THE BRITISH MASTITIS CONFERENCE

JOINTLY ORGANISED BY

MILK MARKETING BOARD

AFRC INSTITUTE FOR ANIMAL HEALTH

and CIBA-GEIGY AGROCHEMICALS

I N D E X

I N T R O D U C T I O N

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The British Mastitis Conference has been set up as a forum for all those concerned with the many aspects of present and future mastitis control. It is hoped that this Conference will become an annual event along the lines of the highly successful National Mastitis Council in the United States.

The Conference will review changes in disease patterns, progress made in control, economic and quality aspects from farm and production angles and review latest developments in research.

Much of what is done to control the disease today stems from the original research started at the National Institute for Research in Dairying more than 25 years ago which resulted in the now widely adopted control routine. The results that have been achieved over that period have certainly proved that the NIRD work has stood the test of time, but despite all the progress made, mastitis is still a major disease problem in UK dairy herds.

However, there has been some concern expressed recently in a number of areas that economical and political pressure on dairy farms has perhaps led to cutting of corners and even abandoning the well proven methods of control. Clearly there is a need to look again at the present position and what may happen in the future to ensure that the substantial benefits gained in the last 25 years can be continued and improved upon and it is hoped that this Conference will provide the necessary forum for this to be achieved.

SESSION 1

Changes in incidence , causes and the nature of mastitis problems

PROGRESS AND PROBLEMS IN THE UK: AN OVERVIEW

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Summary

Over the last 20 years the overall level of mastitis in the UK dairy herd has been more than halved. There has been widespread adoption of the recommended control measures and most forms of the disease have been reduced substantially. Nevertheless, mastitis still costs British dairy farmers an estimated £90 million a year. There remain problems with mastitis due to the so-called environmental bacteria and with summer mastitis. Antibiotic resistance has not been a problem.

Introduction

Imitation is the sincerest form of flattery. This conference has been planned as the first annual British Mastitis Conference, in the image of the National Mastitis Council of the United States (1), in order to bring together as equals and on a regular basis all those with an interest in the control of mastitis. Initially the subject will be mastitis of cows, but there is no reason why goats and sheep should not be included in the future and even some of the more exotic ruminants.

It is highly appropriate therefore to start off with a brief review of the progress made in controlling mastitis and to highlight some of the problems encountered. In this paper I propose to examine progress over the past 20 years, since 1968. As a prelude it is worth recalling the words of C D Wilson almost 40 years ago "Mastitis is similar to the Poor, we shall always have it with us" (2) and, only three years after that, "... even with the additional help of many new antibiotics, it would appear that mastitis is even more with us" (3). The few figures on mastitis incidence available at that time did indicate that this pessimistic view was well founded.

Twenty years ago the average UK dairy herd contained 29 cows and had about 40 cases of mastitis a year. Today the average herd has 65 cows (4) and about 30 mastitis cases a year, in other words herds have doubled in size but the total number of cases has actually gone down. Over the same years average yields have improved from 3,700 litres to 5,000 litres, showing that increasing yields does not necessarily increase the incidence of mastitis. However, mastitis continues to cause substantial financial losses. On a UK basis it is estimated that the loss is approximately £90 million at 1988 prices although, because of restrictions on production, it is unlikely that the whole of this amount could be gained even if mastitis was controlled totally.

The year 1968 is an appropriate time to take for comparative purposes. More figures on mastitis levels were becoming available at that time and, most importantly of all, it was in the middle of "MFE 3" - code for the third Mastitis Field Experiment of the team led by Dr Frank Dodd at the National Institute for Research in Dairying in collaboration with C D Wilson of the Ministry's Central Veterinary Laboratory. It was this series of field experiments which proved the effectiveness of the mastitis control measures subsequently adopted in the UK and in many other countries around the world. Four years later in 1972 another major step forward was the formation of the National Mastitis Awareness Campaign when the six main organisations involved in this field - ADAS, MMB, BVA, NFU, ATB and ABPI -

agreed a joint approach to the control of mastitis (5). This was basically what we know as the "five-point plan", namely 1. annual milking machine test, 2. teat disinfection after milking, 3. dry cow treatment, 4. correct treatment and recording clinical cases, and 5. culling chronic cases.

Progress

Clinical mastitis

Excellent progress has been made in reducing clinical mastitis which is now less than one-third the level it was 20 years ago. At that time the annual incidence was approximately 135 cases per 100 cows according to MFE 3 and other surveys at the time (6, 7). Nine years later the 500 herd national mastitis survey in 1977 found the incidence had been almost halved to 74 cases (8). Today surveys indicate that the average has been reduced still further to approximately 40 cases/100 cows (9, 10, 11). Averages however can be misleading. Some herds now have as few as 5 cases/100 cows whilst there are others with over 100 cases (11).

Subclinical mastitis

Good progress has also been made in controlling the hidden subclinical mastitis although there remains considerable scope for further improvement. Twenty years ago the MFE 3 research workers found 55% of the cows infected in the commercial herds they were working with (12). The national mastitis survey nine years later found 32% of cows infected (8). This type of comprehensive examination is expensive and time-consuming, so unfortunately there are no recent figures. Mastitis cell count figures however indicate a further reduction in infection since 1977, so that it is probably fair to say that subclinical infection has been reduced by half over the last 20 years and that the current level may be estimated at less than 25% of cows infected.

Mastitis cell counts

Comprehensive figures are available on herd milk cell counts over most of the past 20 years. All indicate substantial progress in controlling mastitis. The 30 herds in MFE 3 had an average cell count of 730 thousand cells/ml at the start whilst 100 untreated herds averaged 790 thousand cells/ml (12). Ten years later the average for all herds in England and Wales was around 500 thousand cells/ml (13), with similar figures in Scotland and Northern Ireland. Today the average stands at 360 thousand cells/ml, half the level found 20 years ago.

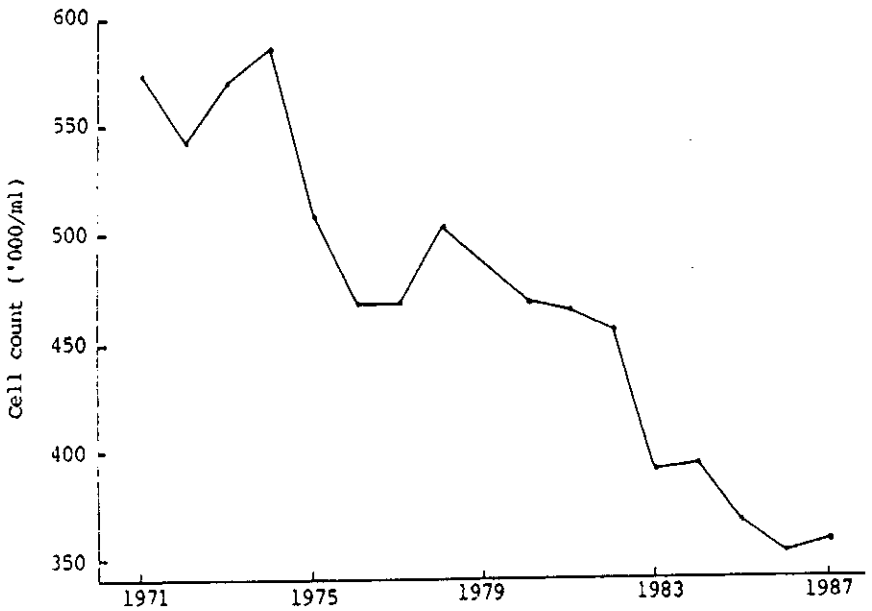

Figure 1: Annual mastitis cell counts in England and Wales

As Figure 1 shows, the major reductions have occurred in two periods. These were in 1975/76, following increased culling of the national herd, and in 1983 after the introduction of payments based on total bacterial counts (TBCs) (13). After both events the lower cell count levels were maintained despite some year-to-year variation.

Summer mastitis

The incidence of summer mastitis is notoriously variable from year to year so that it is difficult to quantify changes. The earliest Milk Marketing Board surveys in 1955 and 1956 found 1.2% and 1.8% of dry cows and in-calf heifers affected in the respective years during the July to October high risk period (14). Ten years ago 2.3% were affected, five years ago the figure was 1.5% (15) and last year it was 2.1% (16). The incidence of affected herds has varied between 35% and 59% with no discernible trend. One can only conclude that there has been no improvement in the incidence of summer mastitis over the past ten years and probably not even over more than 30 years.

The international scene

Good as the progress in controlling mastitis in the UK has undoubtedly been, it is salutary to observe that many other countries have also been making progress. It is neither possible nor appropriate to review progress in this short paper, but a brief comment is possible.

Twenty-three countries replied to the 1983 survey of progress in mastitis control carried out by the International Dairy Federation (17). Six countries in addition to the UK reported a definite improvement: Australia, Belgium, Canada, Denmark, Ireland and Israel, most of them having good evidence to support their claim. Nine other countries reported some improvement: Austria, Czechoslovakia, Finland, Netherlands, Norway, New Zealand, Poland, Sweden and Switzerland. A further five countries reported slight improvement: Germany, Hungary, Japan, South Africa and Spain, although only Germany had any evidence. France reported no change and the United States had no basis for conclusions, although there is recent evidence of a considerable improvement in cell counts - due in no small measure to payment schemes linked to cell count levels which are, for example, operated by half the milk plants in New York state. Bonuses of upto 4% of the milk price can be gained in some states if the cell count is 100 thousand cells/ml or less and the milk meets other quality criteria (1). Most of those countries which reported national average cell counts in 1982 had lower levels than the UK, although it was not always clear how representative were the herds used for this calculation. Ten of the 23 countries in this survey adjusted the milk price according to the cell count. This survey by the International Dairy Federation is to be repeated shortly.

Problems

Adoption of control measures

Achieving widespread adoption of the proven mastitis control measures was the initial problem over the last 20 years. The unity shown by the various organisations in the National Mastitis Awareness Campaign helped a great deal by putting across a single message to farmers and herdsmen. There are still herds where the basic control measures are not practised, but even five years ago more than two-thirds of all dairy farmers were practising teat disinfection and dry cow treatment and having a regular milking machine test (18). Periods of financial pressure, for example in 1973/75 and more recently the imposition of milk quotas, have naturally prompted farmers to

re-examine the continuing relevance of their control measures, but most accept that they are a worthwhile insurance against the losses due to mastitis.

There have been few problems with the basic recommendations for controlling mastitis enshrined in the five-point plan. Research on the design and operation of the milking machine continues and we shall be hearing of this later. Many of the larger herds have progressed from teat dipping to teat spraying (19). An early problem with ineffective teat disinfectant solutions was soon overcome. There is some evidence that more farmers and herdsmen use a full course of antibiotic when treating clinical cases than they did 20 years ago. In general veterinary surgeons are consulted more often about the routine treatment of mastitis although it is probably still not often enough in many herds.

There is now a greater awareness of the need to keep the cow's environment as dry as possible. Cubicles are no longer being designed with a lip which retained both bedding and moisture. Passageways are usually scraped regularly, often more than once a day, and cubicle beds are kept dry and comfortable for the cows.

Environmental mastitis

A problem of more relevance to those practising the control measures has undoubtedly been the emergence of so-called "environmental mastitis". This may be defined as mastitis due to Strep uberis and the coliforms, especially E coli.

It was recognised during the course of the mastitis field experiments that the control measures gave little protection against mastitis due to these bacteria. At that time Staph aureus and the streptococci were the predominant organisms causing both clinical and subclinical mastitis. Twenty years ago the coliforms were the cause of only seven cases of clinical mastitis out of the annual average of 135 per 100 cows (6). Fifteen years later they still caused only seven cases, but this was out of an annual average reduced to 41 cases per 100 cows (9), so the percentage of cases due to coliforms had trebled from 5% to 17%. Clinical cases due to Strep uberis had declined from 24 to 7 cases per 100 cows but as a percentage of all cases they remained constant at 17%. Cases due to Staph aureus fell from 51 to 6, whilst cases due to Strep dysgalactiae and Strep agalactiae were also reduced markedly.

The prevalence of subclinical mastitis showed a similar pattern. As a percentage of quarters infected, Staph aureus, Strep dysgalactiae and Strep uberis had all been reduced by more than half when the 1977 national survey figures (8) were compared with figures for ten years before (6). Strep agalactiae was reduced by less than a quarter. The prevalence of coliforms and other major pathogens however remained the same and, because the overall percentage of infected quarters had been halved, their proportion doubled. Nevertheless they still represented only 5% of all infected quarters.

It is clearly important that research should continue into the causes and control of environmental mastitis and we will no doubt hear more on this subject in the following papers.

Severity of mastitis

Some farmers and veterinary surgeons have suggested that cases of clinical mastitis tend nowadays to be more severe, especially in those herds with low cell counts. In 1980 62% of clinical cases in a national surveillance

scheme were recorded as mild (9). Seven years later we found a very similar figure of 65% of cases with clots only (11). In our survey 4% of cows affected were "sick" as defined by the farmer or herdsman; this is remarkably similar to the 5% of clinical cases with systemic signs found in the third mastitis field experiment 20 years earlier (7). One must conclude that the data do not confirm the suggestion that clinical cases of mastitis have become more severe. In passing it is worth noting that, in our recent survey which was mainly in herds with low cell counts, there was no correlation between the cell count and the incidence of clinical mastitis, ie a low cell count did not predispose to a high incidence of clinical mastitis (11).

Antibiotic resistance and residues

Concern has been expressed that the prolonged use of dry cow treatment could lead to more antibiotic resistance. That there are today no fewer than 22 dry cow and 35 lactating cow intramammary preparations might lend some credence to this. However, there is no evidence whatsoever for increased resistance. A survey in 1961 before the use of dry cow treatment found that 71% of staphylococcal isolates were resistant to penicillin (20); 25 years later another survey found that this had actually declined slightly to 58% (21). Resistance to other antibiotics was low.

The test for antibiotic residues in milk has become very much stricter over the last 20 years (22). The sensitivity of the test has been increased from 0.05 iu/ml penicillin or equivalent to 0.01 iu/ml now, the frequency has been increased from monthly to at least weekly, and the financial penalties are applied at the first failure and are much more severe. Besides encouraging the responsible use of antibiotics and a much greater awareness of withdrawal periods, these sanctions have encouraged milkers to keep records of treatment, which of course has become a legal requirement recently. Recording helps to highlight the chronic, often incurable, cases of mastitis which are most frequently due to staphylococcal infection. These chronic cows, which are always said to be the best milkers, are a source of infection within the herd, but there is now a much greater readiness to cull them than there was 20 years ago.

Conclusions

Good progress has been made in controlling mastitis in the UK over the last 20 years. The incidence of clinical mastitis in the average herd has been reduced by two-thirds and the prevalence of subclinical mastitis has been halved, as has the bulk milk cell count. The widespread adoption of recommended control measures has ensured that once made these reductions are maintained. However there remain wide variations between herds with some still suffering over 100 cases of clinical mastitis per 100 cows in a year and 25% of herds currently having an annual average cell count over 500 thousand cells/ml, some even as high as three million cells/ml.

The main problem encountered has been an apparent increase in so-called environmental mastitis due to Strep uberis and the coliforms. The evidence is that this is not a true increase but that, because infections due to these bacteria have been less affected than the staphylococci and other streptococci by the control measures, they have therefore become more prominent. Individual herds may of course have suffered a genuine increase in infection, although reports of these are less frequent than they were ten years ago. Summer mastitis remains an intermittent problem in some herds. There is no evidence that there has been an increase in the severity of mastitis, nor that there has been any increase in resistance to antibiotics.

