

BRITISH MASTITIS CONFERENCE 1991

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INTRODUCTION

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The Fourth British Mastitis Conference takes place against a background of the long awaited introduction of the cell count payment scheme in England and Wales at the beginning of this month. Our programme today will take a look at the latest position of this scheme and then move on to tackle other items requested by delegates to the 1990 conference.

Amongst the many requests received by the organisers were the farmer's view of tackling a mastitis problem and also the role of alternative therapy.

The popular section on research update will be giving the first results of a large scale field trial on pre-milking teat dipping carried out during the winter of 1990/91, together with the latest information on the effects of more frequent milking on mastitis.

The organisers hope that this wide ranging and very topical programme will continue to provide delegates with a unique forum to both hear and discuss aspects of mastitis control from many different viewpoints.

CELL COUNT PAYMENTS - THE LATEST POSITION

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In this paper I shall describe current trends in mastitis in England and Wales and discuss the effect these may have on cell count payments to farmers.

Introduction

We estimate the current annual cost of mastitis to the British farmers at £70 million. This is based on the direct losses due to clinical mastitis and on an estimated 15% of the UK dairy herd affected with subclinical mastitis, which could well be an underestimate. This represents real progress in controlling mastitis; three years ago, at the time of the first British Mastitis Conference, the estimated annual cost was £90 million.

Cell count payment schemes have been announced by all Milk Marketing Boards (MMBs) in the UK. From 1 October they have been operational in all areas, and some have been operating for several months. In England and Wales it was anticipated that the twelve month warning period would allow farmers time to check their mastitis control measures, and tighten these up where necessary, so that the great majority would be in a position to benefit from the bonus payments. The payment is worth approximately £10 a cow for the farmer who stays in the bonus band throughout the year.

As judged by cell count, this expected improvement has not occurred - at least by the end of June.

Clinical mastitis

With the help of the farmers and herdsman involved, we have been monitoring the incidence of clinical mastitis in a group of dairy herds in Southern England for the last five years. There are now 28 herds in this survey and they receive regular monthly analyses. This does mean that their results are probably somewhat better than the average. Nevertheless, the trend in the incidence of mastitis in these herds is likely to be fairly typical of the national situation.

The 1987/88 results in Table 1 show clearly that the highest incidence of mastitis occurs in the winter period, especially from November to February. The subsequent columns show the changes in the monthly average cases/100 cows compared to the same month in the previous year.

In 1988/89 there was an upward trend in the incidence to 38 cases/100 cows. Since August 1989 the trend has been downwards, to 35 cases in 1989/90 and 30 cases in 1990/91. May 1991 shows the first upward turn in incidence for many months. We will probably know by the time of the conference whether this is significant or not; certainly we have received reports from the field of an increase in outbreaks of clinical mastitis during late spring and early summer of 1991. A further pointer may be the fact that for the first time for many months there were more survey herds which showed an increase in mastitis in May than showed a decrease.

Table 1 Trends in the monthly incidence of clinical mastitis (cases/100 cows)

Month	Cases/mo	Changes in monthly average		
	87/88	88/89	89/90	90/91
Jul	1.5	- 0.1	+ 0.6	- 0.6
Aug	2.0	- 0.4	- 0.2	+ 0.3
Sep	1.7	+ 0.4	- 0.1	- 0.7
Oct	2.5	+ 0.9	- 1.0	- 0.6
Nov	4.2	- 0.3	- 0.2	+ 0.1
Dec	4.2	+ 1.2	- 1.4	- 0.2
Jan	4.4	+ 0.4	+ 0.6	- 0.7
Feb	3.7	+ 0.4	- 0.4	- 1.0
Mar	3.1	+ 1.1	+ 0.2	- 1.2
Apr	2.7	+ 0.9	- 1.3	- 0.4
May	1.8	+ 0.4	- 0.4	+ 0.6
Jun	1.1	+ 0.3	+ 0.3	-

In the group of survey herds *Streptococcus uberis* has been the most frequent isolate, and this appears to be a common finding in herds where the basic mastitis control measures are being practised diligently. Nevertheless, *Staphylococcus aureus* and *Escherichia coli* remain very frequent isolates from the milk samples we receive from cases of clinical mastitis, and even *Streptococcus agalactiae* is not as uncommon as might be expected.

