BRITISH MASTITIS CONFERENCE 1996

Managing Mastitis for Milk Quality

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British Mastitis Conference 1996
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INTRODUCTION

JAMES M BOOTH
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Welcome to the ninth annual British Mastitis Conference. This will be the first without Robert James of Ciba to guide the organisation; we are most grateful to him for all his hard work and the high standards he set.

The conference theme revolves around milk quality once again. The quality of milk, and in particular its hygienic quality, has improved immensely over the past ten years. Bacterial counts in the UK have been reduced to equal the best in the world. Cell counts have been substantially reduced and, with the national average now around 200,000 cells/ml, we can hold our own with the best in the European Union.

The question being asked now is whether this reduction in cell count mirrors a parallel reduction in mastitis. Unfortunately there are no national statistics to resolve this question one way or the other. The suspicion remains that there is more mastitis in the national herd than might be expected from the improvement in cell count.

This conference will address the issues of producing quality milk for the market whilst controlling and reducing mastitis. The correct application of antibiotics is essential to obtain the maximum benefit from treatment whilst minimising the chances of accidental contamination of the milk for sale. It is important too, in these days of cell count bonuses and penalties, to know how soon the cell count falls again after treatment.

Practical advice on controlling mastitis on the farm will be discussed from the farmer's point of view. And the prospects for increasing resistance to mastitis through breeding which would be the best long term solution of all, will be examined.

In the space of one day it is impossible to consider all the intricacies of mastitis. The subjects to be discussed today are drawn from the suggestions made at last year's conference and we welcome your ideas for future years.

TREATING MASTITIS

HOW ANTIMICROBIALS WORK IN MASTITIS

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SUMMARY

Antimicrobials are administered to cows with clinical mastitis in order to effect clinical cure and to try to remove the pathogenic organisms. Their activity depends upon the sensitivity of the target organisms to the drug being administered, and the concentration and duration of persistence of the drug in the environment of the pathogen. Antimicrobials are also used in mastitis control programmes at drying off. In this instance the objective is to remove infection present at drying off and prevent re-infection for part or all of the dry period.

The relative merits of different types of antimicrobials are considered with respect to use for different types of clinical mastitis.

INTRODUCTION

Most cases of clinical mastitis are bacterial in origin and subclinical mastitis is also generally associated with bacterial micro-organisms. Antimicrobial therapy is designed to remove the pathogenic organisms or at least to 'cure' the clinical disease. The therapeutic success of antimicrobial therapy in mastitis depends on:

1. the sensitivity of the organism and its ability to evade the effects of the antimicrobial,
2. the concentration and persistence of the drug in the environment of the pathogen,
3. the contribution made to exclusion of the organism by host immunity which may be influenced by concurrent therapy, disease or age.

SENSITIVITY OF THE ORGANISM

The sensitivity of an organism may in some cases be predicted if the organism is known. However, since therapy in clinical disease should be instituted immediately, culture and identification of the causative organism may only be achieved retrospectively. Nevertheless it is good practice to carry out routine culture of milk from mastitis cases so that the prevalence of particular organisms in a herd can be determined. Furthermore the sensitivity of bacteria varies in different geographical areas and antimicrobial resistance could develop in response to the selection pressure imposed by widespread use of particular drugs. It is therefore useful to carry out *in vitro* sensitivity tests on samples collected routinely and on samples obtained from problem individuals within a herd.

The sensitivity of an organism to an antimicrobial is often expressed in terms of the minimum concentration of the antimicrobial required to inhibit the growth of the organism [minimum inhibitory concentration (MIC)]. The MIC determined *in vitro* may not be an accurate reflection of the actual antimicrobial concentration required for effect within the udder. Antimicrobial activity in the udder may be enhanced by the animal's immune system or reduced by factors which inhibit the drug, such as calcium ions which chelate some antibiotics.

Minimum inhibitory concentrations determined for field isolates are, however, very useful for comparison with known effective concentrations.

On the basis of predictable sensitivity information the selection of first choice antimicrobials may be made (Table 1).

Table 1 Relative sensitivity of bacteria to antibiotics available in intramammary tubes

	β lactamase *S. aureus*	Gram positive *S. aureus* streptococci *A. pyogenes*	Coliforms	Pseudomonads
Penicillin G, Penethamate	-	++++	-	-
Cloxacillin, Nafcillin	+++	+++	-	-
Penicillin G + Aminoglycoside	+	++++	+++	+
Aminopenicillin + Cloxacillin or Clavulanic Acid	+++	+++	+++	-
1st Generation Cephalosporin (Cepacetrile, cephalonium)	+++	+++	++	-
2nd Generation Cephalosporin (Cefuroxime)	+++	+++	++	-
3rd Generation Cephalosporin (Cefoperazone)	+++	++	++++	+++
Tetracyclines	++	+++	+++	+
Erythromycin	+++	+++	-	-
Novobiocin	++	++	-	-

++++ = very sensitive
- = resistant

ANTIMICROBIALS AND THEIR SPECIFICITIES

Penicillin G is still, generally, highly effective against streptococci and *Actinomyces pyogenes* and where these organisms are involved is the logical choice for therapy.

Penethamate is a basic ester of penicillin G which has enhanced distribution properties and similar activity to penicillin G.

Many *Staphylococcus aureus* strains responsible for clinical and subclinical mastitis, produce an enzyme, β lactamase which confers upon them resistance to the unprotected β lactam antibiotics (penicillin G, penethamate, ampicillin, amoxycillin). Cloxacillin and nafcillin are, however, highly effective against such bacteria although they have activity only against Gram positive bacteria and are generally less active than penicillin G against non β lactamase producing bacteria.

Clavulanic acid acts as an irreversible inhibitor of β lactamase but has poor antimicrobial activity on its own. Cloxacillin and clavulanic acid have been combined with ampicillin and amoxycillin respectively and confer upon the combinations activity against β lactamase producing *S. aureus* and activity against some β lactamase producing Gram negative bacteria.

Cephalosporins and erythromycin are also likely to have good activity against β lactamase producing *S.aureus* and cephalosporins have increasing activity against β lactamase producing Gram negative bacteria with succeeding generations. The third generation cephalosporins are particularly effective against coliform bacteria and the recalcitrant *Pseudomonas aeruginosa.*

Novobiocin has a narrow spectrum of activity and is generally used for its effect against *S.aureus*. However it is moderately synergistic with penicillin and combinations of novobiocin and penicillin are justified.

Where coliform organisms are involved the gram negative activity of an **aminoglycoside** (dihydrostreptomycin, framycetin, neomycin, streptomycin) may be exploited. A number of products contain a combination of penicillin and an aminoglycoside. These have been shown to be as effective as any other antibiotic or antibiotic combination for the routine treatment of mastitis. Where sensitivity tests are not available and the aetiological agent is not identified, these are probably the drugs of first choice. Streptomycin and dihydrostreptomycin have very similar properties and are generally less active than neomycin or framycetin. Neomycin is an isomeric mixture and framycetin is one of the isomers.

The **aminopenicillins, cephalosporins** (especially third generation) and **tetracyclines** are all highly effective against coliforms but are likely to have less activity against the penicillin susceptible gram positive organisms than penicillin G.

CONCENTRATION OF THE ANTIMICROBIAL

Antimicrobials must reach the site of infection in sufficient concentration, and remain at the site for a sufficient period of time to effect cure. The concentration achieved will depend upon the amount administered and frequency of administration, the route of administration and the physicochemistry of the antimicrobial and the excipient in which it is delivered.

Since distribution of drugs throughout the body and the udder is principally dependent upon simple diffusion, the concentration achieved at the desired site will be increased if more drug is administered and, for some systemically administered antimicrobials such as oxytetracycline, adequate concentrations will only be achieved in udder tissue if the upper recommended dosage rates are used. Antimicrobials may be administered for mastitis either systemically by injection at a site distant from the mammary gland, or may be administered directly into the mammary gland via the teat canal. Systemic administration is desirable where the teat canal is blocked by the inflammatory response and where the mastitis is associated with systemic signs. Some drugs are not sufficiently bioavailable when given by the intramuscular route and must be given intravenously in order to achieve adequate concentrations in the udder. Furthermore all drugs given systemically must have appropriate distribution characteristics in order to cross the blood milk barrier. Generally highly lipid soluble, basic or non-ionised drugs achieve greatest concentrations in milk if given systemically and some highly polar antimicrobials such as the aminoglycosides are very poorly distributed into mammary tissue if given systemically (Table 2). During clinical mastitis the pH of the milk rises and the integrity of the blood milk barrier is disrupted. As a consequence systemically administered acidic drugs may achieve adequate therapeutic concentrations within the udder.

Table 2 Distribution properties of different types of antibiotic (after 1)

	SYSTEMIC	INTRAMAMMARY
GOOD	Macrolides Florfenicol Trimethoprim Baquiloprim Lincomycin Quinolones	Erythromycin Aminopenicillins Cephalexin Penethamate
LIMITED	Sulphonamides Penicillin G Tetracyclines	Most Cephalosporins Penicillin G Cloxacillin Nafcillin Tetracyclines Novobiocin
POOR	Aminoglycosides	Aminoglycosides Polymyxin B

The intramammary route for the administration of antimicrobial drugs is practical and convenient in animals where the inflammatory response does not occlude the teat canal or cisternae. It is also a suitable route for the administration of long acting antimicrobials at drying off as part of mastitis control programmes. Distribution throughout the mammary gland following intramammary administration is largely dictated by the same physicochemical characteristics as for systemic therapy (Table 2). However drugs do not require to cross the

blood milk barrier and the targeted nature of administration may result in higher local concentrations, particularly of drugs such as the aminoglycosides and polymixin. Formulation characteristics such as particle size and the nature of the excipient can also be used to control the rate of release of the drug. Formulations for use during lactation normally facilitate rapid release with consequent short milk withdrawal times. Those formulated for dry cow therapy may have greatly extended persistence within the mammary gland. For intramammary therapy in lactating cows it has been recommended that drugs which are well absorbed and distributed in the mammary gland should be given 4 times at 12 hour intervals and that poorly absorbed drugs should be given 3 times at 24 hour intervals. However most preparations have recommendations of three treatments at either 12 or 24 hour intervals. Some products, especially those containing cephalosporins, require single administration only, and since the milk withholding period is not unduly extended these may confer advantages over products requiring numerous infusions.

In *S. aureus* mastitis, treatment with these regimens may prove unrewarding and the extended treatment periods required to effect bacteriological cure combined with the milk withdrawal time and potential for relapse may make antimicrobial therapy during lactation economically unjustifiable. Control by identification, segregation and culling has been suggested as an alternative.

DRY COW THERAPY

Dry cow therapy is designed to remove infections present in the udder and to prevent new infections establishing during the dry period. At this time the udder is largely naturally resistant to Gram negative organisms since lactoferrins produced at this time inhibit their establishment. Dry cow preparations therefore require good activity against *S. aureus* (possibly β lactamase producers) and *Streptococcus uberis* and if prophylaxis against summer mastitis is desired they should also be effective against *A. pyogenes*.

Syringes containing cloxacillin, nafcillin or a cephalosporin may therefore be indicated and in addition preparations containing neomycin have good activity against *S. aureus*. It has also been shown that antibiotics in dry cow syringes with persistence in the mammary gland for extended periods (longer than 3 weeks) reduce the number of pathogenic bacteria which can be isolated from the udder at the subsequent calving. These products may only require single administration for protection against summer mastitis and may be preferable to preparations which require administration and consequent breaching of the teat seal during the dry period.

Where products with very long intramammary residence times are used great care must be taken to ensure that the appropriate milk withdrawal periods are adhered to.

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MASTITIS THERAPY - WHAT I AM TRYING TO ACHIEVE

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SUMMARY

The success of mastitis therapy depends on a large number of factors relating to both the individual cow, the herd and the management. Although curing the cow is the primary objective of treatment, other very important aims are to limit the spread of infection to other cows and to reduce the impact on the bulk milk cell count.

Knowledge of the individual cow, the particular bacteria causing mastitis in the individual herd and adequate records are essential to make the best use of the therapeutic tools available. Two different types of mastitis are discussed to illustrate the need to vary the strategy.

THE QUESTIONS

"What am I trying to achieve ?" This question may not be the first that springs to mind when a case of clinical mastitis is discovered and intramammary antibiotic is being administered. It is unclear how much thought goes into routine administration of antibiotics or if the action is a simple reflex developed over years of encountering clinical cases. The intention is obvious - to cure the cow, with, hopefully, the cow responding clinically and the milk apparently returning to normal. This does not mean that the infection has been eliminated. There is a clear distinction between clinical cure and bacteriological cure.

Failure to achieve a bacteriological cure has a number of consequences. If bacteria are still present in the udder then recurrence of the disease may well occur. The cow is an obvious source of infection to the rest of the herd and to other quarters of the same udder. There is, therefore, a need to limit, or better still prevent, spread of infection. This requires good hygienic methods and a milking machine operating efficiently with the milker using it properly. Effective housing management is also necessary so as not to undermine the other efforts at mastitis control. Simply, this is not just treatment of an individual infection for mastitis, but an application of a strategy so that treatment has an influence on the udder health of the whole herd.

Whilst complete cure is the ultimate goal, this may take some time to achieve and in some cases be impossible. Alternative measures may be necessary to limit risk eg by changing milking order, using a separate cluster (1), housing the cow separately. But maybe the cow could receive a different and more effective therapy. Selection of a particular therapy could be by serendipity but could become more specific by better definition of the problem. This starts with background information on the different type of mastitis, causes etc, and some hard information on the types of mastitis usual in the herd in question.

MASTITIS

The Battle

There is a continual challenge of bacteria to invade tissues, colonise and grow. Many bacteria are well adapted to do this and cause as little disruption as possible as that ensures their survival. Others are less well adapted and induce a significant host response which results in disease. A large number of strategies are adopted by the bacteria and the host in an effort to achieve supremacy. The successful outcome for the host is elimination and for the bacteria to establish low grade infections. Neither wins with disease. *Streptococcus agalactiae* is the best example of the most balanced outcome with only a low grade chronic disease occurring. This compares with *Escherichia coli* mastitis when peracute disease may occur, the host may die and the bacteria are eliminated before the clinical conditions is most severe.

The contest between bacteria and host is therefore a battle in which significant intervention is possible. This intervention is strengthened by good intelligence about the opposition - the type of bacteria and the problems it will create, and weapons available - the antimicrobials.

Information

Intelligence gathering starts with information on the bulk tank milk; the cell count and TBC give an overall picture (2). Bulk tank samples may be analysed further to identify particular pathogens involved in herd mastitis. This approach is usually refined by then examining individual cows. Selection of the cows for sampling is best made from monthly cell count records and treatment records. These help to estimate the amount of infection in the herd and give information on its duration. If necessary the response of the bacterial strains isolated can be tested against particular antibiotics although the variability in these results to helping to chose alternative products for therapy. The 'enemy' has been defined.

Approaches to treatment

Once the detailed information on the herd mastitis problem is available the correct strategy can be adopted. This will depend on the particular problem. Two differing examples can be given although many variations are available.

Staphylococcal mastitis

Any clinically significant problems will be caused by *S. aureus*. The coagulase negative staphylococci cause frequent infections but of relatively little impact compared with clinical disease. *S. aureus* is a particularly well adapted pathogen for the mammary gland. It frequently causes clinical mastitis but more often this is a sporadic occurrence resulting from a chronic subclinical infection when the balance tips in favour of the bacteria and against the host. The clinical infections are usually not too traumatic with a swollen udder and clots in the milk. The cell count will be raised to a few million cells/ml. Treatment with an antimicrobial will usually result in an apparent cure but not necessarily. The antimicrobial helps to restore the balance between the host and pathogen. *S. aureus* adopts a number of strategies to avoid elimination from the udder, some strains are resistant to penicillins and all have an ability to survive inside white blood cells and to outlive these cells and thus survive in the udder (3). This is manifested as occasional minor clotting of milk, a persistently high and fluctuating cell count in monthly samples and some yield reduction. Bacterial recovery from milk samples is intermittent.

