normal, are now known to be sub-clinically infected. Success is no longer to make the milk visually normal. Elimination of causal bacteria and return of cell count to acceptable levels are essential if the long term aim of herd mastitis control, to

produce consistently milk to the high standards required by today's market place, is to be achieved. Even when a clinical cure has been achieved, "Post Treatment Checks" are an important part of monitoring whether treatment has been completely successful. These should involve bacteriological screening of milk, say one week after the milk is returned to production, and careful monitoring of individual cow SCC after treatment. Repeating bacteriological sampling may be necessary if the SCC response is transient or poor.

SELECTING COWS FOR TREATMENT

In addition to the "normal" new clinical infection which receives three tubes of antibiotic and is cured, there are two areas which justify special attention, persistently infected cows and sub clinical infections.

Persistently infected quarters - carrier cows

These cows are infected with a chronic long term infection. Persistently elevated SCC or repeated isolation of the same pathogen despite treatment will help differentiate these cows from transiently infected cows, which may have either self cured or responded to treatment. Carrier status is most likely to be caused by *S. aureus* or *Str. uberis*. Other streptococci such as *Streptococcus dysgalactiae* or *Streptococcus agalactiae* can be relatively easily eliminated from individual infected quarters with antibiotic therapy. *Str. agalactiae* can also be eradicated from a herd as it is virtually an obligate parasite of the bovine mammary gland, whereas most other mastitis pathogens have other reservoir sources and so herd eradication is not an achievable goal. However these carrier cows pose a significant infection risk to the rest of the herd and as culling is the only alternative they will often justify the increased expense of a specifically tailored treatment regime.

These regimes are either a form of "Extended Therapy", where treatment is prolonged in an attempt to improve success rates, or "Combination Therapy", where parenteral therapy (usually by injection) is combined with intra-mammary tubes to improve the penetration of the udder tissue and improve success rates.

S. aureus

If a new *S. aureus* infection is not eliminated, the bacteria may penetrate the mammary gland tissues inducing a response involving walling off the area and forming scar tissue. These areas of scar tissue are difficult for drugs to penetrate to an effective concentration. The invading bacteria may also avoid the killing effects of most, if not all, antibiotics by surviving within the white blood cells (neutrophils). Many organisms become inactive but are not killed by the neutrophil or even by antibiotics which are reported to penetrate the neutrophil. It is thought that the bacteria "hide" within the mitochondria of the neutrophil (a structure within the cell) and thus are still inaccessible to the action of even these antibiotics. The bacteria may remain inactive in the neutrophil until the cell eventually dies, in infected tissue this may be within 5-7 days. They are then released to resume cell division and the infection process.

These factors make *S. aureus* a difficult infection to treat and are cause enough to result in the usual dismal bacteriological cure rates. It has been suggested that of the *S. aureus* infections which have been successfully clinically cured (the milk looks normal and there is no swelling

or heat of the udder) only one third (33%) will have been successfully bacteriological cured. Of more concern are the two thirds (66%) which are still infected and pose a risk to the rest of the herd.

Most specific regimes for *S. aureus* involve "Extended Therapy" in an attempt to prolong treatment beyond the life of the neutrophil and hopefully avoid this reason for treatment failure.

Str. uberis

Minimum inhibitory concentrations (MIC) of antibiotics may be four times higher for *Str. uberis* as compared to other streptococci which cause mastitis. This is further complicated by the fact that *Str. uberis* and other faecal streptococci such as *Streptococcus viridans* can be quite resistant to antibiotics as judged by *in vitro* testing. Other streptococci such as *Str. agalactiae* or *Str. dysgalactiae* are very sensitive to antibiotics and in particular to penicillin. Carrier cows are identified by repeat isolation of *Str. uberis*, in pure growth, from the affected quarter. These cows may be previous conventional treatment failures identified on "post treatment checks". When treating resilient cases of *Str. uberis*, aggressive therapy using penicillin, erythromycin or framomycin can be useful, especially when given parenterally both by intra-muscular injection and in an aqueous presentation infused in to the infected quarter prior to infusing a compatible intra-mammary tube. Prolonged treatment courses may also improve success rates. These forms of treatment obviously have implications for the milk withholding period to be applied and appropriate steps must be taken before the milk can be returned to the bulk tank. Post treatment sampling (say one week after the milk has returned to the bulk tank) is essential to monitor bacteriological success rates. These persistent *Str. uberis* infections may still fail to respond to even such intensive therapy, and have on several occasions resulted in the cow being culled. Also choice of the most appropriate dry cow therapy by antibiotic sensitivity testing is essential to afford the best protection of the dry udder and to eliminate infections present at drying off.

Sub-clinical infections

The new *clinical* infection rate, if good records are available, is easy to calculate and is based on no previous clinical case being reported for in that quarter. However identifying a new *sub-clinical* infection accurately is more of a problem. Realistically, to be applied practically, it can only be based on available data. Individual cow SCC are available on many farms and can give a good insight in to the apparent new sub-clinical infection rate on a farm. As SCC can fluctuate almost daily, accurate identification of new sub-clinical infections is not possible when using monthly composite sampling. However as the limitations of monthly recording remain constant the trends can still be usefully monitored.

An apparent new sub-clinical infection may be identified in a cow with a previously low monthly SCC which has had an elevated SCC at the latest milk recording. The level of SCC indicating infection is obviously open to debate. A SCC of 200,000 cell/ml in composite milk is becoming increasingly used as the threshold for change of status from uninfected to infected. It is useful to consider cows with a previous SCC below 200,000 cells/ml and having a significant rise to over 200,000 cells/ml as newly infected.

Apparent new subclinical infection

The number of apparent new sub-clinical infections at each recording may be expressed as a proportion of the cows in milk. If this is expressed as a percentage experience suggests that 5 to 10 % would be acceptable. In fact as this is monitored on a herd basis, just counting the number each month is useful and any changes can be noted. On a practical note the easiest way to monitor the number of apparent new sub-clinical infections is to mark them with a highlighter pen on the cell count records each month and note the total for the herd. This gives an indication of how many cows are going from "clean" to "infected".

Apparent new subclinical infection rate

Taking this one stage further and "moving the goalposts" rather than "trying to keep up with moving goal posts", monitoring the new sub-clinical infection rate can give an indication as to the "background noise" of infection within a herd. If the number of new sub-clinical infections increases, is this an early warning for clinical cases? If nothing else it must identify that pathogens are increasingly being allowed to infect previously uninfected cows. Care must be taken interpreting the data, and samples from these new sub-clinically infected cows can be very revealing. By using epidemiological knowledge of the predominant bacteria involved, management changes can be made to reduce the chance of continued new infections within the herd. If new sub-clinical infections are predominantly caused by environmental pathogens then attention should be spent "outside the parlour" (cubicle hygiene etc). If contagious pathogens predominate then "cow to cow" spread is worthy of attention (parlour routine, teat dipping etc).

CONSIDERATIONS FOR THE FUTURE

Accurate early identification of infection is key. Although early treatment of any infection always gives a better chance of success, an open mind is essential when interpreting the results as treatment is likely to get the credit for all the "self cures". Clearly a compromise is needed. If monitoring and identification of the causal organism of apparent new sub-clinical infections is followed through to its logical conclusion much can be done in the prevention of infection rather than resorting to treatment. However, treatment of apparent new sub-clinical infections (identified by culturing milk from individual quarters from cows with a rise in cell count) may be justified in certain circumstances and will help eliminate the infections from these cows at an early stage. The decision making process has to take many factors into account to decide if, in this instance, treatment is to be undertaken (e.g. the type of pathogen involved, the number of similar SCC rises in the herd and risk of infection spreading to other cows in the herd, cost implications with reference to cost of treatment and discarded milk versus potential financial penalties for elevated BMSCC). Cows with a persistently elevated SCC can be considered similarly.

Assuming early detection is accurate and false positive diagnoses are few and rare then there are advantages from early treatment

- Improved welfare (mastitis is painful – even sub-clinical)
- Reduced chance of spread within the herd. (most significant with contagious pathogens)
- Lower production losses.
- Increased chance of clinical, bacteriological and cell count success.

However, there are potential disadvantages of early treatment

- Treatment of cases which will self cure.
- Treatment of false positive diagnoses

Monitoring in the future

Monitoring predominant pathogens in samples from cows with a persistently high SCC, and cows with a recent rise in SCC, will indicate the dynamics of the infection rate. Prevalence may thus be controlled more effectively and, as a result, bulk milk somatic cell counts will also be controlled more effectively. A clinical case of mastitis is best seen as a failure of the control mechanisms in place on the farm. They will occur, but hopefully not very often. Prevention will remain better than cure.

HOMOEOPATHIC MASTITIS CONTROL: A STUDY ON THE UPTAKE AND EFFICACY OF PRODUCTS IN THE REPUBLIC OF IRELAND

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SUMMARY

Over the past ten years there has been an increasing number of non-antibiotic treatments for mastitis, to treat clinical cases or to reduce somatic cell count available on the Irish market. More than 20% of farmers report having used homeopathic remedies for mastitis and almost half (43%) of the users claimed they had worked.

A number of Irish and international trials are reviewed and the results reported. No significant benefits were found from treating subclinical mastitis with homeopathic herbal products in three trials. Use of a homeopathic nosode to prevent mastitis, in a double blind trial, showed no significant difference between the number of new cases of clinical mastitis or the frequency of isolation of individual pathogens. The Irish trials indicate no benefits from treating animals with some of the commercially produced remedies on the market.

INTRODUCTION

Bulk milk somatic cell counts (SCC) in Irish dairy herds have fallen considerably over the past eight years. Dairy co-operatives have encouraged a greater uptake of mastitis control measures on farms and applied severe penalties on milk supplied with high SCC to ensure milk complies with the quality requirements of national and EU legislation. Individual monitoring of milk SCC levels in cows has allowed farmers to identify chronically infected animals for treatment or culling. While antibiotics have always played a central role in mastitis control on farms, more dairy farmers have become aware of their limited efficacy in controlling and preventing subclinical infections. Meaney (1) drew attention to the limited efficacy of intramammary antibiotic therapy and more recent reviews (2,3) have reappraised the role and efficacy of antibiotics in mastitis treatment.

Over the past ten years there has been an increasing number of non-antibiotic alternative remedies on the Irish market for the treatment of animal diseases. The growth in homeopathic remedies in veterinary medicine has to some extent paralleled that in human medicine. Mastitis would seem to be the animal disease most frequently targeted with many products available and some with specific claims for efficacy in reducing milk SCC. While manufacturers of these products may be able to present some case histories indicating the efficacy of their products there are no scientifically based independent studies to validate the claims made for products.

This paper reviews the results of a number of such studies undertaken on products in the Republic of Ireland

UPTAKE OF HOMOEOPATHY ON FARMS

A questionnaire survey detailing the uptake of homoeopathic mastitis treatments in the Republic of Ireland has recently been published (4). Questionnaires were completed on a total of 234 herds. The herds surveyed in the study were above the national average for herd size and were considered to be among the more progressive of farming groups in terms of farm management practices. Questionnaires were completed in the presence of the farmer by either veterinary surgeons or milk recording personnel visiting farms in the course of their work.

A total of 52 (22.2%) of the 234 farmers surveyed, responded that they had used homoeopathic mastitis control remedies in their herds. Only 34 of the farmers using homoeopathy named the product used in their herds. In 26 cases the products used were marketed as homoeopathic products by their manufacturers. In the other eight cases the manufacturer's classification was not known by the author. Although all the products were classified as homoeopathic by the farmers themselves, the products might have been more aptly described as alternative medicines. The methods of administration of products by the farmers is shown in Table 1. Oral administration was the most common route either by addition to the drinking water or directly through dosing.

Table 1. Route of administration of homoeopathic remedies in the herds surveyed

Route of administration	Number (%) of herds
Adding to drinking water	26 (50.0)
Parenterally	14 (27.0)
Oral dosing	3 (5.8)
Intra vaginally	3 (5.8)
Adding to feed	2 (3.8)
Other	2 (3.8)
Not stated	2 (3.8)

Mastitis was regarded as a problem by 19.2% of the respondents in herds using homoeopathy compared to 25.6% of respondents in other herds. The main reason given by the farmers for using homoeopathy was to reduce milk SCC (83%). Fourteen herds were using homoeopathy at the time they were surveyed and seven of these herds had been using homoeopathy for at least one year. When questioned if homoeopathy had worked in the herd, 21 (43.8%) of the 48 respondents replied that it had worked. Almost 83% of farmers surveyed had heard of homoeopathic mastitis control. Salesmen, other farmers and the farming press were the main sources of information on homoeopathy with only 7.3% of respondents stating veterinary surgeons as the source of information. The reasons given by 123 farmers for not using homoeopathy were; unfavourable reports (53.7%), too expensive (40%) and insufficient information (26.7%).

EFFICACY OF HOMOEOPATHIC HERBAL PRODUCTS

Three commercial herbal homoeopathic preparations marketed in Ireland have been evaluated over an eight year period under experimental conditions at the Central Veterinary Research Laboratory (CVRL), Abbotstown. All the preparations were recommended for use in herds to control mastitis and reduce high SCC. Two of the products were for oral administration and one for subcutaneous administration. A homoeopathic udder cream was supplied and recommended for use with one of the products (5).

The cows used in the experiments were generally in mid to late lactation and were selected from the dairy herd at the research station. A mastitis control programme operated in the herd and quarter milk samples were routinely tested for bacteria and milk SCC. The cows were selected on the basis that quarter milk samples collected prior to treatment had indicated that they were infected subclinically with *Staphylococcus aureus* or coagulase negative staphylococcal mastitis in at least one quarter. Sample collection and mastitis definitions were as outlined by the International Dairy Federation (6).

The results of the treatment are shown in Table 2. None of the preparations showed any significant efficacy in reducing infectious or non-specific subclinical mastitis.

Similar trials on two products have been conducted at the Teagasc Research Centre, Moorepark (7). Product A in their trial was similar to that used above at the CVRL, Abbotstown. In the first trial (7) using product A, 25 cows affected by subclinical mastitis were treated with an oral spray for 20 days and an homoeopathic cream applied to the udder after each milking for five days in 11 of the cows. Quarter milk samples were monitored for 22 days after treatment was completed. No significant benefits from treatment were observed (Table 2). In a second trial (7) using the same remedy modified to a higher potency (product D), 19 subclinically infected cows were treated and udder quarters monitored for 53 days after the treatment was completed. No significant benefits from treatment were observed (Table 2).

EFFICACY OF HOMOEOPATHIC NOSODES

In a joint study between the research centres at Abbotstown and Moorepark the efficacy of a homoeopathic nosode was evaluated in three herds, one each attached to the research stations at Abbotstown and Moorepark and one commercial herd (J. Egan and W.J. Meaney, unpublished). A total of 188 lactating cows were assigned at random, in a double blind trial, into two groups and treated with either a nosode or placebo for a period of 12 months. The homoeopathic preparation used in this experiment was formulated at 30c potency and contained a combined nosode for *Streptococcus agalactiae*, *Streptococcus dysgalactiae*, *Streptococcus uberis*, *S. aureus* and *Escherichia coli*. A placebo was also used.

Table 2. Results of efficacy trials on four commercial homeopathic preparations in the treatment of subclinical mastitis in cows

Product	Ref.	No. of cows treated	Duration of treatment (days)	Route of administration	No. of quarters affected by infections or non-specific mastitis	
					Pre-treatment	Post-treatment
A	5	15	17	Orally	19	23
A	7	25	20	Orally	26	26
B	J. Egan	8	12	Subcutaneous	19	18
C	J. Egan	8	5	Orally	21	21
D	7	19	20	Drinking water	19	16

Table 3. Clinical mastitis in animals treated with a homoeopathic nosode or a placebo (J. Egan and W.J. Meaney, unpublished)

	Treatment	
	Homoeopathic nosode	Placebo
No. of cows on treatment	94	94
No. of cows which developed clinical mastitis (%)	37 (39.4)	33 (35.1)
No. of quarters infections	47	57
No. of cases of acute clinical mastitis	31	36

A total of 148 cases of clinical mastitis cases developed in 70 (37%) of the cows on experiment (Table 3). One hundred and four (70.3%) were new infections and the remaining 44 (29.7%) were recurrent episodes which occurred in a number of cows. Sixty seven (45.3%) of all the infections were considered as acute. All but 12 (17.9%) of these cases occurred during the lactating period. There were no significant differences between the two treatments in the number of new cases of clinical mastitis either within herds or in all herds collectively. There was also no significant difference in the frequency of isolation of individual pathogens from animals on each treatment.

In a separate study Meaney (7) evaluated the efficacy of a nosode formulated for *Str. agalactiae*, *Str. dysgalactiae*, *Str. uberis*, *S. aureus*, *Staphylococcus albus*, *Staphylococcus epidermidis*, *Arcanobacterium pyogenes*, *E. coli* and *Mycobacterium bovis*. One group of 13 animals was treated with the nosode and a further group of 13 cows acted as an untreated control. The nosode was administered using a vulva spray technique and the experiment was conducted over an eight month period. It was concluded that the nosode had no effect in reducing the mastitis incidence or milk SCC.

DISCUSSION

Homoeopathic mastitis control remedies have been used on about 22% of the more progressive Irish farms with the primary objective to reduce milk SCC. Over 43% of herdowners using homoeopathy claimed that it had worked. While most preparations were administered in drinking water or feed some were administered by subcutaneous injection or into the vulva. As most of the products and information on homoeopathy came from non veterinary sources, it would be interesting to have an assessment from the herds' veterinary practitioners on the usage and efficacy of these products in the animals. In an assessment of 12 herds using homoeopathy in the UK(8), it was found that there was little evidence to indicate that the homoeopathic preparation used had any effect in controlling the incidence of mastitis and the authors concluded that the perceived success of the product by farmers derived in part from their attitude and commitment to homoeopathy. It was also found that in a limited number of

cases the farmers using homoeopathic mastitis control had stopped using one of the standard mastitis control recommendations.

The controlled trials conducted to date in Ireland have show no benefits from homoeopathic mastitis products. Most of the products tested have been commercially produced and sold directly to farmers. While manufacturers of these products can usually supply individual case history data to substantiate their claims for efficacy, full data on all other factors that may be affecting mastitis incidence in the herds is generally not provided to make a realistic assessment of the claims. The financial outlay for farmers using homoeopathic medicines in some herds has been considerable and it could be realistically assumed that other proven control measures are rigorously applied in these herds. As the recommendations for using these products are generally not as clearly defined as conventional therapies it is important to discuss with manufacturers, protocols for evaluation prior to any trials on efficacy. To date there is little evidence of the "homoeopathic" industry funding independent studies of these products and as there are generally no adverse effects from their use they are unlikely to become a major issue for regulatory authorities.

Distinct from the large scale homoeopathic treatments are the selective homoeopathic remedies provided on an individual animal or herd basis by veterinary homoeopaths. While many of these treatments remain scientifically unproven there is no doubt that there is a high level of individual animal care provided and the current negative results should not be extrapolated to such homoeopathic treatments.