The range in annual incidence per herd is considerable, from 10 cases/100 cows up to 99 cases/100 cows, despite the present overall average of 30 cases/100 cows. The annual usage of antibiotic syringes per 100 cows ranges from 39 to 568, and the days of milk lost from 38 to 691.

Cell counts

The variation in monthly cell counts for 1987/88, shown in Table 2, is typical of most years and shows clearly that the highest bulk milk cell counts are to be expected in the July to September period. However, this is the national average which is probably most representative of an autumn calving herd, so herds with different calving patterns will have different periods when cell counts are at their highest.

The table shows the changes in monthly average cell count compared to the same month in the previous year. The 1988/89 and 1989/90 cell count changes show an almost consistent downward trend. However, the most recent twelve month period, since the announcement of the cell count payment scheme, has not shown a consistent trend at all, although there has been a slight decrease over the whole year.

Table 2 Trends in monthly cell counts (thousand cells/ml)

Month	Monthly average 87/88	Changes in monthly average		
		88/89	89/90	90/91
Jul	391	0	- 6	- 50
Aug	414	- 15	- 2	- 14
Sep	391	- 12	- 14	+ 2
Oct	332	+ 13	- 19	- 54*
Nov	326	- 11	- 23	- 11
Dec	315	- 2	- 37	+ 11
Jan	314	- 11	- 15	- 18
Feb	302	- 7	- 11	- 3
Mar	313	- 15	- 17	+ 10
Apr	334	- 10	- 25	- 9
May	343	- 5	- 30	- 11
Jun	361	- 23	- 27	0

* Change in instruments used for cell counting

Table 3, which shows the percentage of herds having a monthly cell count below 400 thousand cells/ml and the change compared to the same month in the previous year, is more surprising still. In passing, it should be noted that these figures will not correspond precisely to the cell count bonus band which applies to herds having an average of 400 thousand cells/ml or less, based on the geometric mean of all cell counts carried out over a three month period.

Table 3 Trend in herds with cell counts below 400 thousand cells/ml (% of all herds)

Month	% herds 87/88	Changes in monthly %		
		88/89	89/90	90/91
Jul	53.6	0	+ 1.8	+ 9.4
Aug	48.9	+ 3.2	+ 0.8	+ 3.2
Sep	53.4	+ 3.1	+ 2.8	- 0.5
Oct	64.4	- 2.2	+ 4.7	+ 4.6*
Nov	64.8	+ 2.0	+ 4.7	- 1.8
Dec	66.4	+ 1.1	+ 6.4	- 6.1
Jan	67.4	+ 2.0	+ 2.3	- 0.9
Feb	69.2	+ 1.4	+ 1.8	- 3.1
Mar	67.6	+ 2.6	+ 2.7	- 4.9
Apr	64.7	+ 1.6	+ 4.3	- 1.6
May	63.4	+ 0.9	+ 6.6	- 2.1
Jun	59.9	+ 5.0	+ 5.2	- 3.8

* Change in instruments used for cell counting

As might be expected from Table 2, the percentage of herds with cell counts below 400 thousand cells/ml is lowest in July to September. However, there was a consistent improvement in the situation throughout 1988/89 and 1989/90. That has not been the case in 1990/91, indeed the first six months of 1991 show an average of almost 3% fewer herds in this category.

Cell count payments

For the last 20 years, bulk milk cell counts have been used in the UK as a measure of subclinical mastitis and hence of the effectiveness of control of mastitis infection within the herd.

From now on the emphasis will increasingly be on their use as a measure of milk quality, although we should not forget their value as a measure of disease, together of course with the incidence of clinical mastitis.