With an estimated annual loss of £90 million, mastitis remains the single most costly disease of the UK dairy herd. Undoubtedly some farmers could reduce their losses by more conscientious application of the recommended control measures. There are others however, probably well represented at this conference, for whom the longer term answer lies in research into the epidemiology of the disease, including the influence of the milking machine, and into immunological approaches and possibly breeding for resistance. Later today we will hear progress reports of the research into some of these areas.

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Herd Problems, Investigation and Advice

by

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Herd mastitis problems investigated by veterinary surgeons in practice, in the Veterinary Investigation Service or from the Central Veterinary Laboratory, Weybridge are associated with the same bacteria which were causing mastitis problems ten years ago. Their relative importance, in herd problems has changed over the years. These changes are mostly due to alterations in husbandry and management. The adoption of teat disinfection and dry cow therapy significantly reduced the incidence of mastitis caused by Streptococcus agalactiae, and reduced the incidence of mastitis caused by Staphylococcus aureus. The widespread adoption of loose housing, allowed environmental bacteria such as Escherichia coli and Streptococcus uberis to increase in importance as causes of mastitis. We now see problems associated with S.aureus, Str.uberis, fewer E.coli mastitis herd problems than five years ago, and a few, but an increasing number of problems in which Str.agalactiae is the predominant mastitis organism.

Almost invariably investigations are requested during an epidemic of clinical cases of mastitis. A high or rising bulk milk cell count alone seldom triggers an investigation.

The Mastitis Surveillance Scheme organised by the Central Veterinary Laboratory, Weybridge and the Veterinary Investigation Service, monitored the incidence of clinical mastitis in a cohort of well managed herds in England and Wales over a three year period ending in March 1983. Nearly 33,000 clinical quarters were investigated during this period and the

incidence of clinical cases in the herds involved was about 45 cases per 100 cows per year with between 25 and 30 per cent of cows being affected at least once. Just over 60 per cent of all clinical cases occurred during the first 120 days of lactation which in the predominantly autumn calving herds in the scheme, coincided with the beginning of the housing season. It was not surprising therefore that in the scheme herds the predominant organism associated with clinical mastitis cases was E.coli, with Str.uberis being the second most common and S.aureus the third. The proportion of cases caused by these three organisms differ only slightly from those recovered from mastitis milk samples submitted to VI Centres during the 18 months ending 30 June 1988. The exception to this is the isolations of Str.agalactiae which have increased steadily from about 3.5 per cent of submissions to VI Centres in 1982 and 1983 to 4 or 4.5 per cent of submissions in 1984, 1985 and 1986 and this organism accounts for 4.6 per cent of submissions in 1987 and 4.9 per cent for the first six months of 1988. Str.agalactiae has been associated with some or the herd problems investigated recently. This apparent increase in the Str.agalactiae incidence is worrying because there is no evidence that the organism itself has changed. It can be very well controlled by the conscientious application of the five point mastitis control plan, it is fully sensitive to penicillin, and the bacteriological cure rate following intramammary treatment is usually very good. The bacterium has a restricted life style, living in infected quarters and on the teats of cows with the infection. It is transferred from infected to uninfected cows at milking times. The implication of these trends in Str.agalactiae mastitis is therefore that the application of mastitis control methods, especially teat disinfection and dry cow therapy are now being employed with less vigour than was the case a few years ago. If this trend continues, we would expect that the incidence of S.aureus mastitis will also increase, as this organism lives in the same sort of environments as Str.agalactiae.

The life styles of the environmental organisms contrast with those of S.aureus and Str.agalactiae. These environmental organisms such as E.coli and Str.uberis can live in soil and in bedding materials and on the cow and in the udder, so therefore can be spread between milkings as well as at milking time.

The level of mastitis in any herd depends upon the rate at which quarters become infected and also on the rate at which quarters become free of infection. Control of mastitis will be achieved when both the new infection rate is reduced and the rate of elimination of infections is increased. Spontaneous recovery (self cure) causes some reduction in infections, and we have so far been unable to exploit this. The majority of infections are cleared by culling, and by antibiotic therapy. Mastitis Surveillance Scheme results show that therapy is unsuccessful in completely eliminating the organism from the quarter in around 40 per cent of cases. Treatment failure is more common for S.aureus than for other bacteria. Prevention of infection, or reducing the rate at which quarters become infected is therefore a very important part of mastitis control. Both experimentally and in the field, it has been shown that the degree to which teats are exposed to bacteria is important in influencing new infection rates. The greater the amount of bacteria presented to the teat end, the greater the chance of bacteria entering the teat, so more exposure leads to more mastitis. Thus the stages in formation of a mastitis case are exposure, entry, and establishment. We are able to influence the first two of these, the third component will be addressed in a later part of this meeting.

Exposure

Reducing exposure to those bacteris which reside in infected quarters and on teat skin and teat sores, is carried out by a number of methods which act most powerfully when all are used together in a mastitis control system. Reducing the number of infected quarters in the herd by culling those cows which are chronically affected is essential. The 60% cure rate

mentioned earlier in this paper, is reduced to less than 20% when therapy of persistent mastitic quarters is attempted. If the herd contains a high proportion of chronically infected cows which excrete infection all the time, infection will be spread at milking time, even under the highest standards of milking routine. If the milking machine is properly cleaned, this will minimise the chances of bacteria being carried from cow to cow on milking units. During mastitis investigations, we often identify imperfect milking machine cleaning, due to insufficient quantities of cleaning fluid, too low cleaning temperatures, and/or excessive cleaning times. Teat disinfection is vital to reduce the bacterial population of teat skin. Incorrect dilution of teat disinfectants, or a failure to clean out teat-dip cups are frequently encountered on problem farms. Post milking teat disinfectant spraying is harder to carry out properly than manual dipping, so faults are more common on farms using this method.

Exposure to infection between milkings is important in relation to the bacteria which are able to survive in the cow's environment. At Weybridge we have shown that Str. uberis can survive for 85 days in bedding material, and E.coli can survive for more than 140 days in bedding. Therefore at least daily replenishment of litter in cubicles or yards, and scraping of passageways and loafing areas is essential to enable cows to remain clean and to reduce the exposure of teats to bacteria. The aim must be for cows to have clean teats at milking times.

There has been much debate about washing and drying teats before milking. Surveys have shown that herds in which teats are washed in clean running water containing disinfectant, and then dried with individual paper towels, have lower levels of mastitis than herds which use other methods of pre-milking teat preparation.

Entry

Teat skin damage, especially near to the teat orifice, is often associated with mastitis problems. These injuries reduce the efficiency of

the orifice and canal as natural barriers to the entry of infection, and scabs on teat skin act as potential reservoirs of infection. Ensuring that the milking machine is maintained in good mechanical order, and handled properly will help to prevent teat damage and prevent the machine implanting infection into teats, by creating droplets of milk which can be impacted onto and through the teat orifice. We expect to encounter teat orifice abnormalities in around 20% of teats especially on cows in early lactation. More numerous, or very severe abnormalities are not always the result of faulty milking machine action alone, but may be due to housing and bedding problems. Using teat disinfectants containing emollients will help to keep teat skin in good condition.

Establishment

Evidence from investigations of herd problems and from research, shows that mastitis infections can become established more easily during the early weeks of the dry period, in the period just before calving, and also during the first six weeks of lactation, than at other times. Dry cow therapy has a part to play in combatting the early dry period susceptibility, but the springer, and the cow in early lactation can at present only be helped by reducing her exposure to pathogenic bacteria.

Current advice on mastitis control can therefore be summarised as

1. Manage cows to obtain clean teats at milking times.
2. Use milking equipment which is clean, working correctly - and handle it correctly.
3. Teat disinfect thoroughly and always.
4. Manage clinical cases correctly.
5. Use dry cow therapy on all cows.
6. Cull realistically.

MASTITIS CONTROL IN A DEVON VETERINARY PRACTICE

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Summary

The approach to a herd mastitis problem is discussed with particular reference to a Mastitis Control Scheme offered to dairy clients within the practice. Examples are used to illustrate the type of problem encountered and how the results can be used for diagnostic purposes as well as for disease monitoring and the setting of individual farm target levels.

Over a 5 year period (November 1983 - May 1988) the incidence of clinical mastitis in the herds participating in the control scheme decreased from 69.1 to 39.1 cases (1/4 treatments) per 100 cows per year and the fraction of the herd affected in a 12 months period fell from 31.1% to 21.5%. During the same period the mean herd milk cell count fell from 376,000/ml to 295,000/ml and usage of intramammary antibiotic tubes fell from 2.5 to 1.4 per cow per year.

Assuming an average cost of a case of mastitis to be £40.00 the farms involved have reduced their losses from mastitis by 30 cases or £1200.00 per 100 cows per year (i.e. a saving of £12.00 per cow per year).

The traditional role of the veterinary surgeon in practice is the "Fire brigade" service to attend the emergency cases and the sick animals. However more recently there has been a change towards a more structured service with routine visits becoming more common. The aim of these visits is to improve the herd performance by minimising production losses. Changes in performance, improvements or otherwise, can be monitored by keeping records of various parameters and analysing the data periodically. In the case of fertility monitoring the parameters and targets are well known e.g. calving index, conception rates etc. However in the case of mastitis the figures are only just becoming recognised.

An integral part of any disease control programme is choice and use of drugs. In our practice, for some years now, approximately 40 herds, including the survey herds, have taken part in a Bulk Tank Monitoring Scheme. Every 3 months, or more frequently if necessary, a bulk tank sample is collected by the farmer in a sterile bottle. Potential udder pathogens ("contagious" pathogens) are selected from the mixed growth and subjected to antibiotic sensitivity testing. These results together with sensitivities of any clinical samples sent in are used to select suitable intramammary preparations for each farm (environmental contaminants of any bulk sample are ignored and a clean sample is requested). Both bulk and clinical sample bacterial identification and sensitivities are then transferred to an individual herd file for future reference.

Each farm is given a 1st and 2nd choice milking cow preparation, together with a suggested Dry Cow preparation (preferably differing in action from 1st and 2nd choice preparations). During periods when summer mastitis is a high risk, the Dry Cow selection may be modified temporarily.

We are very aware of the limitations of this system of selecting suitable intramammary preparations but feel it serves it's purpose by bringing some control into the usage of intramammary antibiotics and also acts as a vital link in keeping us in touch with the mastitis situation on each farm.

It must be remembered that despite mastitis being one of the most economically important diseases affecting the dairy industry today, veterinary surgeons in practice see a very small proportion of the clinical cases of mastitis in the national herd. To suggest how much mastitis is acceptable one needs to know the present incidence and then set suitable targets. Clearly some sort of recording needs to be done. Individual farm usage of intramammary antibiotic tubes can be monitored in practice by checking sales invoices. Tube usage can give some indication of the number of cases treated, by making the assumption that each case receives 3 tubes. (This can be misleading, for example the cow receiving 12 tubes of assorted brands in one quarter in the hope that something will work eventually.)

To discover what the incidence of clinical mastitis was in our practice I set up a Monitoring Scheme in November 1983 involving 21 of our more conscientious herds. All cases of mastitis were recorded on individual cow record cards. Mastitis dates, calving dates, lactation numbers, quarters affected, type of tube used and response achieved were all recorded. The files from each herd are collected every 6 months (November and May) and the previous 12 months records analysed.

MASTITIS MONITORING SCHEME

EXPLANATORY NOTES

Definitions

Case:-	one quarter affected once (1/4 treatment)
Cow-case:-	one cow affected once in one, two, three or four quarters (cow treatment)
Quarter recurrence:-	a case requiring one or more repeat treatments during the 12 month survey period (not necessarily the same infection since bacteriology is only performed on selected cases).

Explanation of data fields

Whole herd

1. % cows affected:- proportion of cows in herd affected in one or more quarters during 12 month survey period
2. Cases per 100 cows:- total number of cases (i.e. Quarter treatments) occurring during 12 month survey period divided by total number of cows in herd x 100
3. Tubes per cow:- total number of tubes used divided by total number of cows in herd

Mastitis cows

4. Tube per case:- total number of tubes used divided by total number of cases - (tube usage is checked against invoices and some discrepancies occur)

5. Average number of cases (1/4) treatments) per cow affected
   On a herd basis both a down calving multi-quartered mastitis problem or a high incidence of repeat treatments would result in this figure being high

6. % 1/4 recurrence:- this figure represents the proportion of cases requiring one or more repeat treatments during the 12 month survey period. (It does not give any indication as to the total number of repeat treatments - this information is included in No. 5, average number of cases per mastitis cow).

7. 1/4 per cow:- this figure would be high in a multi-quartered down calving mastitis problem and low if chronic cases involving repeat treatments were a problem.

RECORD CARDS

There are two types:- 1. Individual cow record card

2. Herd record card

The records are collected and analysed at 6 monthly intervals (November and May giving approximately 6 months housed and 6 months at grass). A league table is then constructed and sent to each farmer together with an individual letter discussing progress or otherwise - problems are highlighted and advice on suitable changes given where possible. When progress is made this must be recognised and credit given. Information from a herd visit or bacteriology is included in the letter where relevant.

1. INDIVIDUAL COW RECORD CARD

Calving Date	Mastitis Date	Quarter LF	RF	LH	RH	Treatment	No. of Tubes Used	Response	Sample	COW NO. 27 LAB. USAGE
12/8/88 (4th)	13/8/88	*	*			Spectr-zole	6	Good		One Cow-case Two Cases
	28/8/88			*		Spectr-zole	2	Poor		One Cow-case
	29/8/88			*		Synulox	3	Good		One Case

Fig 1. Individual cow record card

Each case (1/4 treatment) is represented by a * in the appropriate column for the quarter affected.

Calving dates, lactation numbers, dates of each case, type and number of tube used are also recorded. The response to treatment is recorded subjectively e.g. + = good, o = reasonable and - = poor and a change of tube is likely. If a sample was taken for bacteriology, this can also be recorded in the appropriate column.

The individual record card can be used in two ways:-

(a) Day to day for individual cow information regarding treatment and as an aid to culling. Repeat treatments are very obvious - and chronically infected cows, once identified, can be placed on a cull list.

Generally:-

3 cases in one quarter)
or) in one lactation is sufficient to
5 cases in mixed quarters) put a cow on the cull list

If these chronically infected cows are not picked up during "day to day" use of the records they will be identified during the 6 monthly analysis and appropriate advice given.

b) Retrospective information using calving dates, dates of mastitis etc. for herd analysis.

From the data, calculations can be made on a herd basis as to the lactational age and stage of lactation of cows affected, as well as the quantitative data shown in the league table (% cows affected etc.). Stage of lactation is split into 1) 1st week, 2) end of 1st week to end of 1st month, 3) end of 1st month to end of 1st 3 months, 4) over 3 months calved, 5) dry period. Lactational age = parity number.

Checks can be made for over representation of any of the above categories (i.e. stages of lactation and lactational ages) with respect to numbers of cows affected, number of cases and number of cow cases.

e.g. A high proportion of early lactation mastitis, especially if multi-quartered, tends to suggest an environmental mastitis problem. Whereas a more even spread over lactation with predominantly single quartered infections and perhaps a high recurrence rate would suggest contagious udder pathogens as the source of the problem. Confirmation of the type of infection involved by bacteriology of a representative number of clinical cases is recommended. (This can be done cheaply on "divided" agar plates - often sensitivities of the bacteria are not required unless response to treatment seems poor).

Once the bacteria have been identified, appropriate advice can be given. Control measures being based on epidemiological knowledge of the bacteria involved.