The debate on the efficacy of homoeopathic medicines transcends both veterinary and human medicine. Central to the debate is the role of the placebo and the extent of the self cure phenomenon. In mastitis it is clear that a significant number of infections will be eliminated without any treatment or with minimal intervention such as quarter stripping. Ideally, if it was ethically and practically feasible, it would be desirable to structure trials on the efficacy of both conventional and alternative medicines to allow for quantification of this factor. The self cure rate for *S. aureus* mastitis is approximately 25% within the three week period after treatment, giving a true elimination rate for antibiotic treatment of about 17% (9). In the present trials some of the homoeopathic products were evaluated in chronic cases of mastitis where there is little doubt that the response rate from some conventional therapies would have been similar.

It is sometimes difficult to assess adequately conclusions drawn from some experiments due to poor experimental design and/or incomplete data. Day (10) claimed to find some benefits from use of a nosode for unspecified periods in three herds while Sonnenwald (11) found homoeopathic preparations were more successful than antibiotics in treating mastitis cases caused by Gram-negative bacteria but less effective than antibiotics in treating mastitis cases caused by Gram-positive bacteria. In a field study of 100 cases of acute clinical mastitis (12), it was found that homoeopathic treatments gave similar cure rates to antibiotics. The authors also reported that homoeopathic treatments were more successful in mastitis cases caused by Gram-negative pathogens whereas antibiotic treatments were more effective in the treatment of mastitis caused by Gram-positive bacteria. Dorenkamp (13), in a study of homoeopathic treatment on 580 subclinical cases of mastitis, achieved a cure rate of 97.9% after 4 days with only 28 (4.8%) showing relapses after 8 weeks. The same author reported that homoeopathic

treatment of 218 clinical infections achieved 100% cure rate after 3 days with only 5 (2.5%) showing relapses after 8 weeks.

In a review of the use of homoeopathic treatments Hamann (14) concluded that scientific information on the effectiveness of homoeopathic remedies for the treatment of bovine mastitis was too limited to justify a definite conclusion. The results of the trials to date in Ireland indicate that there are no benefits from treating animals with some of the commercially produced remedies on the market. The work highlights the need for further investigations of similar preparations.

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MASTITIS THERAPY IN ORGANIC DAIRY HERDS

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SUMMARY

A total of 16 organic dairy farms were surveyed for 12 months and data were collected on prevention and treatment of clinical cases of mastitis. Comparative data from seven matched conventional farms were also collected. Organic regulations prevent organic dairy farmers from using antibiotic dry cow therapy, and milk withdrawal periods after antibiotic therapy during lactation are prolonged under organic regulations. It was assumed that alternative therapies would be more common in the organic herds. The main alternative to antibiotic therapy on the organic farms was homeopathy, which was the treatment of choice for clinical mastitis on 12 out of 16 farms. Homeopathy was used on 52% and topical udder liniments on 7% of clinical mastitis cases treated on organic farms. Treatment frequency of the alternative therapies differed little from that of routine antibiotic use, but the duration of treatment was on average longer when using alternatives to antibiotics. Milk withdrawal periods were on average shorter when using alternative therapies and were usually shorter than the treatment periods. Clinical mastitis incidence on the organic farms was similar to that recorded on conventional farms. Dry period mastitis was responsible for over 15% of all recorded mastitis cases on the organic farms.

INTRODUCTION

Organic dairy farming in the UK has expanded in recent years. This is due to an improved marketing infrastructure and a favourable milk price compared to the conventional sector. In June 1998, there were 66 organic milk producers in the UK. An estimated 90 dairy farms were in conversion then (United Kingdom Register of Organic Food Standards [UKROFS], personal communication). Presently, new farms are entering conversion at a growing rate, induced by the introduction of higher conversion subsidies by the Government and sinking producer prices for conventional milk. Whilst the organic sector remains marginal in the British dairy scene, it has an important role in exploring alternatives to conventional practices.

Mastitis has been identified as one of the major concerns affecting potential converters to organic dairy production (Mark Measures of Organic Conversion Information Service; personal communication). Two surveys investigating animal health problems on existing organic farms also identified mastitis as a research priority for most organic dairy farmers (1,2).

The treatment and control of bovine mastitis on organic dairy farms differs from the practices on conventional farms mostly in the approach to dry cow therapy (DCT). A routine use of DCT is forbidden under the organic regulations (3). Clinical cases of mastitis can be treated by conventional antibiotic therapy, but prolonged withdrawal periods for milk discourage this practice.

A current MAFF-funded study, being conducted at the University of Reading, is determining mastitis levels and risk factors, treatment and control practices and economic implications of mastitis on 16 organic farms in the South of England and Wales. One aim of the study is to identify good practices and to develop advice for converting and established organic farmers. The results outlined in this paper are based on a preliminary analysis of mastitis treatment data collected during the first year of the two year field study.

METHODS

Data were collected for 12 months from 16 organic (average herd size 100 cows) and seven matched conventional farms in South of England and in Wales. The data included farmer interviews on husbandry and management practices, environmental observations, farm mastitis data and data on treatment and milk withdrawal periods. National Milk Recording (NMR) data on production and milk composition and quality were also used. The farms were visited every three months to collect data and to interview the herdsman during milking.

The data on mastitis treatments were mainly collected from on farm records. The organic dairy farms are obliged to keep detailed records of all animal treatments and milk and meat withdrawal periods. They often keep separate records for conventional and alternative treatments. All conventional farms, except one, kept mastitis treatment records at the beginning of the survey. Only minor adjustments were made to these records to serve the purposes of the survey.

The reliability of the treatment data was confirmed by interviewing the herdsman during the regular farm visits and by checking the veterinary invoices for quantities of antibiotics used. The veterinarians, who had care of the animals on the farm, were also consulted and were given feed-back on the survey results regularly.

RESULTS

A total of 960 cases of clinical mastitis were recorded on the 15 organic and seven conventional farms in 1997. Out of these cases, 615 (64.1%) were treated with antibiotics. All mastitis cases on the conventional farms were treated with antibiotics, whilst, on the organic study farms, antibiotics were used in only 40.7% of the cases (Table 1). More than half of the antibiotic treatments (55.7%) on organic farms were used on one farm alone.

Homeopathic treatments of clinical mastitis constituted 52.4% of all treatments on the organic farms. Other treatments, mainly with topical udder liniments, were used in 6.9% of mastitis cases in the organic herds.

Table 1. Treatment types for 960 clinical mastitis cases on 15 organic and 7 conventional farms in 1997

Type of treatment	Number of treatments (% of all treatments)
Antibiotic treatment on conventional farms	378 (100%)
Antibiotic treatment on organic farms	237 (40.7%)
Homeopathy on organic farms	305 (52.4%)
Other treatment on organic farms	40 (6.9%)

In three out of 15 organic herds, all clinical cases of mastitis were treated by non-antibiotic treatments (Table 2). On all but four of the organic farms, the majority of mastitis treatments were non-antibiotic. The treatment of choice for most organic producers was homeopathy. Nine out of 16 farms used homeopathy alone or combined with non-antibiotic support therapies (Table 3.). Further, three farms used homeopathy to treat a majority of mastitis cases but resorted to antibiotics regularly for various reasons. Two of the organic farms used antibiotics in a majority of cases but expressed interest in homeopathy. Two organic producers used antibiotics by choice in virtually all cases.

Table 2. Number and proportion of non-antibiotic treatments for clinical mastitis on 15 organic farms in 1997

Organic farm	Number of mastitis treatments	Number of non-antibiotic treatments	% of non-antibiotic treatments
1	56	56	100
2	34	34	100
3	14	14	100
4	129	128	99
5	37	36	97
6	43	39	91
7	7	6	86
8	36	30	83
9	62	49	79
10	83	52	63
11	18	11	61
12	19	5	26
13	7	2	29
14	33	2	6
15	131	2	2

Table 3. Treatment of choice for clinical mastitis on 16 organic farms

Method of Treatment	No. of farms
Antibiotics by choice	2
Antibiotics, but would like to learn homeopathy	2
Homeopathy with "lapses" to antibiotic use	3
Homeopathy with mastitis remedies and topical treatment	2
Homeopathy or cold water massage	1
Homeopathy by prescription	6

In the 378 cases on conventional and 237 on organic farms treated with antibiotics, 86.2% and 77.6% respectively (Table 4) were treated with intramammary antibiotics only. A combination of intramammary and injectable antibiotics was used in approximately 13% of the cases on both types of farms. Treatments with injectable antibiotics only and treatments by veterinarians were more common on organic than on conventional farms. The conventional farms used a higher number of intramammary tubes per treatment than did the organic farms.

Table 4. Types of antibiotic treatments of clinical mastitis cases on 15 organic and 7 conventional farms in 1997

	Conventional Farms	Organic Farms
Number of clinical cases treated with antibiotics	378	237
% of cases treated with intra mammary antibiotics only	86.2	77.6
% of cases treated with a combination of parenteral and intra mammary antibiotics	13.5	12.7
% of cases treated with parenteral antibiotics only	0.3	9.7
% of cases treated by a vet	0.5	2.5
Number of intra mammary tubes used/treatment	4.7	3.0

Duration and frequency of antibiotic treatments were similar on the organic and conventional farms (Table 5). Average duration of both homeopathic and other non-antibiotic treatments was, however, longer than that of antibiotic therapy. Maximum duration of up to three weeks of therapy was recorded. The average frequency of non-antibiotic therapy was slightly greater than that for antibiotic therapy. Some homeopathic therapies required very high frequencies of administration.

Table 5. Average duration and frequency of treatments for clinical mastitis cases on 15 organic and 7 conventional farms in 1997

Type of treatment	Average duration of treatment (days)	Average frequency of treatment (times/day)
Antibiotic treatment on conventional farms	2.9	1.7
Antibiotic treatment on organic farms	2.3	1.6
Homeopathy on organic farms	4.6 (max. 20)	2.3 (max. 12)
Other treatment on organic farms	5.4 (max. 14)	2.0 (max. 4)

There were differences in milk withdrawal times between the organic and conventional farms. The withdrawal times were much longer on organic farms, when antibiotics were used (Table 6). When homeopathic or other non-antibiotic treatments were used, the average withdrawal periods were shorter than the treatment periods.

Table 6. Average milk withdrawal times (milkings) during and following different clinical mastitis treatments on 15 organic and 7 conventional farms

Type of treatment	Average withdrawal time <u>including treatment</u> (milkings)	Average withdrawal time <u>after treatment</u> (milkings)
Antibiotic on conventional farms	10.4	4.7
Antibiotic on organic farms	17.5	12.9
Homeopathy on organic farms	6.6	0
Other on organic farms	6.0	0

Homeopathic nosodes (vaccines) in drinking water were used on 13 out of the 16 organic study farms, mainly during the housing period. DCT was used on two farms on targeted animals, under special permission from the UKROFS inspectorate, to attempt a cure for chronically infected udders. All conventional herds in the study used blanket DCT at drying-off.

A preliminary analysis of mastitis related data on the organic study farms during 1997 gave an incidence rate of 41 cow cases/100 cows, with 26% of the herd affected on average. The average recurrence rate was 14%. Notably, 15.6% of all mastitis cases on the organic study farms occurred during the dry period, while the corresponding figure for the conventional study farms was 0.01%.

DISCUSSION

There is a tendency among the organic dairy producers to move away from antibiotic use in the treatment of clinical mastitis. Whilst the organic regulations allow the treatment of sick animals with antibiotics, the prolonged withdrawal periods are an effective incentive to seek alternative therapies.

Homeopathy seems to be the most popular alternative. This is probably due to its resemblance to antibiotics with easy administration and medicine-like qualities. Provision of pre-prepared remedies for different disease entities by homeopathic pharmacies allows novel users to gain confidence with homeopathy rapidly. Preference for homeopathic therapy is, however, not restricted to organic farmers. The Ainsworth's Homeopathic Pharmacy has some 6,000 farming customers (Tony Pinkus, Ainsworth's Homeopathic Pharmacy; personal communication).

Udder liniments, containing mint or anti-inflammatory agents, were used often as support therapy with homeopathy. On one of the organic farms, an anti-inflammatory udder liniment was often used as the treatment of choice.

There was very little adoption of other alternative therapies among the organic farmers. Cold water massage, frequent stripping and herbal remedies were only used occasionally as a support therapy for homeopathy. Many of these therapies were seen as time-consuming and unsuitable for the herring-bone parlour, where it is important to maintain a steady throughput of cows.

Parenteral antibiotic therapy and treatment of mastitis by the veterinarian was more common on the organic farms than on the conventional ones. This is probably a direct reflection of the unavailability of antibiotics on farms that do not use them routinely. It is difficult to draw any conclusions on the number of tubes used per treatment, as the present analysis does not take into consideration the type of tubes involved.

It was expected that the frequency of use would be higher when using alternative therapies than when using antibiotics. The most significant differences appear, however, in the duration of the therapies. Most alternative therapies do not offer recommended treatment regimes, and most herdsmen state that they treat until the symptoms disappear. The nature of supportive, "constitutional" treatment in homeopathy, which requires administration of remedies long after clinical symptoms of mastitis have disappeared, is another explanation to these long treatment periods.

As the alternative therapies do not have statutory milk withdrawal periods, it was expected that the withdrawal periods used on organic farms would be shorter than the statutory withdrawal periods following antibiotic treatments. Milk from cows in treatment is often included into the bulk tank while a "constitutional" homeopathic treatment is being continued.

Targeted DCT was used on a number of cows on two of the organic farms during the observation period. Organic farmer can apply for a special dispensation for the use of DCT, when the farm has a recognized high SCC problem or is unable to cull cows with chronic infections. The latter was the case in 1997, when BSE cohorts were culled from most herds,

leaving little scope for strategic culling. The allowance for the dispensation to use DCT on organic farms seems to be based on similar requirements as used in Sweden to choose cows for DCT (4).

The preliminary analysis of clinical mastitis incidence data from the study farms does not show any real difference between the organic and conventional study farms and a larger sample of conventional farms (5). Similar results on mastitis incidence in organic herds have been published in other studies in the UK (6) and in other European countries (7,8). The high incidence of dry period mastitis on the organic farms was an unexpected result. The uneven distribution of dry period mastitis among the farms suggests that some producers have found successful methods of drying-off without DCT, while others are still struggling to establish good practice. An in-depth study of the drying-off practices and dry cow management is being carried out during the second year of data collection. As available mastitis data from conventional farms does not always differentiate between lactation and dry period mastitis, it is difficult to make comparisons.

During the second year of data collection, the research project will focus on identifying "good practice" herds, calculating the cost of various mastitis control and treatment strategies and the impact of mastitis on organic dairy farms. Further analysis of dry period mastitis and drying-off practices will be conducted. An in-depth analysis of somatic cell count management on organic farms is also required.

As the survey was not designed as an efficacy study, it would be unwise to draw any conclusions on the efficacy of the various therapies used by the organic farmers. However, the preliminary analysis of mastitis levels and treatment practices on organic farms provides interesting data on udder health management without antibiotics and will hopefully be valuable in developing sound advice suitable for milk production under organic regulations.

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RECOGNISING AND CONTROLLING PAIN AND INFLAMMATION IN MASTITIS

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SUMMARY

The aim of this project was to investigate if cows with clinical mastitis suffer pain. This was assessed by comparing the response of cows with mastitis to normal cows, on application of a pressure stimulus. The results strongly suggest that cows with mastitis do have increased sensitivity to pain, even when the mastitis is only mild, or moderately, severe. Treatment of cows with mild mastitis using a single, intravenous injection of a non-steroidal anti-inflammatory drug, flunixin meglumine (Finadyne, Schering-Plough Animal Health; 2.2mg/kg) combined with intramammary antibiotic therapy, reduced sensitivity to pain, but was not effective in moderately severe cases. This beneficial effect on sensitivity to pain was seen one day after the start of treatment which coincides with the duration of activity of flunixin. These results indicate that intravenous flunixin reduced sensitivity to pain in mild mastitis cases, but that repeated doses of the drug may be required to extend this beneficial effect. As cows with mastitis are in pain, improved methods are required to reduce pain and to return the udder to normal function as quickly as possible.

INTRODUCTION

Most farmers and veterinary surgeons agree that cows with severe toxic mastitis often appear to be distressed and in pain. However, it is much harder to decide whether cows affected by less severe forms of mastitis are also in pain. This may be due to our lack of ability to perceive signs of pain in stoical species such as cattle. It may be to ruminants' advantage, in an evolutionary sense, as a herd animal, and as prey rather than predator, to avoid displaying signs of weakness and pain. Development of objective, rather than subjective, methods of measuring pain are urgently required and have the potential to be used in future farm assurance schemes where adverse welfare associated with disease is assessed. While pain suffered by cows affected by diseases such as mastitis is an obvious welfare issue; cows in pain often have a reduced appetite, are less willing to move to sites of available food and have reduced milk yields, with concomitant deleterious effects on farm economics. Recognising pain and developing methods to reduce pain in mastitis, therefore, should benefit both cows and farmers.

Antibiotics are routinely used for mastitis therapy, but non-steroidal anti-inflammatory drugs (NSAID) can also be employed, although they are often reserved only for severe cases, primarily to treat systemic signs. They act to reduce inflammation (anti-inflammatory), to reduce pain (analgesic) and to reduce body temperature (anti-pyretic). There are currently two NSAID available commercially which are licensed for use in

mastitis, flunixin meglumine (Finadyne Injection, Schering-Plough Animal Health) and ketoprofen (Ketofen 10%, Meriel). Both of these products must be administered by parenteral injection, and their use is restricted to 5 consecutive days for Finadyne Injection, and 3 consecutive days for Ketofen 10%, due to potentially serious side effects, which can be fatal, resulting from prolonged use of these products. Since the withdrawal of phenylbutazone, which was commonly used by vets and farmers to treat painful conditions in cattle, from use in food producing animals, the NSAID remain the sole pain-killing drugs available. The other groups of analgesic drugs are not ideal for pain relief in cows with mastitis: the opioids are not licensed for any food producing species; corticosteroids may have immunosuppressive effects and should not be administered to pregnant animals, although they are incorporated into some intramammary antibiotic preparations; and the alpha2-adrenoceptor stimulant, xylazine, has a prolonged sedative effect. Thus, no drug is now available for controlling prolonged pain in cattle. To date, there have been few studies investigating pain in mastitis, although proteins such as bradykinin, known to mediate severe pain in humans, have been detected in milk from clinical and subclinical mastitis in cows (1).

Mastitis is defined as inflammation of the mammary glands. Inflammation is induced, in response to udder infection, by mediators that are produced to help clear infection, but that also result in damage to the milk secreting tissues of the udder. Unfortunately, damage to the udder may occur before signs of disease become obvious to the dairyman, or before somatic cells begin to rise in milk. Certain inflammatory proteins, termed acute phase proteins are present in altered milk during mastitis and may reduce milk quality (2). By measuring these proteins in milk it will be possible in future to monitor their concentration as a rapid diagnostic test for mastitis with a reduction in concentration indicating effective therapy.