Table 4 Cell count payment scheme in England and Wales

Band	Cell count (thousands/ml)	Payment (pence/litre)
1	400 and below	+ 0.2
2	401 to 700	0
3	701 to 1,000	- 0.2
4	Over 1,000	- 0.4

Table 4 reminds us of the payment scheme in England and Wales. The other MMBs have individual variations both in cell count levels, in which some aim straightaway for an improvement on the EEC Step 2 standard, and in bonuses and penalties. However, all have the common theme of payment according to cell count, and this has proved to be highly effective in other countries in bringing down cell count levels. The evidence is not yet clear as to whether mastitis levels and new infection rates have been reduced equally effectively.

Table 5 shows the distribution of herds according to their three month geometric mean cell count since these first became available from the MMB central testing laboratories. Over the first seven months an average of 71% of herds have been in Band 1, 20% in Band 2, 6% in Band 3 and 3% in Band 4. Table 3 indicates that the percentage of herds in bonus Band 1 is likely to be rather lower than this for the first two months of the payment scheme, since the geometric mean will include the months of August and September. Of greater concern is the fact that, at present, between 8% and 10% of herds are in the penalty bands every month. On present calculations this will cost them over £1 million a year in direct cell count penalties, a definite incentive for improvement.

Table 5 Percentage distribution of herds according to three month geometric mean cell count*

Month	Band 1 (400 or less)	Band 2 (401-700)	Band 3 (701-1000)	Band 4 (over 1000)
1990:Dec	72.4	19.1	5.6	2.8
1991:Jan	71.9	19.0	5.9	3.2
Feb	71.6	19.1	5.8	3.4
Mar	71.3	19.3	6.0	3.4
Apr	70.7	20.2	5.9	3.2
May	70.7	20.9	5.6	2.9
Jun	70.4	21.6	5.5	2.5

* All cell counts are in thousands/ml

It is worth noting that the 71% of herds currently in Band 1 produce 81% of all milk, whilst the 9% of herds in the two penalty bands produce only 4% of all milk.

All previous cell count data have shown consistent regional variations throughout the country. These have ranged from the lowest levels in northern England to the highest levels in south Wales. The three month geometric mean cell counts for March, which is a typical month, show that their pattern has not changed.

Table 6 Regional distribution of herds according to cell count band

	% of herds			
Region	Band 1	Band 2	Band 3	Band 4
North Western A	81.1	13.8	3.4	1.7
Northern	78.9	15.1	4.0	2.1
South Eastern	76.5	16.5	4.1	2.9
East Midlands	74.0	16.7	6.1	3.2
Southern	74.0	18.5	4.9	2.6
Eastern	71.8	19.7	5.7	2.8
North Western B	71.3	20.1	5.8	2.8
Mid Western	71.2	20.3	5.4	3.1
West Midlands	70.8	18.6	6.1	4.5
North Wales	67.3	21.8	7.6	3.3
Far Western	65.1	23.1	7.5	4.2
South Wales	61.5	23.7	9.1	5.7

At this conference last year I discussed trigger points for action if cell counts exceeded certain levels. On present performance there are still many farmers who could pay attention to these to their immediate financial benefit. The action levels are:

1. Any three month geometric mean over 300 thousand cells/ml, and
2. Any weekly cell count over 400 thousand cells/ml.

Besides checking their own mastitis control measures, farmers will almost certainly need to seek advice either from their veterinary surgeon or from specialist mastitis advisers such as in Genus or ADAS. Individual cow cell counts can be helpful in this situation, provided it is recognised that they are not a substitute for action to control mastitis at the herd level.

Conclusions

Despite the excellent progress made by British dairy farmers in controlling mastitis, the disease still costs them a great deal of money - £70 million a year on current estimates.

The cell count payment schemes are likely to improve this situation. However, although the one year advisory period has undoubtedly raised awareness, the results of this have not yet shown through in reduced cell counts at the national level in England and Wales.

With the TBC payment scheme, improvements in national TBC levels came about as soon as payments started. However, this cannot be a close model for cell counts as most mastitis control measures take time to act. Removing high cell count cows from the herd is a short term expedient only and all farmers, if they are to receive the cell count bonus on a regular basis, must have an effective mastitis control programme in operation in their herds.