When checking the spread of mastitis cases over lactational ages it is important to take into consideration the total number of cows of that lactational age in the whole herd.
For example if heifers seem over represented, then 5 heifers affected in a herd with 5 milking heifers is more significant than 5 heifers affected in a herd with 15 milking heifers.

By comparing the number of cases and cow-cases at various stages of lactation and lactational ages, an assessment of the proportion of multi-quartered infections can be determined, giving an indication as to the prevalence of environmental mastitis. In early lactation the number of cases and cow-cases is often high, cows being more susceptible to mastitis around the time of calving. For the same reason, if environmental challenge is high the disparity between cases and cow-cases is likely to show in early lactation. In herds where environmental challenge is not a problem, cases and cow cases are often numerically equal at all stages of lactation.

2. HERD RECORD CARD

TOTAL COW-CASES = 30 GILES THE FARM TOTAL CALVINGS = 107

TBC	CC	Rolling Cell Count	Date	Month split into three 10 day blocks			No. of Calvings
				1st ---- 10th	11th ---- 20th	21st ---- End	
			11/87	*	**	**	24
			12/87	**	**		14
			1/88			***	15
			2/88			*	10
			3/88	*	***		13
			4/88	***			4
			5/88			**	1
			6/88				1
			7/88				1
			8/88	**	*	**	-
			9/88	*	*		17
			10/88			*	8

Fig 2. Herd record card

Each cow case (cow treatment) is represented by a * in the appropriate month. Each month is split into three 10 day blocks for ease of recording.

This record can also be used in two ways.

a) Early warning system

It is easy to see if there is a sudden increase in the number of * in any month, and an investigation into why an increase is occurring can be instigated or at least questions asked - increased awareness of day to day incidence. The herd record book can be checked by the farmer as it is filled in and/or by his vet at routine monthly herd health visits. A record of TBC, monthly and rolling cell counts can also be entered on the record card and checked in the same way.

b) Retrospective information

Check for any seasonality. In some herds one can predict the problem months for mastitis. This again tends to increase awareness and may well improve detection and early treatment of clinical cases. Preventative measures where possible, can also be taken during high risk periods. A record of the number of calvings in each month is recorded on the far right of the herd record card. This gives some indication as to the number of more susceptible early lactation cows in the herd during each month. The calving pattern can have a marked effect on the seasonal incidence and in some instances can give rise to a cyclical pattern in the bulk milk cell counts. Housing and turn out dates can also be important in explaining sudden increases or decreases in the number of cases per month.

RESULTS FROM MASTITIS MONITORING SCHEME

Table 1. Survey results showing progress over 5 year period (Nov '83 - May '88)

Figures represent average for all herds during relevant 12 month period.

	NOVEMBER '83 - OCTOBER '84	MAY '87 - APRIL '88
Rolling mean Cell Count (x 1,000)	376	295
% Cows affected	31.1	21.5
Cases per 100 cows	69.1	39.1
Tubes per cow	2.5	1.4
Tubes per case	3.6	3.5
Average number of cases per cow affected	2.1	1.7
% 1/4 recurrence rate	14.2	13.7
1/4 per cow	1.5	1.4

Each farmer receives a copy of the "league table" of results containing the above information. Individual herds are assigned a code number to maintain confidentiality and the figures are listed for each herd. Herd size, yield and mean annual milk cell count are also included for each herd. The cost per 100 cows is listed a) For each herd, b) As a percentage of the average of all the herds cost per 100 cows for that survey period.

The "league table" does not however contain any information relating to the incidence of cases and cow-cases at different stages of lactation or different lactational ages. Too much unnecessary information makes the "league table" cluttered. This information is recorded with all the other data, but is only included in the individual letter to the farmer if it is felt that it will highlight an area of concern. For example, if all the heifers in a herd had mastitis, or if 50% of all cases in a herd occurred in the first week of lactation. (In fact both these examples really occurred - it is difficult to believe, half the quarters treated were in the first 7 days of a potential 305 day lactation! - many were multi-quartered, the farm in question having a considerable environmental mastitis problem.)

Analysis of these records not only reveals the number of cases treated (as one could estimate from sales invoices of intramammary antibiotic tubes) but also the proportion of cows affected, their stages of lactation, lactational age and the proportion of cases needing repeat treatment in a 12 month period. From the results obtained from this type of analysis vital clues can be gained as to the nature and extent of any mastitis problem present within these herds. Equally well, having discovered the range of results one can set target levels for each herd and show that some herds can in fact achieve the optimum targets and have little to worry about. Most herds however have room for improvement and it is best to set targets which are attainable, enthusiasm rapidly wanes if the targets seem out of reach. As the herd performance improves the targets can be gradually reduced.

SUGGESTED TARGET AND INTERFERENCE LEVELS

	TARGET	INTERFERENCE
% cows affected each year	20	25
Cases per 100 cows per year	30	35
Milking cow antibiotic tubes per cow per year	1.2*	1.5
% cases requiring repeat treatment during 12 month period	10	15

* With an incidence of 30 cases per 100 cows per year and assuming an average of say 4 tubes per case, the number of tubes used per 100 cows would be 120 or 1.2 tubes per cow.

TWO HERDS WHERE DEGREE OF ENVIRONMENTAL AND UDDER PATHOGENS ARE IN EXTREME

Usually the results shown in Table 2 are not so clear cut as in the following two herds. Often there is more of a mixture of types of infection making it less easy to show such a convincing trend. However the following results help to highlight the way data can be used diagnostically without visiting the farm. Once alerted to the problem bacteriology and a herd visit are used for confirmation and further investigation.

Table 2. Examples of pattern of udder disease in 2 herds representing an "environmental" and a "contagious" mastitis problem

"Environmental" mastitis problem herd				"Contagious" mastitis problem herd		
Yield 6950 litres						Yield 5800 litres
CC 194,000	*	56.0%	= % Cows affected	= 31.0%	*	CC 407,000
Seasonal-Winter		180.0	= Cases per 100 cows	= 50.0		No seasonality
60% Cow-cases		6.7	= Tubes per cow	= 1.4		Cow-cases spread
Nov-Feb		3.7	= Tubes per case	= 2.9	∇	throughout year
Herd Size = 60	*	3.2	= Av. no. of cases per cow	= 1.6		Herd size = 54
		22.0%	= % 1/4 recurrence rate	= 26.0%	∇	
	*	2.5	= 1/4 per cow	= 1.0	*	

Cases			Cow-cases	
Δ 51	Ø	1st week	+ 17	Δ
20		1st month	10	
9		1st 3 months	9	
22		> 3 months	12	
6		Dry	+ 4	
108	Ø		+ 52	

Cows	Lact	Cases	Cow-cases
** 13	1	32 Δ---Δ	19
-	2	-	-
8	3	28	10
6	4	22	9
4	5	18	10
2	6	6	3
1	7	2	1
34		108	52

* - Over ½ herd affected - More than 3 1/4 treatments on av. in 2.5 quarters
Δ - Multi-quartered
Ø/+ - Early lactation
** - Only 16 heifers in herd

PROBLEM: Environmental especially near to calving
ADVICE: New calving boxes built - relieving pressure on existing boxes
RESULT: Over 50% reduction in all parameters

Cases			Cow-cases	
Δ 2	Ø	1st week	+ 2	Δ
1		1st month	1	
5		1st 3 months	5	
18		> 3 months	18	
1		Dry	1	
27	Ø		+ 27	

** Cows	Lact	Cases	Cows-cases
2	1	3 Δ---Δ	3
5	2	7	7
1	3	4	4
2	4	3	3
1	5	1	1
3	6	4	4
2	7	3	3
1	11	2	2
17		27	27

* - High % cows affected and cases per 100 cows (ref. Targets)
Δ - All single quartered
Ø/+ - Even spread throughout lactation
** - All ages evenly represented
∇ - Tube usage per case low resulting in high recurrence rate (carrier cows)

PROBLEM: Contagious mastitis (carrier cows) - Subclinical + clinical mastitis
ADVICE: Cull chronic cows, machine test increase tubes used per case
RESULT: CC and recurrence have fallen

MY ROUTE TO CONTROLLING MASTITIS

Lawson Northey, Castle Farm, Lifton, Devon

The herd

At Castle Farm we run a herd of 55 Friesians (not Holstein) with 12 heifers calving in October and 7 in February. Calving starts in July and ends February-March.

All heifers are got in-calf to nominated M.M.B. 100% Friesian bulls to ensure as many as possible replacements from heifers, as recommended by ADAS. The remainder of the cows are in-calf mainly to Limousin, Charolais and a few to Belgian Blue bulls. We rear all calves and try to take them through to the finished beast.

This year, because of quota reduction we have started a Charolais suckler herd with a pedigree cow and calf and some Friesian X Charolais heifers calving in October.

The Farm land use

We have 43 acres and an additional 43 acres of grass kept for young stock to graze. Silage is made for the cows and older cattle, while about 15-20 acres of hay is made for calves. During winter the cows have 24 hour access to the silage face and we grab out silage daily into feeders to ensure that young cows cutting teeth have adequate intake of silage since concentrates have been reduced to 900 kg per cow. We also grab out silage for the store cattle. A few big bales are made for use before opening the clamp.

At turn out, which is usually about early/mid April, I never cut the concentrates drastically before the cows have settled into grass and also give access to silage for as long as they will eat it. Then I give them cake, each cow for up to eight weeks after turn out, with enough magnesium and minerals in 4 lbs. It may cost me £300-400, but I know each cow has had its daily requirement of magnesium and this is a lot cheaper than a dead cow.

Milking parlour and regime

Cows are milked through a 3-6 Alfa Laval abreast parlour with a Duovac system, installed in 1979. When the cluster is placed on the udder, milking starts on a short milking phase providing good stimulation from the cow and not putting undue strain on the teats before let down. As soon as the milk flow increases to 0.2 kg/min the pulsation to a wider ratio until milk flow drops to below 0.2 kg/min when it reverts to low ratio again to reduce overmilking damage.

Alfa Laval liners fitted with shields are used and are renewed every 6 months, but I think they ought to be changed every 4 months to ensure they are always in perfect condition. The shield in the liner avoids teat impaction. Instead of the milk shooting right up the teat, the shield deflects the milk to the side of the liner.

During milking we always wear rubber gloves and an apron in the interest of hygiene. All cows are washed with clean warm water from a udder wash unit, foremilk tested, and individual paper towels used to dry them.

As soon as the cluster is removed we teat dip with Iosan Superdip which we have used for many years. It covers the teats well and keeps them in good condition. Dry cows are also put through the parlour to be checked twice daily and teat dipped - even then I have had one summer mastitis this year. This was a second calver with no history of mastitis. It will probably lose the quarter even after intensive therapy.

After each milking the clusters are brushed with a cold water solution of Rinsan detergent/disinfectant before circulation cleaning begins. This is followed by a cold water rinse then 10 gallons of 71°C hot water, plus 10 fluid ounces of Rinsan circulated for 10 mins (NO LONGER or residues will build up) and followed by a cold water rinse. The bulk tank is sprayed with Ciba-Geigy tank cleaner and brushed every day.

The milking machine is tested by the M.M.B. every 6 months. This is money well spent when you consider it is used twice daily 365 days of the year. The test will pick up any faults in the plant, which can soon be rectified. The last test showed that there was not enough vacuum reserve, so a larger electric motor was needed to run the vacuum pump faster. Additionally a rubber joint was leaking. Very importantly the pulsation is checked to see each pulsator is functioning properly. As the vacuum must stay constant we clean the vacuum controller at least fortnightly.

Housing

Before the cows are brought in for the winter, all the cubicles are washed out thoroughly and disinfected down with Centaur. Some say this is a waste of time because the bacteria only build up when the cows are in them. But I maintain it is better to start off clean as opposed to dirty - at least it makes you feel good to see them all lying in clean cubicles.

The cubicles are concrete and have plenty of good barley straw added daily if needed, the back half we lime once a week to keep them drier and, hopefully, keep the bacteria at bay. All passage ways and feed areas are scraped twice a day. If we get a heifer not lying in a cubicle, she gets tied last thing at night and this usually gets her trained quickly.

Cows are kept out of the cubicles immediately after milking, so that the teat orifice has time to close up after milking. An additional benefit is that giving access to silage after eating cake in the parlour provides a more even rumen mixture.

So that is the routine - what are the results?

Table 1. Cell counts and total bacterial counts 1987-88

		Cell Count 000's/ml	T.B.C. 000's/ml	Hygiene Band
1987	May	65	0	A
	June	85	2	A
	July	74	1	A
	August	65	2	A
	September	73	1	A
	October	84	0	A
	November	41	2	A
	December	68	2	A
1988	January	58	2	A
	February	55	0	A
	March	45	1	A
	April	74	2	A
	Average	65	1-25	A

Dealing with mastitis cases

I have recently noticed our cell count rising in June-July to 98,000. This can occur when drying off a lot of cows i.e. milking once a day apparently can cause the cell count to rise.

Cows to be dried off are milked once a day until their yield drops to 2 gallons. Then they are dried off with Cepravin. Sometimes it takes longer to drop to 2 gallons. Because we will have a shorter dry period then we use a tube with a shorter withholding period. A red tag is then placed on the tail to indicate the cow is dry, but she is still seen in the parlour. I will never stop using dry cow antibiotic as the dry period is an ideal time to clean up cows for the next lactation. Mastitis incidence can double by stopping dry cow therapy and teat dipping. No short cuts. If a cow has mastitis we milk it last and milk the offending quarter by hand until it responds to treatment. All cows with mastitis have milk samples taken which are cultured by our local vets at Launceston. They have their own lab and are very much on the ball and ready to give advice anytime. Bacteriological testing of these milk samples is a must; then you know what you are treating. If the cow does not respond to treatment and you have not taken a milk sample, it is too late, you are shooting in the dark. Also you know what bacteria are about in the herd, and if there are ways of combating it.

All cows that have had mastitis and recovered are milked last until the end of that lactation. Milk is not sold from cows that have had treatment until another 24 hrs after the withhold time has elapsed. I drink the milk from the tank everyday and I would not sell something to anyone that I would not consume myself. If we can produce a low cell count band A quality milk which the consumer wants, then we should receive a premium price for such a quality product.

If a cow has mastitis when it is in the cubicles we take it out immediately and put it in an isolation box, and it does not return to the cubicles

until it is fully recovered, the last thing you want is a cow leaking mastitis milk onto cubicles.

We cull mainly because of mastitis, the occasional poor breeder or lame cow. If we need to cull extra cows, the ones that have a history of mastitis are the first to go. This applies even it is a heifer that has only had one calf. We never serve a bulling heifer if it has had mastitis - it is never worth the bother risking if it is going to be a three quarter cow or not.

Fly control

Flies are a big problem, we have tried ear tags with little success since Friesian heifers have had New Forest Eye, the same side as the tag. All cows were tagged one year, but this only killed some of the back flies. The belly flies are the ones to kill and to do this we have used the Knapsack sprayer to spray an insecticide solution under the bellies and udders of in-calf heifers. This seems to have to be repeated nearly every week because flies soon return, particularly in wet weather. This year we have used a pour on insecticide, but I am not impressed with the results.

In Table 2 I have listed our mastitis cases over the last year.

Recently we have had a dry cow with a hard swollen quarter and a high temperature which we culled. We took a sample for bacteriology, but nothing grew. I understand that when a cow has a quarter like this and a high temperature, she produces blood serum which goes to the udder and sometimes gives a negative result on a culture plate. Similarly we have had a couple of lactating cows showing mild signs of mastitis in the foremilk, which were negative when cultured.