The primary aim of this study was to investigate whether cows with naturally occurring clinical mastitis exhibited allodynia, a decreased pain threshold, by measuring their sensitivity to pain induced by a mechanical stimulus using methods described previously (3,4). Another aim was to compare the effect of pain-killing drugs given to mastitic cows by the intravenous or intramammary route on sensitivity to pain. The levels of haptoglobin, a sensitive bovine acute phase protein in blood and the somatic cell count (SCC) of milk from mastitic cows were measured and the response to the NSAID therapy assessed.

METHODS

Animals and sampling

Holstein-Friesian cows from three institute farms and one commercial dairy farm, milked twice daily, were used. Natural cases of clinical mastitis were recognised by the dairy staff on each farm in the usual way, by observation and palpation of the udder. Each cow was visited by the same researcher within two hours of the mastitis being detected. Blood samples were taken, and after clotting serum was removed for measurement of haptoglobin. Milk samples were taken from the mastitic quarter for bacteriology, somatic cell counting and mediator analysis. Samples were also taken from an unaffected, non-mastitic, quarter on the opposite half of the udder, as controls.

Responsiveness to pain

The pain threshold of cows was assessed using a mechanical device attached to each hind limb, just below the hock, and pressure was applied via a gas-driven device that pushed a blunt-ended pin against the cow's leg. When the cow responded to the pressure by shifting her weight or moving her leg, the pressure was immediately turned off. Two or three measurements were taken from each leg at each time point. The cows were re-assessed on six occasions during the study: once on the day of diagnosis and before treatment was given, and on five occasions one, two, four, 20, and 40 days following initial treatment.

Mastitis grades and therapy

The mastitis was classified as "mild" when clots were detected in the milk and the udder appeared normal, or "moderate" when clots were detected in the milk and the udder appeared red, hot or swollen. Any cows with more severe mastitis, which required systemic treatment or veterinary attention, were excluded from the study. All cases of mastitis received treatment. Control cows received a course of amoxycillin/clavulanic acid (3 syringes of Synulox Lactating Cow; Pfizer Ltd.), the second group received the same antibiotic course and an intramammary infusion of flunixin in a specially prepared vehicle created by Schering Plough Animal Health for this study, and the third group received the same antibiotic course and an intravenous injection with flunixin meglumine (Finadyne, Schering Plough Animal Health; 2.2 mg/kg). The first two groups of cows also received an intravenous injection of saline to act as a control for the intravenous flunixin.

Bacteriology and somatic cell counting

Bacteriological examination and somatic cell counting were performed at SAC Auchincruive using standard bacteriological identification and automated cell counting using a Fossomatic Counter. Milk samples bacteriology were taken prior to therapy being administered. Somatic cell counts were re-assessed at the same time points detailed above.

Haptoglobin

Haptoglobin levels in the blood were measured with an automated assay in the Department of Veterinary Clinical Studies University of Glasgow Veterinary School. Haptoglobin was re-assessed at the same time points detailed above

Statistical analysis

Somatic cell count data were log transformed due to being non-normally distributed. Responsiveness to pain was calculated by subtracting the threshold response in the leg on the same side of the cow as the mastitic quarter from the threshold response in the leg on the opposite side of the cow. Data were analysed using multivariate and mixed model analysis of variance (SAS, SAS Institute Inc., North Carolina).

RESULTS

Responsiveness to pain

The technique of measuring responsiveness of cows to pain using a mechanical stimulus presented no problems. All cows tolerated the attachment of the machine to their hind limbs, and their response of shifting weight or moving the limb as a result of the stimuli was easily recorded.

The responsiveness to pain in normal, non-mastitic, cows was similar on both hind limbs. In mastitic cows, increased sensitivity to pain in the leg on the **same** side as the mastitic quarter compared to the leg on the other side to the mastitic quarter, was observed for both mild and moderate mastitis. The increased sensitivity in cows with mastitis was significantly greater than in normal, non-mastitic cows at all sample days up to, and including, that taken at 4 days after start of treatment for mild cases, and up to, and including, that taken at 20 days after the start of treatment for moderate cases.

When mild mastitis cases were treated with antibiotics alone, the responsiveness to pain was greatest two days after the start of treatment and was still present 20 days later. Similarly, mild mastitis cases treated with antibiotics plus intramammary flunixin showed a similar effect, although the responsiveness to pain appeared to be less marked in these cows. When mild mastitis cases were treated with antibiotics and intravenous flunixin, a sharp decrease in responsiveness to pain was seen one day after the start of treatment. This coincides with the expected clinical activity of flunixin administered by the intravenous route. However, increased responsiveness to pain was again observed from the next sample day, two days after the start of treatment, and this effect persisted again until the sample on day 20 (Figure 1).

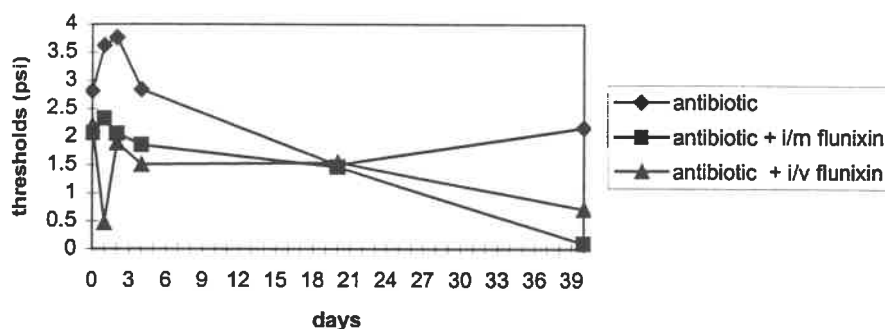


Figure 1. Threshold responses of cows with mild mastitis

With moderate mastitis treated with antibiotic alone, increased responsiveness to pain was seen throughout the study. The moderate cases also showed increased responsiveness to pain when treated with antibiotics plus intramammary flunixin at all time points sampled, and the response was greater than with antibiotic treatment alone, indicating that the intramammary flunixin may have induced an adverse effect. The responsiveness to pain in moderate cases treated with antibiotics plus intravenous flunixin was similar to those given

antibiotic alone, and no reduced responsiveness to pain was seen on day one after treatment, as was the case in mild mastitis cases (Figure 2).

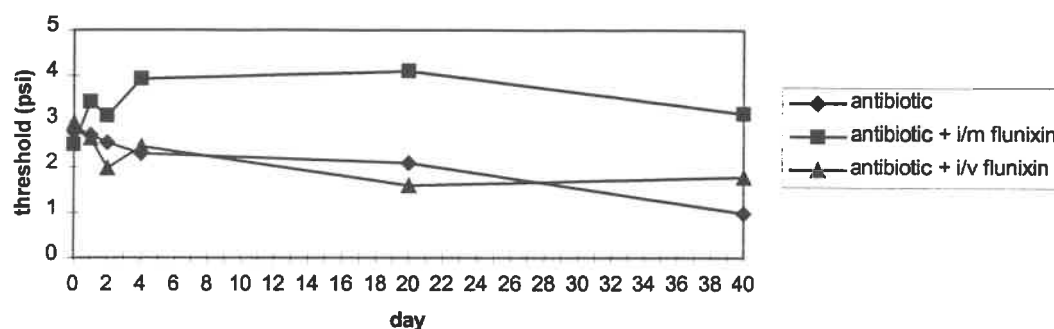


Figure 2. Threshold responses of cows with moderate mastitis

When normal, non-mastitic, cows were given the same treatments as the mastitic cows to act as controls for possible effects of the drugs themselves on responsiveness to pain, the groups given antibiotics alone or antibiotics plus intravenous flunixin, showed no increased responsiveness to pain. However, with groups given antibiotics plus intramammary flunixin, a significant increase in responsiveness to pain did occur, although not until the samples taken on day 20 and 40 following the start of treatment (Figure 3).

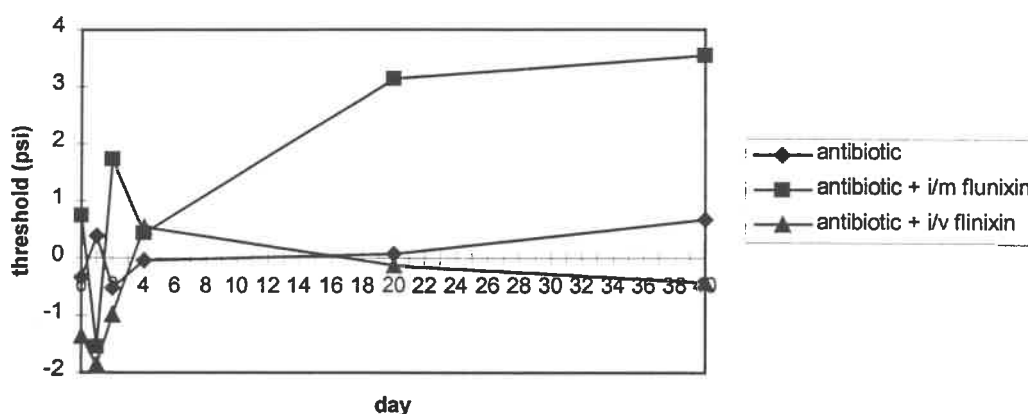


Figure 3. Threshold responses of normal, non-mastitic cows

Statistical analysis showed significant differences in sensitivity to pain among groups (mild mastitis, moderate mastitis, normal non-mastitic cows; $p < 0.0001$), among treatments (antibiotics alone, antibiotics plus intramammary flunixin, antibiotics and intravenous flunixin; $p < 0.05$) and there was a group-treatment interaction; $p < 0.05$) on responsiveness to pain.

Haptoglobin

The moderate mastitis cases had greater levels of haptoglobin than mild mastitis cases at all time points, and both mastitic groups had higher levels than the normal, non-mastitic cows throughout the study. The haptoglobin was found to be increased in both the mild and moderate mastitis groups at the sample taken before treatment, and the shape of the curve suggests that, particularly in the mild mastitis cases, the haptoglobin level may have been raised prior to the mastitis being noticed by the dairyman. The haptoglobin level was still significantly raised on day 4 following start of treatment in moderate cases but had returned to near the level seen in normal cows in the mild mastitis cases by the same time. The haptoglobin levels in moderate mastitis cases returned to normal levels (0.1g/l) by day 20 (Figure 4).

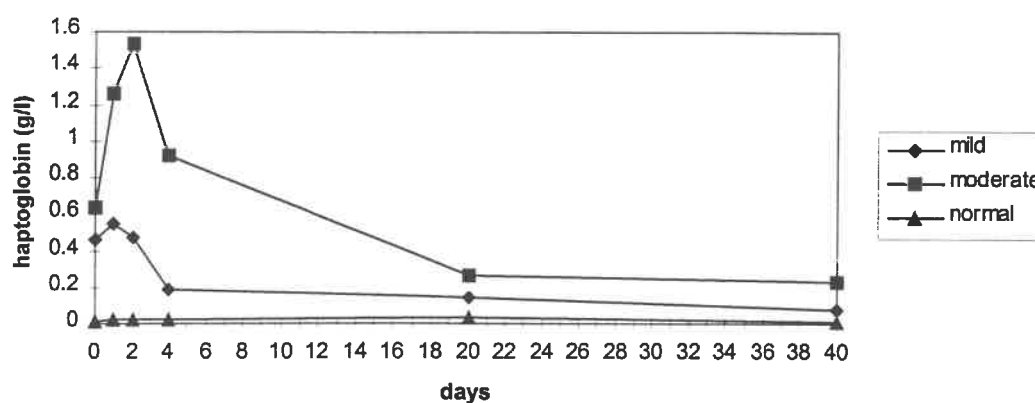


Figure 4. Serum haptoglobin levels in cows with mild mastitis, moderate mastitis and in normal, non-mastitic cows.

Statistical analysis showed a significant effect of group (mild mastitis, moderate mastitis, normal non-mastitic cows; $p < 0.0001$), and time ($p < 0.0001$) but not treatment, on levels of haptoglobin in blood.

Bacteriology and somatic cell counting

Bacteria isolated from the clinical cases included the usual range of pathogens including *Escherichia coli*, *Staphylococcus aureus*, *Streptococcus uberis* and *Arcanobacterium pyogenes*. A number of minor pathogens and contaminants, including *Staphylococcus epidermidis*, micrococci, and *Bacillus* sp. were also recognised. A small number of cases showed no growth on bacteriological examination.

The SCC increase was greatest with the moderate mastitis cases and peaked at approximately 13,000,000 cells/ml milk on day 1 after initiation of treatment. The mild mastitis cases also showed an increase in SCC compared to normal non-mastitic cows and again the peak levels were recorded at the same time point (Figure 5). The SCC in mild mastitis cases declined to normal levels by day 4 after the start of treatment but were still elevated in the moderate mastitis cases at day 20. Cell counts were also increased in normal non-mastitic cows following all treatments, but the SCC levels did not rise above

4,000,000 cells/ml milk in these cows. The SCC was high in a number of the normal cows, some of which had bacteria isolated from them and were, therefore, subclinically infected.

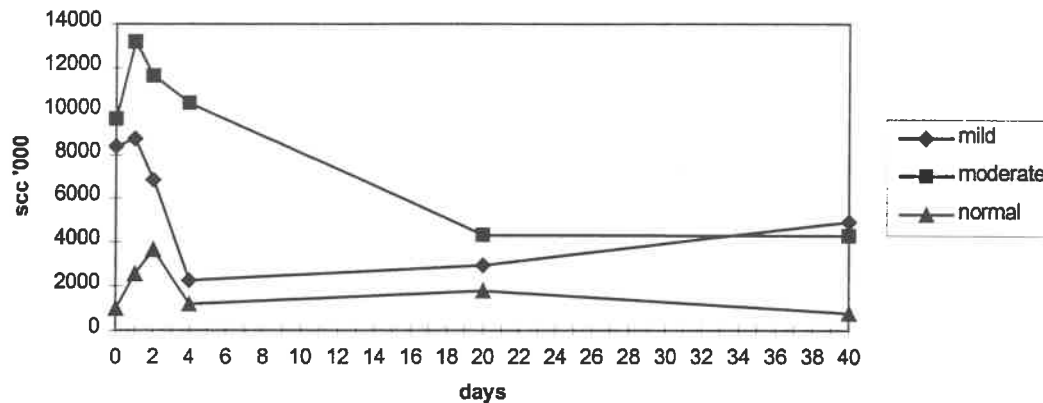


Figure 5. Somatic cell counts from quarters with mild mastitis, moderate mastitis and normal, non-mastitic quarters.

Statistical analysis showed a significant effect of group (mild mastitis, moderate mastitis, normal non-mastitic cows; $p < 0.0001$), and time ($p < 0.0001$) but not treatment, on SCC in milk.

DISCUSSION

The bacteriology results from the clinical cases of mastitis were typical of the bacteria associated with mastitis in the UK.

The increased responsiveness to pain was very marked and of a similar level in both mild and moderate cases of mastitis. The duration of this increased sensitivity was long, and extended to day four following detection of mastitis for mild cases, and day 20 for moderate cases. This implies that cows with mastitis may find painful stimuli that would normally be innocuous, with the potential that everyday activities may cause the cow more pain than usual.

Sensitivity to pain was reduced when mild mastitis cases were treated with a single intravenous injection of flunixin, in combination with intramammary antibiotic. This response was seen one day after the start of treatment, which coincides with the clinical activity of flunixin, after which the cows returned to a state of increased sensitivity to pain. This suggests that a single intravenous injection of flunixin altered pain mechanisms for a short time and indicates that repeated doses of this drug may potentially maintain this beneficial effect. These data also suggest that changes in pain perception are occurring away from the site of inflammation, the udder, because flunixin given locally showed no beneficial effect in mild mastitis cases, whereas the same drug, administered intravenously, successfully reduced sensitivity to pain. Mastitis cases, classified as moderately severe, did not show a similar beneficial effect of treatment with flunixin as the mild mastitis cases, and

it is possible that either an increased dose of flunixin or alternative drugs are required to relieve pain in cows with moderate, or more severe, mastitis. The intramammary flunixin appeared to increase pain sensitivity a few weeks after treatment and it is possible that either the drug or the vehicle in which the drug was suspended induced a delayed inflammatory response in the udder.

The detection of haptoglobin in both mild and moderate mastitis cases indicated that an acute phase response was occurring and that this protein has the potential to be used for an early warning marker of mastitis. This suggests that especially milder cases of mastitis may have measurable inflammatory changes prior to recognition of milk clots or udder changes by the dairyman.

The SCC showed predictable responses to mild and moderate mastitis, with the SCC being greater and increased for a longer period in moderate cases than in mild cases. The SCC in mild mastitis cases had almost peaked on the day of detection, whereas the SCC in moderate cases continued to rise for another 24 hours. The pattern of SCC in mild and moderate mastitis was similar to that seen from the haptoglobin results, again suggesting that inflammatory changes precede clinical detection particularly in mild cases. The cell counts in normal cows, even when not subclinically infected, were higher than would be expected by normal sampling regimes. This is due to the milk sample being collected after the udder had been emptied at milking. At this time, the cells are concentrated in the secretion remaining in the glands. This means that the SCC in this study can not be directly compared to SCC taken routinely from cows or milking jars.

The treatments given in this study had no effect on either haptoglobin levels or on SCC indicating that a different therapeutic approach is required to increase speed of recovery of inflammation. It is possible that different types or doses of NSAID may be more effective in reducing udder inflammation, or it may be necessary to develop novel drugs to treat mastitis in the future.

CONCLUSIONS

- It is possible and practicable to measure the responsiveness of cows with clinical mastitis to pain
- Cows with mild and moderate mastitis have increased responsiveness to pain on the leg nearest the side with the mastitic quarter
- Increased responsiveness to pain, persists for many days after mild mastitis and some weeks following moderate mastitis
- A reduction in responsiveness to pain resulted from treatment of mild mastitis cases with intramammary antibiotic and intravenous flunixin one day after treatment. This indicates a beneficial effect on pain relief.

- The reduction in responsiveness to pain which resulted from treatment of mild mastitis cases with intramammary antibiotic and intravenous flunixin was shortlived and the increased responsiveness to pain returned to former levels and persisted. This indicates that repeated doses of intravenous flunixin may be required for prolonged pain relief.
- Treatment of mastitis cases with intramammary antibiotics and intramammary flunixin caused an adverse effect with increased responsiveness to pain being recorded
- Mild and moderate mastitis induced an increase in blood haptoglobin levels and were unaffected by treatment
- The somatic cell counts were raised in mild and moderate mastitis cases and were unaffected by treatment
- The haptoglobin levels and SCC in mild mastitis cases may have been elevated significantly prior to clinical detection

ACKNOWLEDGEMENTS

This work was funded by the Milk Development Council. The authors would like to thank Shering-Plough Animal Health for providing the intramammary preparation of flunixin, and Dr. Mike Stear of the Department of Veterinary Clinical Studies, University of Glasgow Veterinary School and Dr. Rob Berry of the Scottish Agricultural College, Auchincruive for statistical advice. The help of the staff at all farms where this study was performed is gratefully acknowledged.