The best approach to *S. aureus* mastitis is undoubtedly to take prompt action when the initial infection and disease occur. This means good identification of the early signs of mastitis with examination of foremilk, mastitis detectors, udder palpation, awareness of susceptible cows and knowledge of the prevalence of *S. aureus* in the herd. Prompt and effective treatment can result in a high rate of elimination of the initial infection whilst established infections respond very poorly to antibiotics with only 30-50% being cured bacteriologically. The earlier the identification and treatment the better.

If identification is only made when the infections are chronic e.g. from monthly cell count records, then the prognosis is much poorer. Cure rates will decrease with increased cell count, age and the number of quarters infected (4). Intramammary antibiotic preparations then are limited in penetration of the diseased, possibly abscessed, tissue and higher enough concentrations may not be achieved at the focus of infection. Penetration may also be a slow process through this abnormal tissue. Prolonged treatment may well be necessary to achieve an effective concentration of antibiotic. It has been shown that a higher bacteriological cure can be achieved with multiple intramammary treatments (5). Multiple intramammary treatments may not always be practical for management reasons and they can create problems in calculating milk withdrawal times to avoid antibiotic residue problems.

If antibiotics are administered by injection as well as by the intramammary route then the concentration of antibiotic at the site of infection can be increased (6). It has been shown that a daily injection of antimicrobial for three days plus three days of intramammary tubes produced a better cure rate of chronic infections than intramammary tubes alone (7). Combination of parenteral treatment and intramammary injectors may be more realistic but this requires specialist advice.

Whichever approach is used, it is necessary to continue monitoring *S. aureus* cases for clots and cell count to ensure the standard of cure achieved and any recurrence or new infection. Once infected the cow can be considered as always susceptible.

Traditionally this type of case has been treated in the dry period, the single most effective method shown so far. Use of dry cow antibiotics is essential on all cows.

***Streptococcus uberis* mastitis**

S. uberis is now of increased importance as much of the contagious mastitis is better controlled. Clinical cases caused by *S. uberis* can present quite different problems. This organism is highly sensitive to penicillin G. In theory, treatment should be no problem. Administer a course of a penicillin intramammary tubes and the infection should be eliminated. This is true for many cases but a significant proportion are difficult to cure.

The strategy adopted by *S. uberis* to be a successful pathogen is different. *S. aureus* becomes intracellular whilst *S. uberis* produces a chemical which stops the white blood cells engulfing the bacterial chains. Understanding the significance of this requires appreciation of the action of antibiotics.

First, antibiotics are not disinfectants, they do not kill all bacteria. Some antibiotics will kill some bacteria but their main action is to stop bacterial growth and multiplication. This has two effects. The toxins produced by the bacteria are reduced limiting tissue damage and the

slower multiplication allows the white blood cells to remove the bacteria. If white blood cells are unable to engulf and kill bacteria, then at the end of the limited period of treatment the bacteria start growing again. This is what can happen with *S. uberis*.

It is important to treat promptly for the best success. Although primarily an environmental organism, *S. uberis* can be spread at milking time and this can be reduced by early action. Again the cure needs to be monitored.

S. uberis commonly invades the udder in the dry period. Dry cow therapy is effective in preventing this (8). Knowing that *S. uberis* is a herd problem indicates that all cows must receive dry cow treatment. Prevention is better than cure.

Other management points are a need to cull cows that cannot be cured clinically and to manage the exposure to *S. uberis* from the environment especially bedding (9).

These two examples illustrate the need for intelligence about the herd mastitis problem. This is essential for effective use of antimicrobials. These products are rarely 100% effective but usefulness can be optimised by understanding what they can do, which to use and how to use them.

SUMMARY OF APPROACH

- Identify the problem and the bacteria involved. This means examine treatment records, cell counts, bacteriology on milk samples and possibly antibiotic sensitivity tests.
- The treatment strategy for the individual cow must be dictated by understanding of the problem including the bacteria likely to be involved and if the infection is new or established.
- The best strategy is to treat as soon as possible.
- Treat for a long enough period of time and, if necessary, combine intramammary and injection methods
- Do not forget the cow, there must be post-treatment monitoring. There may be a need for follow-up treatment and always a need for dry cow treatment.
- Consider what to do with high cell count cows - treat or cull.
- Most mastitis causing bacteria have evolved to survive in the udder and usually succeed. Prevention is better than cure.

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CELL COUNT RECOVERY FROM MASTITIS

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SUMMARY

Major changes to milk cell count result from mastitis. Subclinical cases raise cell counts modestly but persistently whilst clinical cases cause immense changes. The duration of higher cell counts is poorly understood. Cell count recovery to pre-infection levels is only possible if the infection is eliminated, it only occurs in 50% cases and make take weeks to be achieved. Additionally there are changes in the proportions of macrophages, neutrophils and lymphocytes comprising the cells in milk during the different stages of mastitis. This may also have an impact on milk quality but these changes are even less well understood at present.

INTRODUCTION

Throughout much of the developed agricultural world the somatic cell content of milk is a major descriptor of the health of the cows producing milk, the quality of that milk and an influence on the value of the milk. Changes in price paid relative to cell content have been a fairly recent innovation but effective in the impact on bulk supplies (Fig. 1). It is unclear if this reflects any significant changes in the level of mastitis in the dairy herd. Milk is sold from recently mastitic cows as long as it appears visually normal and does not contain antibiotics. Some attempt at manipulating bulk milk cell count is made by withholding milk even in the absence of any detailed information of what the milk cell count might be. The cell count of milk, and information on the types of cells, in the aftermath of mastitis, contributing to bulk tank milk quality is reviewed.

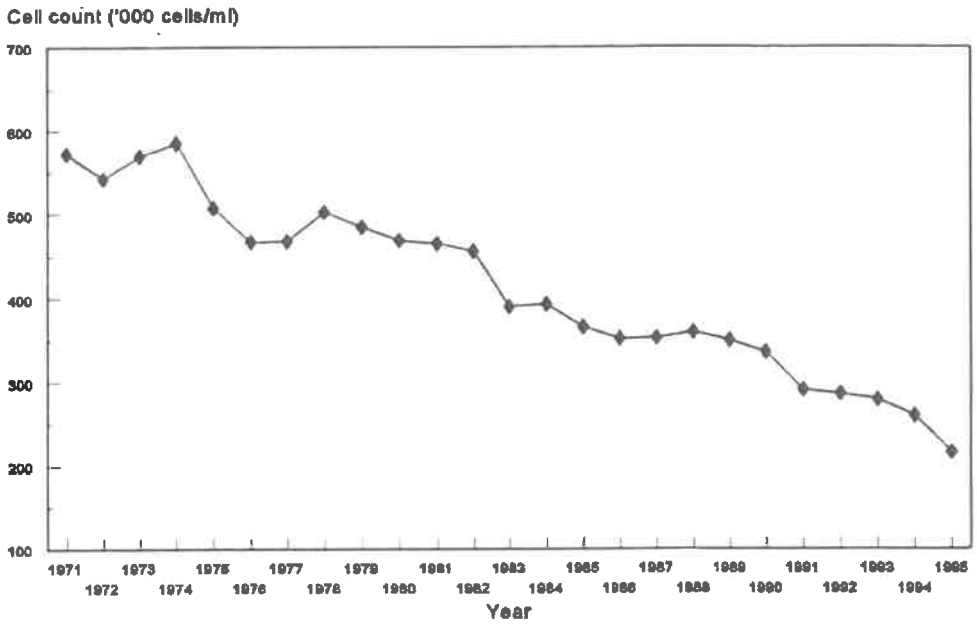

Figure 1 Annual average cell counts from the MMB and Milk Marque.

CELL CONTENT OF 'NORMAL' MILK

The cell count of milk varies throughout lactation for a number of reasons. It is high early in lactation as the udder develops production capability and high late in lactation as productive cells age and the tissue is remodelled. Fluctuations during lactation are caused by inflammation (mastitis), usually as a response to infection and very occasionally some physical or physiological trauma.

There are several types of cell making up a normal complement in the health udder. The total amount should be less than 100,000 cells/ml and even fewer in animals with no experience of mastitis. Some of the cells are epithelial debris, they are a small proportion in the milk productive phase and much more frequent in late lactation. Most cells are leukocytes (white blood cells) of various types. Normally 60% are macrophages. These are mature cells which can engulf bacteria. They predominate in the ducts of the gland and act as sentries to infection. If they detect foreign materials they produce alarm cytokines which induce polymorphonuclear cells (neutrophils) to invade. Neutrophils are usually 30% of the total count. They are phagocytes whose function is to remove debris and foreign materials. They produce some highly reactive chemicals, proteases and oxygen radicals, which are intended to kill bacteria but which also cause significant damage to the milk secretory tissue. The other common type (about 10%) is a mixture of lymphocytes which are stimulatory and antibody producing cells. Small fractions of other cells may exist and they can be common only in unusual circumstances eg a large presence of eosinophils indicates an allergic reaction.

CHANGES IN MILK CELL CONTENT WITH INFECTION

Infection, usually by bacteria, creates mastitis. The bacteria are recognised as foreign by the macrophages or via antibody attachment to the bacteria. This stimulates the macrophages to induce more leukocytes, predominately neutrophils, to invade from the blood circulation. The cell count rises. In the sub-clinical case, where the defences and the bacteria achieve some sort of balanced existence, the level may increase to 0.5-1 million cells/ml. Achievement of full disease, clinical mastitis, occurs when cell count rises further and clots appear along with other signs in the milk and the animal. The peak cell count achieved varies with the bacterial pathogen. The clotting of milk may mean that cell counts are not accurately determined. Approximately 4 million cells/ml is an average for *Staphylococcus aureus* clinical mastitis and over 20 million cells/ml for *Streptococcus uberis* or coliform disease. In the endotoxin mastitis model, where milk does not clot readily, cell counts of over 100 million cells/ml have been reported

The proportion of the different types of cell changes too (Table 1), neutrophils become predominant. The severity of the mastitis varies with the magnitude of the neutrophil response and the activity of their antibacterial systems (the proteases and oxygen radicals) which kill bacteria but also damage secretory tissue and so suppress yield. Yield depression with clinical mastitis is less with *S. aureus* mastitis than with *S. uberis* mastitis (2) because of the different cell counts which result.

Table 1 Change in cell count and percentage of different types of leukocytes in foremilk in endotoxin created mastitis (from 1)

	Cells at time (h)			
	number (cells/ml)		%	
	0	12	0	12
macrophages	112000	23 M	70	18
neutrophils	35000	101 M	23	80
lymphocytes	11000	2 M	7	2

(M = million)

CHANGES IN MILK CELL COUNT AFTER TREATMENT OF SUBCLINICAL MASTITIS

Generally subclinical infections respond poorly to antibiotic treatment with bacteriological cure rates of approximately 20% (3) and only sustained and more specialised treatments improve this (4). Treatment of subclinical mastitis is not considered practically or economically effective. One experimental study of the effect on cell count has been made (4). When bacteriological cure had been achieved the count occasionally dropped to 'normal' in 7 days, usually it took up to 5 weeks to become normal although this occurred in only 70% cases. In 20% of these 'cures' the effect was transient as these animals were soon reinfected.

Cell count remained high in 20% of the 'cured' quarters (Fig. 2). The remaining third of the quarters showed a partial drop in cell count to a late lactation level. Interestingly, in these quarters the cell type changed with epithelial cells being more common and so the quarter appeared to be drying off. The one significant change, whether cell count dropped or not, was that neutrophils declined from 60% to 30% of the total by day 7.

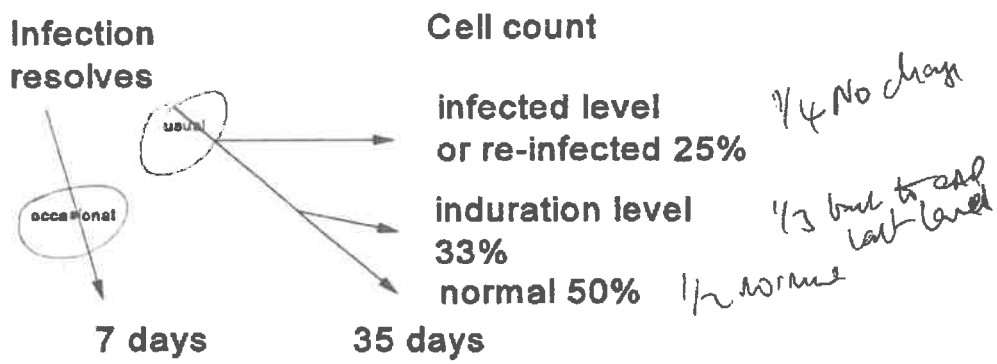

Figure 2 Timing and proportion of quarters with cell count recovery following treatment of subclinical mastitis.

CHANGES IN MILK CELL COUNT AFTER CLINICAL MASTITIS

The changes which occur depend on the bacteriological cure rate achieved which appears to be pathogen dependent, some 30% for *S. aureus* and up to 70% for *S. uberis* in lactation (Table 2). Resolution of clinical signs, without elimination of bacteria, converts the case to a subclinical infection with sustained cell count and sustained high number of neutrophils. If bacteria are eliminated, cell count is reduced and the rate of reduction varies with the severity of the disease, pathogen and cell count level.

Table 2 Cure rates for clinical mastitis by conventionally applied intramammary antibiotic

	S. uberis	*S. aureus*
Bacteriological cure	70%	30%
Clinical cure	90%	~100%
Cell count recovery (for bacteriological cures)	~100%	50-70 %

Conventional treatment methods may lead to problems as these methods are not introduced until relatively severe disease has occurred and cure rates can be low. Cell count cure rates have been rarely studied. The farmer is concerned with the recovery to visibly normal milk and only some time later with the individual cow cell count. The pattern of recovery in response to treatment is similar to that for cured subclinical infections.

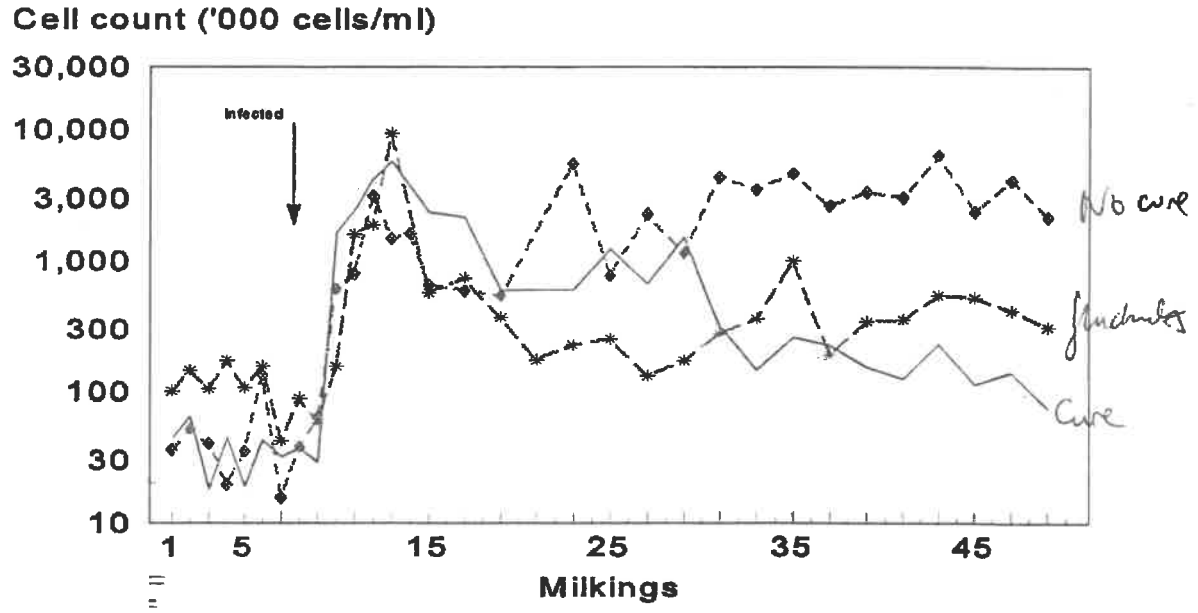

Figure 3 Cell count recovery curves for *S.aureus* infected quarters, cured bacteriologically and clinically, showing cell count cure, no cure (◆) and fluctuating count (✳) examples.