With freshly calved cows we have seen, not only this year, but in the past, clots or tissue debris in the milk for up to 10-12 days after calving. We always remove the calf away at birth (if we are there) so that the calf does not suck the cow. I prefer to milk the cow by hand (or machine if at milking time) and then give the calf its milk by bucket or a teat over a plastic bottle. The cow is then milked through the parlour leading to even milking of all quarters. The calf just sucks one or two, and it is a fiddle to milk two or three quarters out and milking all will overmilk the sucked ones. Then the cluster and jar are flushed through with cold water, and if there are any clots these can be seen.

At calving all cows have a bottle of Calcium Borogluconate 20% with magnesium and phosphate.

Table 2. Mastitis cases at Castle Farm

Cow	Date	Quarter	Treatment
Rachael	12.6.87	B.R.	Milimycin + Tetra Delta - Dried off + L.A. Penicillin
Viv	20.6.87	F.R.	Clamoxil + Nafpenzal (E. coli)
Viv	22.6.87	B.L.	Aprazin + Targot
Badger	28.6.87	B.R.	Tetra Delta
Romany	3.8.87	B.L.	Tetra Delta possible debris from last lactation
Dutch	27.8.87	F.L. F.R.	Tetra Delta with Nafpenzal
Rene	24.10.87	B.R.	L.A. Clamoxil + Ampiclox (Strep.)
Rosie (MH)	17.11.87	B.L.	Tetra Delta (Str. uberis)
Judy	30.1.88	B.R.	Oxymedia + Tetra Delta then Ampiclox
Judy	17.2.88	B.L.	Tetra Delta
Rosie III	1.2.88	B.R.	Aprazin + Leo Yellow (goat mastitis)
Badger	15.2.88	F.L. B.R.	Ampiclox (tested widely sensitive)
Jenny	1.3.88	B.R.	Tetra Delta (" " ")
Jenny II (MH)	4.4.88	B.R.	Tetra Delta dried off Cepravin + Embacycline L.A.

F.L. - front left
F.R. - front right
B.R. - back right
B.L. - back left
L.A. - long acting

SESSION 2

Mastitis - consequences for farm incomes and product quality

Mastitis - counting the cost.

Harvey S. Beck, Department of Agricultural Economics and Management, Reading University, and Frank H. Dodd, Newbury, Berks

Mastitis is a complex series of diseases with both clinical and sub-clinical forms that affect the productive capacity of dairy cattle in a number of ways. It has proved difficult to measure the loss in production caused by mastitis and this is one of the main reasons why many of the published estimates of the economic losses caused by the disease are unsatisfactory. The direct costs and losses from clinical mastitis have been measured relatively simply at around £40 per case. This results in a national loss to farmers of about £40 million per year and on top of this the effects of sub-clinical infection and other systemic effects increase the estimate to around £3500 per 100 cow dairy farm or £100-120M in a year for the national herd. These estimates are not helpful to individual farmers who will not have an average herd with average levels of either sub-clinical or clinical mastitis. We have made an analysis of costs which gives a systematic framework in which the costs of different incidences of mastitis can be estimated under different economic circumstances.

The estimates given below are based on existing or datum conditions. This approach means that rather than estimating what would be the benefit if mastitis had never existed an estimate is made of the economic effects of current levels of clinical and sub-clinical mastitis. Establishing the current datum conditions will involve errors but this problem has been overcome by measuring the effects of a range of mastitis incidence levels and a range of effects of mastitis on milk yield.

In examining the effect of mastitis on farm cost three conditions have been considered:

1. Past EEC policy or where extra output is not penalised. (Extra output).
This is taken as the situation where all output, including any additional output, is sold at the same price per litre. It can be thought of as the the position milk producers were in under the old EEC policy when effectively they had a guaranteed price on all production. The costs of mastitis are similarly calculated if the farm considered is well below quota.

2. Quota regime, management adjustment through variable inputs. (Reduced costs).
Where a strict quota is applied any additional output is valueless. Under these circumstances a farm producing the full quota can only benefit by reducing inputs. By reducing cow numbers there is a general reduction in variable inputs, e.g. less concentrates, less forage, less veterinary services are used.

3. Quota regime, adjustment through a reduction in concentrates. (Reduced concs)
Another means of staying within quota given the potentially greater yield of non-mastitic cows is for a single input to be reduced. As concentrates are the most expensive feed input, and can readily be adjusted, this is the usual input reduced.

Measurement of the costs of mastitis.

The potential annual cost of mastitis to the farmer is measured as the annual benefit that could be obtained from moving from the position where mastitis is at datum levels to a position of no mastitis present. As indicated above there are several options in moving between these positions and the economic benefit, and its corollary potential cost, will vary with the method adopted. The means may be a matter of choice or necessity. Some farmers may prefer to maintain higher yields on fewer cows, however, where concentrate feed is already at a low level this may be the only option to obtain benefits. These different approaches are applied to a model farm. The model farm is assumed to have a milking herd of 100 cows and to be producing milk at a cost/litre of 13.8p (including fixed and variable costs), this cost is based on Farm Business Survey costings for 1986/7. Quota is 520,000 litres and current production/cow 5,200 litres.

Measuring benefit with an expansion of output.(Extra output).

The economic benefit is measured as the sum of the value of the additional milk produced, the savings in the cost of tubes for treatment of clinical cases, the saving of associated veterinary expenses, the saving of fatalities, and finally a saving in herd depreciation.

Value of milk = Milk loss caused by mastitis x milk price (15.9p)
Saving in cost of tubes = No. of clinicals x tubes/case (5) x cost/tube (90p)
Saving in cost of veterinary input = No. of veterinary visits x cost of veterinary visit (£20)
Saving in loss of cows = No. cows dead x value of cow (£500)
Saving in herd depreciation = Datum herd depreciation - Mastitis free herd depreciation

It could be argued that against these savings should be set any additional cost of concentrates to produce extra milk. However the relationship between the presence of mastitis and feed efficiency is not established, and it is possible that additional milk is being produced from energy that was initially used in fighting the mastitis infection. Alternatively the extra milk could have a similar energy requirement to other litres of production. In the absence of any information the former position is taken in this paper.

Reduction in concentrates. (Reduced concs).

The economic benefit in this case consist of the value of concentrates saved, plus the savings in the cost of tubes for the treatment of clinical cases, the saving of associated veterinary expenses, the saving of fatalities, and a saving in herd depreciation. All except the first saving are calculated as in the 'extra output' method. The value of concentrates saved is calculated using the assumption that the response to a reduction of 1kg of concentrates is a loss of 1 litre of milk, but if 1kg of concentrates is removed then an additional 0.5 kg DM of grass or silage will be consumed.

concentrates saved = milk loss x 1 (kg conc/litre) x cost of concentrates (14p/kg)
additional forage cost = milk loss x 0.5 (kg forage DM/litre) x cost of forage (6p/kg DM, average value of grass and silage)

Reduction in cow numbers. (Reduced costs).

The economic benefit in this case consists of the value of variable inputs saved. It is calculated on the basis that if controlling mastitis potentially allows milk yields to increase by a factor k (e.g. if k=0.24 then there is a 24% increase in yield) then this is compensated for by a reduction in cow numbers to the proportion 1/(1+k) of original cow numbers. The value of k is found from the combined values of milk produced but rejected from clinical mastitis, and the loss in actual production associated with clinical and sub-clinical mastitis expressed as a percentage of datum output. Specific savings including the cost of tubes for the treatment of clinical cases, the saving of associated veterinary expenses, the saving of fatalities, and a saving in herd depreciation are calculated as above. An additional deduction is made for the loss in calf revenue associated keeping fewer cows. The loss in calf revenue = (1-1/1+k) x No. of cows x survival rate of calves (.95) x calf price (£150).

Benefit = total variable costs - (total variable costs- specific savings)/(1+k)- loss in calf revenue

The benefits depend on the proportion of costs that are reduced. Two cases are considered. In the short term adjustable costs can be approximated as the variable costs used in Gross Margin analysis but in the long run all costs can be considered variable. In the short run the additional profit from any alternative use of released land is included in the benefit estimated and in the model this has been approximated by assuming an enterprise with a £500/ha Gross Margin.

Technical aspects of mastitis

a) Prevalence of mastitis.

Two estimates of the prevalence of mastitis are required, one to measure the occurrence of clinical mastitis which required intramammary therapy and another to measure the level of sub-clinical infection. The best available national data are these obtained by the Central Veterinary Laboratory, Weybridge. In both measures there is an enormous variation between herds and the correlation between them in individual farms is slight. Herds can have a high level of infection (e.g. more than 30% of quarters infected) and yet have little clinical mastitis (e.g. less than 20 clinical cases/100 cows/year). For the model herd it is assumed that 25% of quarters will be infected at some time in the year which would give an average of 15% of quarters infected at a herd test carried out at a specific time in the year. The assumption on clinical mastitis is 50 clinical cases per 100 cows/year. The infections that occur in the dry cow are included in the levels of infection and clinical mastitis given above.

b) Effects on milk yield and composition.

The milk from cows that have clinical mastitis and are receiving antibiotic therapy cannot be added to the milk sold from the farm. In addition it is generally accepted that the yield and composition of milk of sub-clinically infected cows are reduced, though the measurement of these reductions is difficult. There is good data that in infected cows the average yield reduction of an infected quarter is around 30% whilst the yield of the uninfected quarters shows a compensatory increase. One experiment indicates that this compensation provides a complete recovery in total cow yield. However, most workers who have attempted to measure the reduction in the lactation yield of infected cows have found an average decrease of around 10%. Our calculations have been made on a quarter basis accepting some compensation. They assume a depression in the yield of an infected quarter of 20% after allowing for compensation and that on average an infected cow has 1.5 infected quarters.

The main effect on milk composition is to reduce the lactose content of the infected quarter by an average of 0.2%, the protein percentage is unaffected and some workers have measured a reduction in fat content of the same order as that of lactose. These changes will give a relatively small effect on the calculation of losses and have not been included.

c) Cows culled for mastitis

Various wastage surveys have found that farmers attribute 10-15% of cows culled to mastitis but this is an underestimate since some of the 20% of cows culled for low yields will have yield depressions because of mastitis. We have assumed that on average 20% of cows are culled for mastitis directly or indirectly.

With the advent of antibiotics cattle deaths due to mastitis are infrequent and for a herd of 100 cows a mastitis death will occur, on average, only once every four years.

d) Cost of treatments of clinical quarters.

The immediate cost of clinical mastitis are the purchase price of antibiotics, the number of tubes infused, the days of production that milk has to be rejected due to antibiotic contamination and occasional veterinary visits to acutely infected cows. Our calculations indicate the immediate cost for routine treatment without a veterinary visit is around £18 per clinical case (e.g. 85 litres @ 15.9p/l + 5 tubes @ 90p) and a veterinary visit could be expected to increase this by a further £20. If rejected milk has an economic value say as calf feed then this immediate cost is reduced to around £11.

Results

The results of our analysis are shown for the main scenarios in the tables below. Several graphs also illustrate the sensitivity of estimates to some of the key assumptions. In the graphs the short term case is taken as representative of the benefits from reduced cow numbers. The figures in the tables are for the model herd described above and can be put on a per cow basis by dividing by 100. The measurements are of potential annual costs and it is unlikely that such a complete elimination of mastitis could actually occur. However, the tables can be used to estimate the costs that could be saved in moving from a higher incidence of mastitis to a lower incidence. For instance, consider the case of a farm where current incidence of clinical mastitis is 70 cases/year, and the incidence of sub-clinical mastitis is 40% of quarters per year, if this incidence can be reduced to 40 cases of clinical mastitis, and a sub-clinical incidence of 20%, and benefits can be utilised through reducing concentrates, then the annual potential loss has changed from £7080/100 cows to £3780/100 cows, i.e. a benefit would be gained of £33/cow.

Figure 1 illustrates that, when mastitis is reduced, if output can actually be increased and the same price/litre received then benefits are greater than in the other situations. The yield reduction/quarter associated with mastitis is a major determinant of the level of costs. Figure 2 illustrates how annual costs of mastitis change according to the yield reduction experienced. Figures 3 and 4 illustrate the sensitivity of the standard farm, with standard conditions to variation in the incidence of clinical and sub-clinical mastitis respectively. Based on our assumptions the cost of mastitis is highly dependent on the incidence of sub-clinical mastitis. This suggests that the main cost to the farmer is, in most cases, the level of this hidden aspect of the disease. Few farmers will know their herd sub-clinical incidence level, but the herd cell count gives a reasonable indication.

Some of the mastitis costs have not been included in this analysis. None of our calculations include labour costs in treating existing levels of mastitis, the rationale being that small time savings cannot be adequately equated with cost savings. Nevertheless mastitis in its clinical form produces considerable disruption to the steady pace of the modern milking routine. It must also be borne in mind that the calculations assume that inputs and resources can be divided into small units. In practice small fractions of land, for instance, cannot be transferred to other uses.

Discussion

In making these assessments of the annual costs of mastitis to farmers various assumptions were necessary. The figures chosen for the model farm for levels of clinical mastitis are less than half those common 20 years ago and the assumption on yield loss from infection is modest. Nevertheless the analysis shows that mastitis is still an important disease increasing the cost of an average 100 cow dairy herd by £3-5000/year. The introduction of quotas has lowered the potential financial benefit that can be obtained by reducing mastitis. It appears that the benefit is greater if farmers react to the higher yields by reducing concentrates rather than by cutting cow numbers and reducing other costs. This conclusion, is however, sensitive to the assumed milk response per kg of feed, and so this should not be taken as prescriptive.

It is important to stress that the estimates are approximate and that changes in the assumptions for the model herd would result in considerable differences in the estimated losses (see Figs 2, 3 and 4). If the assumption made on yield loss is too low then the real cost of mastitis could be double the estimate. Perhaps more important is the effect of between herd variation in levels of clinical and sub-clinical mastitis. Commercial herds cover the whole range in levels illustrated in Figs 3 and 4. Furthermore there is good evidence that by adopting the simple economic control methods now recommended that most farmers could be at the low levels of infection that are illustrated. By doing so they would make substantial cost savings.

Table 1. Annual value of mastitis losses of 100 cow herd, assuming a yield loss/quarter of 20% and adjustment through extra output.