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FEATURES AND UPDATES

UPDATE ON SUMMER MASTITIS

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SUMMARY

Summer mastitis is an acute mastitis typically occurring during the summer in non lactating cattle. The incidence of summer mastitis varies between years and has been reported, in England and Wales, at 39% to 54% of herds affected with 2.1 to 4.1 cases per affected herd. The reported incidence for 1997 was 46% of herds affected, with 2.2 cases per affected herd. An updated estimate of the costs of summer mastitis in 1997 suggested a loss of £270 per case or a total cost of £6M per year in England & Wales. Risk factors for summer mastitis include a lack of dry cow therapy, exposure to flies, autumn calving and teat damage. Good attention to remedy these has contributed to a likely reduction in incidence from 10% of cows at risk to the 1997 figure.

INTRODUCTION

Summer mastitis has been reported from northern Europe, Japan, USA, Greece, Australia, Zimbabwe and Brazil. Regional variations occur in incidence and the classic area of occurrence is considered to be a sandy soil or low lying, wooded areas with poor drainage.

Summer mastitis is a clinical mastitis that occurs in non lactating cattle, generally at pasture, during the summer usually from the end of June to September although occasionally cases occur in October. The signs are characteristic and initially include lethargy and a swollen teat or quarter rapidly progressing to a hard, swollen and painful quarter with a thick yellow secretion and a typical unpleasant smell. Without rapid treatment, the infection progresses to induce an elevated temperature, septicaemia, oedema of the hind legs, accompanied by lameness and can result in abortions or perinatal mortalities, and sometimes death of the affected animal. The secretory function of the affected quarter is usually lost. The term, summer mastitis, can be extended to include a similar disease outside the above time period but does not include the peracute, systemic, non suppurative mastitis that occurs just prior to calving. Summer mastitis is also seen in beef cows, calves and bulls.

Edmonds and Welsh (1) reviewed cases in Yorkshire herds and found that nearly three quarters of cases occurred in August and approximately 15% in July and 12% in September. This is important for identifying the risk periods for implementing control measures. Generally June is also included in this control period.

There is an increased prevalence of summer mastitis with age and this continues to a maximum for the dry cow period between the fifth and sixth lactations (2). The lower incidence in older cows may be due to these animals being less susceptible to summer mastitis.

Bacteriological analysis of secretion from the affected quarter often reveals an infection involving several bacterial pathogens. Usually *Arcanobacterium pyogenes* (formerly *Corynebacterium pyogenes*) predominates. *Streptococcus dysgalactiae*, *Peptococcus indolicus* and Stuart-Schwan cocci are frequently isolated. *Streptococcus uberis*, *Staphylococcus aureus*,

Pasteurella haemolytica, *Escherichia coli*, *Bacteroides melaninogenicus* and *Fusobacterium necrophorum* have also been isolated. This latter group may be secondary invaders.

RISK FACTORS

There are various risk factors identified for summer mastitis in herds in the south of England (3) including

- High bulk milk cell count possibly indicating a lower standard of management
- An animal effect possibly related to a greater susceptibility for lower milk fats and lactose
- Grazing on permanent pasture, possibly related to greater numbers of flies hatching
- Trees around fields, but not hedges
- Topography, low lying and less windy areas reporting more cases
- Management factors relating to poor control and not meeting targets such as quota and milk quality levels

PREVENTIVE MEASURES

In the Farm Animal Welfare Council report on the 'Welfare of Dairy Cattle' (4), summer mastitis is highlighted as a welfare problem for both cows and heifers. Where the incidence is high, the consulting veterinary surgeon should be involved in applying a preventive programme. Effective preventive measures include

- ◆ dry cow therapy, this may also be necessary in high risk organic herds
- ◆ fly control, preferably starting before the risk periods
- ◆ pasture management, including cutting plants which can result in abrasions on teats
- ◆ avoiding high risk pastures for susceptible animals
- ◆ treating teat wounds promptly and possibly using teat sealants
- ◆ inspecting animals at least twice daily to identify cases promptly and to reduce the spread of disease
- ◆ changing calving pattern, this may be the only alternative in some organic herds or on some farms with limited possibilities for changing grazing

Generally it is assumed that the sheep head fly, *Hydrotaea irritans*, is involved in the transmission of infection. The circumstantial evidence is that

- the peak incidence of disease occurs when flies are most frequent on cattle
- *Hydrotaea irritans* is a frequent visitor to cattle, including teats, and is known to carry the bacteria associated with summer mastitis (5)
- the epidemiological spread of the disease coincides with the geographical distribution of the fly
- infections are commoner in the front quarters which flies can reach more easily
- fly control is known to reduce the incidence of summer mastitis.

However, infection can occur outside the fly season and in areas where the *Hydrotaea irritans*, or another suitable vector, does not occur and attempts to experimentally transmit the disease have only shown limited success (6,7,8).

Aehnelt (9), in Germany, recorded successful control of summer mastitis, reducing the incidence to 0.7% by spraying insecticides on cattle at 3 weekly intervals from the end of May to mid September susceptible animals, compared with the incidence of 9.4% in the control group.

In the 1950s it was shown that antibiotic infusions into the dry mammary gland could protect against summer mastitis. Pearson (10,11) used single and multiple infusions of penicillin in an oily base and reduced the incidence to 0.7% in treated cattle compared to 10.6% in untreated controls. However, inoculating the teats of frisky young heifers at any time is no easy task. Weigt and others (12) in Germany described an infusion method for heifers restraining the heifers in a crush and holding the nozzle over the teat orifice whilst depressing the plunger of the tube. Reasonable protection from infusing heifers has been reported (12,13). Edmonds and Welsh (1) showed a benefit from multiple infusions of an amoxycillin dry cow preparation at 3 week intervals and also had no problems with antibiotic residues even when infusion had occurred 3 days prior to calving. A cost benefit was found from use of multiple infusions compared to use of a single infusion of dry cow therapy.

ECONOMIC COSTS

In a 1992 estimate of the costs of summer mastitis (14), some 49% of losses were due to lost milk production, 37% from the lost animal value, cull and calve losses and labour, drugs and veterinary charges accounted for 14%. The average loss was £192 per case and the cost to the dairy industry in 1987 was £6.22 million. This was at a time when milk price was 16.25 p/litre and cull values were £335 to £435 depending on age. These costs would equate to a figure nearer £270 per case in 1998. Taking account of the number of animals currently in the national herd and the higher cost per case there remains a total annual cost in England and Wales of £6M.

INCIDENCE IN ENGLAND AND WALES

Since 1978 the Milk Marketing Board for England and Wales, then Genus and subsequently AxiEnt have carried out surveys on summer mastitis experienced by users of their field service in England and Wales. Data collected included the number of cows, dry cows, pregnant heifers and non pregnant heifers and the incidence of summer mastitis. Use of dry cow therapy usage and fly control measures in place were also recorded.

Wide regional variations occurred every year, both for percentage of herds affected and the number of cases per affected herds. The south east of England generally reports a lower incidence and the north and western areas of England and Wales a higher incidence. The results over the period 1978 to 1997 are presented in Table 1.

Table 1. Incidence of summer mastitis during the period 1978 - 1997 (including results from references 15 and 16)

	No. of herds	No. of animals at risk	No. of summer mastitis cases	%	No. of herds affected	%	Average number of cases/affected herd
1978	248	-	-		107	43	3.5
1979	393	-	-		157	40	2.4
1980	416	-	-		179	43	2.6
1981	391	-	-		231	59	4.1
1982	499	-	-		269	54	2.6
1983	484	48106	548	1.1	203	42	2.7
1984	226	26199	244	0.9	91	40	2.7
1985	275	22339	207	0.9	97	35	2.1
1986	210	22910	250	1.1	97	46	2.6
1987	182	17508	290	1.7	108	59	2.7
1988	287	27100	427	1.1	133	46	3.2
1989	317	30686	406	1.3	147	46	2.8
1990	322	26564	237	0.9	113	35	2.1
1991	350	27007	356	1.3	146	42	2.4
1992	358	27419	262	0.9	128	36	1.9
1993	360	28463	337	1.2	141	39	2.3
1997	345	24643	358	1.5	144	46	2.2

Table 2. Summer mastitis incidence per group at risk from 1983 to 1997

Year	No. of dry cows	% affected	No. of pregnant heifers	% affected	No. of non-pregnant heifers	% affected
1983	19464	1.2	10864	1.9	17778	0.6
1984	11691	1.2	5222	1.1	9286	0.4
1985	9584	1	5379	1.5	7476	0.5
1986	11000	1.4	4563	1.6	7347	0.3
1987	8496	2.1	3383	2.2	5629	0.6
1988	12675	2.2	5573	2.3	8852	0.3
1989	13268	1.8	6437	2.2	10981	0.2
1990	13838	1.1	5055	1.2	7671	0.4
1991	12865	1.7	5379	1.7	8763	0.5
1992	12660	1.3	6357	1.4	8348	0.1
1993	13276	1.7	6706	1.5	8481	0.1
1997	9064	2.3	7264	2.0	9916	0.1

The incidence of summer mastitis in heifers and dry cows in northern Europe has been reported to be as high as 10% (17), similar to that reported from the UK and Germany for animals receiving no protection (9,10,11). In protected animals in England and Wales over 2 % of dry cows and pregnant heifers are affected. The true incidence of summer mastitis in dry cows is probably much higher, as in most years most dry cows at risk receive dry cow therapy.

DRY COW THERAPY

In most years surveyed over 99% of herds used dry cow therapy in cows, few used dry cow therapy in heifers and few re-tubed dry cows. The herds carrying out the latter two practices tended to be herds that had experienced a high incidence in susceptible animals.

Since 1991, the percentage of herds using longer acting dry cow therapy (lasting more than 32 days) has steadily increased from 59% to 75% in 1997 (Table 3). There appears to be no difference in incidence rates for summer mastitis between those using short or long acting therapy.

Table 3. The incidence rates for the different tubes used in the 1997 survey

Preparation	Herds using this tube		% of herds affected	Cases per affected herd using that dry cow therapy
	No.	%		
Cepravin	181	52	33	1.9
Orbenin Extra	64	19	30)	2.1)
Boviclox Extra/	4	1	60)-34	2.3)-2.0
Kloxerate Gold	10	3	50)	1.0)
Preparation lasting less than 32 days	86	25	29	2.1

Table 4. Fly control measures used from 1983 to 1997

Year	Pour on	Spray	Ear tags	Tar
1982		43	6.2	.2
1983		43	9.3	.5
1984		16	32	
1985	2	22	21	
1986	7	23	18	
1987	8	32	16	
1988	30	21	12	
1989	38	15	10	
1990	39	16	9	
1991	44	17	5	
1992	43	15	5	1
1993	47	10	4	3
1997	63	14	4	5

FLY CONTROL

Table 4 shows the changes in fly control measures from 1982 to 1997. There was a rapid uptake on the use of insecticidal pour-ons, once introduced probably due to their ease of use. Table 5 shows the fly control measures used and the numbers of herds affected and cases per herd. There were marginal differences in the incidence of summer mastitis in herds using insecticidal sprays, tags or pour-ons but no one method seemed more effective than any other and only small numbers of herds used sprays and ear tags. Stockholm tar and insecticidal

creams seem the least effective but there were only small numbers of herds using these methods.

Table 5. Fly control measures used and percentages affected for the survey 1997

Fly Control	Herds using this method		Percentage of herds affected	No. of cases per affected herd
	No.	%		
None	44	13	36	2.6
Insecticide tags	15	4	40	2.0
Insecticide sprays	49	14	41	2.0
Pour-on insecticides	218	63	48	2.4
Stockholm tar	18	5	78	3.5
Insecticide cream	2	1	100	2.0

Only 13% of herds used no fly control with 36% of this limited sample affected by summer mastitis. It may be that these herds are perceived as being of low risk. There were wide regional variations in those herds not using fly control, with the South and Mid South West, South Wales, North East, North Wales and North West Midlands and North West all having more than 10% of herds using no fly control. In the other regions fewer than 3% of herds used no fly control.

CONCLUSIONS

There was significant progress in reducing the numbers of summer mastitis cases from the levels reported in the 1950s, taking the figures given by Pearson, to the 1970s. Since then there have not been any significant or sustained changes in the incidence rates for the different groups at risk. Compared with the Scandinavian countries, there is a lower incidence of summer mastitis in pregnant heifers in England & Wales but, in some herds, attention still needs to be paid to control measures for these animals. The 1998 FAWC report highlighted summer mastitis as a welfare issue and that control measures must be used to prevent cases, especially in those herds with greater risks. Dry cow therapy and good fly control are still two of the most important and effective control measures available.

ACKNOWLEDGEMENTS

The author would like to thank the Milk Development Council, all the Axient Mastitis and Hygiene consultants and Mark Scott.

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BULL PROOFS FOR SOMATIC CELL COUNTS

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SUMMARY

In 1997 the Animal Data Centre (ADC) began to publish bull proofs for Somatic Cell Count (SCC). These proofs cover bulls with daughters in the UK, as well as converted proofs from other countries. How these proofs are calculated, how they might be used in sire selection, plus some of the associated research being undertaken on SCC in the UK and elsewhere are described.

UK SCC PROOF CALCULATIONS

The procedures used to calculate SCC proofs (1), are essentially the same as used for production traits.

The ADC receives the monthly cell count test information from the Milk Recording Organisations (MRO). For each lactation, the geometric mean is calculated for all individual test results, with cows requiring six records per lactation for inclusion. As is the case for the individual test results, these means are not normally distributed, and the data are transformed to logarithms prior to analysis.

The model used by the ADC to predict genetic merit is based on their earlier investigations of UK data, but the procedure is generally similar to that used in other countries.

Non-genetic factors considered in the model include month and age of calving, and lactation number. Data on up to five lactations are used in the analysis, with a repeatability of 0.35 between lactation means. The heritability of SCC is assumed to be 11 percent, lower than for production traits, and this has an impact on the reliability of the resulting bull proofs. Because of the low heritability, there is little point publishing SCC proofs for cows.

As with Animal Models generally, the analysis uses all pedigree information. While proof information on cows is not published, SCC records on dams are used when predicting the merit of their sons. Foreign proofs on sires are included in the calculations where no UK proof is available.

The resulting proofs are presented as percentages. Those with negative values are good, in that they are expected to have daughters with a lower than average cell count. On the other hand, bulls with positive proofs are expected to increase cell count in their daughters. The larger the "number", the bigger the expected impact.

CONVERTED PROOFS

The UK dairy industry is unusual in that it is a major importer of dairy semen. Major supply countries include the USA, the Netherlands, France and Canada. The normal

procedure is that the best bulls tested in these countries are then marketed as soon as their home country proof is available. This applies to proofs for SCC, as well as for production, type and whatever other traits are available.

In addition to calculating proofs using UK daughter records, the ADC is also responsible for establishing conversion formulae, so that foreign proofs can be converted into UK equivalents. These formulae are derived using proof information for bulls with daughters in both the supplying country and the UK. This is a retrospective exercise. However, if these two proofs are highly correlated, then a formula which relates the two proofs can then be used to “predict” how his future UK daughters will fare, based on his home country proof. In short, the formula is then used prospectively.

To date, the ADC has looked at bulls from a number of countries, where they have both a home country and a UK proof for SCC (Table 1). In each case the correlation between the two proofs is very high. In simple terms this means that rankings of bulls for SCC in their home country is a good indication as to how they will ultimately rank in the UK.

Table 1. Correlation (as at February 1998) between SCC proofs in foreign countries and the UK

Foreign Country	No. of Bulls	Correlation with UK proof
Canada	46	0.88
France	24	0.95
Germany	40	0.86
Netherlands	66	0.90
USA	36	0.88

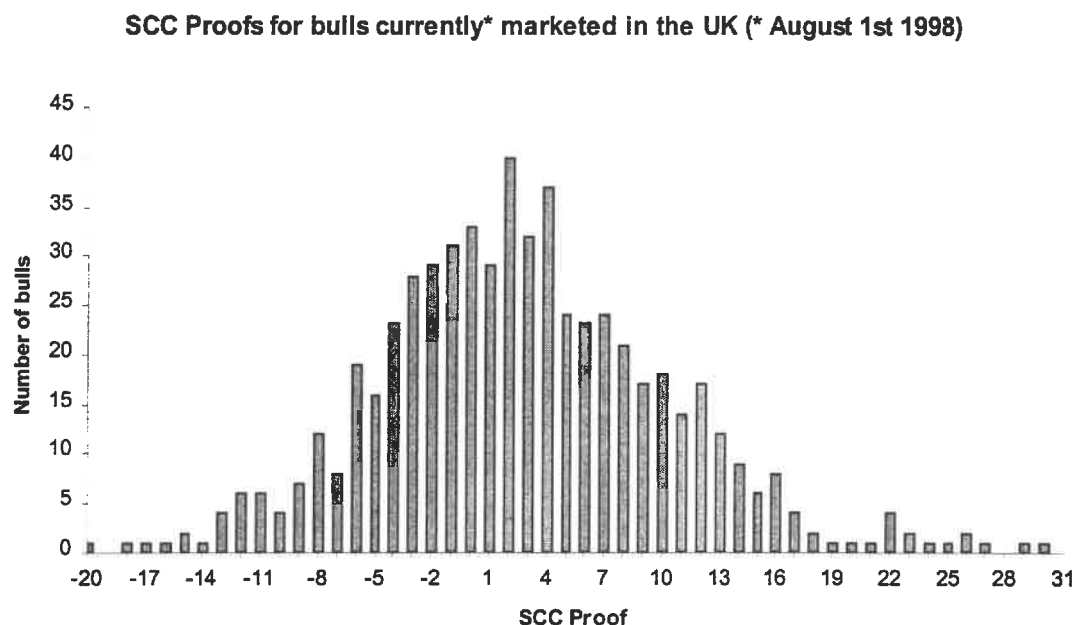
ACCESS TO SCC PROOF INFORMATION

One of the challenges facing the dairy industry is how to get relevant and up to date genetic information out to the potential users. This has become a particular problem, now that proofs are coming out commonly four times a year, from supplying countries whose proof release dates do not always coincide, and where proofs are appearing on a growing and often bewildering array of traits.

An important development has been the creation by the Holstein Friesian Society (HFS) of a web page, where one of the options is a list of bulls currently marketed in the UK, with their up to date proof information. The maintenance of such an accurate bull file has also required considerable input from AI organisations.

There is wide variation in the distribution of SCC proofs for bulls currently marketed in the UK, with either UK or converted proofs. While the distribution is essentially bell-shaped, with most values around the mean, the extreme values vary between minus 20, which we can see as “improvers” for SCC, to plus 30. Thus it is clear AI companies are offering UK farmers a very wide choice as regards genetic merit for SCC.