We can expect that 1992 will witness a significant improvement in national cell count levels. In the meantime, 10,000 dairy farmers are likely to get an unpleasant surprise in six weeks time when they will miss out on the cell count bonus, worth approximately £1 a cow on the monthly milk cheque, and a quarter of these are likely to suffer a direct financial penalty.

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CAN CELL COUNTS BE TOO LOW?

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The short answer is: No.

Obviously this needs some explanation!

The argument against low cell counts

Some people fear that if the bulk cell count becomes too low, then the herd will lose its resistance to mastitis. This will result in outbreaks of clinical mastitis which are often severe and difficult to clear up. Although there is no general agreement on what the cell count should be to "keep some resistance in the herd", a lower limit of 250 thousand cells/ml is often mentioned.

The scientific argument is that too much emphasis on mastitis control, especially dry cow therapy and teat disinfection, removes the minor mastitis bacteria from the herd. These minor pathogens are said to give resistance against major mastitis pathogens like *Escherichia coli*, *Staphylococcus aureus* and *Streptococcus uberis*.

The difference between low cell count herds and high cell count herds

In high cell count herds a high proportion of cows are usually infected with subclinical mastitis. The clinical flare-ups often manifest themselves as mild infections, unless at calving when the mastitis may be acute or even gangrenous. The large number of infected cows means that heifers or "clean" cows coming into the herd get infected soon after entering the herd. The damage to the secretory tissue is severe and the herd production is considerably lower than it should be.

In low cell count herds most of the "contagious" mastitis bacteria have disappeared. They cannot live in the environment and the infected cows which act as reservoirs have been cleared up by treatment or culling. The reservoir for new infection is the environment. The environmental bacteria are not very invasive, but will be harmful if they are present in huge numbers, i.e. due to dirty wet bedding or poor ventilation, or if the cow's natural defence has been weakened, i.e. due to teat end damage or stress. They cause clinical mastitis which may be peracute, acute or mild. Cases tend to be peracute or acute around calving and mild in mid and late lactation. Temporary milk loss in the affected quarter is common after a peracute case, but rarely lasts into the next lactation. Response to treatment is usually good, although it may be slow.

To over-simplify: mastitis in low cell count herds is usually very noticeable. Mastitis in high cell count herds is more frequent and much is subclinical, but still highly damaging.

Some of the typical characteristics of high and low cell counts herds are shown in the table below.

High cell count (>400,thousand)

Many animals infected with contagious organisms:-

Streptococcus agalactiae
Streptococcus dysgalactiae
Staphylococcus aureus

Mainly persistent subclinical mastitis causing major damage to gland

Long term milk loss

Low cell count (<200,thousand)

Few cows infected with environmental organisms:-

Streptococcus uberis
coliforms

Mainly short duration clinical infections causing damage which is usually repairable

Short term milk loss

The role of the minor pathogens

The two minor pathogens most frequently mentioned are *Corynebacterium bovis* and coagulase negative staphylococci (CNS). *Staphylococcus epidermis* used to be the main CNS, but recent research has shown that there are many sub groups with different names and different characteristics. There is some experimental evidence that infection with *C. bovis* and some strains of CNS offer protection against infection with the major pathogens (1,2), but in other trials the minor pathogens did not offer any protection against infection (3). Jackson (4) reported that the presence of untreated micrococcal infections did not protect quarters during the dry period and even seemed to predispose to infection after calving. Besides, minor pathogens are not harmless. Some workers attribute considerable milk loss to the presence of these bacteria in the udder (3) and they can cause clinical mastitis which, certainly in the case of CNS, can be hard to cure.

What does the bulk milk cell count measure?

It does not measure resistance. The cell counts from individual cows make up the bulk milk cell count. A high cell count in an individual animal means that infection is present, not that resistance against infection is present.

The cow's defence mechanism against mastitis

A normal healthy quarter contains milk with a low cell count, usually well below 100 thousand per millilitre. The cell count is made up of cells shed by the lining of the alveoli, where milk is produced, but the vast majority are white blood cells. Some of the white blood cells act as watch dogs - these are the macrophages. They recognise foreign substances, like those found on the outside of bacteria, and send out a signal to the bloodstream and the bone marrow.