Incidence of clinical mastitis (cases/year)	Incidence of subclinical mastitis, % quarters/year									
	5	10	15	20	25	30	35	40	45	50
10	1550	2580	3620	4650	5680	6720	7750	8780	9820	10850
20	1740	2770	3800	4840	5870	6910	7940	8970	10010	11040
30	1930	2960	3990	5030	6060	7090	8130	9160	10200	11230
40	2120	3150	4180	5220	6250	7280	8320	9350	10380	11420
50	2310	3340	4370	5410	6440	7470	8510	9540	10570	11610
60	2500	3530	4560	5600	6630	7660	8700	9730	10760	11800
70	2690	3720	4750	5790	6820	7850	8890	9920	10950	11990
80	2870	3910	4940	5980	7010	8040	9080	10110	11140	12180
90	3060	4100	5130	6160	7200	8230	9270	10300	11330	12370
100	3250	4290	5320	6350	7390	8420	9450	10490	11520	12560
110	3440	4480	5510	6540	7580	8610	9640	10680	11710	12740
120	3630	4670	5700	6730	7770	8800	9830	10870	11900	12930
130	3820	4860	5890	6920	7960	8990	10020	11060	12090	13120
140	4010	5050	6080	7110	8150	9180	10210	11250	12280	13310
150	4200	5240	6270	7300	8340	9370	10400	11440	12470	13500

Table 2. Annual value of mastitis losses of 100 cow herd, assuming a yield loss/quarter of 20%, and adjustment through reduction in costs (long term)

Incidence of clinical mastitis (cases/year)	Incidence of subclinical mastitis, % quarters/year									
	5	10	15	20	25	30	35	40	45	50
10	1190	1890	2570	3240	3890	4520	5140	5750	6340	6920
20	1340	2030	2710	3380	4020	4660	5270	5880	6470	7050
30	1480	2180	2850	3510	4160	4790	5400	6010	6590	7170
40	1630	2320	2990	3650	4290	4920	5530	6130	6720	7290
50	1770	2460	3130	3790	4430	5050	5660	6260	6840	7410
60	1920	2600	3270	3920	4560	5180	5790	6380	6970	7530
70	2060	2740	3410	4060	4690	5310	5920	6510	7090	7660
80	2200	2880	3540	4190	4820	5440	6050	6640	7210	7780
90	2340	3020	3680	4330	4960	5570	6170	6760	7330	7900
100	2490	3160	3820	4460	5090	5700	6300	6880	7460	8020
110	2630	3300	3950	4590	5220	5830	6430	7010	7580	8140
120	2770	3440	4090	4730	5350	5960	6550	7130	7700	8260
130	2910	3580	4220	4860	5480	6080	6680	7260	7820	8380
140	3050	3710	4360	4990	5610	6210	6800	7380	7940	8490
150	3190	3850	4490	5120	5740	6340	6930	7500	8060	8610

Table 3. Annual value of mastitis losses of 100 cow herd, assuming a yield loss/quarter of 20%, and adjustment through reduction in costs (short term).

Incidence of clinical mastitis (cases/year)	Incidence of subclinical mastitis, % quarters/year									
	5	10	15	20	25	30	35	40	45	50
10	1100	1720	2330	2920	3500	4060	4610	5150	5680	6200
20	1240	1850	2460	3050	3620	4190	4740	5270	5800	6310
30	1370	1990	2590	3180	3750	4310	4860	5390	5910	6430
40	1510	2120	2720	3300	3870	4430	4980	5510	6030	6540
50	1640	2250	2850	3430	4000	4550	5100	5630	6150	6650
60	1770	2380	2980	3560	4120	4680	5220	5740	6260	6770
70	1910	2510	3100	3680	4250	4800	5330	5860	6380	6880
80	2040	2640	3230	3810	4370	4920	5450	5980	6490	6990
90	2170	2770	3360	3930	4490	5040	5570	6090	6600	7100
100	2300	2900	3490	4060	4610	5160	5690	6210	6720	7220
110	2440	3030	3610	4180	4740	5280	5810	6330	6830	7330
120	2570	3160	3740	4310	4860	5400	5930	6440	6950	7440
130	2700	3290	3870	4430	4980	5520	6040	6560	7060	7550
140	2830	3420	3990	4550	5100	5640	6160	6670	7170	7660
150	2960	3540	4120	4670	5220	5750	6280	6780	7280	7770

Table 4. Annual value of mastitis losses of 100 cow herd, assuming a yield loss/quarter of 20%, and utilisation through concentrates reduction.

Incidence of clinical mastitis (cases/year)	Incidence of subclinical mastitis, % quarters/year									
	5	10	15	20	25	30	35	40	45	50
10	1190	1900	2620	3330	4050	4760	5480	6190	6910	7620
20	1340	2050	2770	3480	4200	4910	5630	6340	7060	7770
30	1480	2200	2910	3630	4340	5060	5770	6490	7200	7920
40	1630	2350	3060	3780	4490	5210	5920	6640	7350	8070
50	1780	2490	3210	3920	4640	5350	6070	6780	7500	8210
60	1930	2640	3360	4070	4790	5500	6220	6930	7650	8360
70	2070	2790	3500	4220	4930	5650	6360	7080	7790	8510
80	2220	2940	3650	4370	5080	5800	6510	7230	7940	8660
90	2370	3080	3800	4510	5230	5940	6660	7370	8090	8800
100	2520	3230	3950	4660	5380	6090	6810	7520	8240	8950
110	2670	3380	4100	4810	5530	6240	6960	7670	8390	9100
120	2810	3530	4240	4960	5670	6390	7100	7820	8530	9250
130	2960	3680	4390	5110	5820	6540	7250	7970	8680	9400
140	3110	3820	4540	5250	5970	6680	7400	8110	8830	9540
150	3260	3970	4690	5400	6120	6830	7550	8260	8980	9690

Figure 1: Reduction in the costs of production in a 100 cow herd with the elimination of mastitis. The estimates are made assuming various management decisions to utilise the potential savings. The initial mastitis level was 25% of quarters infected during the year and 50 clinical cases per year.

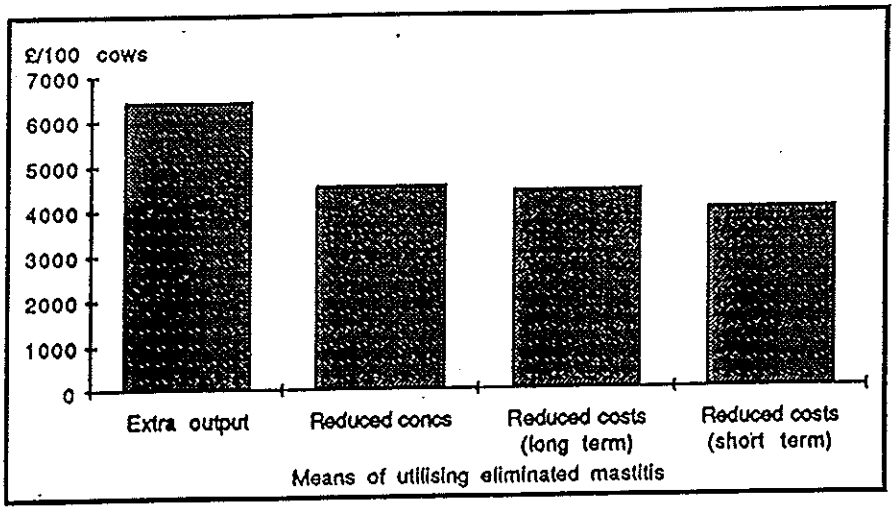


Figure 2. Variation in annual cost of mastitis with different assumptions of the yield loss/quarter. Calculated for a 100 cow herd with a clinical incidence of 50 cases/year, and a subclinical incidence of 25% of quarters/year.

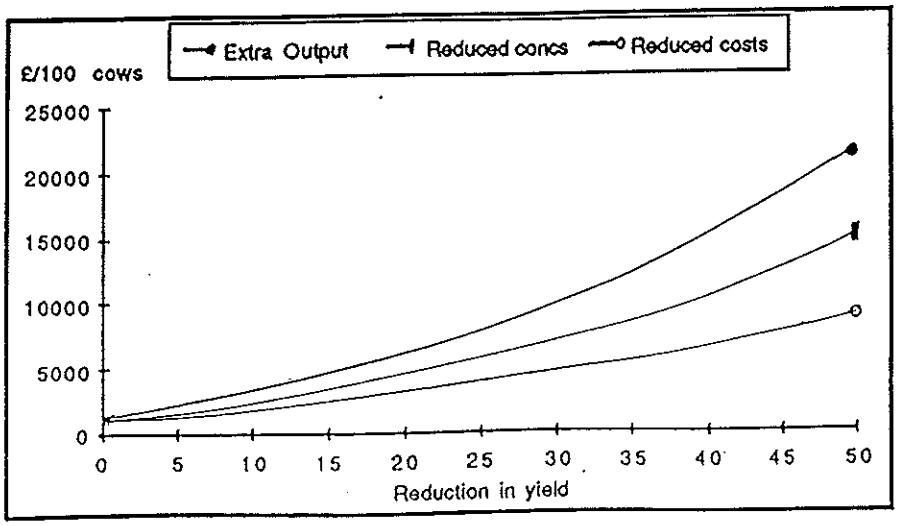


Figure 3. Changes in the annual cost of mastitis with variation in the incidence of sub-clinical mastitis. Calculated for a hundred cow herd with a clinical incidence of 50 cases/year and a yield reduction/quarter of 20%.

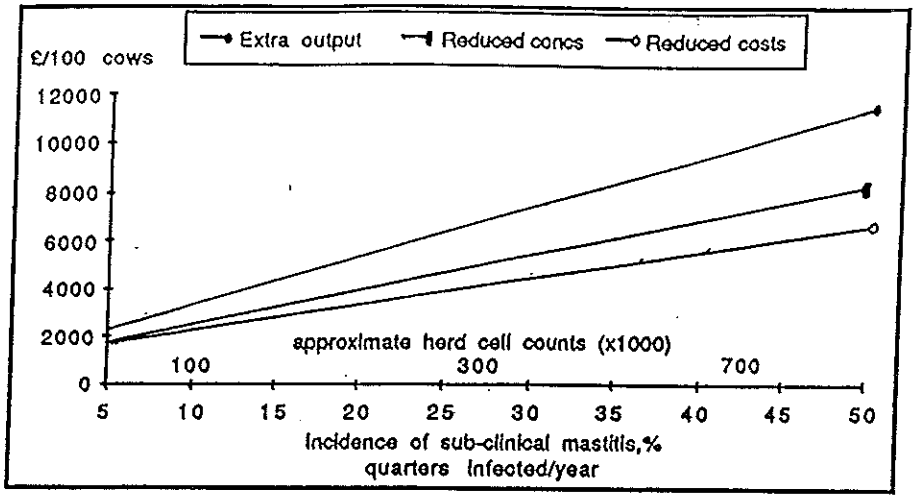
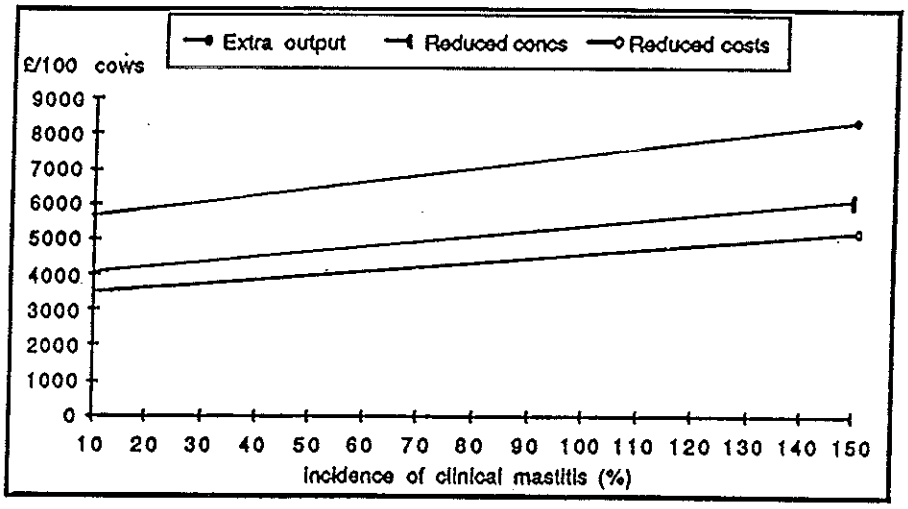


Figure 4. Variation in the annual cost of mastitis with the incidence of clinical mastitis. Calculated for a 100 cow herd, with a sub-clinical incidence of 25% of quarters/year, and a yield reduction /quarter of 20%.



"Mastitis and Milk Quality"

Frank Harding, Technical Director
Milk Marketing Board of England and Wales

Summary

The effect of high somatic cell counts (SCC) milk on the yield and quality of milk are well reported in the scientific literature and are briefly discussed here. Quality Payment Schemes in the UK are explained. These do not include SCCs. At present the reasons being that:

(a) the commercial impact of SCCs in the expected range (200,000 to 600,000) is not clear

(b) it is difficult to devise a simple and equitable quality payment regime on SCCs and

(c) producers are already indirectly penalised through yield, compositional quality and bacterial count if they do not adopt good mastitis control.

The EEC Heat Treated Milk Directive and the requirements for SCCs are discussed.

1. The effect of mastitis on milk quality

The effect of mastitis on both milk quality and yield are well reported in the scientific literature (1) (2) (3).

The conclusions are:

(a) mastitis has numerous effects on the quantitative and qualitative composition and microbial flora of the milk secreted

(b) mastitis may affect the processing properties of milk and influence product quality

Kitchen (4) reviewing published data concluded that fat, protein and especially lactose tend to be depressed.

The level of free fatty acids may be increased in mastitic milk - probably due to higher levels of lipase from somatic cells and blood serum, this increases the potential for "off flavour" development in liquid milk and milk products.

The heat stability of milk is affected by an imbalance of minerals, by protein composition and pH. The heat stability of mastitic milk is lower than that of normal milk. Poor heat stability commercially impairs the quality of condensed milk and the suitability of milk powder for recombination into liquid milk.

There are contradictory reports on the impact of mastitic milk on starter cultures. However, it is suspected that high cell count milk can give starter inhibition. Much of the information on mastitis and milk and product quality is based on data obtained from milk from quarters, individual cows or selected milk samples. There is not a lot of data related to bulked milk where changes are less pronounced. The strongest scientific evidence relates to cheese yield and quality where it is reported that counts in excess of 400,000 cells/ml affect renneting and clotting time, firmness, rate of acid development and cheese yield.

Total whey protein increases in mastitic milk at the expense of the caseins - this gives rise to a decrease in cheese yield since smaller molecular weight whey proteins are not captured in the cheese but are lost to the whey. This change in protein type, however, may not be reflected in producer payments based on total protein content of milk.

2. Microbial quality

Numerous bacteria cause infections in the mammary gland. Mastitic milk has very much higher levels of bacteria than "normal" milk and some of these are pathogenic to man. The most important pathogenic organism is Staphylococcus aureus which not only causes mastitis in cattle but food poisoning and infections in man, and can produce enterotoxins in products such as cheese. Levels of pathogens such as Streptococcus agalactiae Enterobacteriaceae and Listeria may also be elevated in some mastitic milks. From a human health point of view this is only significant where milk is consumed raw since pasteurisation kills pathogenic bacteria.

Looking at a farmer's income, an outbreak of mastitis can cause a dramatic increase in the TBC of milk since an infected quarter can have a count of over 20 million bacteria/ml. Mastitis can therefore dramatically increase the herd TBC and is often the cause of a sudden unexpected TBC increase with the resultant financial penalty.

3. In view of the effect of mastitis on milk quality why is it not a quality payment parameter in the UK?

Quality Payment Schemes have two objectives:

The first is to pay farmers differentially according to the market value of their milk.

The second is to influence improvements in quality by means of financial pressure - be it bonuses or penalties. The cost of the raw material, milk, is the largest single cost affecting the value of dairy products. Those factors affecting the yield or quality of products must therefore be controlled.

Taking the simple case of butterfat, Table 1 shows the effect the butterfat content of raw milk has on butter yields.

Table 1 The Effect of Compositional Quality on Product Yield

Producer A with milk of 3.00% fat requires 30,555 litres of milk to make 1 tonne of butter
Producer B with 3.90% fat requires 23,350 litres of milk (23% less than Producer A to make 1 tonne of butter

The above simple example shows the impact of compositional quality on product yield. Clearly from the farmer's viewpoint Producer B should be paid more for his milk and from the Processors viewpoint there should be an incentive to encourage the production of milk with a higher fat content.