Figure 1



To assess the likely impact of individual bulls, assume a UK herd which has an average bulk milk cell count close to 200,000 cells/ml, which is close to national average. Against this herd average, bulls with a SCC proof of -15 would be expected to have daughters with average counts of 170,000 cells/ml (15 percent less than the population average). By contrast, the daughters of bulls with a +15 proof might be expected to have an average cell count of 230,000. These figures are approximate in that they assume the herd is close to the national genetic average for SCC, but they indicate the sort of impact that bulls can have on herd cell counts.

CORRELATIONS WITH OTHER TRAITS

A considerable amount of research has been done, to look at genetic correlations between cell count and production, type or disease traits, and especially mastitis incidence.

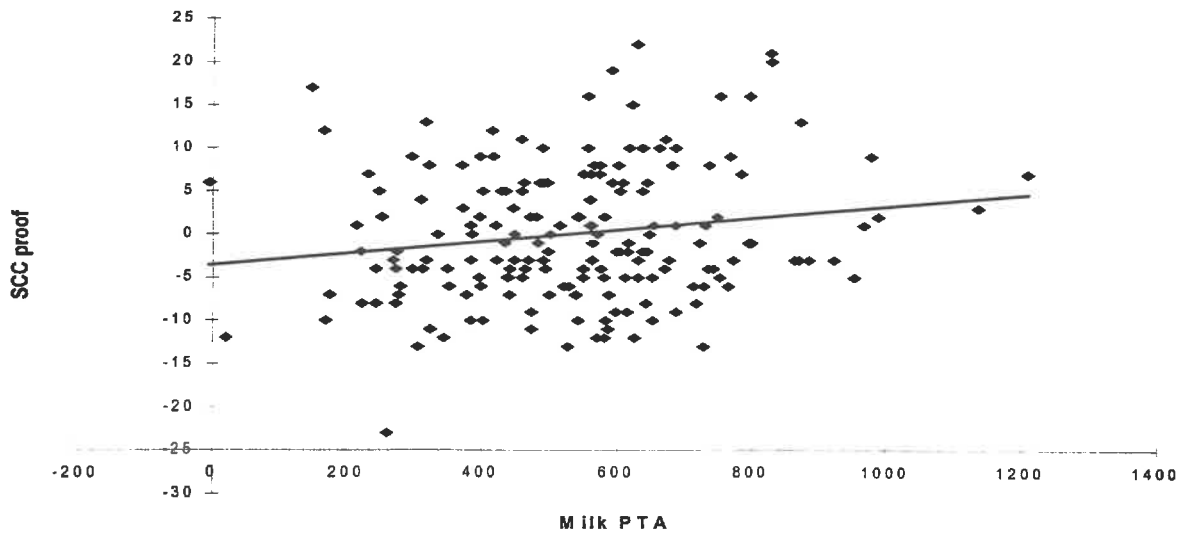
These days, dairy improvement programmes are in the hands of breeding companies, and the major emphasis in most programmes is to increase yield. It is fair to ask what effect this goal might have on cell count.

In almost all studies, higher yields of milk, fat or protein have been found to be associated with higher cell counts. While the correlations are not strong, generally in the region of 0.1 to 0.2, they are quite consistent. Thus a breeding programme geared to improving yields, or a production index such as PIN, would lead to a higher cell count as a correlated response.

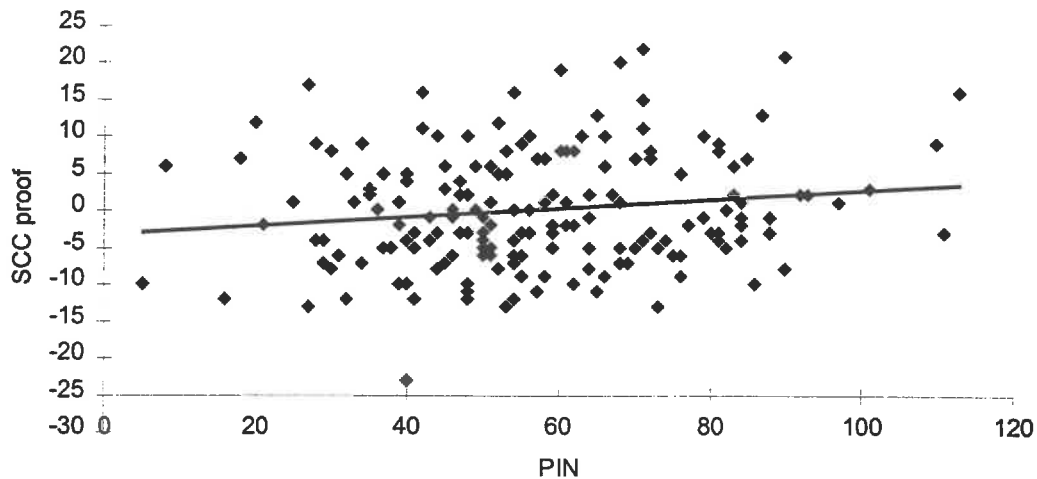
This association between PTA for production and SCC is nicely illustrated in Figs. 2a and 2b, which show associations with PTA for Milk and with PIN for several hundred Genus bulls that were recently progeny tested. The correlations with the SCC proofs are in the range 0.15 to 0.20, which is in line with other research evidence. While the correlation with production is not strong, it is unfavourable. Farmers will obviously have to work even harder on management strategies to keep cell counts at current levels if they select on PIN alone.

Figure 2 Regression of SCC proofs on (a) Milk PTA and (b) PIN

a)



b)



In the UK it has been found (1) that a bull's PTA for SCC is associated with his standardised PTAs for fore udder attachment, udder depth, teat placement side and teat length (Table 2). With sire and dam information available on the trait (SCC) itself, parental assessments on udder and teat traits are probably of limited extra value in

preselecting bulls for progeny testing. Nevertheless it is still reassuring that a favourable (negative) proof is correlated with characteristics thought to indicate sound udders and teats.

Table 2. Correlation between UK bull proofs for SCC and Type Traits

Type Traits	Correlation	Favourable SCC proof associated with
Fore udder attachment	-0.19	Strong attachment
Udder Depth	-0.19	Shallow udders
Teat placement side	0.11	Close teat placement
Teat length	0.13	Short teats

One of the major reasons for an interest in SCC is the high genetic correlation with susceptibility to mastitis. For example, a genetic correlation between SCC and clinical mastitis in Denmark of the order of 0.70 has been reported (2). Given the absence of adequate recording schemes for mastitis in many countries, including the UK, SCC provides an appropriate indirect selection criterion.

Further evidence of this genetic association between SCC and mastitis comes from another recent study (3). This looked at SCC proofs in the US, and the subsequent proofs for clinical mastitis for those same bulls, when their daughters were milked in Scandinavia. The correlations between the proofs were -0.66 and -0.49 in Denmark and Sweden respectively (the negative sign is simply an artefact of how proofs are expressed in the different countries), so that this association spans countries.

HOW MUCH EMPHASIS TO GIVE TO SCC?

In discussing current dairy breeding programmes in the Holstein/Friesian, it is relevant to point out that the direction and pace of genetic changes are determined by selection decisions made by AI organisations. Commercial producers will follow the same general “trajectory”, with some scope for a departure to this pattern, mainly as a consequence of sire selection decisions.

How much attention are AI studs paying to SCC proofs? That is not known at present, but probably not much. The main area of competition between AI studs is in genetic merit of their bulls for production, provided they also meet an acceptable standard for type. The emphasis given to any trait will be driven by market demand. Given the emphasis on production in breeding programmes, it is probable that we can expect a gradual deterioration in genetic merit for SCC.

How much attention should SCC receive in a herd's breeding programme? This should depend both on its economic importance and scope for change, relative to other traits that might be considered.

At present it is necessary to identify the economic importance of SCC. The ways in which it achieves economic significance are clear:

- Through price penalties for milk with high cell counts.
- From the negative association between cell counts and daily milk production (4), presumably because high cell counts indicate mammary tissue damage, which then results in lower production, and
- Through its strong association with clinical mastitis.

While it is possible to arrive at reasonable estimates of these latter two effects, if only from the literature, the benefit of breeding for lower cell counts in terms of milk price penalty depends very much on herd bulk tank counts. In the UK, with current penalty bands, the choice of bull was shown to be of little consequence for herds of very high or very low cell counts (5), as it would not move the herd from one penalty band to another. However, herds with bulk cell counts in the intermediate range, of around 200,000 cells/ml, could benefit substantially from even small changes in cell count, as choosing a bull with a low SCC proof could move the herd to a lower penalty band.

As if this non-linear association was not a sufficient problem, it is necessary to define the economic importance of a trait when daughters start milking, which is about three years after semen purchase decisions are made. It is not easy to predict price penalty schemes for bulk tank SCC that far ahead.

Work on the economic importance of SCC is continuing in the UK within a project on Sustainable Breeding Goals, funded by MAFF, the Milk Development Council and the HFS. The plan is to include SCC information along with production and type proofs, probably into an index such as ITEM, where the aim is a ranking on overall economic performance.

What might be done in the interim? The initial screening of potential AI sires should still be on an index of overall merit, such as PIN or ITEM. Bulls meeting those standards can then be subject to further selection on traits such as SCC, just as they might now for particular type traits, calving ease etc.

HOW FAR TO GO IN REDUCING CELL COUNTS?

The reasons why SCC proofs should be of interest to individual farmers and, more importantly, to breeding organisations have already been outlined. However, a frequently asked question is how far to proceed along that route, and whether in fact breeding for low cell counts may not lead to animals which are immunologically incompetent. Specifically, animals with very low cell counts may in the end have a higher incidence of mastitis.

Several studies from Scandinavia give little support to this concern.

Several years ago, Philipsson, Ral and Berglund (6) looked at Swedish data, where there are good records on both SCC and clinical mastitis, and where bull proofs are calculated independently for these traits. They looked at the regression of the Mastitis Proofs on the SCC proofs, and found a straightforward linear relationship. Within the current population of bulls, selecting those with extreme low SCC proofs would lower the incidence of mastitis in the next crop of daughters.

Similar evidence along the same lines has now emerged from a recent study (3). This also looked at the regression of Mastitis proof on SCC proofs. However, in this case the Mastitis proofs were either Swedish or Danish, while the SCC proofs were from the US. The 80 bulls included in the study were all born and tested in the US, and subsequently sired Scandinavian daughters when marketed there.

Both of these studies indicate that selecting sires with favourable SCC proofs should reduce both SCC levels and the incidence of mastitis in their daughters. Of course it is still possible that individual cows with extremely low SCC may be particularly susceptible to udder infection. But at the level of daughter average performance, which is all that a sire proof describes, this is certainly not the case.

ACKNOWLEDGEMENTS

Raphael Mrode of the ADC provided useful discussions and Matthew Winters helped with the HFS web site. The R and D Project on Sustainable Breeding Goals is jointly funded by the MDC, MAFF and the Holstein Friesian Society.

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MILKING MACHINE ASPECTS

MACHINE MILKING AND THE WELL-BEING OF THE DAIRY COW

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SUMMARY

A number of observable changes in cow behaviour during milking and visible changes in teat condition immediately after milking have been noted. They are probably related to a combination of milking conditions, equipment and operator technique. There is a need to understand the causes of these changes in order to evaluate the effects on the well being of the dairy cow.

In a series of on farm observations it was noted that heavy milking clusters and the associated technology gave more complete milking out and less liner slip but at the cost of an increase in agitation of the cows seen as fidgeting, unsuccessful attempts to remove the cluster by kicking and increased defecation and urination during the period of cluster attachment. Similar changes were observed with light clusters when pulsation faults were diagnosed. Indexing of cows in parlours produced benefits in terms of cow loading, cow position and cluster position during milking.

Under 'normal' conditions cows milked with the heavy cluster were more likely to exhibit red or blue teats with a palpable ring at the base of the teat after cluster removal.

INTRODUCTION

International standards have recently been revised by experts from 15 countries on behalf of the International Standards Organisation and these describe minimum specifications of design, installation, maintenance and testing of milking machines (1). The standards are not mandatory and in some countries variations in interpretation and even disagreement can lead to differences in operating conditions of the milking machine.

One type of milking machine has many features which are different from all other types. The most significant differences are a smaller claw bowl volume (150 ml), a narrower long milk tube diameter (14 mm), greater cluster weight (>3.2 kg), simultaneous pulsation with no ACRs used on a midi - line plant milking at a recommended system vacuum of 49 kPa.

An independent field study was undertaken to compare this milking philosophy with systems more frequently noted. These results can form the basis for practical advice to dairy farmers. Effects on cow behaviour, milking performance and teat condition were recorded. The comparisons have been made in line with International Dairy Federation recommendations (2).

METHODS

Agreement to co-operate was obtained from twenty farms to allow inspection of the milking equipment and observation of the milking within six months of installation with a second visit six months later. All the milking machines were intended to be compliant with the 1996 ISO standard (1). Twelve of the plants were fitted with a light cluster (approx. 2.4 kg) with a large claw bowl volume, 16 mm diameter long milk tube, alternate pulsation and milked with a system vacuum of 41-47 kPa. The other eight plants were of the alternative type using a heavier cluster.

Thirteen of the plants assessed were constructed with some degree of cow indexing, while seven of the plants were un-indexed having a straight breast and rump rail.

On the first visit a full static test was performed in accordance with ISO 6690 (3). The plant was fully inspected and the whole afternoon milking observed. An examination of the orifice of all teats of 50 cows was made during the visit for teat orifice hyperkeratosis (4).

Following the first visit all farmers received a verbal and written report of maintenance problems or major faults. Only a small number of selected items on a few farms were addressed before the second visit (5). The detailed assessments reported are therefore for parlours operating to the farmers selected criteria and reflect normal operating conditions and applications of the standards in practise.

During the second visit, full observations were made of the performance during milking. This included scoring on a basis of zero (could not be improved) to three (could not be worse) various parameters including cow entry, cow positioning, cluster positioning, liner slippage, cow behaviour during milking, cow behaviour during teat inspection and completeness of milking.

A more detailed assessment of teat condition of 50 or more cows on each farm was made. The most significant parameters of colour, response to touch, palpable ringing at the base of the teat and degree of openness of orifice were recorded after cluster removal.

RESULTS AND DISCUSSION

The total and average scores for each parameter assessed during milking are shown in Table 1, sorted according to relative cluster weight or presence of indexing. Lower scores indicate better performance.

Table 1. Total and average parlour performance sorted by cluster weight and indexing of cow position

	Cluster weight				Indexing of cow position			
	Light		Heavy		Indexed		None	
Parameter	Total	Average	Total	Average	Total	Average	Total	Average
Cow entry	11	0.9	10	1.3	11	0.8	10	1.4
Cow position	8	0.7	10	1.3	3	0.2	15	2.1
Cluster position	13	1.1	11	1.4	13	1	11	1.6
Cow behaviour								
During milking	4	0.3	12	1.5	11	0.8	5	0.7
After milking	12	1	11	1.4	15	1.2	8	1.1
Liner slip	8	0.7	3	0.4	9	0.7	2	0.3
Milking completeness								
High yielders	5	0.4	0	0	5	0.4	0	0
Low yielders	14	1.2	3	0.4	7	0.5	10	1.4

Cow entry and loading were achieved more easily into an indexed parlour. It was common in non indexed parlours for the operator to leave the pit to bring cows from the collecting yard. Eleven of the farms used a crowd gate which eased loading. Cow position during milking was better in indexed parlours. Although cows appeared to be better positioned with light weight clusters, this effect is partly related to these parlours being more likely to have indexation and usually having a milking point per standing with the cow standing adjacent to the available cluster.

Cluster position during milking was better in indexed parlours as the cow position was more controlled. A heavy cluster did not ensure an adequate cluster position. Cluster position with heavy clusters was adversely affected in many cases by excessive length of long milk tube and position of the long milk tube riser.

Cows milked with a heavy cluster showed more agitation during milking than cows milked with a light claw. The behaviour was marked and included at least one cow in twenty defecating or urinating during milking and at least three cows in twenty stepping or kicking at the cluster. This effect may be related in part to average system vacuum applied (45 kPa for light claws and 47.3 kPa for heavy claws). Exceptions were noted with certain operators where overmilking was minimal.

The effect of cluster weight on cow behaviour after milking was less marked. Cows milked with the light claw were generally more amenable to handling although behaviour was more variable in this group.

Less liner slip was observed with heavy clusters.

Heavy clusters achieved more complete milking especially in low yielding (late lactation) animals. It was difficult to discover if only completion of milking was being achieved or to what degree over milking might have occurred. No determination of over milking was made although occasional cluster-on times of 15-20 minutes were observed in several parlours using heavy clusters. Extended unit on time was more noticeable when the operator had to leave the pit to load cows from the collecting yard, ensure the correct number of cows were loaded or spend time on teat preparation before cluster attachment. The under milking of low yielders noted with light clusters tended to be predominately front quarters. This may be a product of poor cluster position and poor end of milk flow determination.

Clusters and liners can only achieve complete milking and low strip yields under optimum conditions of cow behaviour and cluster position. Table 2 clearly shows a reduction in completeness of milking as cluster position deteriorates.

Table 2. Relationship between completeness of milking and average cluster position for High (H) and Low (L) yielding cows

		Completeness of milking score			
		0	1	2	3
Cluster position					
	0	1L			
Heavy	1	7L, 5H	2L		
	2	1L, 2H	1L		
	3				
	0	1L			
Light	1	4L, 4H	3L		2L, 2H
	2	3H		1L	1L
	3				

The physical condition of the teats after milking is related to the operating conditions within the milking cluster. The most significant of these are likely to be milking vacuum, cluster weight, liner type and duration of cluster attachment.

There are several easily observable changes to cows teats that occur during milking. These changes are transient but may be useful indicators of milking conditions within the clawpiece. These can be observed and broadly quantified without disruption to the milking routine.

Teat colour changed rapidly with time after cluster removal except where the teats showed a normal pink colour. Eleven of the twelve farms using a light claw recorded less than 50% of cows showing red or blue teats. Seven of the farms with the light claw had less than 25% of cows showing red or blue teats. In contrast, seven of the eight farms using a heavy clawpiece

recorded between 76 and 100% of cows showing red or blue teats. The remaining farm using the heavy clawpiece showed only 59% of cows exhibiting red or blue teats. This farm employed an efficient milking routine with less units installed therefore reducing the opportunity for over-milking.

Teat discolouration immediately after cluster removal could indicate impaired circulation and constriction to fluid transfer. It is not possible to say categorically whether this is causing pain or discomfort although experience sleeping on an arm would suggest the sensation is far from pleasant.

Wide variation was found between herds in the proportion of teats with a palpable ring at the base of the teat. 23% of teats showed no mark while more than 60% had a palpable ring. A number of farms using the heavy clawpiece had more than 90% of examined teats showing a palpable ring. This ring indicates localised oedema which accumulates during milk extraction and can take many minutes to disperse.

The exact significance of this oedema is not known although it has been suggested that if the ring develops early in the milking, milk flow can be adversely affected. One farm with a light weight claw was using a liner which caused severe ringing. When the liner was replaced and the ringing removed, daily yield increased by 300 litres. There was a consistency in scores for teat colour and palpable ringing at the base of the teats of cows milked with the heavy clawpiece.