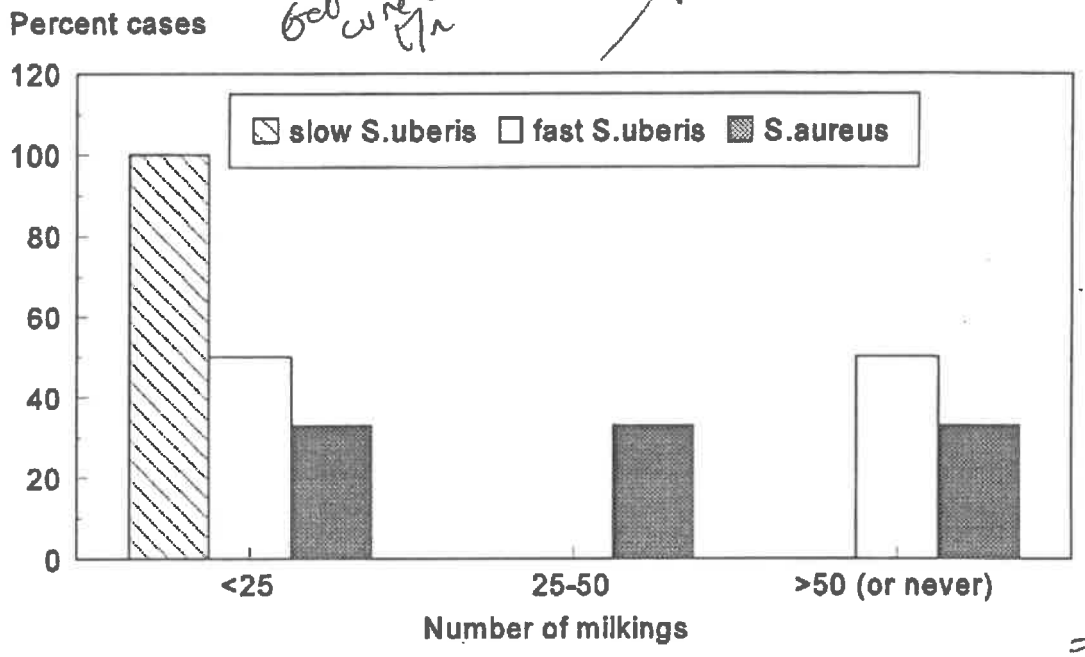

Figure 4 Relative rate of cell count recovery for *S.aureus* and *S.uberis* infections

Quarters apparently cured following a *S. aureus* clinical mastitis can shed quite different amounts of cells into the milk for a long time. A 'good' cure will result in cell count recovery, at least to below 400,000 cells/ml in approximately 20 milkings (Fig. 3) although pre-infection cell count may not be recovered in some animals within 50 milkings. Quicker recoveries are possible but there may be some fluctuation in cell count for a considerable time (Fig. 3). Some quarters appear clinically cured and bacteriologically cured yet continue to shed millions of cells/ml into milk for the rest of the lactation (Fig. 3). With *S.aureus* quick, slow and 'never' curing quarters may occur in equal proportions (Fig. 4). *S.uberis* clinical cases seem to resolve quickly or not at all (Fig. 4). The poorly resolving quarters are those in which clinical disease developed rapidly and this may reflect the state of the cow at the cow at the time of infection.

The only detailed study on the changes in cell count type during mastitis and the recovery process have been made in experiments with the endotoxin mastitis model, not with infectious agents (1). Initially there is a rapid rise in the number, and proportion, of neutrophils in milk (Fig. 5). This is accompanied by a smaller increase in the size of the macrophage and lymphocyte population. As the mastitis resolves the absolute number of cells declines with neutrophil and macrophages proportions returning close to normal but the lymphocyte numbers and proportion reduce more slowly and remain high for some time. These data are limited to the endotoxin model where mastitis is resolved in 5-7 days. Resolution of a bacterial mastitis takes much longer. No data are available to show the relative changes in cell type but when cell count recovery is up to five time slower then the milk must contain a high content of neutrophils and macrophages for much longer.

The cell count response to infection and disease can be immense and sustained for as long as infection is present. Otherwise it declines, often slowly, and in many cases never returns to the pre-infection level. When it remains high it stays so for at least the remainder of the

Time to cell count recovery
very variable

often 7-12 days
maybe over 25 days —
Do not know because SCL only
done monthly;
So [illegible] can have a graft on [illegible]

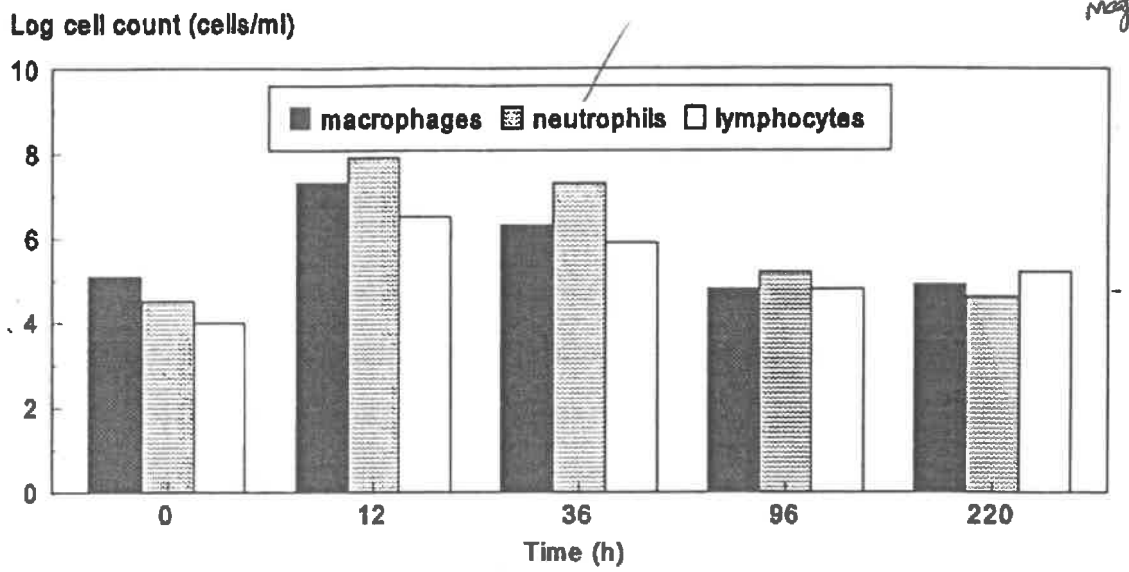

Figure 5 Relative proportions of different cell types during endotoxin mastitis (after 1)

lactation. There are also medium term changes in cell type which may be related to development of immunity. Whilst the absolute cell count has been the focus of interest for milk quality and price the effect on milk usefulness is more likely to depend as much on cell type content. Normal milk is obviously acceptable but milk containing a higher content of neutrophils might be considered less acceptable if the destructive effect of these cells on milk components and tissue continues.

CONCLUSION

Milk cell count and the cell types vary with the health of the udder. In the convalescent udder there are limits to the recovery possible.

There are two persistent problems

1. Post infection there are more cells in milk indicating poorer quality. This has consequences for the bulk tank supply, tissue damage and milk production levels

2. For a minimum of 7 days, and possibly for the rest of that lactation, after a return to saleability, milk contains significantly more neutrophils. This is not true for all quarters. Those that have a higher neutrophil content may produce milk with a much shorter keeping time and a lower processing value if it degenerates by action of extracellular proteases etc. This will be much less of a problem in the involuting gland.

Perhaps it is becoming timely to determine udder and milk health by use of cell type proportions and abundance rather than simply on abundance of cells. This could indicate better the fitness of the milk, the effect of infection on the udder and the effectiveness of the treatment regime applied.

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MASTITIS CONTROL - A TEAM EFFORT

MASTITIS CONTROL - A TEAM EFFORT

THE CONSULTANT

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SUMMARY

How a consultant can contribute to a mastitis management effort is described. An example of this illustrates the progress possible.

A herd with an average cell count of more than 400,000 cells/ml in November 1992 is now achieving results of 100,000 cells/ml and less while maintaining a low clinical incidence (22 cows affected in a herd of 149 during 1995). This was done by the farmer, his herdsman, the veterinary surgeon and the consultant agreeing a mastitis management plan and ensuring that the components were acceptable to all and applied routinely given the usual constraints on time suffered by any commercial unit. Regular (three monthly) visits to the farm and contact by telephone, if problems arose, ensured the close support to and co-operation of the team. Careful monitoring of the results at each meeting reinforced the purpose of good application of control.

INTRODUCTION

Mastitis is dynamic changing from season to season and reacting to even small alterations in herd management. It is not one problem responding to a single solution. On the farm, because of heavy penalties attached to high cell counts, there is a tendency to work on cell count management rather than tackle the underlying problem of infection level within the herd. It is clear that over the last 25 years there has been a remarkable decrease in the national average cell count (Table 1) and that the NIRD/CVL 5 point plan for mastitis control has played a major role in these improvements.

Table 1 Changes in the national average cell count of bulk tank milk

Year	Average cell count ('000 cells/ml)
1971	573
1976	467
1981 a	465
1986 a	352
1991 - 1992 a	289
1992 - 1993 a	282
1993 - 1994 a	268
1994 - 1995 b	218

a = MMB b = Milk Marque

The decline in the amount of clinical mastitis is rather different, Booth (1) reported that thirty years ago there were 135 cases per 100 cows. By 1980 - 1981 this had been reduced to 70 cases per 100 cows and in 1990 a MMB survey recorded 39 cases per 100 cows (2).

In the last five years there appears to have been little change and this is possibly a result of inadequate attention given to the prevention of new infection and poor understanding of the limitations of the 5 point plan in controlling environmental (coliform and *Streptococcus uberis*) infection. If cell counts are to be consistently low regardless of calving pattern, and the incidence of clinical cases is to be reduced, greater priority must be given to preventing cows becoming infected in the first place.

This is also a welfare issue. No one observing milking cows will be in any doubt that mastitis causes pain.

Some 62% of clients on the Genus Mastitis Control Service for two years have achieved bulk tank cell counts consistently below 250,000 cells/ml (3). It is the steady progress achieved by a continuing relationship between mastitis consultant and farm staff that gives the low bulk sample results that milk buyers are looking for and on which profit from milk depends. Lea Grange Farm has always been a 'well managed and resourced herd'. Its progress in further controlling mastitis is not a 'rags to riches' story but reflects fairly the majority of farm businesses in the East Midlands using advisory services.

THE ROLE OF THE CONSULTANT

Most farmers want good results for the lowest possible cost. Most herdsmen want good results but not to have to work 24 hours every day to get them. My job is to see that they get good results and remain friends!

Every farm is unique with a different system of management. An experienced adviser is probably well placed to decide which measures will bring about the greatest benefits on individual units in the shortest time. This is especially important given the number and range of products now on sale aimed at the mastitis control market.

However, the British climate is the biggest single problem faced by a mastitis consultant. It is a continual effort to stay one step ahead of what the weather is about to throw at the poor cows and interpret what this may represent in terms of bacterial challenge.

The first visit to any farm is the best time to get everyone working on the farm together, explain the mechanics of disease along with the control strategy and, in case there is any doubt, the financial implications of not controlling udder infection. It is important for the consultant to listen. The staff know the farm better than you ever will. Building the team, including the veterinary practice concerned, begins straight away.

Key points for this might be

1) Include everyone working with the cows, don't forget the relief milker.
2) Make sure everyone understands the aim of visits.
3) Discuss and explain face to face. Written reports are not always read.
4) The dairy staff must be given access to all sample results.
5) Access to current thinking, articles, meetings etc all stimulate interest and further discussion.

GETTING THE SYSTEM RIGHT AT LEA GRANGE FARM

It is important to understand the reasons for some of the changes that were made and why greater emphasis was placed on certain parts of the control programme there.

From the beginning the farm development plan included an ambition to increase herd size. Like many others the farmer was unable to change or enlarge everything at the same time and it was unlikely that replacements would be bought in. This meant that culling had to be limited back in late 1992. The first priority was that most effort be put into ensuring that heifers and young cows remained free from infection for as long as possible. It was not possible to segregate this group, which would have been the ideal, so it was back to the dedicated application of the 5 point plan plus a very critical look at the winter housing with continual assessment of this aspect. The approach succeeded.

Table 2 Average cell counts by lactation age (NMR Summary)

Lactation age	Sept 95	Oct 95	Nov 95	Dec 95
1	129	123	80	81
2	98	85	85	82
3	100	81	104	65
4	348	223	213	404
5 +	222	189	170	156

[The herd mainly calves between July and November which explains transient peaks.]

Table 2 illustrates that most infection is confined to cows with four or more lactations.

Dry cow therapy

Previously all cows had been dried off abruptly using a 30 day activity dry cow antibiotic. Dry cows are usually housed in the straw yard where they calve. To extend the period of protection, as cows are dry 6-8 weeks, a change was made to a longer activity dry cow tube, after consultation with the vet. Mastitis at calving is a rare event in this herd which also indicates good dry cow management.

Culling

The combination of good clinical records and the correct use of individual cow cell counts has ensured that only minimal, but necessary, culling for mastitis has been done. Cows suffering frequent recurrence of disease, probably the most infectious cows in any herd, have been given priority. Where possible cows with infections not responding to dry cow therapy, indicated by a consistently high cell count and a cell count of over 200,000 cells/ml in the first two months of the new lactation, are not served. These are usually older cows.

Following the first two visits to the farm two cows were added to the culling list and four more were to be monitored. These cows went at the end of their lactation. Culling for mastitis now averages 2-3 cows per year.

It is clear that anyone looking now to reduce cell counts quickly because of penalties should follow individual cow cell counts with more bacteriology, consider treating non-clinical cows, drying-off early, feed high cell count milk to calves, and probably, the culling of more cows.

The milking machine

The milking machine was a 12/12 hybrid. Throughput was good but it was difficult for the milker to give much individual attention to the cows and it was strenuous work; worse for the relief milker. The plant was tested by a Genus technician every year and improvements had been carried out as necessary. Although vacuum reserve and pulsation rate and ratio were to British Standard 5545 there was variation between the pulsators in the 'b' phases, 33-41%, and in the 'd' phase, 18-25%. The vacuum reserve was 630 litres/min but vacuum recovery was slow at some units. There were two sizes of claw piece in use.

The milking machine is about the only thing over which the dairy farmer has complete control and full advantage has to be taken of this. Therefore as the farmer was able to consider changes (in 1994) the old parlour was replaced with an 8/16 eye level unit with automatic cluster removers. It is still a hybrid and can be enlarged easily if the herd is increased again.

There is now a vacuum reserve of 760 litres/min, the pulsation 'b' phase is 46-47% and the 'd' phase is 22-25%. Large capacity claw pieces are fitted throughout. The working environment for the herdsman has improved and the cows are content.

The plant had been washed after every milking but checks showed that the water temperature was too low. The plant was, therefore, a possible source of bacteria apart from any mechanical influence. Attempts failed to make the boiler operate satisfactorily so the boiler was replaced.

Clinical records and treatment

Good clinical records have always been kept and treatment started promptly. Clinically infected cows are usually milked with a separate cluster into a dump bucket.