Both the bone marrow and the blood stream hold millions of neutrophils, the killer cells that ingest and subsequently digest bacteria. They are attracted in huge numbers to the site of infection. Where bacteria have managed to damage the tissue, substances are released from the dead cells that cause extra blood flow to the udder (more neutrophils!), zinc and iron are removed from the environment so that bacteria cannot grow and there may be a rise in temperature, which also inhibits bacterial growth. All this is visible on the outside

because the animal has clots in the milk, which consist of damaged tissue, fibrin and dead neutrophils; a red and swollen quarter from the extra blood flow and sometimes an elevated body temperature and a depressed appetite. A swollen quarter and a fever are not bad signs in acute mastitis! They are signs that the cow is defending herself. When the invading bacteria have been eliminated, the neutrophils start cleaning up the dead tissue. The cow's cell count usually drops to close to its previous level within two or three weeks, when the cleaning-up has been finished. It may remain somewhat higher in the quarter that was infected.

Failure to eliminate the bacteria triggers a second defence mechanism. The bacteria are segregated by a wall of dense tissue and neutrophils in a small abscess. Some bacteria survive in the neutrophils themselves, especially *S. aureus*. A chronic mastitis develops. The presence of bacteria in the udder continues to attract neutrophils and the cow has a permanent high cell count.

The whole process of elimination of bacteria from a healthy udder depends on the speed of recruitment and activity of the neutrophils which enter the mammary gland in response to infection, not on the number present in the udder at the time of infection.

Even large numbers of neutrophils may not eliminate or protect against infection. A few examples include:

- 1 The most severe cases of mastitis occur at or just after calving when the udder is full of colostrum. Colostrum contains millions of neutrophils.
- 2 Intramammary device, which raises the cell count to close to one million cells/ml and still fails to give protection.
- 3 Cows with subclinical mastitis and a cell count of several million, as frequently happens with *S. agalactiae*. Bacteria are still present, regardless of the cell count.

Factors affecting the cow's defence against infection

The speed and activity of neutrophils are influenced by many factors. The stress; the hormone cortisol has a severe depressing effect on neutrophil activity. This may partly explain the severity of the disease around calving, when cows are under stress and cortisol levels are high. It is not important which pathogen causes the infection. The so-called "*E. coli* infections" with severe systemic involvement can also be caused by staphylococci or *A. pyogenes*(5).

As was explained at this conference two years ago (6), vitamin E and selenium are important for neutrophil activity, and there may be other dietary factors. A high energy diet may cause fluid dung and cause severe contamination of the environment with coliforms.

The milking machine can damage teat ends and create an entry for environmental bacteria. Milk fever and lameness both increase the time spent lying down and thus the risk of environmental mastitis. Concurrent contagious disease may drain the reserves of neutrophils and slow down response.

It is possible that neutrophil activity is inherited, but that is extremely difficult to establish. What is inherited is the first line of defence: the teat shape, size, tightness of the sphincter and udder shape. We should be looking for cows with strong supporting ligaments, rounded teat ends and not too easy milking. Mastitis is almost unavoidable with cut teats, which demonstrates the practical role of the teat in defence.

Factors affecting bacterial numbers

Environmental bacteria show explosive growth in a warm humid environment. Housing, bedding, ventilation, scraping, stocking rate and milking machine cleaning should all be managed in such a way that bacterial numbers are kept as low as possible. The time and money spent on this will be gained during milking, because cows come in clean and have less mastitis. Obviously this is especially important for calving and freshly calved cows, because they tend to get the more severe cases. I refer to the papers given by John Sumner and Philip Francis at the British Mastitis Conference in 1989 for details on management (7,8).

What to do during an outbreak of clinical mastitis?