Teats which are unresponsive to touch can also indicate impaired circulatory action. After milking teats should remain soft and tactile. While 75% of teats examined were normal, 24% of teats examined were considered firm and 54% of teats on one farm were hard and 'wooden'.

CONCLUSIONS

The objective of any milking machine, irrespective of manufacturer or configuration, must be to milk cows safely, gently, quickly and completely. The results of this field study provide further evidence that the milking process and its interaction with the cow is complicated and multi factorial.

While it is impossible to be precise as to when milking causes pain or discomfort to an animal, there are a number of key parameters which should give an early indication of an animal being milked in less than optimum conditions.

Many of the reported observations relating to the heavy clawpiece including increased agitation towards the end of milking, dunging and urination, higher proportion of red and blue teats and palpable ringing at the base of the teat were rare on farms using the heavy clawpiece where a smooth milking routine was employed or fewer units were installed per operator. This suggests the compounding effect of over-milking must be considered.

As herd size increases, there is a move towards more units per operator to improve efficiency. However, this increase in throughput must not be at the cost of a reduction in the well being of the dairy cow.

The market place increasingly demands that the industry responds to changing welfare requirements. An awareness from operators of the influence of the milking machine on sensitive teat tissue and how some of the highlighted effects may adversely affect the well being of the dairy cow will allow the UK dairy industry to maintain it's competitive edge.

ACKNOWLEDGEMENTS

The forbearance of the twenty farmers who co-operated, and their staff, has been gratefully appreciated. The study was funded and supported by the Milk Development Council.

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MILK HARVESTING SYSTEMS FOR HIGH-PRODUCING COWS

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SUMMARY

The principles for milking the high-producing cow are the same as for any other dairy cow: her teats should be clean and dry for milking; she should be milked gently, quickly and completely with minimal machine stripping or overmilking. However, these principles need to be applied in different ways because high-producing cows have: 1) a lower pre-milking stimulus requirement compared with low-producing cows, 2) higher peak milking rates and higher average flowrates (in spite of this, high producers take longer to milk), 3) poorer teat-end condition, 4) higher risk of new mastitis infections.

Although less time and effort is needed for deliberate manual stimulation in high-producing herds, there is a progressively greater need for meticulous care and attention to ensure teats are clean and dry before milking to minimize the increased risk of mastitis. Healthy teat skin provides the best defence against all types of mastitis pathogens. Teat condition can be improved, and milking times per cow reduced by use of excellent milking procedures and adjusting the take-off settings of automatic detachers.

The new performance-based ISO international standards for milking installations can be used, with confidence, to evaluate the "system" components of existing milking systems. The results of milking-time tests (measurements or observations made while milking cows) are the best and most direct indicator of the performance of milking units.

INTRODUCTION

This presentation is a reappraisal of a paper produced for a seminar on "Managing the 30,000-pound Herd" organized by the American Dairy Science Association in 1992 (1). At that time, it was stated that existing national and international standards for milklines and milking units might not be adequate to cope with the higher milk flow rates from high-producing herds. Compared with the challenges of feeding and breeding management for a herd averaging 45 kg milk per cow per day throughout lactation, however, it was concluded that milking was likely to be the easiest part of managing such a herd. The top cows in excellent herds were producing at or above this level six years ago, and they were being milked successfully.

A number of interesting papers on the science or practice of milk harvesting have been published in the intervening six years. The new information supports some of the conclusions presented in 1992 but other conclusions need to be modified now.

What's new since 1992? This paper provides a brief outline of the advances in knowledge, experience and understanding which support or challenge the 1992 recommendations and which will continue to change milk harvesting practices and systems.

Pre-milking udder preparation

In 1992, it was concluded that present-day, high-producing Friesian/Holstein cows appear to need little or no manual stimulation to maximize their milk yield. Because the "half-life" of oxytocin in the blood is about 4 min, oxytocin concentration is seldom a limiting factor. Therefore, good pre-milking udder preparation should ensure that teatcups are applied to visibly clean dry teats that are "plump" with milk, with a minimum of time and effort.

Subsequently, an excellent review and analysis of research herd studies in the US and Europe by Reneau and Chastain (2) concluded that:

- although some cows may come into the parlour dripping with milk, less than 10 s of cleaning and teat manipulation is not an adequate stimulus for consistent letdown response in all cows, especially those in late lactation;
- a teat cleaning and drying procedure that results in a quality stimulus of 10-20 s is adequate to sanitize teats and achieve consistent milk letdown in most cows ;
- the optimum "window of time" to apply teatcups is 60-90 s after the cow's teats and udder are first touched by the milker (commonly referred to in USA as the "lag-time"). This window allows time for milk letdown to occur in most cows and makes optimum use of the milk ejection hormone.

Analysis of the combined data from five independent US studies (2) indicated that a stimulus of 10-20 s per cow and a time-delay of 60-90 s after the teats are first touched by the operator reduced average milking time per cow by 0.6 min (a gain of more than 10%), increased mean milk yield per cow by 0.3 kg/milking (a gain of 1%), and resulted in optimum parlour throughput. Reducing the machine-on time has important consequences for improved teat condition, as we shall see later.

Put Cups on when Teats become Plump with Milk: Picking the best time to attach teatcups has benefits for cleaner quicker milking out, improved teat condition and slightly higher milk yield per cow (2,3,4). Putting cups on too soon usually results in teatcup crawling during the first minute of milking. Teatcups will crawl higher up the teats if milk flow slows or stops at this time because "drainage" milk has been removed from the udder cistern and teat sinuses before the main milk is ejected from the alveoli into the milk ducts and cisterns. When teatcups crawl early in milking, then milk harvesting is less complete and less efficient near the end of milking because the milk pathways between the cistern and teat sinus become more restricted more quickly.

In many herds milked with an otherwise good milking routine, the simplest way to match the timing of cup application with milk ejection would be to delay the time of cups on by 30-60 s. On rotary platforms, this change might require nothing more than moving the cups-on operator to a position about 60 s past the cow entry point.

As a simple check, watch the claw bowls during the first minute of milking. When teatcups are applied too soon, milk flow into the claw bowl typically slows or stops after about 15-20 s of initial flow, then full flow does not start (or restart) until about 1 min after cups on.

Peak milking rates, average flow rates, and average milking time per cow

Contrary to the expectations of many farmers, it is not reasonable to expect high-producing cows to milk out in 5 minutes flat. Although peak milk flow rates for top cows in early lactation averaged 5.5-5.7 kg/min, their average milking times were relatively slow. Top cows milked twice per day took nearly 10 min/cow to milk (average yield = 25 kg/milking) while the group milked thrice daily took almost 7 min for an average yield of 16 kg/milking (1).

Other studies published since 1992 (2,5,6,7) provide much better evidence for the broad guideline that:

- cows with an average yield of 10 kg milk per milking should milk in about 5 min (plus or minus 1 min),
- cows averaging 15 kg milk per milking should take about 6 min (plus or minus 1 min),
- add about 1 min to average milking time per cow for each additional 5 kg of milk per milking.

If teatcups are applied too soon, before milk letdown occurs, these guidelines for typical average milking times should be extended by about 1 minute.

Teat condition

Healthy teat skin provides the best defence against all types of mastitis pathogens. Furthermore, smooth healthy teat skin is easier to clean and easier to keep clean compared with rough or damaged teat surfaces. In 1992, comprehensive data from Sieber (8) was used to illustrate the general deterioration in teat end condition associated with increasing 305-day milk production. Cows which produced less than 5500 kg of milk in a 305-day period had a much higher proportion of normal teat ends (30%) than higher producers. The proportion of teat ends classified as "normal" fell to less than 9% for cows which produced more than 8200 kg of milk in 305 days. Sieber's results showed that the severity of teat end abnormalities increased and the proportion of normal teat ends decreased as the average milking time of individual cows increased from 4 min or less to 6 min or more.

Based on these highly significant changes, poorer teat end condition seemed to be an unavoidable and inevitable consequence of milking high-producing cows (1).

This conclusion was premature! A Danish study (9) has provided the springboard for a "quantum leap" towards better teat condition for high-producing cows milked in US dairy herds. Milking time was reduced by 0.5 min per cow with no loss of milk yield when the end-of-milking setting for automatic cup removers was raised from a flow rate threshold of 0.2 to 0.4 kg/min. Teat condition improved markedly in the early detachment group of cows. Incidence and prevalence of sub-clinical mastitis were not affected but significantly fewer cows in the early detachment group developed clinical mastitis (9).

During the past 2 years, the threshold flowrate has been increased from a default setting of 0.3 kg/min (for BouMatic detachers) to 0.4 kg/min for herds milked twice per day, and to levels as high as 0.7 kg/min for some herds milked thrice daily. At the same time, the typical setting

of 13 s time delay for cup removal has been shortened to 0-5 s. The net effect, in the commercial herds involved in the field study, has been to reduce milking times by up to 1 min or more per cow with no loss of milk yield, no change in SCC or mastitis levels. In addition to quicker milking, the major benefits have been improved teat condition and calmer cows, especially the fresh cows. The most surprising discovery of this on-going experimental work has been to find so little milk left in a typical udder following early removal. For example, average strip yield is a mere 25 ml per cow in the herd with the highest threshold setting of 0.7 kg/min and 3 s delay time.

It is tempting to conclude that cows milk out more quickly and completely if the teat tissues can be maintained in a soft, supple and compliant condition. It is likely that other factors have contributed to these excellent results, however. To date, the results have been obtained in herds with good pre-milking teat preparation, calm consistent milking routines, narrow-bore liners, and milking units that are positioned carefully on the udder by the operator(s) at the start of milking.

New mastitis infections

A simulated genetic selection study in Germany indicated that incidence of mastitis will increase with increasing milk production (10). Given the present rate of genetic gains for milk production, new infection rates will increase by about 1% per year without positive selection for udder health in a progeny-testing program (M. Goddard, personal communication).

Many factors contribute to an increased infection risk for high-producing cows. Apart from the greater disease risk associated with their faster metabolic rates, the teats of these top cows tend to leak more easily and more frequently when they walk or when they lie down. Leaky teats probably result in higher infection risk. Results of a series of milking experiments involving high bacterial challenge showed clearly that cows which milk faster have a higher risk of infection (11). These results supported earlier work showing that cows with more patent teat canals also had a higher risk of infection during their dry periods (12). Thus, high-producing cows may have a higher rate of new infection because of the indirect relationship between high production and high milking rates (i.e. more "open" teat canals).

Another contributing factor may be that liners slip more frequently on high-producing, tight-uddered cows (13). It is well-known that liner slip is linked with increased rate of new mastitis infections (14). Furthermore, work in progress in The Netherlands suggests that the rate of clinical mastitis may be higher in cows with poorer teat-end condition. Teat end condition deteriorates as milk yield increases (8) unless the settings for automatic cup removers can be optimized (9).

IMPLICATIONS FOR DESIGNING MILKING HARVESTING SYSTEMS

Units per operator

The likely combination of longer milk-out times and shorter pre-milking udder preparation times in high-producing herds means that milking systems should be designed with more units per operator for more efficient labor utilisation. The effects of daily milk production on cow throughput are illustrated in the modeling results of Reneau and Chastain (2). If milk yield is increased from about 14 to 18 kg per cow per milking, for example, the predicted steady

state throughput falls from 68 to 61 cows per hour in a Double-8 parlour, from 80 to 71 cows per hour in a Double-10 parlour, and from 91 to 82 cows per hour in a Double-12 parlour.

In high-yielding herds, highly automated parlours should be planned to provide 20-24 units per operator, i.e. a D-10 or D-12 for 1 operator, or a D-20 for 2 operators. Parlours with one unit per stall are preferred for high-producing herds so that low-level milklines can be installed. The basis for this will be explained later.

The "system" components of a milking system

In the six years since 1992, national and international standards for the construction and performance of milking machine installations have been revised extensively. The revised standards incorporate new performance specifications to provide a common basis for evaluating the great variety of types and sizes of milking systems used throughout the world. For the first time, the main performance specifications are the same for international (15) and American (16) standards. The new performance criteria can be used, with confidence, to evaluate the "system" components of existing milking systems. For designing new installations, each of these standards includes guideline tables of recommended sizes for system components such as vacuum pumps, airlines and milklines. Because of subtle variations in the specified design criteria, some of the guideline tables in the ISO and ASAE informative annexes differ markedly.

These new performance-based standards have led to cost-effective improvements in milking and cleaning performance in many milking systems. In general, vacuum pump capacities, airline and milkline sizes have been reduced in USA but increased elsewhere. In USA, fewer 100 mm (4 inch) milklines are being installed and significantly lower amounts of hot water and chemicals are required to clean milking systems that have been correctly designed. In existing installations, the common problem of inefficient vacuum regulation has been resolved by eliminating unnecessary vacuum pump capacity and by mounting the regulator sensor closer to the sanitary trap.

What is the "right" vacuum level for milking?

According to guidelines given in ISO 5707, the system vacuum should be set to achieve a mean claw vacuum within the range 32-40 kPa during the period of peak milk flow for a representative sample of cows. A recent Danish report on milking at lower vacuum, using mean claw levels within the range 26-39 kPa (17), concluded by endorsing the ISO guideline. On the other hand, market pressure from US dairy farmers is for higher vacuum levels so they can milk more cows per hour and per day. To increase milking speed, some US veterinary consultants are now advising their farmer clients to set the operating vacuum for a milking system with a high-level milkline to 54-57 kPa so that the mean claw vacuum is maintained close to 40-42 kPa.

It is common knowledge that increasing the system vacuum level results in faster milking times. Carefully conducted research studies have shown that milking can be successful with vacuum settings as high as 70 kPa. An added bonus is that the frequency of liner slip is reduced as vacuum level is raised. On the other hand, outbreaks of clinical mastitis, high cell counts, or poor teat condition frequently are linked with an unsuspected high vacuum problem on dairy farms. It is necessary to reach a compromise between machine settings for fast

milking and for maintaining healthy teats and udders because the benefit of higher vacuum level may be offset by higher strip yields, higher incidence of teat end abnormalities, and more machine-induced teat congestion and oedema.

Dealers are understandably cautious about implementing advice from a veterinary consultant to set the system vacuum above 50 kPa. Generally system vacuum should be set so that mean claw vacuum during peak milk flow is about 40-42 kPa to milk cows as quickly as possible while still maintaining gentle milking conditions. Nevertheless, provided that cow preparation procedures are excellent and the milking system is adjusted to minimize the low flowrate period of milking, it is possible to experiment with raising the mean claw vacuum level above 42 kPa. This implies that parlors with low-level milklines could be set to a system vacuum above 45 kPa and highline systems could be set above 50 kPa under certain circumstances. Individual milking systems should be modified, before raising the system vacuum, according to the following steps.

- a) Reduce the vacuum drop between claw and milkline. Low milking vacuum results from problems such as excessive milkline height, restrictions in the milk tubes, excessive vacuum drop across ancillary components, blocked air vents, excessive air admission through air vents or air leaks into the cluster. Raising system vacuum to compensate for unnecessarily high vacuum drop between the claw and milkline results in a greater increase in claw vacuum when milk flow falls near the end of milking, which is the riskiest time for teat damage, discomfort and new mastitis infections. Therefore, the aim is to keep the milkline as low as practicable. The implication is that low-level milklines are preferred for milking high-producing herds. Furthermore, milk tubes should be as short as possible and any kinked or flattened hoses replaced because they cause slightly greater vacuum drop and slightly slower milking.
- b) Minimize detacher take-off delays (preferably less than 5 s) and then optimize the flowrate threshold (that is, reduce the resistance setting). Both scientific evidence and recent field experience suggest that changing the detacher settings will produce much bigger improvements in teat condition and milking speed, compared with the effects of raising the system vacuum or increasing the diameter of the milk hose. When changing detacher settings, remember to monitor strip yields by hand-stripping a representative group of cows before and after making changes.

When the detacher settings are optimized for a given farm, the system vacuum could be raised about 1-2 kPa per week. The system vacuum should NOT be set above 50 kPa if detachers are not installed and/or not optimized and if milking procedures are inadequate.

The veterinary consultant should be responsible for deciding if pre-milking hygiene and management procedures are adequate to reduce the greater risks associated with milking at higher vacuum level.

A competent observer (the dealer or the veterinary consultant) should be responsible for monitoring any changes in the mean strip yields per cow within one week after the system

vacuum is raised, and watching for any changes in teat condition during the month after such a change.

Performance of the "milking unit" components of a milking system

The new ISO and ASAE standards become less specific as they get closer to the "cow end" of the machine. There are no specifications for liner bore, effective liner length or liner tension, for example, and no guidelines for weight of the cluster or uniformity of weight distribution between the four teatcups. Similarly, field tests of milking systems usually stop short of any systematic analysis of performance of milking units because most of us tend to measure the things which are easy to measure (such as vacuum pump capacity, system vacuum level or vacuum stability) rather than those which may be more important. As a result, "milking is not going well in some modern milking installations which meet or exceed International Standards Organization standards" (18).

Biological responses of the teats and udder to the milking unit and/or operator.

Details of four of the most practical and useful short-term measures of performance of the milking unit have been described (19). These measures are:

- Mean milking time per cow (relative to the mean yield per cow per milking).
- Frequency of liner slips and falls requiring corrective action by the milker (per 100 cows).
- Amount of available milk left in the udder when cups are removed (mean strip yield/cow).
- Teat condition scored just after milking.

Ideally, teats should be as soft and supple just after milking as before milking. Usually, teats are thicker after milking with wide-bore liners, or after milking at a high vacuum level (20). Teats that are slightly swollen or hard after milking (due to congestion or oedema), or slightly blue or purple in colour (cyanotic) result from machine-induced circulatory impairment. A simple procedure has been developed by for systematic measurement of short-term changes in teat colour, teat firmness, swelling of the teat base, and degree of closure of the teat orifice after milking (21).

Behavioural responses to milking.

Frequency of flinching, stepping or kicking (the "FSK" response) has been proposed as an indicator of comfort/discomfort while the milking unit is on the cow. Preliminary results of cow behaviour in 20 UK parlours indicate that differences in cow behaviour between parlours can be assessed by systematic observation of dunging/urination, paddling/stepping, cow reaction to having her teats touched after milking, and cow entry/exit times (22). The trickiest part of this type of study is the difficulty of distinguishing between environmental effects (such as flies or stall design), operator effects, interactions between machine and operator, and milking machine effects. Furthermore, results in commercial herds will depend to a large extent on whether cows are accustomed to having their teats touched after milking.

Mean claw vacuum measured with an artificial udder as a flow simulator.

A flow simulator (23) provides an easy, convenient and reliable method of measuring mean claw vacuum, and vacuum drop through the milk hose and through ancillary equipment such

as sensors and milk meters. Results can be compared with ISO specifications for the maximum additional vacuum drop caused by fittings in the milk hose, i.e. not more than 5 kPa at a milk flow rate of 5 kg/min and an airflow of 8 l/min. Furthermore, results can be compared with the mean claw vacuum recorded during milking to estimate the peak milk flow rate of individual cows.