One of the arguments sometimes used against reducing cell count is that the clinical incidence will increase. It has not happened at Lea Grange Farm and probably will not as long as the emphasis remains on prevention of infection

Table 3 Annual clinical incidence

	Herd size	**No of clinical cases**
Dec 1993	121	21
Dec 1994	138	23
Dec 1995	149	22

Table 3 illustrates a continuing low clinical incidence at Lea Grange while herd size has increased.

Teat disinfection

Post milking teat disinfection is carried out by spraying. Good coverage is achieved and the herdsman is careful to spray as soon as possible after the units come off. At present an iodophor is being used but hypochlorite and chlorhexidine products have been used previously.

Teat condition is monitored closely before and after milking to keep chaps, sores and mechanical damage to a minimum. Healthy teats with skin in good condition reduces bacterial contamination (4) and this leads to a reduction in the number of new infections (5)

Housing

Most winters there are 2-3 coliform type infections. Up to last winter all the milking cows were housed in cubicles or kennels. The aim has always been to keep the cows clean between milkings so that a dry wipe is usually all that is needed as teat preparation for milking. In recent years the farm staff have put more emphasis on bedding management. In the winter of 93/94 a straw chopper was introduced to make bedding up easier but wet or soiled straw is always removed first. Passages are scraped twice per day in an effort to keep bedding dry. Straw is stored under cover.

Repairs to cubicle divisions, roof or guttering are carried out during the summer to make the daily management over the winter easier.

WHOLE HERD MANAGEMENT

Whole herd management seems to be the key. If any one aspect of mastitis control is ignored then more infections result. Just one extra infected cow makes the battle for lower cell counts that much harder. Paying attention to nutrition, comfort of the cows and handling with care, all make for a contented herd. Stressed cows and stressed workers do not give of their best for 365 days of the year.

Planning ahead and reacting to adverse conditions can make a real difference.

CONCLUSION

Today milk buyers demand, and pay more for, low somatic cell count milk (250,000 cells/ml or less) and low bacterial counts. From January 1998 milk with a three month geometric mean of more than 400,000/ml will not be acceptable.

Farmers wanting to remain in dairying must be sure now that their daily system of managing cows at milking time, and their cattle housing is geared to producing the low bulk milk counts that their buyer is looking for.

This requires a thorough appraisal of infection control measures and everyone concerned (farmer, manager, herdsman) must be in agreement, and work together, if milk production is to remain profitable.

The system must take into account the size and skill level of the workforce as well as the hours available including weekends.

What has made this farm such a pleasure to work on is that everyone wants what is best for the cows. Having realised that they were not progressing they were willing to take advice and act on recommendations.

A mastitis consultant cannot wave a magic wand over any farm but can only suggest ways of getting better results from the business.

Farmers have the right to demand the full co-operation of their staff - everyone's future depends on it after all - but in return they must provide the facilities and encouragement for the staff to do their job well.

ACKNOWLEDGEMENT

I am grateful to Elizabeth Berry for help in preparing this paper.

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MASTITIS CONTROL - A TEAM EFFORT

THE FARMER

A F HAYWARD, Lea Grange, Twycross, Atherstone, Warwickshire.

SUMMARY

The approach of a farmer, working with a mastitis consultant, to tackling an increasing bulk milk cell count is described. The plan adopted includes all aspects of mastitis management but with less emphasis on culling. Major changes were made to dry cow treatment by use of longer acting preparations, a milking machine update and especially support by the consultant to the farm staff. The support included interpreting the results on milk quality and explaining the strategies being applied.

INTRODUCTION TO LEA GRANGE FARM

The farm is rented from the Crown Estate Commissioners and is part of the Gopsall Estate at Twycross on the Leicestershire/Warwickshire border. It is staffed by a full-time herdsman, son Michael who is a partner, and two long serving part-time self employed skilled men. In addition to my involvement in the business, I have some commitments which take me away from the farm three or four days a month.

Farm Details : Spring 1996

328 acres :	90 acres winter wheat
	10 acres set-aside
	34 acres forage maize
	184 acres grass, temporary leys and permanent pasture
Stock :	150 Friesian/Holstein dairy cows
	70 dairy replacements

The farm is a good mixed farming area, being mainly medium to heavy soil type. The buildings which have of course been improved and extended over the years are adequate, but by no means 'state of art'. On part of the farm adjacent to the village alongside the main road, we have developed a small Garden and Aquatic Centre. Production from the cows is currently 6600 litres per cow and heifer in the herd at 4.24% butterfat and 3.40% protein. The TBC averages 4000 and cell count averages 155,000 cells/ml

BACKGROUND TO THE EXERCISE

In the year ending March 1993 the farm was progressing. Yields were moving up satisfactorily, TBC's were consistently in Band A, but the cell count averaged 349,000 cells/ml and was creeping ever higher.

At this time it was becoming obvious from reports in the farming press, and meetings attended, that in the not too distant future the buyers and processors of milk were going to demand higher standards throughout the milk producing industry. It was obvious that the problem had to be tackled.

The herdsman, who is still with us, was young and keen so we embarked on the task of achieving an improvement in the situation by a team effort. At the end of 1992 we joined the Genus Mastitis Monitoring Scheme. This brought Kate Allen on to the farm every three months to advise us and to monitor our progress.

CHANGES MADE TO THE MANAGEMENT OF THE HERD TO KEEP MOVING FORWARD

The high somatic cell count was the problem, so we decided to improve our overall standard of management throughout the herd. In our approach to the problem of high cell counts several factors had to be taken into consideration. As Tenant farmers on a pretty high rent which is reviewed every three years (always upwards), the financial effects were to be of great importance. I did not feel a drastic cull of those cows with the highest cell counts to be justified.

Dry cow therapy

The dry cow therapy programme was the first to be given attention. We had for over twenty years used dry period antibiotic on recalving cows. We infused each quarter of every cow at 'drying off' with a long acting antibiotic tube, such as Orbenin DC or Leo Red. A change to Cepravin Dry Cow tubes was made immediately. Cows are milked twice a day until the drying off date, usually 8 weeks before calving. The teat ends are wiped with cotton wool soaked in surgical spirit and a tube is placed in each quarter. The udder is then sprayed with our usual teat spray, a yellow tail tape attached and the cow is then removed from the milking herd into a separate group. If cows are dried off in the spring or summer, they will spend a few days in a well bedded yard, fed on a simple dry feed before being put out to graze in a convenient group, perhaps with in-calf heifers.

Culling programme

Although cell count readings were high, actual clinical cases of mastitis were not particularly numerous. Our response was that those cows which had the highest cell counts were not served again and eventually sold as cull cows, but some others were kept if they were still producing well. At no time did the culling programme exceed 20-22% overall, and of this percentage of total culls, only 2-3% were directly attributable to mastitis.

This less drastic culling programme has been carried out on similar lines over the years creating the present situation which is by no means excellent, but has put us in a more comfortable position. This may not have been the best way, but it has worked and it has done so without a severe programme of culling which would have involved considerable financial expenditure.

The milking machine

The milking machine is the most important piece of equipment, by far, on the farm and we try to ensure the best results by regular servicing. The plant is tested by a Genus milking machine technician annually and any faults rectified immediately. Two years ago we changed from an elderly 12/12 Fulwood Herringbone parlour with ACRs, to a hybrid which has been most successful. It is an 8/16 Herringbone with jars, Vaccar pulsation is fitted, Westphalia ACRs and Soffi claws, shells and liners. New sets of liners are fitted every three months. This combination may sound somewhat confusing, but it milks gently and quickly. We have recently used a very experienced relief milker for a few days and he rates it to be one of the best parlours in which he has worked.

The milking routine

Our daily routine for the dairy cows is probably very similar to many other farmers, and over the years we have found systems that work for us in our constant campaign against udder infections whether clinical or sub-clinical. The number of cows suffering annually with clinical mastitis has been around 22. Generally our daily routine and hygiene appeared good enough.

Milking is carried out at 6.00 am and 4.00 pm. When the cows enter the parlour they are fed part of their concentrate ration. The udders are wiped with clean paper towels before the clusters are attached. The only time we wash udders is when we obviously have to. I came to the conclusion many years ago that the less water that comes in contact with a cow's udder, the better. After milking, the udder is sprayed with Deosan Super Excel teat spray. This is a ready to use product, not cheap, but it is effective. The cows never get chapped or sore teats.

A bucket of clean soapy water is kept in the pit for the milker to rinse his hands from time to time. After milking, all units are scrubbed to remove dung etc. before the washing process. In the morning the usual type of hot wash, followed by cold is circulated. Deosan circulation cleaner is used. In the evening, cold water with hypochlorite added is circulated and run to waste.

Lactation therapy

Like everybody else we get our disappointments, but hopefully not too often. When a case of clinical mastitis does occur, our general approach is to use Tetra Delta tubes once per day for three days. If the cow is running a temperature we inject 20 ml of Engamycin for three days as well. Invariably the cow will be isolated and so we can strip out the infected quarter several times during the day. This last procedure is remarkably effective. The more severe the infection the greater the need for frequent stripping by hand. We have found Synulox to be very effective also and will use as recommended on stubborn cases which don't respond to Tetra Delta, or on first time severe cases which experience tells us need that treatment.

Housing and grazing

In the summer the cows return to their grazing fields as soon as possible after milking. In the winter when the cows are housed in cubicles and kennels, we try to keep them loafing in the outside feed area for at least half an hour before letting them in to lie down in the cubicles.

During milking, morning and afternoon, the cubicles and kennels are very thoroughly scraped. Also the concrete feed areas are kept as clean as possible. My first job in the morning is to do the scraping out. This enables me to check on the condition of the cubicle beds. The management of these is of paramount importance. We are lucky in that it is very rare for us to have a cow or heifer which refuses to lie in the cubicle or kennel. We have a 90 cubicle shed erected some 25-30 years ago and a 45 cow kennel house erected shortly after. The cows have access to both buildings and both seem equally popular. The original beds were made up of rammed soil/clay and are still the same today. We are fortunate in having a good supply of straw produced on the farm and that is what we use for the beds. Three times per week the Teagle Big Bale straw chopper is used to bed the cubicles and kennels and makes an excellent job. We use less straw with this machine and it makes a much more satisfactory job than when we used small bales spread by hand. The cows remain clean all winter, which of course is a benefit to them and also to us who have to work with them. Once per week hydrated lime is sprinkled on the beds before the chopped straw is blown in. We aim to prevent any build up of muck on the beds as this can be a major source of infection. At weekends when we don't use the Teagle, we just check the beds, remove any dung and rake a little clean straw from the head of the bed to cover any dampness. Our biggest enemy in the two sheds is wet! This is why we pay so much attention to this department.

In November 1995, a new covered yard was completed enabling us to reorganise our housing for the dairy herd. This building, which holds 30 cows, is at present used for stale milkers nearing the end of lactation. We are keeping this as a loose housed straw bedded area and so far it has proved to be a great success. We feel there is considerable advantage in having this type of housing to give us flexibility in the future perhaps to hold older cows or freshly calved heifers.

All the buildings enjoy good ventilation and during the winter the cows have a considerable area of open concrete on which to feed and exercise. They obviously enjoy this freedom because even on the roughest winter night a number of cows will be outside when I visit on my last look around.

CONCLUSIONS

I have endeavoured to convey my attitude and actions to the major problem of mastitis in our dairy herd. So many factors can have an influence for good or ill on the problem. Housing, milking technique, handling, the age of the herd and weather, to name but a few. The route we have attempted is one which has had as little detrimental financial impact as possible on the business. We can never afford to be complacent, but I am pleased to say we now have an improved herd which is profitable, pleasing to the eye and a pleasure to manage.

ACKNOWLEDGMENTS

My thanks go to Kate Allen of the Genus Mastitis Monitoring Service for her help over the period, particularly for the time taken with the herdsman on her visits to the dairy unit. The Cockburn Veterinary Group are always willing to give assistance and discuss relevant problems. Finally the improvements would not have been possible if our herdsman and the rest of the farm staff had not taken an interest and contributed to the exercise.

QUALITY FORUM

MATCHING YOUR MILK TO THE MARKET

PETER NICHOLSON, Robert Wiseman Dairies, Lake Road, Old Trafford, Manchester

SUMMARY

The path followed by Robert Wiseman Dairies to develop its current position as a major UK milk buyer is outlined. This includes close association with the milk producer and the retailing needs. Changes in the milk market have led to development of new milk purchase contracts requiring farmers to plan their output. It is now possible to sell to two different main markets, liquid and processing, which require raw product of different specification. Other markets will develop. Common to all markets will be the highest hygienic quality of milk and the image of the production system. Farmers can now chose a market and plan to produce the correct product for that market.

INTRODUCTION

Robert Wiseman Dairies

Robert Wiseman senior farmed in the central belt of Scotland, milking 100 Ayrshire cows. He watched as, through compulsory purchase orders, the development of the "New Town" of East Kilbride gradually reduced the size of his family farm. With a family of five to keep, it was necessary to change the direction of the business and so in 1948 he decided to meet the demands of growing population on his doorstep and went out to sell his product - liquid milk. Robert Wiseman Dairies was born.

The company has tried to pre-empt market requirements, keeping pace with, and wherever possible ahead of our competitors never losing sight of the core market, and always being prepared to move with it's demands. Actions taken include

- moving into the wholesale market when it became obvious that the consumers' buying habits were changing.
- tendering for supermarket contracts in the early eighties when all the talk was the protection of the doorstep delivery.
- together with Tetra Pak, pioneering the reclosable tetra top container to resolve the main concern associated with cartoned milk.
- having seen the change in buying habits in Scotland, migrating south and investing in the development of a brand new, state of the art liquid processing site at Manchester, totally dedicated to the production of quality milk in disposable containers for the supermarkets of England and Wales.
- working with Tesco to develop a 1 pint polybottle to meet their requirement of 100% plastic bottles for fresh milk.

- and latterly developing a contract to buy directly milk which reflects the quality of raw material the business requires for the future.

The UK milk market

Over the last 25 years the UK milk market has changed dramatically. Even ignoring the abolition of the Milk Marketing Boards in November 1994, the change has been significant.

In the milk year 1969/70, 62% of the milk produced in the UK was sold into the liquid market. Ten years later it was 41%. Had it not been for the introduction of quotas in 1984 the proportion would have continued to decline at the expense of a very new, expanding UK manufacturing market. The reduction in actual and potential production brought about by the quota regime stabilised the liquid milk market at around 50% of the total milk produced, where it remains to this day.

As far as the manufacturing market was concerned, the UK was very much the apprentice of Europe. A strong liquid milk market had always meant that the structure of the manufacturing market was based on balancing liquid requirements. The production was seasonal and rarely matched market requirements. Its increased importance from the early eighties onwards, meaning that the major companies involved in manufacture had to learn quickly from their counterparts in Europe in order to produce product that was attractive to the market. The arrival of quota restrictions in 1984 created further problems with over-capacity, making it necessary to speed up this process. Following a sharp learning curve, these companies rose to the challenge and UK manufactured products now compete successfully in European and world markets.

Milk payment methods followed these changes. The gradual increase in the significance of the manufacturing market meant that more importance was placed on the total solids element of milk as the yield of butter/cheese etc. affected the return on nearly half of the milk sold in the UK. This led to a significant change in the way milk was bought from farms in the early eighties. A price per litre dependant on total solids was replaced by the value being reflected in a pence per percent of butterfat, protein and lactose. This was aimed at concentrating the minds of farmers on increasing the compositional quality of milk in an effort to give the manufacturing market the quality of product they required.

The reducing popularity of butterfat throughout the eighties resulted in another change within the milk market. Low fat milks became popular, butter was replaced by low fat spreads and margarine and yoghurts, desserts and other niche market products began to increase in popularity. This was gradually reflected in the prices that the UK Milk Marketing Boards offered to farmers, with the value of the protein element increasing at the expense of butterfat. In 1984 the butterfat fraction represented 47% of the value while the protein was only 45%. In 1990 the balance changed and protein represented 48% while butterfat only 45%. By 1994 the gap had widened to such an extent that protein represented 60% of the value of milk and payment for lactose had been eliminated completely. This change reflected the market as the quantity of milk sold for cheese over the same period rose from 16% to 21% while that for butter and cream dropped from 32% to 19%.