Similar calculations can be made for the impact of fat and proteins on cheese yield, and for protein and lactose on skimmed milk powder yield.

It is against this background that we adopt payment systems for compositional quality .

Table 2 Producer Payment:Compositional Quality

	*pence per %	National Average Value	pence per litre
Butterfat	2.086	3.96	8.26
Protein	2.128	3.27	6.96
Lactose	0.314	4.63	1.45
			16.67

*England & Wales MMB as at 1.9.88

It can similarly be argued that the bacteriological quality of milk offers an indication of the production conditions on the farm and may influence the quality of products, particularly in the case of heat resistant bacteria or enzymes which survive pasteurisation. Again with antibiotics for which we have a penalty scheme, their presence contravenes the Food and Drugs Act.

Quality payment to date in the UK is based on these three parameters: Compositional Quality, Hygienic Quality (TBC), and Antibiotics.

On current evidence it is our view in the UK that a SCC penalty would not justify differential payment to farmers in terms of the market return for that milk and there is doubt that it would effectively lead to milk quality improvements.

In my opinion cell counts are not included as a quality payment parameter in UK because:

(1) The somatic cell count is a useful indication of the quality of milk and the lower the cell count the less chemical and microbiological changes will have occurred. It is difficult, however, to judge the importance of such changes in commercial practice in the range 200,000 to 600,000 cells/ml.

(2) It is difficult to devise a simple equitable quality payment scheme for SCC. Many countries link SCC into a points or classification system with sediment, bacterial counts and other factors. Others set rolling geometric means as the classification parameter but until now few are able to implement a simple scheme which really has an impact.

(3) Lastly producers with a mastitis problem are already penalised, albeit covertly, in terms of existing payment systems; they lose out by low yield, low lactose and high TBC. One may argue therefore that they are already aware of the financial impact upon them of producing mastitic milk.

When Central Testing started we showed an immediate approximately 20% decrease in somatic cell count of market milk which we believe was initially due to producers keeping mastitic milk out of the bulk tank in order to protect their TBCs(5). In many individual cases of high TBC, mastitis is suspected and producers are advised to look at mastitis control measures in order to improve their TBC payment.

Whilst at present we have no immediate plans to introduce SCC into our quality payment schemes the subject is under regular review.

As far as Europe is concerned SCCs play a part in the long awaited Health and Hygiene Directive which lays down the parameters for the Intra Community Trade in heat treated milk.

This directive which comes into force in January next year gives quality requirements for heat treated milk for Intra-Community Trade and for raw milk going into heat treatment establishments. There is no SCC quality criteria for heat treated milk but there are standards for the raw milk used (Table 3).

Table 3 EEC Health and Hygiene Directive

Standards for Raw Milk		
	Step 1	Step 2
Plate Count @ 30°C* (Microbiological)	≤300,000	≤100,000
Cell Count#	≤500,000	≤400,000
Freezing Point	≤ 520°C (539°H)	≤ 520°C (539°H)
Antibiotics		
Penicillin	<0.004µg/ml	<0.004µg/ml
Others	undetectable	undetectable

Untreated milk from each holding

*Averaged over 2 months

#Averaged over 3 months

In the UK we wish to opt for the higher step 2 standard. This means that we will have to demonstrate that we achieve the higher step 2 microbiological standards for our domestic pasteurised and raw milk. As far as SCC is concerned there will be no specific requirement for our domestic market but anyone wishing to export heat treated milk within the Community will have to use raw milk from farms with <500,000 cells/ml if they are exporting to a step 1 status country and <400,000 cells/ml if it is to a step 2 status country.

As we wish to be a step 2 status country anyone exporting to us would have to meet the <400,000 cells/ml standard.

In summary therefore the effect of the EEC requirements on UK SCCs will only affect those intending to export heat treated milk within the Community. We do however have to have an eye on 1992 and beyond when a greater freedom of intracommunity trade may well herald an even greater freedom of milk movement when further harmonisation of milk quality standards across the EEC might follow. The Board will therefore continue to review the quality payment parameters used to improve quality and differentially pay producers.

References:

(1) Effects of Mastitis on Milk Yield, Milk Composition, Processing Properties and Yield of Milk Products Review Article: Munrow et al; The Australian Journal of Dairy Technol March 1984 pp 7-16.

(2) Progressive Changes in individual milk protein concentration associated with high somatic cell counts. Anderson A.T. J. Dy. Res (1977) 44, 223-2335

(3) International Dairy Federation: IDF Bulletin Doc 114 (1979)

(4) Kitchen B J J. Dairy Res 48, 167-188 (1981).

(5) The Impact of Central Testing on Milk Quality. F Harding Dairy Industries International 1987, 52 (1) 17.

(6) Testing for Antibiotic Residues in Milk. J M Booth & F Harding, Veterinary Record (1986) 119, 565-569.

SESSION 3

Research update

REVIEW OF UK AND INTERNATIONAL RESEARCH ON MASTITIS.

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SUMMARY

Work in progress on mastitis prevention in various research centres of the world is considered with respect to the control of bovine mastitis. It is concluded that significant future opportunities exist to build upon current successs by improved management, new generation vaccines and modified milking machines. In the long term, possibilities do exist for increasing disease resisatance by using transgenic technology.

In the UK and many other developed dairying countries schemes for the control of bovine mastitis have been in place for many years. Usually these have been based upon the use of the "5 point plan" which encompasses:

1. Post-milking teat disinfection
2. Antibiotic therapy at drying-off
3. Therapy of clinical mastitis
4. Milking machine testing
5. Culling of chronically infected animals.

Elsewhere in this conference the progress made by applying these techniques is considered as is the changing patterns of disease. Despite considerable progress which amply repays the money invested in research, mastitis remains a significant economic and welfare problem and research continues at several centres. It is impossible in a short paper to consider all these studies so I shall discuss some recent findings and indicate their potential for improved control.

Broadly speaking research efforts fall into 3 main areas: epidemiology, disease resistance and machine milking. The topics of immunology and machine milking are being considered in detail by other speakers so my consideration of them will be brief.

Epidemiology

Epidemiology in this context refers to patterns of disease, its seasonality, sources of infection and the effects of management practices upon them. It has been well-established that mastitis is a multifactorial disease in which the practices employed on farms have a major influence on incidence (1). The study that the Institute for Animal Health (IAH) and the Milk Marketing Board have been engaged in over the last 2 years shows rates of clinical mastitis vary between herds by more than 20 fold (2). This variation occurs despite the fact that these herds applied the recommended measures, albeit with varying reliability. Thus, poorly understood or poorly controlled mechanisms still operate on farms to influence mastitis rates.

Table 1. Between herd variation in rates of clinical mastitis despite the application of control measures.

Herd	No. of cows	No. of cases/100 cows	Days lost milk	Annual mean cell count
271	111	5	41	311
420	140	6	61	133
187	120	9	63	216
698	217	11	42	332
307	106	13	76	194
644	192	15	127	428
-	-	-	-	-
-	-	-	-	-
712	104	50	178	615
563	143	50	358	492
705	135	56	440	228
708	51	57	179	346
648	117	72	495	174
699	56	102	339	169
Average	122	34	195	275

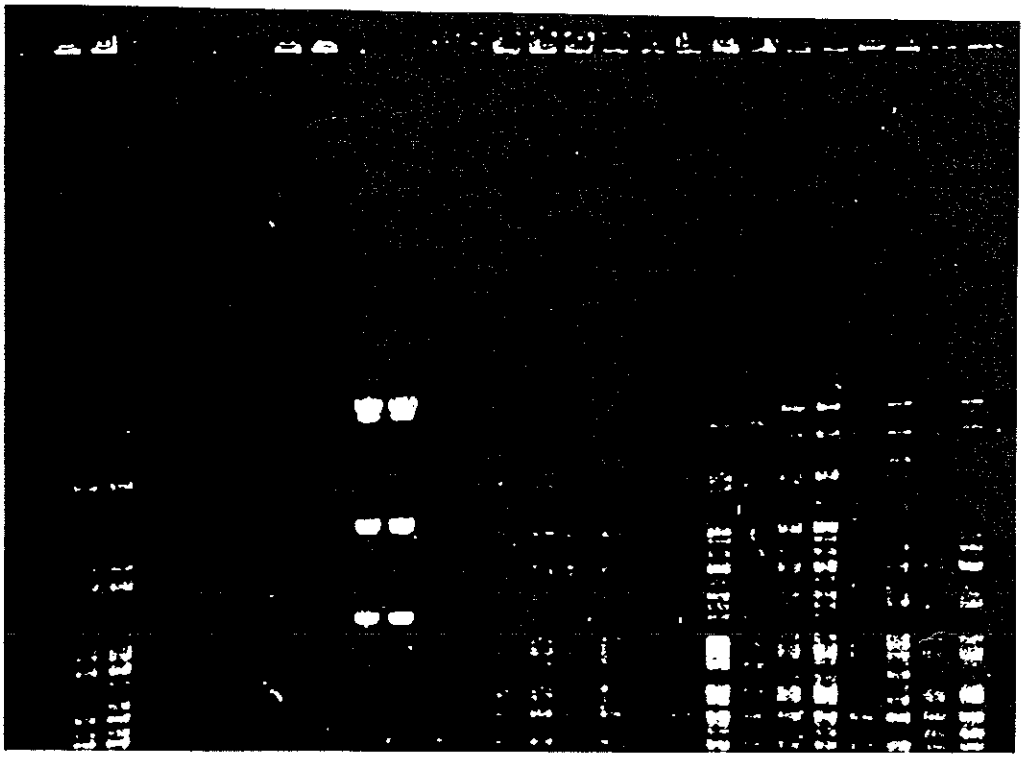
From Ref 2.

Although the patterns of disease will also vary between herds, it is generally the case that high incidence herds show a similar distribution of pathogens to low incidence herds - they simply acquire many more infections.

Prominent among these herds will be infections due to Streptococcus uberis and coliform bacteria. In both cases there are clear data that environmental and herd factors are important. Housing of cows increases infection and data from both the UK and USA show links between types of litter used and the incidence of mastitis. Sawdust and wood shavings present a higher risk factor for coliform mastitis and straw bedding is more likely to be associated with Str. uberis mastitis. These increased risks occur because the bacteria are able to multiply to high numbers in the litter material. It has also been shown that daily replacement of bedding can contribute to lowered rates of coliform mastitis by reducing levels of challenge to the bacteria (3). This is the start of work which will lead to improved housing systems which are cost-effective, provide comfort, clean cows and reduced disease levels.

A limitation to studies of the epidemiology of Str. uberis has been the absence of suitable typing systems to compare isolates. This is important because without such techniques sources of infection or the mechanisms of pathogen transfer cannot be confidently identified. We have recently developed a genetic fingerprinting technique which we hope will greatly facilitate these studies and thereby aid in the identification of critical factors in the infection process (Figure 1).

Figure 1. Restriction endonuclease "fingerprint" of Streptococcus uberis.



We have also been studying the patterns of infection with Str. uberis and these studies highlight the importance of the dry-period in infection. Cessation of dry-period therapy leads to increased rates of new infection with Str. uberis, even among groups of cows free of infection at drying-off. However many of these infections are eliminated by the cow, and it is important to understand the mechanism of this elimination in the hope that it can be stimulated.

Recent data from the USA has indicated that increased control of Str. uberis and coliform infection might be achieved by the use of "pre-milking teat dipping" (4). In experiments done in New York and Louisiana teats of cows were washed and dried before milking after which half the teat were immersed in iodophor or chlorhexidine teat dip. The dipped teats were left for 30 seconds and then wiped with a paper towel prior to attaching the cluster. All teats were teat dipped after milking. Rates of environmental mastitis were reduced by 50% in the pre-dipped teats. More work needs to be done before such a technique can be recommended and careful consideration needs to be given to milk residues.

There is also increased interest in nutritional effects on mastitis incidence. Data from Ohio State University has shown that feed supplementation with vitamin E and selenium can lead to reduced mastitis. There are also correlations with reduced herd cell counts (5). Trials on vitamin E and selenium supplementation are starting shortly at Compton.

Disease resistance

The effects of vitamin E/selenium are related to various activities of the defence mechanisms of the cow. In particular the polymorphonuclear leukocyte (PMN) is a critical feature of the defence of the lactating udder. PMN need to enter the udder rapidly in response to infection and it appears that this process and possibly the bactericidal effectiveness of the PMN is influenced by vitamin E/selenium.

A further technique which has been used to stimulate the speed of PMN entry into the udder is the "intramammary device". This consists of a plastic loop which provides a mild irritation to stimulate PMNs to enter the milk. This is intended to increase protection against infection. Results suggest that rates of coliform infection are reduced but that there are severe reactions in some cows and blood in milk.

Efficient neutrophil killing also requires antibody to the bacteria to be present in milk. Studies in USA, Australia, France and the UK are showing that with some bacteria specific surface antigens which stimulate antibody production may only be elaborated under carefully controlled conditions. It may be that these antigens were not present in most of the vaccines which have been unsuccesful in the past. If these antigens are important in protection then the failure of such vaccines is not surprising.

An exciting new approach to modify resistance to mastitis may involves transgenic technology. The technique is to place genes from one species into the fertilised eggs of another. The consequence of this is that the animals become transgenic i.e. they contain genes from more than one genus. The best known agricultural examples relate to work of the AFRC at the Institute for Animal Physiology and Genetics Research in Edinburgh who have made sheep transgenic for human blood clotting factors. The sheep secrete blood-clotting factor in large quantities in their milk and can act as "biological factories". Work at Compton is starting to use this technology to investigate the possibility of increasing resistance to mastitis by using a bacterial gene which codes for a substance specifically lethal to staphylococci.

Milking machines

Current research on the role of milking machines and mastitis is restricted to a handful of research groups world wide. However it remains likely that the milking machine and the forces it applies to teat tissue are a factor contributing to herd differences in infection rate. We now have a generation of milking machines in which the cross-transfer of milk is effectively prevented by various means (6). This milk may contain pathogenic bacteria and the use of such devices has led to reduced rates of infection.

New research is revealing very much more information about the effects of loads on the biological responses of teat tissues and the consequences for mastitis susceptibility. Increasingly the view is that these local effects may provide much more useful information than more remote tests on milking machine function. Techniques are now becoming available that will assess the effects of different milking conditions on teat end oedema and

muscle tone. This will lead to modifications to machine operating conditions to leave the teat in a similar condition after milking to before milking.

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PROSPECT FOR DEVELOPMENT OF EFFECTIVE MASTITIS VACCINES

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In common with other mucosal surfaces the bovine mammary gland is protected from infection by a combination of specific and non-specific defence mechanisms. Since the normal route of infection of the mammary gland is via the teat, it is not surprising that major non-specific defence mechanisms are associated with the teat canal. Thus teat duct keratin has a mesh like structure which it has been suggested impedes the progress of micro organisms at the external orifice. Keratin also contains a variety of fatty acids and basic proteins which have an anti-microbial effect.

Milk also contains a variety of proteins with non-specific anti-microbial activity. These include lactoferrin, lysozyme, complement factors, cationic proteins and the lactoperoxidase, thiocyanate, hydrogen peroxidase system. A number of phagocytic cells are also present within the mammary gland, Milk from healthy bovine udders may contain up to 10^5 cells of which the predominate cell type from both dry and lactating lands is the macrophage. The precise role of the macrophage in mammary gland defence is not fully understood but it is likely to extend beyond simple phagocytosis and include antigen processing and presentation as well as providing a signal for the recruitment of other phagocytic cells during infection. The protective role of neutrophils in the lactating udder has also been demonstrated, and defects including defective mobilisation, phagocytosis and intracellular killing have been implicated in increased susceptibility to disease.