No claims are made for the accuracy of, or the implications of, claw **vacuum fluctuations** measured with the flow simulator (23). The over-riding effects of tiny air leaks past the artificial teats makes such measurements misleading, frustrating and unreliable.

Changes in vacuum measured in the liner mouthpiece chamber.

High mouthpiece vacuum is considered to be uncomfortable for cows but there is not much published information. Recent results (24) link high mouthpiece vacuum to increased teat thickness measurements, poorer cow behaviour and higher incidence of clinical mastitis.

Uniformity of weight distribution between the four teatcups on an artificial udder.

Uneven weight distribution between the four quarters of an udder is one of the most common causes of incomplete milking, uneven milk-out, and liner slips. Ideally, the milking unit should hang squarely on the udder so that about 25% of the total cluster weight is applied to each udder quarter throughout milking. This rarely occurs in practice (22). Incomplete milking has been related to light cluster weight, poor cow position, poor cluster positioning, and lack of indexing for cows in the parlour (22).

CONCLUSIONS AND RECOMMENDATIONS

The new performance-based ISO international standards for milking installations can be used, with confidence, to evaluate the "system" components of existing milking systems. However, these new standards and test procedures are less useful for evaluating the performance of the milking unit. This is partly because most of the ISO test procedures are conducted as "dry tests", that is, tests conducted with the machine running but not milking, and with only air flowing through the machine. This type of test has been described loosely, but incorrectly, as "static testing".

The results of milking-time tests are the best and most direct indicator of the performance of any milking system. Milking-time tests describe measurements or observations made while milking cows. Immediate additional testing and service is recommended if:

- cows appear to milk slowly, unevenly or incompletely;
- teatcups slip or fall frequently;
- teat condition is poor;
- or if cows appear nervous or uncomfortable.

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POSTERS

SURVEY OF CLINICAL MASTITIS INCIDENCE

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The clinical mastitis incidence in 269 herds in England and Wales and possible factors related to this were investigated.

This survey was carried out between October 1996 and September 1997 and involved herds subscribing to the Axient mastitis consultant service. Approximately 30 herds, with varying cell count results and clinical mastitis incidence, were selected from each of 10 geographical regions in England and Wales. All herds had 12 months continuous data, used dry cow therapy on all cows and were not housed continuously.

Each area of data was sub-divided, in order to analyse the data, to obtain the relevant results. The specific objectives were tested by using Anova tables and then with a Student's *t* test.

The average clinical incidence was 34 cases per 100 cows per year, with individual herds ranging from 2 to 103. Average herd size was 108 with a range from 16 to 386.

The arithmetic mean of the monthly geometric bulk milk cell count was 184('000 cells/ml), with a range from 48 to 470 and average bactoscan 33('000 impulses/ml), with a range from 8 to 124.

The small herds (below 50 cows) had the lowest clinical incidence, but there was no statistical significance between herd size and clinical mastitis incidence.

Table 1. Clinical incidence rates in relation to herd size groups

Herd size	No. of herds	Mean rate	Range	Mean bulk milk cell count ('000 cells/ml)
0-49 cows	34	30	2-103	230
50-99 cows	99	33	3-99	193
100-149 cows	77	35	4-92	173
150 + cows	59	34	1-67	158

Most clinical cases occurred in January, and fewest in June, a significant difference.

The clinical mastitis incidence was significantly higher ($P < 0.01$) for the months when cows are generally housed (October to April) than for when the cows are usually at grass (May to September). No adjustments were made for the month of calving.

There were no significant differences in incidence for herds in the different cell count bands, except for those herds greater than 300,000 cells/ml which had a significantly higher clinical incidence ($P < 0.01$).

Table 2. Clinical incidence per 100 cows per year by average cell count bands

Cell count range '000 cells/ml	0-99	100-149	150-199	200-249	250-299	300+
No. of herds	28	71	75	52	20	23
Clinical incidence/100 cows/year	31	34	33	32	32	41

There was no statistical relationship between bulk milk bactoscan and clinical incidence. The herds were grouped into four categories according to their winter housing type:- cubicles which included eight with kennels, yards, byres, and a combination of cubicles and yards. Byres had the lowest clinical incidence but there were not enough herds for the difference to be statistically significant. There was a highly significant difference between the mastitis incidence in cubicles and yards ($P < 0.001$), with the clinical incidence 11 cases/100 cows lower in cubicle housing.

The clinical mastitis incidence was significantly lower ($P < 0.001$) in the far south west of England (25 cases/100 cows/year) and significantly higher ($P < 0.001$) in the south east (46 cases/100 cows/year). This appears to be related to housing. In the south east, 75% herds were housed in yards and 19% in cubicles whereas in the far south west, 73% herds were housed in cubicles and only 24 herds in yards.

Acknowledgements

To the Milk Development Council, Axient mastitis and hygiene consultants and Mark Scott.

CLINICAL MASTITIS IN SIX SOMERSET DAIRY HERDS

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Herds were selected on the basis of location (Somerset), bulk milk somatic cell count (<200,000 cells/ml), a non-seasonal calving pattern and likelihood of owner compliance with the study protocol. A mastitic quarter was defined as a quarter with an abnormal secretion with or without signs of inflammatory udder changes. These quarters were sampled by the herds person, following training. All repeat cases (defined as a quarter case more than 5 days after a previous mastitis in that quarter, in which the milk had returned to normal in the interim) were sampled. Sampling has been carried out for a period of one year to date and is ongoing.

Samples were submitted to Langford VIC for bacteriology. Organisms were identified and quantified using standard laboratory techniques.

If an organism was isolated in pure growth, or was the predominant growth with little growth of other organisms, it was considered to be the cause of mastitis. A sample was labelled 'contaminated' if there was a mixed growth including environmental/faecal organisms. A sample was called a 'mixed growth' if there was a significant growth of 2 or 3 known mastitis pathogens.

Some 302 cases of mastitis in 810 cows occurring between 1 April 1997 and 31 March 1998. The causes of mastitis, in decreasing order of incidence, are shown in Table 1.

Escherichia coli was the most significant cause of mastitis. It was responsible for 32.8% of mastitis cases in which a pathogen was identified. Enterobacteriaceae were responsible for 40.5% of all mastitis. Table 2 shows that significantly more *E. coli* mastitis occurred from July to March than April to June ($p < 0.05$) and significantly more cases of mastitis occurred in hind (60.4%) than fore (39.6%) quarters ($p < 0.05$).

Table 1. Mastitis Incidence

Pathogen	Cases/100 Cows/year	% Total Isolates
<i>E. coli</i>	12.2	32.8
<i>S. uberis</i>	4.6	12.1
<i>S. dysgalactiae</i>	1.7	4.6
Coag +ve Staphylococci	1.7	4.6
Coag -ve Staphylococci	1.6	4.3
Corynebacteria spp.	1.5	4.0
Klebsiella spp.	1.1	3.0
Serratia spp.	0.9	2.3
Bacillus spp.	0.9	2.3
Yeast spp.	0.6	1.7
Pseudomonas spp.	0.5	1.3
<i>A. pyogenes</i>	0.5	1.3
Citrobacter spp.	0.1	0.3
Morganella spp.	0.1	0.3
Enterobacter spp.	0.1	0.3
<i>S. agalactiae</i>	0.0	0.0
All Enterobacteriaceae	15.1	40.5
Mixed Growth	1.7	4.6
No Growth	6.5	17.5
Contaminated Samples	0.9	2.3
Total	37.3	

The seasonal incidence is in Table 2.

Table 2. Number of cases of mastitis by season

	April-June	July-September	October-December	January-March
All mastitis	52	72	87	92
<i>E. coli</i>	11	26	39	23

LONGITUDINAL STUDIES OF CONTAGIOUS MASTITIS IN A HIGH SOMATIC CELL COUNT DAIRY HERD

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Investigation of a sixty cow dairy herd with persistently high bulk milk somatic cell count (BMSCC) was initiated following notification to the farmer by the milk purchaser of possible termination of the milk contract if the BMSCC were not reduced to an acceptable level. The BMSCC had been greater than the EU standard of 400,000 cells/ml of milk, since the winter of 1995. Investigation of the high BMSCC was started in December 1996 by assessing the milking routine and by the use of individual cow somatic cell counts (ICSCC). Cows were then selected for California Mastitis Testing (CMT) on the basis of ICSCC >400,000 cells/ml. Individual quarter samples, positive on CMT, were submitted for bacteriological culture. The results showed 67% of cows sampled to be infected by *Streptococcus agalactiae* and 43% to be infected by *Staphylococcus aureus*. The milking routine and general management on this farm were poor. Improvements in record keeping, general management and strict adherence to the "Five Point Plan" were advised. Whole herd "blitz therapy" was then implemented to eliminate *S. agalactiae* infections from the herd and to produce a rapid reduction in the BMSCC, thereby allowing the farmer to continue to market the milk. The "blitz therapy" comprised of intramammary treatment Nafpenzal MC (Intervet UK Ltd), and parenteral treatment with Depocillin (Intervet UK Ltd), of all lactating cows; and parenteral treatment Synulox Ready-To-Use Injection (Pfizer Ltd) of the dry cows and maiden heifers. The longest milk withdrawal period for the antibiotics used was 84 hours. The milk from the herd was tested for antibiotic residues before collection for commercial sale was allowed resulting in the milk being withheld from the bulk tank for 4 days.

Following "blitz therapy", bulk milk samples were taken for bacteriological culture weekly for the first 12 weeks and then monthly for the next 9 months, and the BMSCC were monitored for one year. Bacteriological culturing of individual quarter samples from all the lactating cows in the herd was carried out on four occasions following the "blitz therapy", and from all cows which calved within one month of the "blitz therapy". Individual quarter somatic cells (IQSCC) were carried out on the cows sampled during the herd screenings and the IQSCC were mostly within acceptable levels, i.e. <250,000 cells/ml.

The BMSCC was reduced to below 400,000 cells/ml almost immediately and remained below that level for at least one year after the "blitz therapy". *S. agalactiae* was not isolated from any of the bulk milk samples or any of the cows with the exception of one cow which had been dry at the time of the herd treatment. This cow calved one week after the herd treatment and was identified as being infected with *S. agalactiae* three weeks later. She was immediately isolated and treated with the antibiotic regimen used for the lactating cows during the "blitz therapy". Repeated sampling from this cow failed to detect *S. agalactiae* infection and to date, the herd has remained free of *S. agalactiae* infection for one year.

The isolation of *S. aureus* from 10/20 bulk tank samples and also from 21/75 (28%) of the cows on one more occasion during the herd screenings following "blitz therapy" has led to work being initiated in an attempt to reduce *S. aureus* infection in the herd.

A STUDY OF THE EPIDEMIOLOGY OF *Staphylococcus aureus* IN THE COW USING PULSED FIELD GEL ELECTROPHORESIS

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Aims and Objectives

A study of the epidemiology of *Staphylococcus aureus* mastitis within dairy herds using modern molecular typing methods, to determine the reservoir of infection, e.g. nasal carriage or mastitis infected cows, in incidents of *S. aureus* mastitis.

Introduction

In this study Pulsed Field Gel Electrophoresis (PFGE) was used as an epidemiological tool for population genetic studies of *S. aureus* to determine: (i) which *S. aureus* clones, isolated using classical bacteriological techniques, are responsible for mastitis (ii) how long these strains persist in bulk milk from particular dairy farms, and (iii) whether relapses of infection are caused by the same *S. aureus* PFG electrophoretic types (ET) as those isolated from previous infections or by ET originating from other sites on the cow.

Material and Methods

Bulk milk tanks on 10 commercial dairy herds were sampled at twice monthly intervals for one year. The milk (0.1 ml) was spread onto Kranep agar and sheep blood agar plates. All typical *S. aureus* colonies, up to a maximum of five per plate, were subjected to confirmatory testing, by coagulase test (Oxoid Staphytest), Gram stain and API Staph. Individual cow quarter milk samples, nasal, vaginal and axillary swabs were taken on a high somatic cell count dairy herd on three occasions at monthly intervals. Milk samples were treated as above, swabs were enriched in 10 ml staphylococcus enrichment broth and plated on to Kranep agar. *S. aureus* isolates were also obtained by swabbing different sites (axillary, nose, teat, vagina) on dairy and culled cows. Thus, a total of 259 isolates of *S. aureus* were collected over a two year period and these have been analysed using PFGE. The protocol for the preparation of chromosomal DNA was modified from that described by Thomson-Carter F, Carter PE and Pennington TH (PFGE for the Analysis of Bacteria Populations. Soc. App. Microb. 1993). All isolates were evaluated by digesting their chromosomal DNA with the low-frequency-cutting enzyme *Sma*I and separating the restriction fragments by contour-clamped homogeneous electric field gel electrophoresis.

Results

The bulk milk tanks isolates were assigned to 9 different restriction fragment profiles (A-I), (Table 1). Of the total 117 strains of *Staph. aureus* tested, 48 were identified as type A strains, 29 as type B, and 25 as type C. The remaining isolates were D (5), E (2), F (4), G (1), H (1) and I (1).

Table 1. Source of and Pulsed Field Electrophoretic type diversity of 259 *S. aureus* isolates from cows

	No. of isolates	ET
1. Fortnightly bulk milk tanks from 10 dairy herds over a period of one year	117	9
2. Individual cow body swabs and quarter samples from a HSCC dairy herd	94	6
3. Nasal, teat, vaginal and axillary swabs from individual cows	48	16
Total	259	31

The most commonly isolated *S. aureus* ET types from bulk milk tanks were also the most persistent. Type A being consistently found on farms 1 and 2 and Type C from farm 9 over the period of one year. The DNA patterns of the other farms were different during the year. For example, farm 4, gave six different ET, ranging from 60% to 4% of the total (Table 2).

Table 2. *S. aureus* isolates from bulk milk tanks over a one year period showing PFGE type diversity.

	A	B	C	D	E	F	G	H	I
Farm 1	100								
2	100								
3		70		30					
4		9	60	9	9	9	4		
5		74							
6	26								
7			89			11			
8	86							4	10
9			100						
10									

The 94 isolates of *S. aureus*, from quarter milk samples and a body swab from a HSCC dairy herd, were assigned to six different ET. The majority of the milk isolates (81%) fell into a single ET. The patterns of the remaining isolates were similar showing differences in only one or two bands. The body swab strains varied by more than two bands and could be considered distinct. The rates of isolation of *S. aureus* from various body sites of 38 culled cows was nares (58%), vagina (24%), axillary (24%) and teat (21%). PFGE typing subdivided the 48 isolates into 16 ET's. Among the 38 cows, 3 were presented with two or three positive sites with the same ET. Another 4 cows with 2-4 positive sites were identified with different ET.

Discussion

Other studies on the genetic diversity and structure of natural populations of a number of human and animal pathogens have revealed that (i) most species are clonal in nature, (ii) the number of clones are relatively small and (iii) the majority of cases of serious diseases is caused by a small proportion of the total number of existent clones (Fitzgerald *et al.* (1997), 119, 261-269). Here a predominant ET was identified (A), which was found to share a significant similarity with other ET's (B, C, D, E) in the milk isolates. The isolates from various body sites showed a much greater diversity of ET's suggesting the site may influence the clonal type isolated.

THE TREATMENT OF SUB-CLINICAL MASTITIS IN THE YOUNG DAIRY COW

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Introduction

The early detection of new infections in individual quarters may soon be practicable on many dairy farms using computerised parlour routines involving the daily monitoring of the constituents of milk and changes therein, e.g. conductivity will allow individual cow/quarter changes to be highlighted. This information may then be used to identify and thereby treat new infections before they cause tissue damage within the infected quarter and also to reduce the level of exposure within the cow and subsequent cows to be milked. Data from previous work show that the prevention of chronicity is a benefit. Chronic cases of staphylococcal mastitis in older cows do not appear to resolve as well as new infections when the same therapy is used.

The efficacy of parenteral and/or intramammary therapy in the treatment of subclinical (not chronic) mastitis in dairy cows in lactations 1 to 3 was studied.

Trial animals were 1500 lactating dairy cows undergoing their first, second or third lactation on 14 commercial dairy farms in the UK. Around 200 subclinically infected quarters were available for treatment. To date, not all participating sites have completed the trial.

Excluding the first month of lactation, NMR data were used to detect a trend towards high or increasing whole udder/cow milk somatic cell count, suggestive of subclinical mastitis in an individual cow. A count of over 100,000 cells ml⁻¹ in any one month triggered further investigation of the cow. Quarter milk somatic cell count and bacteriology were used to confirm the presence of subclinical mastitis in individual quarters on three occasions (days 1, 15 and 22) prior to treatment (day 22 and 23). All quarters (treated and untreated) were then re-examined at days 40-43 and 70-73 by bacteriology and somatic cell count measurement.

Assessment of the efficacy of treatments is being based on the difference between whole udder and individual quarter somatic cell counts between treatment groups at critical time points in the trial schedule and on bacteriological cure rate at the relevant critical time points. Calculations of bacteriological cure rate will be made for all major subclinical mastitis pathogens isolated during the study if feasible. Data assessment is on-going.

Two treatment groups are being used:

- Group 1 Cows treated with an intramammary infusion, after 3 consecutive milkings, of Cefquinome (Cephaguard L C, Hoechst Roussel Vet) combined with two consecutive parenteral injections of cefquinome (Cephaguard 2.5% Suspension, Hoechst Roussel Vet).
- Group 2 Cows treated with an intramammary infusion, after 3 consecutive milkings, of cefquinome.

Table 1 gives the summary results. The following definitions apply:

Bacteriological Cure - a pathogen isolated at day 1 and/or day 15 and/or day 22 was not isolated at either day 40-43 or day 70-73.

Somatic Cell Count/Clinical Cure - a QMSCC on day 1 or 15 of more than 100,000 cells ml⁻¹ which had fallen post treatment and was or remained at fewer than 100,000 cells ml⁻¹ at day 70-73.

Somatic Cell Count/Clinical Improvement - a QMSCC on day 1 or 15 of more than 200,000 cells ml⁻¹ which had fallen post treatment to more than 100,000 cells ml⁻¹ but fewer than 200,000 ml⁻¹ by the final assessment (day 70-73).

Preliminary results only are shown.

	Treatment Group 1	Treatment Group 2
Total quarters	33	33
% quarters showing bacteriological cure	55 (10/18)	73 (11/15)
% quarters showing QMSCC cure	39 (13/33)	47 (15/33)
% quarters showing QMSCC improvements	12 (4/33)	6 (2/33)
% quarters showing QMSCC benefit	52 (17/33)	52 (17/33)

TREATMENT OF PERSISTENT *Staphylococcus aureus* INFECTED COWS AT DRYING OFF WITH TILMICOSIN (Micotil Elanco)

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Summary data

Fifty cow family farm. Average yield 5,500 litres. Cubicle housed. Bulk milk somatic cell count consistently below 100,000 cells/ ml. Generally closed herd policy. Cows 2 and 18 purchased spring 1997.