During this period, dairy farmers had no option as to where they sold their milk, it went to the Milk Marketing Boards of the UK. They in turn had a responsibility to supply their customers with the quality of milk which would yield the best price. The liquid milk market price was volume based only, but there is no doubt that the monthly negotiations for the manufacturing milk prices did reflect the yields obtained and consequently the quality of the raw material. On November 1st 1994 the situation changed. Farmers no longer had to sell their milk to the Milk Marketing Boards and milk purchasers no longer had to buy from them. They had the opportunity to go in search of the quality of milk that their business required.

THE RAW MATERIAL

Milk marketing has been put on the same basis as any other business operating any where else in the world i.e. buying the raw material that suits the end product. However, few purchasers took the opportunity to benefit from the changes. In fact those liquid processors who got closest, writing a contract purely related to volume at a minimal compositional quality level, were unfairly criticised and their contracts ridiculed. Most of the purchasers interested in buying directly from farms had a single objective and that was to obtain as much milk as possible whatever the quality in order to secure their share in the market place. As farmers are fairly conservative it was price that was the main influence on signing new contracts. Many companies developed contracts which were purely variations on a theme with a guaranteed price above Milk Marque/Scottish Milk. Robert Wiseman Dairies was one of those companies. Having very little experience in buying from farms the company initially chose to mirror Scottish Milk's contract with a guarantee to sellers of a penny a litre difference.

The dairy industry is now changing its approach.. The initial phase has passed and companies now consider the raw material that is right for their business. In the lead up to the opening of the new factory at Manchester, with the prospect of launching a direct contract in England and Wales, Robert Wiseman Dairies reconsidered the makeup of it's contract. Changes were made to reflect the new market and for the future as the company saw it. The contract launched in October 1995 not only gave the dairy farmers of Lancashire and Cheshire an additional choice of buyer but it also gave them a choice of matching their milk to a specific market as happens with other agricultural enterprises e.g. a potato farmer will determine whether he wants to supply his product for the crisp market, for chipping or for boiling. If it is boiling then he will grow Maris Bard or Adora varieties, whereas Record would be for crisps and Maris Piper for chipping.

It is probable that an increasing number of companies will follow suit and that the dairy farmers of the UK will be able to consider what they want to produce from the land they farm and the animals they milk, and not be led to produce high total solids to get the best price. After all it is the bottom line that counts.

THE MARKET

The abolition of the Milk Marketing Schemes in 1994 didn't alter that half of the milk produced in England and Wales goes into the liquid market and half into the manufacturing market. What it did change was the method by which milk was sold or purchased. Milk purchasers had the opportunity for the first time to buy milk directly from farms paying them for the quality produced, whether it be butterfat, protein or hygienic quality. Previously they had only bought it by the litre and their alternative to buying it directly still relied on them buying it by volume rather than by quality.

To reflect the value of this option two markets can be compared. The liquid milk market has the priorities of keeping quality and freshness. Providing minimum compositional quality requirements are met, there is little point in paying a lot more money for high protein milk from the farms. It has little value in the end product and while its importance in health and fertility of the animal/herd should not be ignored, consistent protein of above 3.25% for this market is not justified. To a certain extent the same is true with butterfat with over half the milk in the liquid market being sold on a reduced fat basis (skim, semi-skim, standardised). However, the resultant by-product, cream, at present has a significant value and consequently I believe there should be a value to the production of butterfat over and above the minimum requirement. The optimum level of butterfat should be in the region of 4%. TBC's and cell counts are obviously very important in a market where the keeping quality of the product is the difference between retaining a supermarket contract and losing it. Freshness is also important, milk must be collected from the farm, get to the dairy, be processed and out to the supermarkets quickly as possible. Alternate day collection is therefore not a general practice with milk sold directly to Wisemans.

The cheese market however has very different requirements. Whilst the hygienic quality criteria are just as important, providing the milk is kept at the appropriate refrigerated conditions, alternate day collection has an economic advantage. The compositional quality of the milk required is almost the opposite to the liquid market with proteins of 3.25% and above being desirable as the higher the protein the greater the cheese yield. Butterfat should be at least 4%, anything below that resulting in too dry a cheese. However, too high a butterfat can also be a problem with this market making the product too soft and too creamy.

Other manufacturing markets will have different priorities and a picture is beginning to develop whereby certain types of milk are preferred for certain markets. The one common aspect is hygienic quality. Low TBC's and cell count are a requirement of every market and any farmer unable or unprepared to meet these standards has no future in the industry.

IMAGE

The image and perception of milk and its production are requirements of the future not just the scientific analysis of milk quality. Supermarkets are becoming increasingly involved and their interest in visiting farms is increasing. Whatever this industry thinks about this trend, it is inevitable and will affect all sections of the milk industry as the majority of milk and dairy products sold within the UK already end up on supermarket shelves. This percentage will only get bigger.

As a company, Robert Wiseman Dairies recognises this and issues probably the only contract for buying milk directly that reflects it. First impressions are important to the supermarket. Quality of product and service are essential and value for money is paramount. Buyers are quite prepared to pay a good price for the correct raw material. The industry needs to make sure that it's image, not only to the customer but also to the consumer, is good. Animal welfare and environmental issues are very topical with Codes of Practice and guidelines having been issued by three major companies with most of the others finalising drafts. There is still a lot of work to do in parlours and dairies to bring them up to the standards necessary for food production. It is for this reason that Wisemans have an independently assessed scheme clearly outlining standards of cleanliness and hygiene in this area.

The dairy farmers of the future need to be conscientious and have a responsible attitude to the image of their farm. It is no longer sufficient for them only to consider the cleanliness and appearance of areas which come in direct contact with their product. This is not the case in the processing end of the industry and the future will demand similar standards throughout the chain. Perception should not be under estimated.

CONCLUSION

It is two years since vesting day and most dairy farmers are used to the fact that they have a choice as to whom to sell their milk. This choice in the future will not only depend on price but also on quality. Quality will determine the product for which that raw material is most suitable. Image, perception and welfare issues are future requirements, and these need to be addressed now, but it is the production of butterfat and protein which will determine which market is most suited for a farm's milk. Milk with less than 4% butterfat and 3.25% protein will be more suited to the liquid market while milk with a higher butterfat will be more suited for manufacture. The choice is available to the producer with margins, instead of headline prices, becoming the over riding factor. Each and every farmer should have the ability to match his milk to a market, and if he has a choice, then it makes sense to use that choice before selecting the outlet for his milk.

TRAINING FOR MILK QUALITY STANDARDS

ROSEMARY BARFOOT, ATB Landbase Ltd, NAC, Kenilworth, Warwickshire.

SUMMARY

Market demands are changing, requiring dairy farm businesses to change. In order to manage change successfully, training is required to help staff understand and to formalise new standards. There are many benefits of training, which can influence the profitability of the business and improve the daily working relationships on the farm. The quality and success of training, however, involve the participation of far more than the tutor and trainees.

INTRODUCTION

Milk sold off farm must match the market. Information to aid this must get to those at the sharp end of the business - herdspersons, farmers and advisers. These people not only need to know what is required, but also how to achieve it. Training is one solution.

Training for this purpose, is the systematic process through which an individual is helped to master defined tasks or areas of skill and knowledge to pre-determined standards. This may involve actual training courses, or within-company structured development and instruction. The emphasis should be on structure.

The purpose of training is to develop self reliant, skilful workers, who can do the job to the required standard. This may involve technical training such as milk quality, mastitis control or developing herd health programmes, or it may be more fundamental management issues, such as communication skills or business planning.

Good training achieves these aims participatively, cost effectively and enjoyably.

In the current market, milk quality is no longer just about bacterial content, but embraces somatic cell count, compositional quality, antibiotic residues and other contaminants, and in the broadest sense, the farm assurance scheme which looks at overall management quality.

This market demand has changed considerably over the last few years and will continue to change. Just as in mastitis control, maintaining milk quality needs to be a team effort and therefore everyone on the farm needs to understand these changes, their role in achieving them and why management decisions are made to ensure that the milk meets the demand. The involvement in training goes beyond the farm gate and each and every one of us involved in the milk industry has a role to play.

Attitude to training is a fundamental problem in the agricultural industry, as too often it is seen as a cost rather than an investment. Yet companies such as CWS Agriculture are committed to and have used training throughout their estates and can demonstrate many of the benefits.

For training to be cost effective, it has to provide improvement. Training alone will not create that. The skills need to be put into practice, encouraged and developed. Too often training is seen to have "failed", because not enough thought has been put in before and after the training. The concept can be illustrated by the analogy of training as "seeds".

The aim is to plant some seeds, to grow and harvest the crop and to achieve the best return on investment. The task starts long before the purchase of the seed.

1. Decide on the objectives

To grow maize, there is no point buying wheat seed. Particular characteristics may affect the variety chosen. It needs also to be related to what else is being grown.

Training needs to be related to business objectives, as there is no point doing a mastitis course if the need is to improve ration formulation. The priority given to training should be related to the most critical business objectives, and planned in conjunction with all other training.

2. Choose the right place to plant

There is no point putting sun loving plants in the shade or arid loving plants in a wet clay field.

Who attends the training needs to be carefully considered. Is it appropriate to their present level of knowledge or capability? Are they receptive to this type of training? These are some of the questions which need to be asked.

3. Prepare the ground

Few would think of putting seed into unprepared soil.

Trainees need to understand why they are going on the course and what they are expected to get out of it. Valuable time can be wasted on courses while trainees adjust to why they are there, or some have the attitude of "don't know, don't care" and the whole event is wasted.

4. Plant the seed correctly

It is important that the seed is planted at the right depth and spacing and in the right way. Ensure it is quality seed being planted.

Training needs to be geared at the right level for the trainees attending and in time intervals which are appropriate. The training needs to be delivered in an appropriate way for the subject, so it is unlikely that maintenance of milking machines can be learnt solely in a classroom.

For maximum retention, it is important that training is delivered participatively whatever the subject, whether it is communication skills or animal welfare. People remember 20% of what they hear but 90% of what they say and do.

5. Nurture the seeds

No one would expect seeds to survive or do well with no post planting care. The young seedlings need food, water and weed control right up to harvest.

Trainees need to be nurtured on return from training. The role of their manager, or colleagues, is to maintain the motivation and learning created by the course, help it to develop back in the workplace and stop bad habits forming as they may detract from the new skills.

6. Evaluate the Harvest

It is important to determine how well the seeds performed and if there are any changes needed in their management.

Evaluation and management of training is a valuable and integral part of ensuring quality training.

From this it is obvious that all have a role to play and that their roles be agreed. They may be employers/managers setting clear training objectives for staff or selecting the right candidates, preparing the trainees by changing attitudes to training, encouraging its up-take or developing/monitoring/encouraging afterwards.

Whether as veterinary surgeons, advisors or tutors delivering training, the role is to ensure quality delivery, evaluating training and making changes. The follow up, where possible, is to encourage the trainees afterwards as well.

Those involved in commercial organisations, retailers, milk buyers or drug companies, must consider their role in sponsoring development of new courses, and encouraging up-take.

For those involved in research, the role surely is to help those tutoring access the latest information available. This is perhaps an area to improve on in the future with interactive communication technology.

Finally all have a role to play in passing skills learnt on to others and promoting the value of training.

If these roles can be adopted, then quality training does have tremendous benefits to offer, including

BENEFITS TO THE INDIVIDUAL

greater involvement in and commitment to the job - persistence
more job satisfaction and motivation
improved communication with other staff and employers
confidence in own abilities and feelings of achievement
higher status - (National or Scottish Vocational Qualification)

BENEFITS TO THE EMPLOYER

consistent standards achieved - fewer quality penalties
compliance with farm assurance schemes
less breakage, breakdown and accidents
timeliness of operations
low turnover of staff
less need for close supervision
improved profitability

BENEFITS TO THE INDUSTRY

improved image to the outsider
better quality of product
more customer satisfaction

These benefits are available to any business in the milk industry. The key benefits for milk quality are commitment, communication and persistence.

Commitment - Training can help gain commitment, because it can provide the opportunity to step back from the daily routine and business, to analyse what is being done in the presence of peers, share experiences and develop specific objectives. How often does the daily routine allow this opportunity to be taken?

Training can help understanding and someone is far more likely to do something if they understand why they should be doing it. Much of what happens on the dairy farm ultimately still remains in the hands of the herdsperson. It is important that they understand the reason for doing something, otherwise ways round it can be found. There are many instances where a problem cow for the milker just "never seems to be seen bulling" or a new piece of equipment they did not want "never seems to work". Wearing rubber gloves is a classic example, with their purpose rarely explained and so the gloves are never warn.

Communication - Everyone involved must be very clear what is being attempted and where they can help. The team approach is vital but suffers from a lack of communication on some farms. This could be greatly improved between the farm owner and herdsperson, the farmer and veterinary surgeon, and especially between the full time milker and relief milker. Problems can certainly exist between herdspersons and tractor drivers.

Yet the team is only as strong as the weakest link. It is just as important for the tractor driver responsible for bedding up to understand milk quality issues, and his role in maintaining it, as the herdsperson. There are frequently complaints of how a milker could do a better job, if the cows were cleaner, or how things always go wrong the weekend the relief milker is on. Generally too little information is passed on.

Change is inevitable, and yet managing change is perhaps one of the hardest management tasks. Many farmers have never had any management training and especially in this particular aspect. Experience indicates that training in communication skills, business planning and development, managing change and assertiveness training may have a greater influence on maintaining milk quality than many of the technical courses.

Persistence - At present, mastitis cannot be eradicated therefore control is never ending, and maintaining milk quality is an every day event. For the herdsperson milking twice a day, six or seven days a week, whilst maintaining the enthusiasm to carry out a full routine of control measures can be very difficult. Training can help motivation and make them realise that they are not alone with their problems.

In all of these is the word **can**, because it depends not just on the quality of the training, but what effort is applied before and after.

CONCLUSION

To survive, businesses have to change and meet the challenge of the future. In order to do this successfully, they need to improve communications and train. Everyone has a role to play in encouraging, sponsoring, developing and/or delivering quality training.

PRODUCING QUALITY MILK

TERRY DRAYCOTT, Institute for Animal Health, Compton, Newbury, Berks.

SUMMARY

All farms have their own particular targets in achieving optimum performance especially in milk quality. A large number of factors influence these eg animal effects or feeding. In many cases staffing can be equally important. It is essential to decide what to achieve, have a plan to achieve it and to involve everyone in the task. The highest quality achievable at all levels is the goal to secure a future in dairying.

INTRODUCTION

Since the deregulation of milk sales in November 1994, the marketing of the daily harvest of milk has taken on an entirely new dimension. It is necessary to produce milk beyond a minimum quality standard set by buyers to ensure achieving the highest price possible. However, it is the margin which is important and a high price is little good if the costs of production are too high. All farms have their individual problems. Producing milk at the Institute for Animal Health is no exception and poses special challenges.

The institute is unique in having an 800 hectare estate to help to support the science programme, both with the supply of animals of known health and genetic status and financially. The supply and use of animals causes a series of problems, including consumption of all of the calves, except dairy replacements, and the 'loan' of animals for scientific work.

Briefly, the farm consists of 400 Ha cereals, oilseed rape and set-aside; 90 Ha forage maize; 20 Ha lucerne; 160 Ha temporary grass; and 60 Ha permanent grass. This supports 350 outdoor sows, 550 polled Dorset ewes and 1100 cattle of which there are 380 Holstein/Friesian dairy cows. The cows are kept in two herds of 230 cows and 150 cows. Te current M.O.P.F. is £1300 per cow, butterfat is 4.01%, protein 3.3%, Bactoscan 12-20 and somatic cell count 170-195 cells/ml. The herd used for mastitis work has the lower cell count !