Immune defence mechanisms are also important for recovery and subsequent protection from re-infections. These acquired processes act synergistically with the non-specific mechanisms outline above. Most work has focused on humoral defence and it has been shown that bovine colostrum may contain in excess of 100 mg/ml of immunoglobulin of which IgG_1 comprises about 85-90%, IgM about 7% and IgA about 5%. the IgG of colostrum is restricted almost entirely to the IgG_1 subclass which is concentrated from serum. During early lactation the immunoglobulin concentration falls rapidly but IgG_1 remains the major immunoglobulin throughout lactation and is almost entirely serum derived. Very small amounts of IgG_2, IgA and IgM are also present in milk but over 80% of milk immunoglobulin remains serum derived. Lymphocytes have been identified in mammary secretions and are comprised of approximately 45% T cells and 20% B cells. There is evidence to suggest that mammary lymphocytes are a distinct sub-population that are functionally independent of the peripheral blood pool.

The relatively low immunoglobulin levels in milk increase markedly during the first few hours after an acute inflammatory response. Both IgM and IgG_1 antibodies are able to activate complement and control the growth of coliform organisms. In contrast IgG2 is poor at activating complement and promotes its bactericidal activity by virtue of its cytophilic property for neutrophils which together have been shown to be highly effective in, for example, the killing of staphylococci. IgA on the other hand is essentially non-inflammatory and it would appear to exert its effect in the gland by preventing adhesion of bacteria to mammary gland epithelia.

Many attempts have been made to increase the level of antibody in mammary secretions, but they have been generally unsuccessful in providing protection against mastitis. While acute forms such as gangrenous staphylococcal mastitis were prevented, chronic mastitis was not. Two types of vaccination regime have been considered. The majority have involved the injection via subcutaneous or intramuscular routes of killed bacteria or toxoids. Unlike other systemic bacterial vaccines used in ruminants (e.g. Bacteroides, Clostridia, Brucella) the protective response has been disappointing, and this has generally been held to be due to lack of antibody of the correct isotype being transeeded into milk. One notable exception to this has been the use of live Staphylococcal vaccines injected subcutaneously, which result in the appearance of IgG_2 antibodies in milk and which provide apparently good protection in sheep. This has led to the development of a cow vaccine composed of killed organisms from wider culture conditions that promote expression of protective antigens, a collection of haemolysins and other staphylococcal toxins with an adjuvant that promotes an IgG_2 response. How successful this approach will be remains to be determined.

An alternative approach is based upon the observations that mammary infection and vaccination which is intramammary or in the region of the supramammary lymph nodes can stimulate the appearance of antibody in milk. A significant proportion of this antibody is locally synthesised and includes antibody of the IgA isotype. In the light of the properties of IgA described above this provides the potential for protection without the complication of inflammation. The development of this physiological approach to the control of mastitis requires both an understanding of basic antigen handling in the gland and of ways of potentiating local responses. In addition the identification of bacterial antigens critical in the pathogenesis of infection is required.

A protective response following mammary infection and disease with Streptococcus uberis has been found from work at IAH. This appears to involve locally synthesised antibody. Although more work is needed to determine the mechanism of protection this lends support to the possibility that successful mastitis vaccines can be developed using molecular biological and immunological techniques.

MACHINE MILKING AND MASTITIS

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Introduction

The last 20 years or so have seen a marked reduction in the incidence of clinical cases of mastitis from 120 to 40 cases per 100 cows each year. Whilst this improvement is the result of progress made in research, development and the application of advances in mastitis control on farms, annual losses due to this disease still exceed £100 million. Speculation into the extent of the milking machine's contribution to current losses varies from 25% to as much as 50%. Whatever the contribution may be, however, it will be considered unimportant in herds with a very low mastitis incidence. Conversely, a 25% contribution in a high-incidence herd is undoubtedly important.

The infrequency of new infection suggests that the teat canal is normally an extremely effective barrier against the microorganisms which cause mastitis. The muscle fibres surrounding the teat canal close it tightly, retaining the milk within the udder and preventing the entry of bacteria. The teat canal is also lined with keratin which aids the physical seal and has antibacterial properties. For an infection to occur the teat must first be exposed to pathogenic organisms and these must then penetrate the streak canal. Although infection is not inevitable following entry of the gland, the probability is high and depends ultimately upon the effectiveness of the defence mechanisms within the gland and on the likelihood of the bacteria being flushed out by milking before they can become established in the udder.

The frequency and degree of exposure to pathogens is of particular importance in mastitis as with other infectious diseases (Schultze & Thompson, 1980). Contamination occurring between milkings inevitably influences the challenge present at milking time and, therefore, factors not directly concerned with the milking machine but which operate at milking time are included in this review.

Sources, transmission and invasion of pathogens

The practices of post-milking teat disinfection and drying off therapy, extensively evaluated in field trials in the early 1960's, remain unchallenged as providing a sound basis for mastitis control. Applied together, consistently, teat disinfection and dry cow therapy have halved the average rates of new infection (Neave et al., 1969). Antibiotic therapy at drying off eliminates existing infection more successfully than treatment at other times and has the added advantage of preventing many new infections in the dry period. The improved cure rate also serves to reduce levels of bacterial exposure to healthy teats during the following lactation. Bacteria contaminating the teat surface at the end of milking

can be almost completely removed by teat disinfectant which will also prevent the colonization of teat lesions and teat ducts.

It is important to appreciate, however, that while teat disinfection reduces new infection by reducing exposure of the teat to bacteria, it has no effect on the transfer of organisms during milking nor between milkings from the teats' contact with soiled bedding. The presence of Escherichia coli and Streptococcus uberis in the cow's gut guarantees a plentiful supply of these bacteria in faeces and it is these pathogens that are generally associated with mastitis outbreaks due to contamination from the environment.

Teat lesions may arise from physical damage, changes in climate, unhygienic housing or poorly operating milking machines. Whatever the cause, they quickly become infected with bacteria particularly Staphylococcus aureus and Streptococcus dysgalactiae which further aggravate the lesion. The act of machine milking exacerbates all types of lesions as well as being the primary cause of hyperkeratosis (orifice eversion) and chaps. The exposure and risk of udder infection is considerably amplified when lesions are at, or near, the teat orifice because they both reduce the integrity of the streak canal and the scabs, associated with lesions, protect the bacteria from the action of chemicals used during teat disinfection. An outbreak of teat lesions within a herd is often accompanied by increased incidence of clinical mastitis and suggested areas for farm investigation would include inadequate pulsation (Mein, Williams & Brown, 1982), excessive vacuum levels, the use of excessively wide bore liners, overmilking, poor hygiene in winter housing systems, abrasive bedding materials and incorrect mix of teat disinfectant. Teat chapping results from a loss of skin elasticity related to low temperatures and drying weather conditions whilst dirty bedding, poor udder washing and flies all increase their severity. Complete coverage of the teat surface with an approved teat disinfectant is beneficial both in the prevention and healing of lesions; if the disinfectant is formulated with an emollient it can be particularly effective.

Machine milking influences intramammary infection in a number of ways such as flushing out invading bacteria from the mammary gland, mechanically spreading infection and altering the integrity of the teat orifice and canal thereby reducing the resistance to bacterial invasion.

There are only two known mechanisms by which the machine assists penetration of the teat duct by pathogens. The first results from inadequate liner collapse (Reitsma et al., 1981) caused through incorrect pulsation or insufficient liner length. This impairs blood circulation in the teat and increases the susceptibility of the teat duct to colonization and invasion by bacteria. However, provided that the pulsation characteristics of the machine conform to standards (British Standards Institution, 1980) and that short liners are not employed to milk cows with long teats, there appears to be little risk of increased disease due to this mechanism particularly if teats are disinfected after milking.

The second mechanism, which has received much attention in recent years, increases bacterial penetration of the teat duct by means of milk droplets, contaminated with mastitis pathogens, being impacted on the teat end. Fluctuations in vacuum beneath the teat are generated by pulsation and intermittent milk flow. These fluctuations can cause milk, contaminated

with mastitis pathogens, to travel back up the short milk tubes as liners open and contaminate teat surfaces. If these reverse pressures are exaggerated by sudden admissions of air into the vacuum system, particularly when liners slip (O'Shea et al., 1975) and when clusters are removed (Griffin et al., 1982), contaminated milk can strike the teat orifice with sufficient force to penetrate the streak canal and so increase infection.

The transmission of mastitis pathogens between the teats and through the teat canal during milking can be minimized by modifying the design of the cluster and the operating conditions of the machine. The mechanical forces assisting bacterial penetration can be reduced by restricting the amplitude of the vacuum fluctuation and speed of vacuum change at the teat end. This can be achieved by supplying adequate vacuum reserve, pipeline and clawbowl capacity, liners stable on the teats without slip, appropriate cluster removal and good milking practice. A practical alternative is to provide a physical barrier to the reverse movement of milk eg shields (Griffin et al., 1980), or non-return valves (Griffin, Grindal & Bramley, 1988), preventing the adverse consequences of vacuum fluctuations.

Although cross contamination can be minimized during milking by modification of the cluster or plant, liners act as a vector by transferring pathogens from cow to cow. A liner contaminated by milking an infected quarter can contaminate the corresponding teat of the next 4 or 5 cows milked. When clusters are inverted, between individual cow milkings, infected milk from the claw can run back into the liners providing a further source of bacterial contamination. This means of transfer between cows can be reduced by flushing clusters with water at 85°C for 5s or backflushing with water or disinfectant but the effect of backflushing on new infection rate is small compared with post-milking teat disinfection.

The speed at which liners deteriorate or 'age' depends largely on their composition. Those having nitrile rubber as a major component are more expensive than natural rubber liners which they have to a large extent superceded. However, they are more resistant to flex cracking, are easier to clean and have a longer life provided they are not overstretched or subjected to rough treatment. Silicon rubber can resist all forms of deterioration over a long period but its high cost limits its use for liners. It is important to replace damaged liners and renew liners on a routine basis (as recommended by the manufacturer). Extended use will lead to deterioration and distortion, resulting in cleaning problems, reduced milking efficiency and perhaps more frequent liner slip at milking time.

Plant cleaning and disinfection after milking should remove milk residues and significantly reduce bacterial contamination. Insufficient water volume, temperature, contact time or chemicals used during circulation cleaning or the use of deteriorated rubberware can result in a build-up of residues and bacteria. Bacteria surviving the cleaning process will multiply between milkings and high numbers situated in the liners may contaminate teats at the following milking.

With this increased knowledge and the means of successfully controlling pathogen transfer during milking, the contamination and transfer that occurs prior to it becomes more important. Pre-milking udder washing, particularly with water containing disinfectant, might be expected to reduce bacterial numbers on the teat skin but its influence on new

infection rate is usually insignificant. Indeed rates of new infection are generally much lower when udder washing is omitted. This probably reflects an unwillingness to dry teats after washing them as inadequate drying increases the number of environmental bacteria reaching the teat end. The use of a common cloth is not recommended as it can encourage the further spread of bacteria.

A survey by the Milk Marketing Board (MMB) showed lowest somatic cell counts among herds using a dry wipe with paper towels or no udder preparation. Herds employing other methods of udder preparation had higher cell counts although this was marginal for herds in which washing was restricted to the 'dirty cows' using hosed water and drying with paper towels. These results emphasise the importance of management techniques and good housing systems in keeping cows clean and by no means advocate the milking of dirty cows.

Recent work in the USA indicates that premilking teat disinfectant, applied to clean teats and followed with drying, reduces rates of environmental mastitis by more than 50% (Pankey, 1988). This degree of environmental mastitis control, achieved by premilking but not postmilking disinfection of teats, implicates the milking machine to a greater extent than previously supposed. Predipping is likely, however, to increase the risk of disinfectant residues contaminating the milk and controlled studies are required to evaluate the significance of this problem.

Emphasis on control

With the emphasis in the dairy industry on ever greater efficiency the udder may become increasingly susceptible to mastitis. The modern dairy cow has been bred to provide high yields under intensive conditions with little supervision and although benefits from modern technology have clearly been greater than the losses due to mastitis, effective control measures have become increasingly important.

Incidence of new infection are known to vary considerably between herds despite teat disinfection ie 15/100 cows each year to 255/100 cows (Dodd & Neave, 1970). It is not known, however, to what extent this variation was due to differences in exposure to infection. An uneven distribution of new infection also often occurs within a herd. Older cows experience increased incidence of hyperkeratosis and teat lesions. These cows are often more susceptible to mastitis, whilst those considered to be the 'best' cows (high yielders with a fast milking rate) frequently experience a higher incidence of mastitis, presumably due to the reduced integrity of wider bore streak canals. If such characteristics are proliferated and compounded by milking and feeding systems, which increase lactation yields, the need for foolproof application of improved mastitis control measures will be not only desirable but imperative. On the other hand some changes may prove beneficial. For example, further automation of the milking machine could lead to more frequent milking and this may be expected to reduce the mastitis risk through more frequent flushing out of the teat canal.

Maintaining correct operation and routine procedures in mastitis control at milking time are important and this should be appreciated by not only management and 'regular milkers' but also by 'relief' staff. Short cuts or brief periods of 'economizing', for example by inefficient washing

and no drying, 'abrupt' cluster removal, omitting teat disinfection, failure to observe and treat cases of clinical mastitis or teat lesions, milking lower yielding cows once daily, and failing to have milking machines regularly tested and maintained or satisfactorily cleaned after milking will undermine previous concientious efforts.

Reassessment of the machine's influence

It is perhaps encouraging to discover, by extrapolation from current new infection rates and the speculative contribution of the milking machine, that the average annual cull rate is higher than the rate of new infections induced by the milking machine. This must not lead to complacency, for whilst the infrequency of infection currently attributed to the milking machine underlines the generally good performance of present-day milking machines, comparatively little is known of the occurrence, degree and duration of tissue changes due to machine milking (Hamann & Mein, 1988). If impaired circulation of blood in the teat reduces the efficiency of the defence mechanisms or causes internal teat lesions, then those infections previously accepted as 'non-machine' induced may need to be re-examined. Physiological changes to teat tissue which have a protracted effect may influence the incidence of udder infection from environmental pathogens arising both between milkings and in the dry period. If this is the case, previous estimates of the extent of the machine's contribution to the total number of new infections may need considerable revision. Future research will explore the association between teat trauma and mastitis incidence, and lead to much closer control of the forces applied by the liner to the teat. Hydraulic milking is one recent development that provides the opportunity to closely manipulate these forces (Grindal, 1988) and minimize the physiological changes incurred during milking.

It is probably inevitable that a teat during its lifetime will experience one or more of the adverse conditions detailed in this paper and this emphasises the need for constant vigilance in the application of control measures.

The most constructive and effective approach to future control will continue to be a coordinated one enveloping hygiene, management, therapy and milking techniques. But knowledge is of no value without its application and, ultimately, success will depend on maintaining effective links between research and farmers. Any compromising of this liaison will cut this supply line and cause the greatest disservice of all to prospects over the next 20 years.

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RESEARCH ON AUTOMATIC MILKING

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1. Introduction

Current milking practices have developed from experience, new knowledge, and technology, but of recent years have been greatly influenced by the social needs of staff and labour. Much effort has therefore been put into fitting the milking operation to human requirements leading to high throughput parlour systems used for two restricted periods within a working day. The 8 hour day 5 day week may be desired but is not possible because of the absolute necessity to milk daily and to be financially viable.