Date	Sample	Isolate	BMSCC	Action
19 May 1997	Monitoring bulk milk sample	Positive <i>Staphylococcus aureus</i> (20 colony forming units (cfu's))	91,000 cells/ml	No action
13 October 1997	Monitoring bulk milk sample	Positive <i>Staphylococcus aureus</i>	193,000 cells/ml	Advise sample "problem" cows"
28 October 1997	6 cows sampled Cows - 1,2,18,29,70 Cow - 80	Positive <i>Staphylococcus aureus</i> Negative <i>Staphylococcus aureus</i>		Advise SCC test of whole herd & bacteriology on all high SCC cows
4 November 1997	10 high SCC cows ¼ sampled 1,2,10,11,18,29,30,34,45,70	All cows and 17 quarters positive for <i>Staphylococcus aureus</i>	140,000 cells/ml	Cull cows 2,10 & 45
Herd has "batch" calving pattern Whole herd is dried off November/December				7 cows (13 infected quarters) treated with Tilmicosin at drying off

Protocol

- Persistent rather than transient *S. aureus* infection determined by 2 quarter samples at least 1 week apart
- Quarter Somatic Cell Count (SCC) performed on all bacteriology samples
- Tilmicosin (Micotil Elanco) administered at drying off
- Post calving samples taken on 3 occasions

Treatment

All cows received 600 mg Cloxacillin (benzathine salt) as dry cow therapy (Orbenin Extra Dry Cow Pfizer)

All cows treated with Tilmicosin (Micotil Elanco) received 10 mg per kg (1 ml per 30 kg body weight). This was administered as a 20 ml dose given subcutaneously in four divided doses of 5 ml. All cows were subjected to an inhibitory substance test 4 days after calving before their milk was returned to the bulk tank.

No cows failed a milk inhibitory substance test and all cows had a dry period of 8 weeks or greater.

Results

All 7 cows had favourable SCC responses as all cows treated had a composite SCC of less than 100,000 cells/ml at the end of the trial period.

Eleven quarters were apparent bacteriological cures.

Quarter SCC of cured quarters were generally below 50,000 cells/ml.

Two quarters were probably bacteriological failures as *S. aureus* was isolated from all post-treatment samples.

Discussion

Possible reasons for the high success rate.

- Early identification of herd and then individual cow infection
- Possibly low virulence strain of *S. aureus*
- Age of cows - general wisdom dictates older cows are not worth treating. However no 4th or 5th lactation animals were treated

Current Position

Herd results have been maintained. 42 of 49 cows in milk have individual SCC of less than 100,000 cells/ml.

No *S. aureus* have been isolated from monitoring bulk milk samples.

Herd Bulk Milk Somatic Cell Counts are consistently below 100,000 cells/ml (last BMSCC was 81,000 cells/ml).

PREDICTING THE OUTCOME IN CASES OF TOXIC MASTITIS

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Twenty veterinary practices from throughout Northern Ireland were requested to submit milk and blood samples along with clinical details relating to acute and peracute toxic mastitis in cows which they encountered during their farm visits. Milk samples were cultured and antibiotic sensitivities were performed on bacterial isolates. Routine haematology, urea and creatinine measurements were performed on blood samples.

Samples and completed questionnaires were received for a total of 264 toxic mastitis cases from 18 different veterinary practices. Of the toxic mastitis cases for which the outcome was known, 14% cows died from the infection, 22% lost the infected quarter, 21% were culled prematurely and 43% were kept in the herd.

Binomial univariate analyses of clinical and laboratory examinations indicated that recumbency, subnormal temperature ($\leq 101^{\circ}\text{F}$), being less than one week calved, elevated ($> 130 \mu\text{mol/l}$) creatinine, elevated urea ($> 8.3 \text{ mmol/l}$) and high packed cell volume ($> 45 \%$) were all associated with a significantly increased probability of dying from the condition.

Various categorical and categorical/continuous logistic regression models were fitted to the clinical and laboratory data to assess which variables gave the most accurate prediction of the probable outcome of cases of toxic mastitis, that is whether the cow lived or died from the condition. The different models indicated consistently that recumbency and high creatinine levels were the most accurate predictors. When recumbency was excluded, urea levels gave the best indication of the probable outcome.

The close relationship of the probability of a cow dying to the blood creatinine level and the blood urea level are shown in Figures 1 and 2. For example, a cow with toxic mastitis that is recumbent and has a creatinine level of $200 \mu\text{mol/l}$ has only a 1 in 3 chance of surviving.

Figure 1 - Probability of dying from toxic mastitis in recumbent cases at a given blood creatinine level

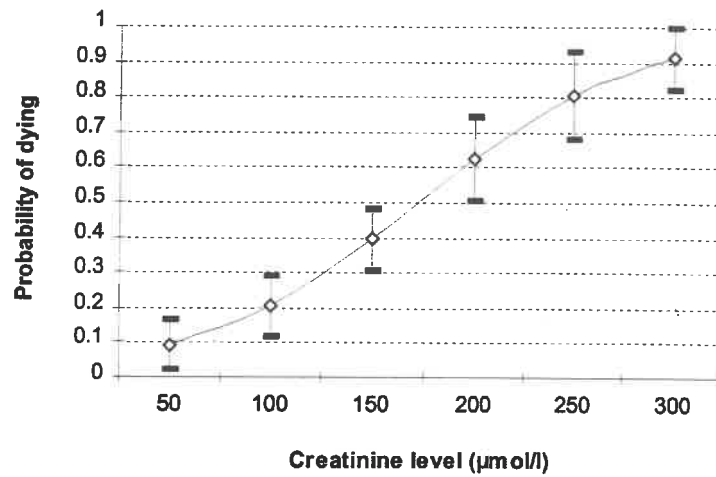
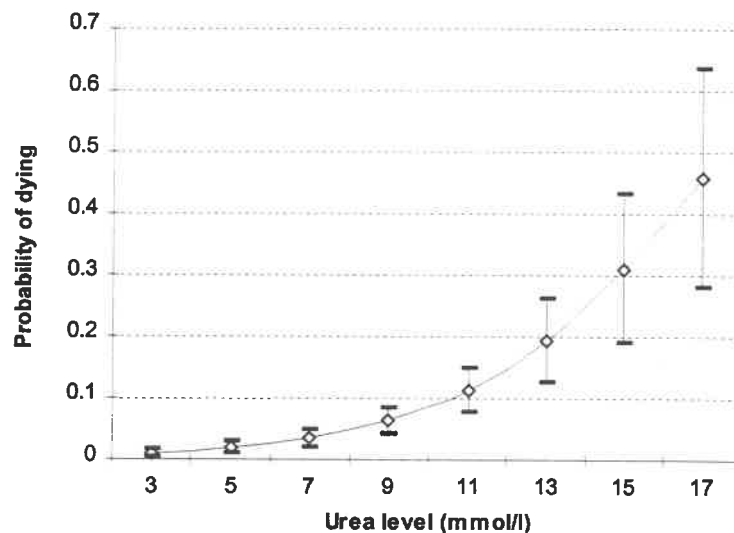


Figure 2 - Probability of dying from toxic mastitis in non-recumbent cases at a given blood urea level



The availability of such decision support models can be of assistance in determining the fate (i.e. treatment or euthanasia) of cows suffering from toxic mastitis. The models enable a quantitative assessment of the probability of survival and, as they can be applied at an early stage of diagnosis, can be used to identify cases where administration of expensive treatments would be of questionable value.

IMMUNOLOGICAL RESPONSES OF HOLSTEIN FRIESIAN CATTLE TO *Staphylococcus aureus*

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Bovine mastitis has both serious economic and welfare implications for the dairy industry. The disease is costly to treat, results in loss of milk quality and quantity, and premature culling of persistently infected animals. A major pathogen responsible for both clinical and subclinical mastitis is *Staphylococcus aureus*. This bacterium has numerous mechanisms for evading the host's immune system resulting in current therapies being largely ineffective, and resulting in chronic infection of the mammary gland.

Bovine peripheral blood mononuclear cells (PBM) were isolated using density centrifugation. These cells were stimulated *in vitro* with formalin-fixed whole *S. aureus*. The kinetics of this immune response were of a primary rather than a secondary nature, with the day of peak proliferation occurring on day nine of culture. Proliferating cells were phenotyped by flow cytometry and shown to consist mainly of CF4⁺, CD8⁺ and $\gamma\delta$ cells. Initially ten cows, five sired by bull A and five sired by bull B were studied, and significant differences ($p < 0.03$) were found in the proliferative response to *S. aureus*.

Milk somatic cell count (SCC) is recognised as an indirect measurement of subclinical mastitis. Bulls are selected at present on an index which includes predicted transmitting abilities (PTA) for type and production traits, and more recently, PTA for SCC have also been available. However generation of PTA for SCC is costly, with an extended time period required before bulls may be scored accurately.

Our preliminary study examined the proliferative response of ten bulls. Five bulls were recorded as having low PTA for SCC, and five bulls were recorded as having high PTA for SCC. These animals represented extremes of the normal distribution within the population. Recent results have shown that PBM from these proven bulls proliferate *in vitro* to formalin-fixed *S. aureus* and that stimulation indices were shown to correlate negatively to PTA for SCC ($r = -0.7$). These results may indicate that *in vitro* immunological assays may be useful in identifying bulls whose progeny would be associated with a higher resistance to intramammary infection with *S. aureus*. Molecular cloning of class II alleles of the major histocompatibility complex using Novagen PT7 Blue Perfectly Blunt™ to enable sequencing of the bulls DNA which will reveal the animals genotype is currently being assessed as a potential genetic marker.

TOXIN PRODUCTION BY *Staphylococcus aureus* FROM CASES OF BOVINE MASTITIS

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This pilot study was instigated following investigation of a large herd where, in the course of a few weeks, 15 cows developed gangrenous staphylococcal mastitis (fatal in some cases) despite prompt detection and intensive therapy. In a second herd investigated, all cases of staphylococcal mastitis were mild and responded promptly to routine intramammary therapy.

It was discovered that the strain of *S. aureus* which caused gangrenous mastitis produced staphylococcal toxin shock syndrome toxin1 (TSST1) and enterotoxin C. (Some strains of *S. aureus* produce enterotoxins, which can cause human food poisoning. These toxins survive pasteurisation). *S. aureus* from the herd with only mild cases did not produce any of these toxins.

Twenty isolates of *S. aureus* from twenty different herds were examined for toxin production, using "Oxoid" reversed passive agglutination kits, and phage type:-

Toxin production by strains of *S. aureus* isolated from bovine mastitis cases

Mastitis Severity	Very severe	Severe	Chronic	Mild
<i>S. aureus</i> strains tested	2	12	4	2
TSST1 production	1	5	1	0
Enterotoxin A production	0	2	0	0
Enterotoxin B production	0	0	0	0
Enterotoxin C production	1	2	1	0
Enterotoxin D production	0	0	0	0

TSST1 was produced by 7/20 isolates in this sample.

Enterotoxins 'A' and 'C' were not isolated from strains which did not also produce TSST1.

Seven different phage types and six untypable strains of *S. aureus* were involved. All three phage type 78 isolates produced TSST1. This phage type has been associated elsewhere with very severe cases of *S. aureus* mastitis. Other phage types also produced TSST1. Strains not producing these toxins also caused severe mastitis.

*It can be misleading to talk about staphylococcal mastitis in general terms because the clinical severity can depend on the strain of *S. aureus* involved.*

DEVELOPING AN UNDERSTANDING OF THE COST BENEFITS OF SUBCLINICAL MASTITIS AND SOMATIC CELL COUNT CONTROL IN HERDS IN SCOTLAND

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Mastitis control programmes for the dairy cow involve a number of separate procedures. The cost and efficacy of each of these is different, and since resources are often limited an appreciation of their relative economic merit is an important management tool. The most common approach to such an analysis is the use of multivariate statistics to build a simple model. This was applied to Scottish field data (1,2,3) to establish the marginal impact of individual mastitis control procedures on bulk tank SCC (BTSCC) control and so by inference upon subclinical mastitis. Estimates of milk yield depression and the probability of herds paying a BTSCC penalty due to the presence of subclinical mastitis were calculated. Subsequently, using a statistical analysis of an economically optimum disease loss and disease control expenditure mix (for subclinical mastitis) interactions between mastitis control procedures and other management variables were tested. The models allowed an assessment of the technical and economic efficiency of mastitis control by Scottish farmers.

Statistically significant interactions were detected between premilking udder preparation methods (UP) and post milking teat disinfection (TD); the milking system of the herds and both the use of dry cow therapy (DCT) and a regular milking machine test (MMT). Any udder preparation involving washing, appeared to encourage the spread of infection and had a detrimental effect on the efficacy of TD. Amongst the herds facing a high BTSCC problem (BTSCC 300,000 to 600,000 cells/ml) herds using TD without UP and those using DCT and MMT with a parlour system obtained returns of £1.4, £3.9 and £1.1 respectively per £1 invested in each of these procedures. These returns were due to savings from milk yield losses and increased milk price from reductions in BTSCC penalties. Using a loss-expenditure frontier approach (4) for high SCC herds the economically optimum disease loss and disease control expenditure mix was attained by a parlour system using DCT, MMT and TD without UP. This in itself was not surprising but the frontier suggested that losses associated with high BTSCC (and by inference subclinical mastitis) should cost £65.50/cow/year (made up of £41.40 revenue losses and £24.10 mastitis control expenditures). However, on average, subclinical mastitis was estimated to cost high BTSCC farms considerably more than this, £100/cow. The difference between these estimates (£34.50) could therefore be saved by more efficient use of mastitis control procedures in these high SCC herds

The above analysis is "comparative-static" in nature. It assumes that the other components of a system remain unchanged. While this is useful as a first step to developing economic explanations for certain control approaches it is clearly simplistic. Subclinical mastitis control takes place in a complex and dynamic environment and thus, in the longer term, needs more sophisticated methods for dealing with the problem. In a second study a Stochastic Dynamic Programming (DP) Dairy Cow Model was used to evaluate separately the economic impact of a

range of alternative mastitis control procedures in the long-term. In this case the model employed all the field data above i.e. all records of all herds not just those with a "high" BTSCC. In this more broadly based approach (more relevant to present conditions where herds generally have BTSCC figures less than 250,000) the DP model predicted that reducing milk yield and somatic cell count (SCC) penalty costs by use of dry cow therapy (DCT), post milking teat disinfection (TD) and milking machine test (MMT) added approximately £13, £10 and £4 respectively to an original annuity, equivalent net to the present value for a replacement heifer (£286). Assuming that these procedures would also reduce involuntary culling by 50 percent gave an additional benefit of £8.90. This latter figure indicates that a significant part of the benefit of mastitis control procedures may come from a reduction in the culling rate due to persistent clinical and subclinical cases (something not taken into consideration in the earlier study as reliable culling data was not available). The model predicted that the proportion of cows that had fewer than 400,000 cells/ml was increased by 1.6, 3.3, and 3.8 percent under MMT, TD and DCT applications respectively. The strength of the DP model in this context was that it provided an integrated evaluation of the various impacts of each alternative procedure. Further work is needed to develop the DP model and data availability in order to provide improved decision support for mastitis (and SCC) control of dairy cows.

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QUALITY MILK PRODUCTION - Meeting the Challenge

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Introduction

The hygienic quality of milk demanded from dairy producers, in terms of both bacterial quality and somatic cell counts, is of increasing importance. The loss incurred by the producer for not matching the top grade can be in excess of 1 penny per litre. This cost, plus the "invisible" losses associated with poorer quality milk, can readily account for an amount equivalent to a 10-15% reduction in milk price.

In addition to the financial incentives, more milk buyers, at the behest of the retailers, are introducing quality assurance and welfare standards. Failure to meet such standards can mean the ultimate sanction of having to find an alternative milk buyer.

The Five Point Plan

Consistent application of the Five Point Plan will almost certainly result in a dairy herd that meets the high quality standards demanded by a competitive market place. Many producers, however, jeopardise their herd health by not following these simple steps.

- Teat management---including teat preparation, post milking teat disinfection and housing management.
- Dry cow therapy---particularly targeted use of additional antibiotic therapy on known problem cows.
- Early identification and treatment---to include foremilk and taking milk samples for bacteriological and antibiotic sensitivity analysis.
- Culling cows with persistent infection---cows with 3 or more cases in a quarter or 5 or more cases in any quarter should be removed from the herd.
- Correct installation, maintenance and operation of the milking machine---including simple tests during operation by the operator and cleaning /hygiene audits.

Parlour Throughput

Although the principles of mechanically extracting milk from a cow have remained largely unchanged over the years, the expectations of the number of cows milked or litres produced per hour have risen dramatically. Twenty years ago the majority of dairy farmers were aiming for a parlour performance of around 60-80 cows per hour. Although not common, it is now not unusual to find throughputs exceeding 150 cows per hour.

But, at what cost?

The welfare of the dairy cow has never been as high on the agenda as now. The FAWC Report on the Welfare of Dairy Cattle identified mastitis as one of the three major scourges within the national dairy herd, although acknowledging that the clinical incidence had fallen since the 1960's. However, as reported at the 1997 British Mastitis Conference, the reduction in the somatic cell count has remained largely unaltered in the last few years, as has the clinical rate.

Is high throughput compatible with high welfare standards and quality milk production? Automation of cow identification, in-parlour feeding, cluster removal and post milking teat disinfection, together with innovations in parlour configurations over time and the introduction of rapid exit parlours and upgraded rotary parlours have undoubtedly allowed higher throughputs without, arguably, adversely affecting cow welfare. Retaining foremilking (a legal requirement), efficient teat preparation and cluster attachment will allow a maximum throughput of about 110 cows per hour. Many working in the field of mastitis would argue that automatic post milking teat disinfection as the cows leave the parlour is far less effective in the control of mastitis compared with dipping or spraying teats immediately after cluster removal. This therefore jeopardises both milk quality and cow welfare. If manual teat disinfection is introduced then throughput falls to less than 100 cows per hour, easily matched with a 10:20 or 12:12 configuration herringbone parlour.

Although there are exceptions, it is difficult to attain high quality milk (i.e. milk with Bactoscans consistently less than 20, with somatic cell counts below 125,000 and the clinical mastitis incidence less than 25 cases per 100 cows per annum), where thorough teat preparation and foremilking are not carried out.

With more demanding tests on the horizon from buyers such as tests to determine thermotolerant and psychrotrophic bacteria, cow throughput may become a less important issue. It is already possible to make out an economic case to reduce throughput, employ an additional milker and so ensure that the top quality benchmarks are met. The associated lower capital investment and running costs will also improve farm profit.

Anecdotal evidence already indicates that re-introducing efficient teat preparation methods and foremilking do not adversely affect parlour performance. Dynamic recordings of vacuum in the short milk tube often indicate that minimal teat preparation delays milk let down. As a consequence the cluster is on the cow for longer. Milk flow rates are therefore often higher with good teat preparation, compensating for the extra labour time required. The benefit also induces higher quality milk with less sediment and improved mastitis control.

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