MILK

First comes the decision to whom to sell the milk. This includes deciding on the market available, the optimum price per litre for the type of product that can be produced and the achievable or target margin. Part of the choice depends on the destination of the milk, into the liquid or processing market, as the specification of milk required can be very different. The decision also includes some ideas on how consistently the product can be produced, in volume, composition and quality over the whole year. Compton is fairly high on chalk where it gets very dry in summer. Rainfall is only 24 inches per year.

Eventually a suitable milk buyer is found and a favourable contract secured. Next comes the planning for the production of the product within the capabilities identified in the farm strategy

Composition

The first problem is usually how to feed the cows to manipulate butterfat and protein. There are lots of opportunities to vary rations, from grass varieties in seed mixtures, through mixed forages with some new or old combinations eg whole crop spring barley with peas or the old favourite oats and vetches.

Composition is also affected by health. This can be health of the rumen, lameness affecting food intake or mastitis. All may depress yield, change salt content of milk, alter fat and, especially, change the types of proteins in milk. Mastitic milk contains many more destructive enzymes reducing keeping quality of the liquid product and lowering processing value. Managing milk composition involves health management as well as rationing. So far, it appears unknown if altering milk composition by diet has any effect on mastitis levels or incidence. It is possible that mastitis-causing bacteria may grow better or less well in different milks.

Quality

The standards used to assess quality are TBC (Bactoscan now for many) and cell count. Achieving the target levels is a continuous process. Momentary lapses are slightly forgiven by the payment systems but if these are due to missed cases of clinical mastitis, eg a high TBC from missing a case of *Streptococcus uberis* mastitis, then the staff are not so forgiven !

Generally, the highest standards of care and attention to detail are needed in managing housing, collecting yards and grazing to control TBC. This is a daily task as well as a long term task involving maintenance and finding time for this on a tightly staffed dairy farm is difficult even if the money is available. Some times the money and time just have to be available.

Controlling hygiene also controls mastitis. One herd at Compton is on straw yards and most mastitis tends to be *Streptococcus uberis*. The other is in cubicles and coliform infections are more common. Rarely do *Staphylococcus aureus* cases of mastitis occur, only 2 of over 1500 quarters have such an infection. This is entirely due to diligence with the five point plan, especially teat dipping, and a bit of extra information from monitoring of clinical cases and cows drying off. There are around 25 clinical cases per hundred cows per year. All clinical cases are treated promptly and thoroughly. All quarters of all animals to re-calve into the herd receive dry cow therapy

ANIMALS

The standard of cow at Compton has improved significantly over the last 5 years. They produce a lot more milk, despite the scientific work. The aim is to breed better cows for production and efficiency and disease resistance. This can only be a long term approach for which more understanding of where to aim precisely would be useful

The welfare of the cows is a primary consideration. Improvement is an ambition of the institute. New welfare codes are all the rage. They suffer from inconsistency although there is a lot of common ground. Realistically the contents of these codes are only common sense and in many cases best practice. Welfare is only one example where the farm relies on a team approach and using the best information to achieve a target.

The target is enshrined in the contract to supply. The plan comprises how to do that in the most efficient way whilst meeting all of the contractual, ethical and quality objectives. The animals produce the output and so require the best attention possible. The whole system depends however on the staff involved, all of the staff, and the management.

STAFF

Getting good staff, and then keeping them, has always been a problem in this industry. It is not getting any easier as the jobs become more technical and the socio-economic conditions less acceptable. It is possibly not very important whether staff are young or old, whether they are formally qualified or not and whether they are ambitious or not. It is important that they are trained, informed and motivated.

Quality of output from staff requires that they know what to do, how to do it and when. Qualified staff may be recruited but they still need to learn new skills whether they are use of technical equipment, animal care or, increasingly data management and information systems.

The best training of staff is a continuous process with them learning about the aims of management and most of all understanding why the farm goals have been set. Staff need to be trained and briefed regularly on what is going on, what the target is, how much success is being achieved and how the individual can contribute.

Success does not come from individuals though. It is achieved by a team. At Compton the animal staff are in teams whether at the individual dairies or the support team who feed, manage the young stock and provide relief to the dairy teams. They all know the target. They have individual roles, with a lot of flexibility, and they rely on each other. Superimposed on all of this is a motivation to achieve.

Motivation has to be provided by management. It is complicated to achieve and methods may differ widely with different individuals. The students are motivated by the long-term goal of a permanent job (out of overalls ?). Whilst others may be motivated by career development, satisfaction at doing a good job (if that is not too old fashioned), occasionally financial reward but for very many appreciation of effort and being part of the team goes a long way. There is no best motivator simply a need to find the appropriate blend for the individual member of staff. Only when they are all working to the best of their ability can the highest targets be set and there be hope of attaining them. Then all may have a rewarding future.

CONCLUSIONS

There is no simple way to produce quality milk and no universal applicability. Dairy management includes

- feeding the cow properly
- using the best hygienic methods to prevent and control problems
- perhaps breeding better cows

- keeping cows under the best possible conditions
- but mostly it depends on using and supporting staff in the best way possible for them and the farm.

The buyer of the future wants a quality product from a quality unit so ensuring a future in dairying for all involved.

THE EVALUATION OF BULLS FOR SOMATIC CELL COUNTS IN THE UK

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SUMMARY

Analyses of UK cell count data indicate a heritability of cell count of 0.11. The known correlation of mastitis incidence with cell count, data readily available from milk recording schemes, suggests a possible indirect means of breeding to reduce mastitis incidence. Genetic evaluations for cell count have now been produced and will be available to the industry in 1997.

INTRODUCTION

Despite a significant reduction in the incidence of both clinical and subclinical mastitis over the last 25 years (3) mastitis remains one of the most costly health problems of dairy cattle and a major source of economic loss to dairy farmers. This improvement was largely due to the success of management control programmes. The continuing economic loss has raised interest in breeding as a means of reducing the incidence of mastitis. However, breeding to reduce the incidence of mastitis has not been possible due to problems in routinely collecting data over the national herd. Herd average somatic cell counts (SCC) have been routinely collected on a monthly basis since 1977. These have been widely used as a monitor for mastitis with a high SCC used as an indicator of potential problem cows within the herd. In conjunction with various management control measures the herd average SCC levels have declined from 573000 cells/ml to 268000 cells/ml in 1993 (2). Since 1991 Milk Recording Organizations (MRO) in the United Kingdom (UK) have introduced an optional SCC recording service. Approximately 80% of all cows are now routinely recorded each year. For the first time data on SCC for individual cows are readily available in sufficient volume to allow the proper assessment of the genetic parameters of SCC and if suitable to enable the production of genetic evaluations for bulls and cows.

The purpose of this paper is to summarise the results of analyses of UK SCC data and to discuss the genetic evaluations produced and the problems identified.

MATERIALS AND METHOD

SCC data were obtained from 3 MROs and records were validated using the general rules applied in the national production system. Additional edits were used for SCC records. Since SCC data are highly skewed a logarithmic transformation was applied to the lactation average (geometric) SCC in order to produce a normal distribution. A minimum of 6 test day records were required before accepting the lactation average. This led to the loss of many records from the first 2 years where this information was not available.

Heritabilities were estimated from first lactations using a univariate animal model restricted maximum likelihood (REML) procedure. The effects included were herd year season, month and age of calving, herd sire interaction with breed proportion included for Holstein Friesians. The analysis was repeated using the first three lactations with the additional effects of permanent environment and lactation number included.

First lactation records for SCC, milk, fat and protein yield were used to estimate the correlations between the traits. A sire model was used with the same fixed effects as described for previous analyses.

The genetic parameters obtained were used to produce genetic evaluations for bulls and cows. Examples are shown for the Holstein Friesian, Ayrshire and Guernsey breeds. The genetic evaluations were carried out using an Animal Model Best Linear Unbiased Prediction (BLUP) procedure similar to that used for the production traits in the UK (1). As with production the first 5 lactations were included. The model included fixed management group effects, month of calving, age within parity (linear) and random effects of herd - sire interaction, permanent environment and animal.

The evaluations were expressed as Predicted Transmitted Abilities (PTA) and were expressed as deviations from a fixed base, the average PTA of cows born in 1990. The Transmitting Abilities (TA's) obtained for Holstein Friesians were correlated with the genetic evaluations for conformation traits for the same bulls.

RESULTS

Table 1 shows the range of herd average SCC found in the Holstein Friesian data. In the case of the first lactations, herd averages for SCC ranged from 21000 to 399000 cells/ml with individuals in these herds ranging from 9000 to in excess of 3 million cells/ml. With all lactations the top and bottom herds averaged 30000 and 713000 cells/ml respectively with individuals ranging from 7000 to 6.8 million cells/ml.

Table 2 shows the number of records per lactation, means and standard deviations for the Holstein Friesian (HOL), Ayrshire (AYR) and Jersey (JER) breeds. The means and variances of Log_{10} SCC (LSCC) generally increased with lactation and were highest in the Jersey breed.

The heritability estimates for first lactation LSCC were 0.11, 0.12 and 0.09 for HOL, JER and AYR respectively. The estimates from lactations two and three were similar to those from first lactation records at 0.14 and 0.10 respectively.

The phenotypic correlations between LSCC and milk (-0.12), fat (-0.11) and protein (-0.11) yields in the first lactation were negative but the genetic correlations positive i.e. antagonistic. The genetic correlations with milk, fat and protein yields being 0.06, 0.14, and 0.09 respectively.

Table 1 Distribution of herds by somatic cell count range

Somatic Cell Count Range (000/ml)	Number of Herds
20.1 - 40	56
40.1 - 60	819
60.1 - 80	1624
80.1 -100	1735
100.1 -120	1633
120.1 -140	1214
140.1 -160	827
160.1 -180	578
180.1 -200	387
200.1 -220	277
220.1 -240	173
240.1 -260	100
260.1 -300	116
300.1 -400	95
400.1 -500	23
>500.1	12
	9669

Table 2 Means and standard deviations for $\log_{10}$ somatic cell count by breed

	Holstein Friesian			Jersey			Ayrshire		
Lactation	N	Mean	SD	N	Mean	SD	N	Mean	SD
1	63,414	4.85	0.33	14,509	4.94	0.35	7,966	4.84	0.33
2	31,412	4.92	0.34	8,071	4.97	0.38	4,237	4.95	0.36
3	11,109	5.02	0.37	2,948	5.06	0.41	1,558	5.03	0.39

The distribution of TAs for LSCC for Holstein bulls with at least 50 daughters and cows with lactation records are shown (Figs 1 and 2 respectively). Nearly all evaluations were between +/-0.13 with 80% and 96% of bull and cow TA's respectively being within +/-0.05. The distributions of LSCC TAs for the AYR and GUE breeds followed the same pattern.

The heritability estimates for first lactation LSCC were 0.11, 0.12 and 0.09 for HOL, JER and AYR respectively. The estimates from lactations two and three were similar to those from first lactation records at 0.14 and 0.10 respectively.

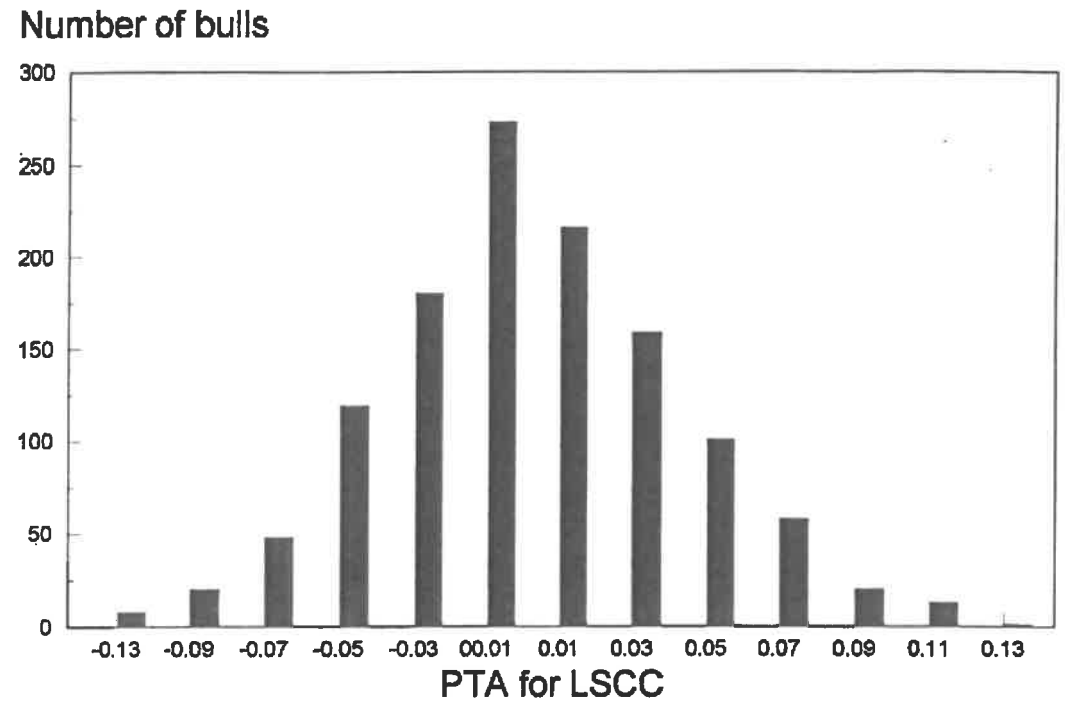

Figure 1 Distribution of bull cell count predicted transmitting ability

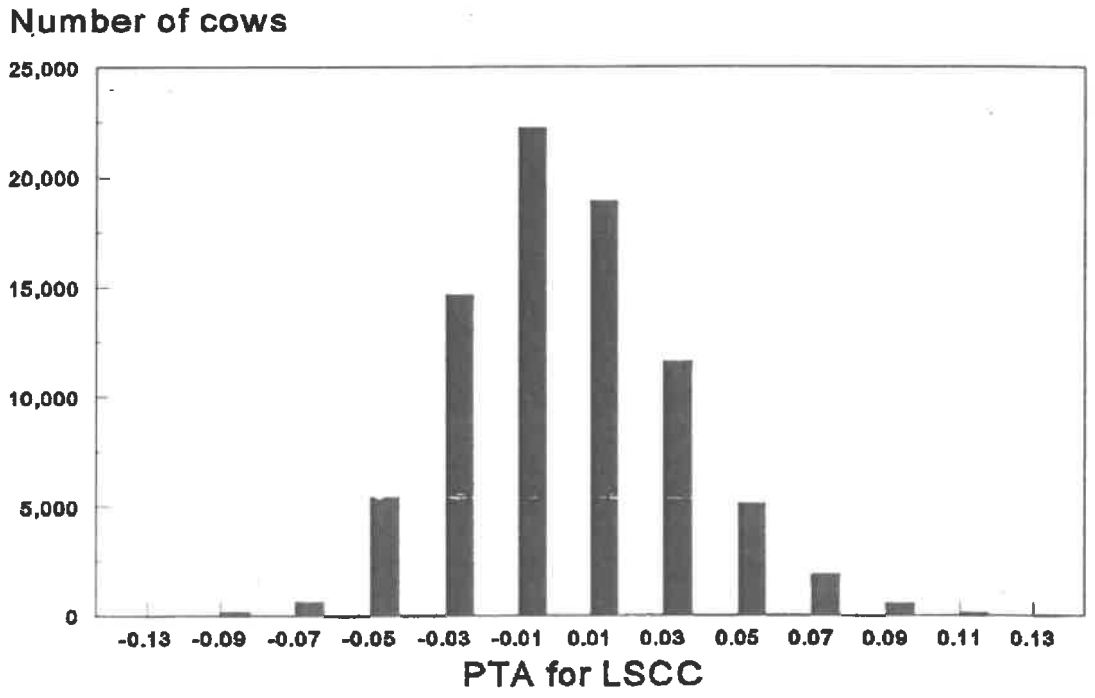

Figure 2 Distribution of cow cell count predicted transmitting ability.

narrower – less cows in data set

The proportion of bulls with a reliability > 50%, the minimum publication standard, was 17%, 5% and 8% for the Holstein Friesian, Ayrshire and Guernsey breeds respectively. For cows, 54%, 10% and 25% respectively had reliability's >30%, the minimum standard.