The cows' needs, although recognised, have been suppressed for the benefit of man, but given the means, the freedom from labour restrictions, most dairy farmers would milk more frequently for longer periods of the day. The benefits of doing this are in increased yields and almost certainly in improved health of the herd. With the right system much of the hassle of cow handling and movement may also be eliminated.

Present robotic technology, disregarding cost, will certainly allow automatic cluster application and the building of automatic milking systems. The research efforts[1,2,3,4,5,6], some now at the stage of prototype systems, are being directed to viable commercial systems. Some effort is also going into improving the milking operation to match the physiological requirements of individual cows. Fully automatic, unattended milking is the target and it not only permits, it demands, a reappraisal of the whole dairy farming operation.

2. The Automatic Milking Concept

Analysis of the operational requirements[3] for automatic milking leads to the rejection of any multi-stall parlours. There is no place for batch systems with their attendant cow movement problems and there are no robots

competent enough to deal with the multi stall situation. All current designs[1,2,3,6] have adopted the single stall as a unit. The single stall when fitted with identification equipment, controlled gates, and cow position sensing simplifies many of the robotic operations.

All systems expect the cow to have free access and turn up at the milking unit without prompting. Automatic identification will enable information about each cow to be retrieved from a computer which can then be used to control the rest of the process. The first decision is that the cow must meet certain entry criteria, such as having sufficient milk, before being allowed into the unit. Rejected cows will be diverted away or could be collected in a holding area if required for treatment etc. The milking unit will adjust in size to match the cow and make sure teats are in an acceptable position. Movements of the teat cups or washing equipment to the teats will be controlled using stored data and aided by local sensors for final attachment.

The milking operation will be monitored so that comparisons can be made with the normal characteristics for each cow. The parameters checked would be flow rates, expected yield, conductivity for mastitis detection, and given suitable detectors the health for each cow. At the end of milking the cow is released, new data is recorded, anomalies reported, and the unit is cleaned for the next cow. A computer will be used for general herd and cow management and must also monitor times of visits and perform roll-call functions to report any cows not attending.

3. Technical Problems

Automatic milking imposes enormous problems that are not present in manufacturing industry. Cows are of different shapes and sizes and move. The important features, the teats, are on an elastic floppy bag that changes shape with milk fill and can be obscured by legs. On the positive side, the workpiece will move into position itself, and accuracy of robotic positioning can be relaxed from fractions of a millimetre to perhaps 5 mm.

Robots can be made fast enough to track the teats but require information to control its movements updated at an appropriate rate (20 ms intervals).

Acquiring this information is a difficult operation. Mechanical touch sensing would be very complex and might irritate the cow. Ultrasonics is a good non-contact method but lacks resolution and speed unless very close. Vision techniques are the most promising and although the image can be taken quickly, the subsequent processing is far too slow unless extremely expensive systems are used.

The current automatic milking systems therefore rely on maintaining the teat position information for each cow in a computer and using this information together with cow position data to steer the robot to the teats. When near to the teats additional short range sensors can aid the final attachment. The cow needs some restriction to limit movements but does not need to be clamped in an immovable position.

4. Current Research

The Dutch Vicon company[1] has the task of co-ordinating the Dutch work and already have a working prototype on trial with a small number of cows. Although the milking unit is still a single stall technique, a pair of units are connected in tandem and share a single robot. The teat cup assemblies which also incorporate teat washing equipment are carried on a sub-frame which is moved into position by the robot. The teat cups are attached one at a time, the sub-frame being moved by the robot for each attachment.

Gascoigne's[6] also have a working prototype unit, in this case a teat cup assembly is set up to match the cow's teat position and swings up between the spread rear legs to attach all teat cups together. The teat cups have a funnel shaped liner to assist teat entry.

French research[2] at CEMAGREF also uses a single stall but has an individual robotic arm for each teat cup. Sensing of teat positions is being tried using laser scanning and a vision system, and therefore stored data may not be needed for attachment, although it will be needed for other purposes.

The AFRC work uses a pneumatic robot upon which a fully adjustable teat cup assembly can be mounted. Trials have so far been carried out in placing a

single teat cup and this is successful for about 70% of the attempts. Work is now being done to improve this and to incorporate the remaining teat cups.

There are other groups with an interest in robotic milking with similar ideas but it is not known if prototype systems are being built.

5. Farm Use

Although no prototype units are ready to operate on farms yet, it is possible to outline a few of the obvious points from design features or research results.

Dutch work[7] has shown that cows on a voluntary milking scheme attended an average 5.4 times a day and did not show any particular diurnal pattern. One milking unit assuming 10 minutes per milking allowing for inactive and maintenance periods will allow 100 plus milkings over 24 hours. As the visits to the milking unit will be limited according to yield and time in lactation, then 30 to 40 cows could be accommodated on each unit. Other work[1] has also shown that cows will travel up to 300 metres to visit a milking unit. From these figures it is possible to suggest that under housed conditions 4 or 5 units may be grouped to serve up to 200 cows. For summer grazing, units would be better separated each serving 30 to 40 cows to minimise travelling distance. Self contained transportable units would clearly be most suited to grazed herds but introduce problems of providing services and milk collection. 24 hour operation requires that the units are supplied with sufficient intelligence to deal with normal day-to-day problems, such as mastitis or cows under treatment, and the computers will then inform the manager of actions taken. Severe problems will need human attention and alarms must be built into the system to alert staff.

6. The Cow and Health

Cow behaviour is clearly the key feature to automatic milking. The Dutch work[7] shows that cows will visit voluntarily and individually and there appears to be no herd behaviour. The most likely effect of free access milking is that cows would adjust by virtue of a natural hierarchy to their own visit pattern, taking account of time slots imposed by the system. The

Dutch work also showed that the cows appeared very contented which may be in part due to the elimination of the disturbance from man.

Within automatic milking there is considerable potential for health improvement beyond just better monitoring and treatment. Cleaning and disinfecting will be consistently done and not be subject to human failings. More frequent, shorter periods of milking are closer to nature and lead to less teat damage and mastitis. The automation deals with each animal individually and this individual approach could easily be extended to adapt the milking process to each cow for optimum conditions, a subject already being studied at the AFRC Milking and Mastitis Centre. With no requirement to collect all animals together for communal milking, it would be possible to maintain small groups each with its own milking unit and so reduce disease transmission. More frequent milking may also stimulate the secretory responses which could possibly be exploited by extending the lactation period.

Purely from the mechanical point of view there are a number of cows that would be difficult or impossible to milk with the present system designs, teats being too close or at awkward angles. Automatic milking will clearly prompt the selection of cows for new factors that lead to ease of automatic milking.

7. Monitoring

The automatic process will depend heavily on sensors to monitor the operation. Many sensing techniques are already available so that the essential measurements of yield, flow rates and animal weight will be easily made. The measurement of animal movement and therefore agitation can be achieved with standard sensors. AFRC is already well advanced in the development of a non-intrusive milk conductivity sensor for mastitis which will need no special cleaning. Body temperature, heart rate and respiration rate have been monitored experimentally and it may well be reasonable to incorporate such techniques into the milking unit. The combination of a number of factors, i.e. activity, temperature, yield etc, could when processed point to oestrus. As the technology of biosensors improves it may well become possible to monitor hormone levels in the milk

and detect oestrus very positively. The reduced number of milking positions makes it possible to consider fitting a quality and quantity of sensors that would be prohibitively expensive in traditional parlours.

8. Economics

Parsons[8] commented that the capital costs are higher than for conventional parlours, but the increased yields offset the extra costs to show benefits of £48 to £72/cow year for herds of 60 or more. Quota limitations reduce the benefits to between £1 and £25. More comprehensive monitoring giving improved health and oestrus detection produce benefits which are more difficult to predict but could be of the order of £17/cow year. The savings in labour if costed outweigh the extra costs in all circumstances. However staff are necessary even with full automation and alternative work may not be appropriate.

9. Conclusions

Automatic milking offers considerable opportunity for not only removing some of the onerous tasks in dairy farming but also for improving the milking process, and animal health and welfare. The human limitations of working hours become less important and allow a much wider choice of farming strategies. Animal stresses due to handling and herding and different techniques by different operators will be reduced.

The reduced number of stalls will make the introduction of improved and better sensors for yield, health, and equipment monitoring easier and less costly. The more effective monitoring will lead to better and more relaxed management.

There is also clearly potential for other improvements in yield and health due to interaction between the system and each animal on an individual basis and for further extension of the cows' production.

Economically most medium to large dairy farms would be more profitable when using automatic systems.

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INTRAMAMMARY MASTITIS THERAPY - SOME NEW DEVELOPMENTS

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SUMMARY

The major problems associated with unsuccessful intramammary antibiotic therapy of the lactating cow are identified and discussed. The ways in which new developments are providing solutions to some of these problems are summarised and the possible future developments or strategies discussed.

1) THE PROBLEMS

a) "82% - No more"

Scanning through the clinical efficacy data from the majority of intramammary products available in the UK, almost all quote a clinical cure rate of 80 - 85% . Averaged out, there appears to be an almost magical barrier of 82%. Whilst nobody could argue that this is not a very good cure rate for cases of mastitis, it can surely be bettered.

b) Antibiotic Resistance

This is still the largest comprehended problem in achieving successful intramammary therapy. Note:- this is not the only problem and probably not even the most important problem.

c) Breakdown of clinical cures

The 82% clinical cure rate mentioned above represents that observed up to 7 days following the initiation of intramammary treatment. With all intramammary tubes currently available this cure rate is significantly reduced by 10 to 20% if measured at 14 or 21 days post treatment. Whilst some of these "breakdown" cases can be put down to another infection being contracted, in most cases they represent a re-emergence of the original infection following apparent clinically successful intramammary treatment. A large majority of these breakdowns can be put down to the following points d) and e).

d) Intracellular and "Intra-alveolar" staphylcocci

Staphylococci are readily phagocytosed by inflammatory cells within the udder. Some of the bacteria may remain viable within the cell and can be protected by the cell from antibiotic action. On the death of these cells, viable bacteria may again be released into the milk. In some cases cellular and other debris produced by the action of staphylococci and their toxins on the alveolar wall, may block the milk ductule draining the alveolus.

As a result there may be poor antibiotic penetration to the alveolus and a microscopic reservoir of infection may remain.

e) Incomplete course of treatment

The temptation to a farmer is to cease intramammary therapy as soon as he observes apparently normal milk. With intramammary tubes requiring a 3 tube course, this may mean that treatment is terminated after only one or two tubes. Such an action may severely limit the potential success of the treatment, particularly against Staphylococcal mastitis.

f) Antibiotic residues/milk withdrawal

Both of these cost a farmer money. Whilst milk failure due to antibiotic residues are avoidable, milk withdrawal following intramammary therapy is not. In the perfect intramammary, statutory milk withdrawal would coincide with a return to normal milk quality. Unfortunately this is not always possible at present.

g) Improper technique of administration

There is considerable scientific evidence to prove that full insertion of an intramammary syringe cannula into a teat canal can increase the rate of new infections in that quarter. In other words, this technique might actually cause mastitis rather than cure it. Partial insertion of the cannula (i.e. only just into the entrance of the teat canal) not only reduces the risk of carrying bacteria from the teat canal into the teat cistern but will also allow antibiotic to be placed nearer the initial site of infection (teat canal colonization).

2. THE SOLUTIONS

a) New Antibiotics

The past 10 years have seen what could be termed the "cephalosporin era". Products containing cephalosporins have been launched in the "multi-shot milking cow", "dry cow", and "single -shot" markets. To a certain extent cephalosporins, because of their low levels of resistance and yet broad spectrums of activity, represent the modern answer to multiple antibiotic "shotgun" therapy.

There is no doubt that the cephalosporins have indeed provided an answer to many of the resistance problems encountered in mastitis therapy. Cephalosporins (in particular the 2nd and 3rd generations) possess extremely broad spectrums of activity and as such represent ideal compounds for successful routine mastitis therapy where a specific pathogen has not been identified.

They have not solved the problem of intracellular and intra-alveolar staphylococci however, and it is perhaps only this which prevents them fully breaking the "82% barrier".

Cephalosporins appear to lend themselves to various treatment regimes particularly well. As a result we now see dry cow tubes with 10 weeks activity (e.g. Cepravin); multi-shot lactating cow tubes with 12 hours activity (e.g. Spectrazol); and single-shot lactating cow tubes with 48 - 72 hours activity (e.g. Vetimast).

b) Single-shot applications

A single shot intramammary tube can be defined as one which provides an adequate concentration, and period of time at that concentration, of antibiotic for the successful treatment of the majority of routine lactating cow mastitis cases.

Basically a single-shot intramammary will provide an equivalent course of antibiotic therapy to, for example, three tubes of a penicillin/streptomycin intramammary administered at 12 hourly intervals.

There are several advantages to single shot intramammaries. Firstly they provide a very easy routine for the farmer to follow. He does not need to remember to repeat treat, he does not have to work out a complicated "equation" for his milk withdrawal, and he does not have to leave intricate messages for the relief milker! Secondly, both the single shot tubes available (Vetimast and Pathocef) offer a saving in a shortened milk withdrawal time over many 3 shot courses. Thirdly, the single shot tubes enable the farmer to strip out the quarters during treatment (up to four times a day with Vetimast) with little effect on the antibiotic remaining to kill the bacteria. Fourthly, and in my opinion most importantly, they ensure all cows treated received a full course of antibiotics (not two of the three tubes because the milk "looked better!").

c) New potentiators

This year has seen the launch of the first intramammary containing a potentiated antibiotic (Synulox). Potentiators are designed to assist an antibiotic in overcoming bacterial resistance. Potentiators offer a useful alternative, theoretically, to the production of "new" antibiotics and may enable the resurrection of some antibiotics which have admirable dynamic qualities in the udder but have become of limited use due to resistance.

d) Education

A dramatic increase in the successful use of intramammaries could be achieved by better education of the farmer on how, when, and what to use.

How often does one hear, "I've used this tube for 5 years now and it seems to be OK." May I ask what became of antibiotic resistance?

Intramammary tubes are very effective treatments for mastitis, but only when used correctly. In days of consumer pressure to reduce indiscriminate use of antibiotics in livestock, we must make every effort to ensure their use is optimized.

3. THE FUTURE

a) New Antibiotics

Ideally antibiotics should be developed with broad spectrum activity, limited resistance, the ability to kill intracellular staphylococci, and still have minimal milk withdrawal. In reality human pharmaceutical research appears to be directed towards ultra-effective narrow spectrum drugs. Because most veterinary antibiotics are offshoots of human research, the future does not look particularly hopeful.

b) New potentiators/non antibiotic additions

Getting more from the antibiotics already available may be the answer to the potential lack of new antibiotics. Potentiators to overcome resistance, and non antibiotic additions to improve tissue penetrations, may well answer many of the problems faced by intramammary therapy at present.

c) "Targeted" drugs

The human pharmaceutical industry hold out great hope for these developments and I see no reason that they cannot perform the same in veterinary medicine. Basically targeting means that you do something to an antibiotic to enable it to get to its intended site of action quicker and in larger quantities than normal. In the intramammary field this could involve "packaging" antibiotics so that they can be active against intracellular staphylococci.

In conclusion, the last 10 years have seen major advances in intramammary therapy. All the problems have not yet been solved, however, and the future may well see a slowing of progress in the field of new antibiotics. Nevertheless, there are still many opportunities for, "more from the same" and it is perhaps in this area we should concentrate for the next 10 years.

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