1. Do not blame the cell count. And do not try to increase it! This may soon lead to even greater losses.
2. Take milk samples for bacteriology from clinical cases to establish the source. Environment or contagious? Act accordingly, i.e. improve environment (7,8) or check mastitis control programme (9).
3. Whatever the source, have the milking machine tested. Check for teat end damage, which may lead to an increase of environmental mastitis. A low vacuum reserve will cause the spread of contagious mastitis.
4. Check for stress factors. Bad weather? Long walk from field to parlour? Impatient herdsman? Underlying disease? Metabolic diseases? Difficult calvings? Bullying? Overcrowding? Lameness? Nutritional changes? Cows are extremely sensitive to changes. Address these factors where possible, good management can usually overcome poor conditions.
5. Treat cows properly, identify treated cows and keep records. Many so-called outbreaks are caused by chronic infections flaring up at the same time, due to a common stress factor. Dry cow therapy on the carrier-cows should be monitored carefully and they should be culled if they do not respond.

Summary

The bulk milk cell count does not measure resistance to mastitis. It provides an approximate measure of mastitis in the herd, mainly in its subclinical form. Individual cows in that herd with high cell counts are likely to be resistant to coliform infections; but they are already infected and certainly cannot be described as healthy.

The desirable resistance against mastitis depends on the teat barrier being intact and the ability of the cow to mobilize large numbers of efficient neutrophils in a short time.

This ability is impaired if the cow suffers from stress, malnutrition or disease.

Resistance can also be overcome if the bacterial challenge is big enough, as is the case in wet and dirty conditions, or if the teat is damaged.

The role of the minor pathogens in protecting against major pathogens is still unclear and research in this area is needed. They may not be as harmless as has traditionally been thought.

Seen as a proportion of clinical cases, low cell count herds suffer more from environmental mastitis because contagious mastitis has largely been eliminated from the herd. Environmental mastitis is occasionally very severe and thus very noticeable. However, cure rates are better than for most contagious bacteria and the damage is rarely lasting.

It should certainly not be an argument to go back to the hugely damaging state of a high cell count herd riddled with mastitis, both from an economic and a welfare point of view.

The only way forward is to decrease the challenge from the environment and to give cows every opportunity to defend themselves against infection.

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ON-FARM TESTS FOR SUB-CLINICAL MASTITIS

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Background

Consumers and buyers of milk rightly demand high quality milk and milk products. Bacterial content is arguably the most important factor affecting milk quality and, as is now widely accepted, mastitis is a major source of bacterial contamination.

There is a strong financial incentive for UK dairy farmers to produce milk of a high quality, particularly in relation to hygiene. In 1982 the MMB introduced a central testing scheme and payment based on the total bacterial count (TBC) of milk. Nine years later, October 1991, payment in England and Wales are also based on the somatic cell count (SCC). In financial terms, milk consistently in TBC band A is worth about £12 per cow per year and about a further £10 a cow can be achieved by producing milk consistently with a cell count of less than 400,000/ml. In the days of declining profit margins an extra £22 per cow per year should not be sneezed at.

ADAS investigations of high TBC cases show that during housing, mastitis is the main cause of the problem in nearly one half of cases. This observation agreed with an analysis of 754 high TBC bulk milks which showed that 44% of samples contained more mastitis related bacteria than non-mastitis types (1).

Milk from a healthy quarter usually has a TBC of less than 1,000/ml. TBCs of milk from clinical quarters vary but can exceed 100 million/ml (2). When a quarter becomes infected the numbers of bacteria present in milk begin to rise and can be high when the infection is close to the clinical stage.

The most dramatic effect on TBC is therefore associated with clinical infections, but if a herd has a high proportion of sub-clinical infections, although lower numbers are excreted into the milk, the TBC is also affected. Where there are high numbers of sub-clinical infections in a herd, there is an increased chance of more cases becoming clinical.

Most researchers would agree that milk from cows free from infection should contain less than 200,000 cells/ml. Once an infection is established an increase in the concentration of cells in milk usually occurs.

For strong economic reasons milk producers must not only minimise mastitis infections, they must also produce high quality milk to meet the needs of the market. Early identification of clinical disease followed by exclusion of contaminated milk from the bulk supply are vital if the objectives are to be met.