The TAs for LSCC were correlated with the genetic evaluations for type traits for Holstein Friesian bulls with at least 50 daughters. In general the genetic correlation's were effectively zero for the body traits. Small but significant correlations were obtained for rear legs side (0.12), foot angle (-0.14), fore udder attachment (-0.19), udder depth (-0.19), teat placement side (0.11), teat length (0.15) and beef shape (-0.19).

DISCUSSION

First and foremost from a UK standpoint what do these results show? Over all breeds SCC was found to have a reasonable heritability of approximately 0.11. Although not as high as production at 0.35 it provides scope for selection. Secondly the genetic correlations between SCC and the yield traits were positive indicating continued selection for yield will lead to higher SCC levels. In the light of the continuing high cost of mastitis and increasing penalties for high herd SCC levels this is a concern.

The UK results are similar to those obtained in other countries. The heritability estimate of 0.11 compares with the same weighted average estimate for 14 analyses from other countries (5). Most importantly this estimate is higher than that of mastitis incidence from various studies of approximately 0.04 (5). A review of the literature confirmed a genetic correlation between SCC and incidence of mastitis of approximately 0.7 (5,6).

The higher heritability for SCC, together with a favourable genetic correlation between SCC and mastitis incidence and with a national scheme for routinely collecting SCC data all confirm SCCs suitability as an indirect trait for breeding to reduce mastitis incidence. Given this and in the light of the continued losses due to mastitis plus pressure to reduce SCC within payment quality schemes all point to the benefit of producing SCC evaluations.

The consequence is that genetic evaluations for SCC are now produced in 7 countries. In Scandinavia and Denmark, close cooperation with the veterinary profession results in the collection of detailed mastitis records and information from both SCC and mastitis is used in genetic evaluations systems. The combination of both measures was estimated to be approximately 20% more efficient than selecting on SCC alone (6). However there seems little prospect of detailed mastitis records being collected in the UK in the short and medium term. It has been reported (4) that indirect selection on SCC produced 93% of the response of direct selection for mastitis resistance emphasising its value as a single trait. However they also found that inclusion of udder traits and milking speed with SCC in index was more effective and produced a 1% higher response than direct selection for mastitis resistance.

POSSIBLE PROBLEMS FROM SELECTING ON SCC

It has been suggested that selection for lower SCC may reduce the cows' ability to respond to infections. A recent study (6) examined the linearity of the genetic relationship between SCC and clinical mastitis. They reported a linear relationship between the bulls Relative Breeding Value (RBV) for SCC and clinical mastitis in 2 Swedish breeds. They concluded that from the size and linearity of the genetic relationship between both, that selection for lower cell counts is desirable and reflected a reduced incidence of infection rather than ability to combat them. In addition, clear evidence is accruing that sires with the lowest PTAs for SCC have daughters with the lowest incidence of mastitis (7). A limited analysis involving ADAS data produced a similar conclusion (Animal Data Centre, ADAS unpublished results).

A further concern is that environmental pathogens such as *E. coli* may be becoming more prevalent. Since these produce a high SCC for relatively low duration monthly SCC sampling may only detect 10 - 20% of infections, thus reducing the effectiveness of selection. However it has been found (7) that sires with the lowest PTA for SCS had daughters with the lowest incidence of mastitis from all pathogen groups including environmental pathogens. They also observed that clinical episodes, in daughters of sires with low somatic cell PTAs tended to be less severe and shorter in duration when compared with clinical episodes in daughters of sires with average to high PTA for SCS.

These studies confirm that selection for low cell counts, could improve resistance to mastitis and that low cell counts do not reflect reduced ability to respond to infectious organisms.

PROBLEMS ASSOCIATED WITH PRESENTATION OF RESULTS

Methods of presenting evaluations vary across countries. Ease of interpretation and understanding of genetic evaluations, is important since SCC, although of interest, is not a primary selection trait but is of value in identifying extremes. Various alternatives are shown in Table 3. In the USA the same method of presentation (SCS) has been adopted by both Milk Recording Organisations and the genetic evaluation service, which has obvious advantages. Other forms of presentation, for sire evaluations, include Relative Breeding Values (RBV) in Denmark and Germany, Estimated Breeding Values (EBV) standardised to a mean of 100 and standard deviation of 10 in Finland and Sweden and PTA after adding the mean of standardised $\log_2$ SCS for first lactation cows born in the base year to all evaluations, in the USA. The EBV for SCS for three lactations from a test day model in Canada are combined to give an overall EBV, based on their economic values. The Netherlands produce Estimated Transmitting Abilities (ETA) transformed back to SCC scale (ie: 1000 cells/ml) and deviated from a base group. At present discussions in the UK favour Transmitting Ability (TA) expressed as a percentage deviation from a fixed base or converted to a financial value. No decision has been taken as yet.

The LSCC, as obtained directly from the evaluation system, whether as TAs or breeding values (TAx2) expressed as deviations, are difficult to interpret in relation to the way cell counts are presented on farm ('000/ml). The addition of the mean, as favoured by the USA and Canada, apart from removing the potential problem of interpreting the sign (+ or -) as good or bad, does not change this situation.

Table 3 Presentation of somatic cell count evaluations

Alternatives	Range of Evaluations	Comments
Log SCC	-0.15 to +0.14	Transmitting ability, (deviation)
Log SCC	-0.30 to +0.28	Breeding value, (deviation)
Log SCC	4.66 to 4.95	Overall mean added
STA LSCC	-3 to +3	Standardized transmitting ability
RBV	90 to 110	Relative breeding value
%TA	-10 to +10	% Transmitting ability
000/ml	52 to 88	Transmitting ability

An alternative favoured for the presentation of type traits is to standardize the evaluations by dividing by a measure of the variation of each trait known as the standard deviation. This results in the evaluations falling in the general range +/-3 for each trait. While this has been accepted for type evaluations this is largely due to the fact that a visual comparison of a large number of traits is required. This is not the case for SCC where some clear understanding of the evaluation is necessary.

The option of presenting the results as published on farm, thousands of cells/ml, was also considered. However, the small range for bulls at +/-15000 cells/ml when compared to the large range of cell counts for herds and, in particular, cows again highlighted problems of acceptance. The favoured option to date is % Transmitting Ability where evaluations are expressed as a percentage deviation from a fixed genetic base e.g. +2%, -4% etc.(see Table 4). This is easy to explain and understand and applies regardless of the average cell count in the herd. Another alternative being considered is to express the result as a financial value following the success of publishing the National Profit Indices PIN and ITEM in this way (8).

PROGENY TESTING

The practical implication of the results for breeding programmes is that the lower heritability of SCC will mean a lower reliability for bull evaluations than for production given the same amount of information. In practice therefore more information is required for the same degree of confidence in PTAs as for yield. For example, whereas 60 daughter lactations per bull would result in a good initial progeny test with reliability of 85%, for SCC 140 lactation records would be required. As a consequence caution is recommended. While individual bull evaluations will be published and extremes identified the most efficient way of using the information for bulls, and in particular cows, will be to include SCC within the national profit

index, ITEM. Work is currently underway to do this by Scottish Agricultural Colleges and Edinburgh University.

Table 4 Somatic cell count evaluations for Holstein Friesian bulls

Top 5

Bull Identity	No. of DTRS	REL %	PTA LSCC	SCC (000/ml)	TA %
64365515	893	96	-0.11	-15227	-11
64379906	68	73	-0.10	-14377	-10
63805426809	58	64	-0.10	-13885	-10
01420709	309	91	-0.09	12762	-9
01432671	1307	98	-0.09	12762	-9

Bottom 5

Bull Identity	No. of DTRS	REL %	PTA LSCC	SCC (000/ml)	TA %
64333836	498	97	0.08	14406	+8
651814930	1376	97	0.08	14406	+8
651874645	1584	98	0.08	14406	+8
64379639	296	90	0.08	14595	+8
64334489	55	70	0.08	15355	+8

CONCLUSION

The analyses on SCC data in the UK agree well with results obtained in other countries. The heritability of SCC is confirmed at 0.11 which is similar to other countries and the correlations with yield traits although low are positive and therefore antagonistic. This together with the known high genetic correlation with mastitis incidence and the ready availability of SCC from MROs confirm its suitability as an indirect means of breeding to reduce mastitis incidence. Fears that selection for low SCC will lead to problems appear unjustified. The continuing high cost of mastitis losses, despite the reduction in incidence over the last 25 years together with increasing penalties for high SCC levels in milk will continue the pressure to reduce SCC levels. Breeding has a useful role to play in addition to management control programmes. Genetic evaluations for SCC have been produced successfully and will be launched as part of a service to the Industry in 1997. Research is underway to incorporate SCC into ITEM, the National profit index since this may be the best way to use the information.

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Select for
Low SCC
Mastitis incide
speed of milking (flow rate)
slower rate lower SCC?
Yield ↑ related how??

ABSTRACTS OF POSTERS

Heywood Velvarus Strep uber
No summary for Proceedings?!

BACTOSCAN - A NEW METHOD FOR DETERMINING THE TOTAL BACTERIAL COUNT (TBC) OF RAW MILK

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Traditionally the total bacterial count (TBC) of raw milk is determined using an agar plate method. A small volume of milk is mixed with a suitable agar medium and incubated, usually for 3 days at 30°C. During incubation, the bacteria in the milk multiply to give colonies which are then counted and the number of bacteria per ml of milk calculated.

This method has three major drawbacks.

1. It takes three days to complete leading to delays in identifying high TBC problems.

2. Some important types of bacteria are not determined either because they do not grow, or, they grow insufficiently to give colonies large enough to be counted. Examples are anaerobes and psychrotrophs.

3. Other types of bacteria do grow sufficiently to be counted but are underestimated because they occur in the milk as either clumps or chains. Each of these multi-cell units produces only one colony and therefore is counted as one individual bacteria. A good example is mastitis causing streptococci.

However, technology moves on and in an attempt to overcome the above problems Foss Electric have developed a new method for determining the TBC of raw milk in the form of a milk analyser called the Bactoscan.

The Bactoscan works by taking a small sample of milk, cleaning it up to remove somatic cells, fat globules etc. and treating the resultant bacterial suspension with a special dye which stains all living bacteria. These stained bacteria are then directly counted under blue light thus giving an instant count of the individual living bacteria in the milk.

Bactoscan overcomes the drawbacks of the agar plate method for the following reasons.

1. It takes only 10 minutes to complete thus identifying hygiene problems very rapidly.

2. It counts all living bacteria irrespective of whether they are capable of growing in agar media under standard incubation conditions.

3. It breaks down all clumps and chains of bacteria in the milk thus counting the individual cells in multi-cell units.

Because agar plating and Bactoscan are fundamentally different they give totally different results. As a very general rule of thumb, the Bactoscan count is five to ten times greater than the TBC as determined by agar plating. However, this relationship varies enormously for individual milk supplies depending on the microflora of the milk.

ADAS has investigated a large number of cases where the Bactoscan count is significantly greater than ten times the agar plate count result. In the majority of cases this has been due to high numbers of psychrotrophs, although in a few instances high counts of Clostridia sp. were the cause.

The Bactoscan is more accurate than agar plating for determining the TBC of raw milk. Therefore, sometimes it will detect hygiene problems previously not revealed by the traditional method.

EC HYGIENE DIRECTIVE - A MAFF SPONSORED CAMPAIGN TO REDUCE SOMATIC CELL COUNTS

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ADAS, on behalf of MAFF, has provided a programme of free consultancy visits to help producers of high cell count milk to meet the requirements of EC Hygiene Directive 92/46. The objective was to communicate the following message to producers of milk with cell counts around and in excess of 400,000 cells/ml.

- EC Hygiene Directive 92/46 was implemented in May 1995. The Directive sets maximum levels for somatic cell counts in milk. Initially, this standard is being implemented at the level of bulk consignments to milk buyers. From 1 July 1997, the standard will apply at individual producer level.

- Supplies with a three month geometric mean somatic cell count higher than 500,000 cells/ml will become unsaleable after 1 July 1997. From 1 January 1998 the standard will be a maximum of 400,000 cells/ml.

- Somatic cells are mainly white blood cells which are the animals first line of defence against invading pathogens. Bulk milk cell counts and clinical incidence are indicators of the effectiveness of mastitis control measures.

- Thorough implementation of the NIRD/CVL Five Point Mastitis Control Plan is the most effective method of controlling clinical mastitis, sub-clinical mastitis and associated somatic cell counts.

An initial programme of producer meetings was held throughout England.

A total of 428 visits have been made in response to requests from milk producers. A copy of the letters confirming advice given has been sent to each farm's veterinary surgeon.

Rapid progress has been made when producers have fully implemented the Five Point Plan. The main reasons for not achieving lower cell counts have been failure to disinfect teats, no dry cow therapy, poor milking technique, inadequate milking machines, and failure to identify and cull cows with recurring mastitis.

Milk buyers are recognised as being amongst the most significant influencers on dairy farmers attitudes. In order to ensure that the somatic cell count message reached all producers, presentations have been made to a number of milk buyers and other influential groups.

Comprehensive information relating to the whole industry is not presently available, following the change in milk marketing arrangements. Information from individual milk buyers suggests that somatic cell counts are now much lower than in April 1994.

USE OF INDIVIDUAL QUARTER SOMATIC CELL COUNTS IN DIAGNOSIS

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Somatic cell counts (SCC) are being used increasingly by milk producers and purchasers to estimate the amount of sub-clinical mastitis in a herd. Further investigation involves thorough study of a series of individual cow cell count records.

The George Veterinary Group considers that a consistent cow cell count of greater than 200,000 cells/ml indicates infection but individual cow cell counts do not identify the quarter(s) or the organisms involved. Bacteriological examination of quarter samples can be used for further information. However, aseptic sampling by clients, especially when taking a lot of samples, frequently results in a high level of contamination. Further repeat sampling by the veterinarian is often necessary. This is expensive as bacteriological examination costs from more than £3 per sample (negative result) to more than £8 per sample for a positive identification of bacteria. These problems have led to a different approach being adopted.

Individual quarters only are sampled. These are selected by first measuring individual quarter cell counts of suspect (high cell count) animals. This method leads to accurate identification of infected cows, identification of causative organisms, requires less laboratory work and halves the cost. An example of the simplicity of this system is shown.

SOMATIC CELL COUNT CONTROL IN SCOTLAND

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The introduction of regulations enshrining EC 92/46 created a need for some Scottish dairy farmers to improve the hygienic quality of their milk (primarily Bulk Tank Somatic Cell Counts or BTSCC). Consequently, they were subjected to a major education drive in 1993-95. The methodology was developed by SAC in an earlier pilot study funded by the 3 Milk Marketing Boards in Scotland. Producers were informed that elevated BTSCC figures are predominantly due to sub-clinical mastitis and that control of this is based on limiting the extent and spread of infection and where possible eliminating pre-disposing factors. This project, funded by the European Union and the Scottish Office but requiring producers to fund much of their individual herd investigations, applied these methods throughout Scotland with the aim of substantially reducing the BTSCC of Scottish producers within 2 years.

Technology transfer was approached systematically. BTSCC and Total Bacteria Count (TBC) profiles were distributed to illustrate individual herd problems, encourage basic mastitis control and initiate herd investigations. Producers coming forwards with problems were then assisted individually. Herd investigations were based on the analysis of Individual Cow Somatic Cell Counts (ICSCC) and bacteriological sampling of selected cows. Subsequent herd-specific advice was tailored according to these results and knowledge of individual management practices. Progress of the project was assessed by evaluation of the data collated.