Examination of fore-milk for clots remains an effective method of detecting clinical mastitis. It is worth remembering that the Milk and Dairies Regulations still require that the fore-milk of each cow is removed, examined and discarded at every milking. In-line mastitis detectors have been used by many producers. But identification before the onset of clinical

disease allows mastitis suspects to be recognised and provides the possibility of keeping milk with a high TBC out of the bulk tank.

Early detection of mastitis has long been recognised as a key element in mastitis control. In terms of contagious mastitis early identification of infected cows helps to reduce the transmission of pathogens during milking to uninfected cows. It also reduces the severity of infection, enhances the prospects of recovery and reduces production losses by minimising the damaging effect on long term yield.

Research Developments

For many years researchers have attempted to develop test methods, including cowside tests, for sub-clinical mastitis. Most of the effort has been directed towards measuring the changes which occur in milk composition when infection occurs. Infection leads to damage of alveolar cells or increased vascular permeability. The capacity of epithelial cells to produce milk is reduced and the increased permeability of damaged tissue results in, amongst other changes, increased bovine serum albumin, increased sodium and chloride content, changes in enzyme levels and an increase in somatic cells. Changes in protein, fat and lactose levels can also be observed and there are other less noteworthy changes. It is worth reviewing some of more important research developments.

Electrical conductivity

For half a century or so, it has been known that the electrical conductivity of milk is related to mastitis. Based on the understanding that sub-clinical mastitis results in ionic changes in milk (sodium and chloride ions diffuse into the milk) it was considered that changes in milk conductivity would be a good indicator of infection.

Many reports can be found in the literature, but not all reached the same conclusion. Early work (3) indicated a high correlation, but later work (4) was unable to confirm the results. Many approaches have been taken including measuring absolute conductivity, relative conductivity between samples and the situation has been complicated further by use of samples taken from fore-milk as opposed to strippings. It has been recorded (5) that conductivity of fore-milk taken at a single afternoon milking was an accurate method of differentiating between infected and non-infected cows and that conductivity generally could be a useful indicator of sub-clinical mastitis (6).

Conductivity levels vary between cows as a result of differences due to age, number of lactations, stage of lactation and mastitis history. Other factors which affect conductivity measurement are fat content of milk, temperature at which the measurement is taken, cell count and milking interval. However, the conductivity levels of milk from healthy quarters tend to move in parallel from day to day for individual cows. In view of this, and provided allowance is made for influencing factors, conductivity levels can be used to detect sub-clinical mastitis, i.e. comparing conductivity levels between quarters "within cow" gives a more specific diagnosis.

There have been numerous attempts to develop in-line systems for automatic detection of conductivity changes, but practical problems have included air bubbles in milk causing variation in conductance and fouling of electrodes by fat and protein. The degree of cleaning required during milking in order to maintain accuracy has been found to be unacceptable.

Recent collaborative work by the Institute of Animal Health at Compton and the Silsoe Research Institute (7) has overcome the latter problem by an inductive and electrode-free sensor. Experimental results indicated that the detection of electrical conductivity changes in individual quarters is possible. Milk yield variation was also taken into account. Conductivity changes were associated with clinical signs of infection, bacterial recovery and increased SCC. Work to date has been based on the hydraulic milking system (non-aerated milk). Any successful in-line system will require automatic cow identification and a computer link for storing and analysing data. Each cow could therefore be given its own threshold value and monitored at every milking. The possibilities are encouraging.

Further work at Compton has reviewed the effectiveness of a hand held conductivity meter (8). Known as The Milk Checker (Eisai Co. Ltd.) the instrument gave higher milk conductivity readings from cows with sub-clinical *Staphylococcus aureus* infections than readings from uninfected cows, but milk from cows with sub-clinical *Streptococcus uberis* infections showed no increase. However both types of clinical infections had an influence on conductivity levels and could be detected at least one milking before clinical signs were observed.

Milk temperature

Observations on fluctuations of milk temperature during milking (9) showed frequent fluctuations were found in cows with sub-clinical mastitis. It was suggested that fluctuations may indicate disorders in milk ejection and that automated measuring equipment may be used for early detection of mastitis.