The co-operation of milk buyers and access to milk quality data were essential for the production of profiles but were severely disrupted by de-regulation. Nevertheless, clear improvements were made in milk hygiene: the number of producers in Scotland consistently in excess of the EC SCC threshold (3 month geometric mean >400,000 cells/ml) was halved, and there was also a substantial improvement in TBC. Many herds were infected with *Strep. agalactiae* and/or *Staph. aureus*. Where *Strep. agalactiae* predominated, response to control measures was generally good but where *Staph. aureus* was the causal organism improvement was slower, as might be expected.

Modelling showed that, in order to keep the BTSCC consistently below 400,000 cells/ml, the annual average must be less than 250,000 cells/ml. The use of a full dry cow therapy programme was the most cost effective sub-clinical mastitis control procedure studied giving a return of £4.60 for each £1 invested. Furthermore, reducing BTSCC from over 400,000 cells/ml to under 250,000 cells/ml resulted in an estimated increase in financial margins of approximately 5%.

ISLE OF MAN SOMATIC CELL COUNT PROJECT (MAY '95 TO MAY '96)

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The single central creamery on the Isle of Man operates as a statutory based co-operative. Following advice from SAC concerning the implementation of EC Directive 92/46, and funded by the Isle of Man Department of Agriculture, Fisheries and Forestry, the Isle of Man Milk Marketing Association began to implement a control scheme for milk quality with particular reference to somatic cell counts. In May 1995 a milk liaison officer was appointed from the local community to work for the creamery as a link between milk producers and all other interested parties including DAFF, NMR, local veterinarians and the NFU. To obtain baseline information about the dairy industry, 97 out of 103 farms were fully surveyed and a database constructed. Analysis showed there were a number of management factors associated with bulk tank somatic cell count. In summary, the average herd size was 62 cows and herringbone parlours, dry cow therapy, post milking teat dipping, record keeping and having a closed herd were all associated with a lower SCC. Analysis of data from all three years confirmed that to avoid exceeding 400,000 cells/ml (the EC threshold), farms required an annual mean of less than 250,000 cells/ml.

A co-ordinated approach was employed using quarterly BTSCC profiles for each individual producer along with individual on-farm contact via the milk liaison technician and the introduction of more stringent hygiene penalties. Herds with BTSCC exceeding 250,000 cells/ml were encouraged to conduct individual cow somatic cell counting to identify problem cows. Bacteriology results from selected groups of these animals was used to give herd specific advice on SCC control. Bacteriology from farms on the Island shows that *S. agalactiae* is less prevalent on the island than in Scotland. *S. aureus* was isolated from samples in the majority (48%) of submissions.

The general progress by producers on the island has been dramatic moving from 350,000 cells/ml in January 1995 to 165,000 cells/ml in July 1996. Almost 40% of producers were in excess of 400,000 cells/ml in January 1995, and in July 1996 this had reduced to 3%! Milk intake to the creamery in 1996 was 6.3% greater than 1995 despite no change in cow numbers. It is believed that this is at least in part due to this improvement in cell count. It is hoped the collaboration cultivated between the creamery, producers, local vets and DAFF will allow the development of this scheme into other aspects of dairy cow welfare.

BOVINE MILK NEUTROPHIL CONTENT AND CHEESE QUALITY

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An ELISA test was developed to detect neutrophils in bovine milk. The neutrophil is the most important leucocyte in acute inflammation and is considered a more specific indicator of mastitis than total SCC. The correlation for 89 milk samples between neutrophil numbers, determined microscopically, and the ELISA was found to be 0.97.

It is known that elevated SCCs in milk are linked to poor cheese making properties such as increased cheese moisture content, longer rennet clotting times and increased proteolysis during ripening. It has also been found that bulk tanks of similar total SCC may contain varying levels of neutrophils. As neutrophils are an important source of tissue-degrading proteolytic enzymes, the ELISA test was used to investigate the effect of variations in bulk milk neutrophil content on cheese quality. A preliminary study found that Gouda-type cheese made from high neutrophil milk had inferior texture and flavour as compared with that made from milk of similar total SCC but lower neutrophil levels. Further studies have shown that neutrophil proteolytic enzymes, elastase and cathepsin B, as well as crude preparations of the cells themselves, may influence casein breakdown in controlled cheese ripening model systems. Relationships between these activities and textural and flavour defects in cheese made from high SCC mastitic or late lactation milk are apparent. These results highlight the significance of the neutrophil and indicate an important role that the immunological measurement of these cells may have for the screening of raw milk in the cheese industry.

SURVEY OF CLINICAL MASTITIS INCIDENCE DURING WINTER HOUSING PERIOD

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Management practices, such as housing hygiene, milk practices and drying off procedures are important risk factors for mastitis occurrence.

A survey was carried out on 544 members of the Genus Mastitis Consultant Service in England and Wales for the years 1995 to 1996 to investigate the clinical mastitis incidence during the winter housing period. Data were collected on herd size, type of housing, clinical incidence for the whole year and winter housing period and reasons cited by the mastitis consultant as to why there was a high clinical incidence. The clinical incidence was calculated only for those keeping accurate clinical records and for those members with records for a complete year. This was calculated per 100 cows for the whole year and winter housing period. None of the farms surveyed practised a total confinement system.

For the 544 herds average herd size was 106, with an average clinical incidence per 100 cows per year of 32 cases (range 0 to 163) and an average of 20.5 cases (range 0 to 74) per winter housing period.

357 herds housed cows in cubicles. Average herd size was 109 and average clinical incidence per year was 29 cases with 19 cases per winter housing period. 36% of farms had 20 or more cases per housing period.

21 herds housed cows in cubicles and yards. Average herd size was 120 and average clinical incidence was 34 cases with 21 cases per housing period. 50% of farms had 20 or more cases per housing period.

7 herds did not house cows over the winter. Average herd size was 62 and average clinical incidence was 30 cases with 16 cases during the equivalent housing period.

The reasons cited by the mastitis consultants as to why farms may have had a high clinical incidence were:- poor housing management (ranging from not bedding up or scraping out frequently enough, to using insufficient or wet bedding), overstocking, poor building construction, dry cow management, and failure to follow the 5 point plan fully.

For cubicle housing of those farms with a high incidence 52% were due to poor housing management, 4% to overstocking, 15% to poor building construction, 21% to failure to follow the 5 point plan and 8% to dry cow management.

For yard housing 54% were due to poor housing management, 17% to overstocking, 7% to poor building construction, 8% to failure to follow the 5 point plan and 14% to dry cow management.

For cubicle and yard housing 60% were due to poor housing management, 10% to overstocking, 10% to poor building construction, 10% to failure to follow the 5 point plan and 10% to dry cow management.

In this survey, yard housing had both the highest yearly clinical incidence and winter housing clinical incidence, followed by cubicle and yard housing, with cubicle housing having both the lowest yearly clinical incidence and winter housing clinical incidence.

In all types of housing the main reasons cited were poor housing management.

ACUTE TOXIC MASTITIS IN COWS

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A bovine mortality survey carried out in Northern Ireland identified coliform mastitis as the single most important cause of death in dairy cows accounting for 12% of dairy cow mortalities which received veterinary attention (Menzies *et al.*, 1995). As a result of this finding, an epidemiological study into acute toxic mastitis in cattle is presently being performed to identify risk factors associated with the occurrence of the condition on an individual animal and a herd basis. Twenty veterinary practices have been requested to submit milk and blood samples along with information relating to cases of acute toxic mastitis they examine and treat. Milk samples are cultured and antibiotic sensitivities are performed on bacterial isolates. Routine haematology, urea, creatinine and haptoglobin measurements are carried out on blood samples. Follow-up questionnaires are sent to the relevant farmers requesting further information about the affected cow and a matched control from the herd.

Preliminary findings from 75 cases indicate that the majority of cases of acute toxic mastitis are caused by infection with *Escherichia coli* alone (54% of cases). Thirty percent of acute toxic mastitis cases died or were culled.

Data will continue to be collected on individual cases of acute toxic mastitis until information is available on 300 cases. The second stage of this project will involve a further survey which will target identification of herd management practices which are risk factors associated with the occurrence of the disease.

REFERENCE

MENZIES F D, BRYSON D G, McCALLION T & MATTHEWS D I (1995) A study of mortality among suckler and dairy cows in Northern Ireland in 1992. Veterinary Record, 137 531-536

EARLY DETECTION AND EARLY TREATMENT OF NEW MASTITIS CASES

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A high degree of accuracy in detecting developing clinical mastitis, in advance of any visible signs, has been achieved by measuring the electrical conductivity of foremilk. Now work has been undertaken to show how this information can be used.

A comparative study of treating mastitis created experimentally with *Staphylococcus aureus* or *Streptococcus uberis* has shown that starting intramammary antibiotic treatment when conductivity changes as opposed to when clots appear has significant benefits. Early treatment prevented virtually all clinical signs. A bacteriological cure of 97% was achieved. Significantly less damage to udder tissue, as determined by a much lower depression of yield, occurred. Also the average time for cell count to recover to the pre-infection level was at least 7 days less. These benefits were achieved by treating for six consecutive milkings. This required a similar amount of antibiotic to the national average use rate, more than the label recommendations, however, the high bacteriological cure rate achieved leads to a lower rate of recurrence of disease and thus a lower strategic rate of use of antibiotic.

IMMUNE AND INFLAMMATORY RESPONSES IN *STAPHYLOCOCCUS AUREUS* MASTITIS

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Herds of dairy cows infected with the mammary gland pathogen *Staphylococcus aureus* often have a dominant strain present in the majority of infected cows and the dominant strain varies among herds. Four adult Friesian dairy cows suffering from chronic staphylococcal mastitis were studied. Two of the cows, both from the same farm, were naturally infected with strain A, and two cows from a different farm, were naturally infected with strain B. Each of the cows was challenged in a double crossover design such that one cow from each farm was challenged with the indigenous and one cow from each farm was challenged with the non-indigenous strain. The four cows were monitored bacteriologically and immunologically to determine whether the indigenous *S. aureus* strain was displaced by the challenging strian and whether challenge with either strain modified the immunological response. Bacteriological monitoring comprised initial culture and strain identification of *S. aureus* by restriction enzyme fragmentation pattern analysis.

Cows which were challenged with their own indigenous strain of *S. aureus* (either A or B), as expected, only had the indigenous strain identified in the milk post-challenge. This indicated it unlikely that other strains of *S. aureus* occurred in the cows during the experiment. Cows which were challenged with the non-indigenous strain remained chronically infected with the indigenous strain although the non-indigenous strain was isolated from the cow challenged with strain A three days after challenge.

Peripheral blood mononuclear cells isolated from the cow infected with strain A and challenged with strain A, proliferated *in vitro* to both strain A and strain B to a similar degree. The time of peak proliferation varied from day 7 to day 10. Peripheral blood mononuclear cells isolated from the cow infected with strain B and challenged with strain A, proliferated *in vitro* to both strain A and strain B, but the response was far greater to strain A. The time of peak proliferation occurred on the same days as with the other cow. The kinetics of peak proliferation suggest a primary rather than a secondary immune response is occurring in the systemic circulation in spite of the fact that the cows were chronically infected with *S. aureus* at the time of sampling. Phenotyping of the responding cell populations revealed that they were composed of T cells expressing CD4, CD8 and γ/δ molecules.

The inflammatory response following intramammary challenge with *S. aureus* was monitored by quantifying the individual quarter somatic cell counts. No association between somatic cell numbers and the ability of the cows to clear intramammary infection was recognised.

ACKNOWLEDGEMENTS

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HOW DO YOU TREAT YOUR COWS!

MARTIN F H SHEARN, Institute for Animal Health, Compton, Newbury, Berkshire, RG20 7NN

Approximately 13 million antibiotic tubes are used on UK dairy cows every year. How? For obvious reasons antibiotic treatment is carried out in the milking parlour. After the cow is milked, the quarter may be stripped out, the antibiotic tube nozzle pushed into the streak canal, its contents infused into the teat and the quarter massaged. Teats are probably then disinfected and cow let out.

FINE, but how often is the teat end cleaned before treatment? How often are the tubes of antibiotic stored in a dirty cupboard or shelf?

Treatment of the mammary gland should be taken more seriously. The antibiotic should be kept in clean, cool and DRY storage. When required, the tubes should be placed conveniently in the parlour where they will not become contaminated. After cluster removal the quarter should be hand stripped, the teat end cleaned with medicated wipes (one may not be sufficient), the nozzle of the tube should be **partially** inserted into the orifice and the contents infused into the teat canal. Massaging the udder will help to disperse the antibiotic into the quarter.

Treatment of the cow at drying off is similar except that the udder should not be massaged. Dry cow therapy is a preventive as well as a therapeutic procedure. Antibiotic left in the streak canal and teat cistern will disperse naturally around the quarter, leaving a trace in the teat. The early dry period is a time when new infections are likely to occur and dry cow antibiotic will help to prevent invasion of the streak canal by bacteria.

HOW DO YOU DRY OFF YOUR COWS?

MARTIN F H SHEARN, Institute for Animal Health, Compton, Newbury, Berkshire, RG20 7NN

In the recent past drying off cows at the end of lactation was not a problem as yields tended to be low then. Even so many cows were dried off by changing the milking interval from twice to once a day, then missing a day or two. This was what happened for generations of family farmers'. Research in the early 1970's showed that the risk of infection is increased in unmilked quarters compared with milking normally. Continuing to milk cows at less frequent intervals prevents the complete involution of the udder.

Cows today sustain yield until they need to be dried off. They should be milked twice a day up to drying off. Checks made over the next few days should not involve stripping out a little milk to reduce the pressure. The milk will be absorbed in a few days. If at all possible disinfect the teats with `teat dip' for a few days. The udder pressure at drying off from the milk is unlikely to be as high as just prior to calving.

Further treatment in the dry period may be given for prevention of summer mastitis or precalving coliform, however it will have little effect on any infections not cured by the initial dry cow treatment.

MASTIMIND QUIZ

A H ANDREWS, Royal Veterinary College

This competition has been successfully run for four years and provides a lighthearted look at the major problems of mastitis, its control and milk quality. It is hoped that all delegates to the Conference will be prepared to fill in the questionnaire which will take about five minutes. The questionnaire is completely confidential and only the identity of winners will be subsequently known by their declaring their having the winning entry number. Prizes will be awarded to the best and, in particular, it is hoped that farmers will undertake the competition and they will receive at least one prize for their efforts.

ASYMMETRICAL UDDER SYNDROME IN DAIRY COWS

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This condition is not a common herd problem. Six cases have been investigated by Sutton Bonington Veterinary Investigation Centre over a period of several years. Affected cows have one or two quarters on one side of the udder smaller than those on the other. The condition develops slowly. Milk yield in affected quarters is substantially reduced compared with contra-laterals, but total yields do not appear to be markedly depressed. There is no evidence that the condition is caused by an infection - it is not a mastitis. It has been seen as early as two weeks after calving. There is no evidence of genetic involvement.

The condition has appeared in some herds following the installation of new plant. Let-down failure has not been a feature, and affected cattle are not particularly nervous. There is no evidence of 'stray-voltage' involvement. The problem has eventually disappeared in all affected herds investigated following changes in milking plant. However, no consistent faults were found in the plants, and alterations made were not identical.

Whilst the condition could result from several causes, one explanation is that milk is not being removed completely from affected quarters. Consequently less milk is then secreted into these quarters and atrophy or involution occurs.

Involvement of one half of the udder suggests that pulsation might be involved, and in the first herd investigated the situation improved markedly when 2-2 pulsation was altered to 4-0. However, this change was carried out in other affected herds without obvious benefit. Long milk tubes of insufficient length, causing teat cups to hand at an angle on one side of the udder, were seen only in one herd. "Liner creep", in which tissue at the base of the teat is sucked into the teat cup liner, obliterating the connection between teat and gland cisterns was a marked feature in only one herd. It is emphasised the condition is not common as a major herd problem, and the precise cause has not been elucidated. However, it could be more widespread in individual animals.

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