Catalase

The oldest and most widely used enzymatic test, particularly in the USA, is the catalase test (10). The method measures the oxygen liberated when the hydrogen peroxide reagent, which reacts with catalase, is added. The method is not effective at detecting infections caused by streptococci which are catalase negative.

NAGase

The enzyme NAGase can be determined by a relatively simple and rapid calorimetric test. NAGase levels increase in mastitic milk largely due to an outpouring of cytoplasmic material from damaged secretory tissue and the presence of white blood cells. NAGase assay has been shown to be a useful diagnostic test.

Detergent-based tests

A number of tests based on the reaction between somatic cell DNA and alkalis or detergents to form a gel have been developed over the years. Such tests include the Whiteside Test and the California Mastitis Test (CMT). The degree of reaction is roughly proportional to the SCC of the tested milk.

These tests have been used in many countries by veterinarians and others when investigating mastitis problems as a cowside test. Unfortunately the tests are not sensitive enough to reliably detect sub-clinical mastitis and the interpretation of the degree of viscosity is highly subjective.

Comparative studies

A number of studies have compared methods. In discriminating quarters infected with *S. aureus* from non-infected quarters, Sheldrake (11) showed that misclassification for cell concentration ranged from 8-20% among herds, electrical conductivity ranged from 22-32% and a range of 15-48% was found for serum albumin. Fernando (12) reporting on fore-milk samples tested by conductivity showed 9% false negatives and 34% false positives, a mean of 21% false values. Other work (13) recorded error rates of 9.1% for CMT, 13.9% for SCC and 29.4% for conductivity.

The literature produces many contradictory arguments particularly in relation to conductivity - foremilk versus strippings, absolute values versus quarter ratios and so on. The recent Compton/Silsoe work has shown that detection of incipient mastitis in individual quarters can be achieved easily and speedily before visible signs appear. However the difficulties associated with false positives and negatives were not fully resolved.

The above descriptions cover the major milk compositional changes and the general principles behind their detection. The test list is by no means complete. Chemiluminescence to detect leucocyte activity has recently been examined, whilst in the past ATP and antitrypsin have attracted interest. However, while this range of tests is viewed as indirect tests for mastitis, not all can be adapted for cowside use.

Available equipment for cowside tests

In the UK there has until recently been relatively little interest in cowside tests. The Whiteside and CMT tests have been used by veterinarians and specialists (including ADAS) to investigate herd mastitis problems. Over the years a number of attempts have been made to introduce conductivity meters, but their success has been limited by false readings. In the following paragraphs reference is made to a range of devices currently marketed. The list is by no means exhaustive and omission of any product does not imply criticism.

Using the catalase principle, MASTCHECK, an on-farm kit, has claimed advantage of indirect measurement of the level of inflammatory cells reflecting the presence of inflammation in the udder. The test takes five minutes. Experimental data from the manufacturer, Cambridge Veterinary Sciences Limited, claims that although high SCCs were not always associated with a high MASTCHECK reading, where readings were high, 95% of samples had high SCCs.

A battery driven conductivity meter has been developed by AHI of New Zealand. Milk from individual quarters is drawn directly into the device. The presence/absence of high conductivity is shown by red or green lights. The indicator compares conductivity ranges between the quarters of each cow. The device is marketed here by Telsen Products Ltd.

A conductivity meter, originally developed in Israel (Afikim) is marketed by Fullwood. Incorporated into a milk recording meter, absolute conductivity is recorded at every milking and by using rolling averages, deviations in conductivity are detected. Cow identification numbers are recorded either manually or by neck transponder.

The principle of the MILKCHECKER, a hand held conductivity meter, marketed by Deosan has been discussed. The company recommend the kit to be used to detect SCCs of individual cows in excess of 800,000/ml in addition to checking suspect cows.

The "BLACK BOX" designed originally by Action Plan Limited, is an example of a further attempt to detect sub-clinical mastitis by conductivity. Situated in the milkline between cluster and jar, it constantly monitors milk as it leaves each cow. The device includes a collection dish with electrodes inside and records absolute conductivity levels by digital display.

A number of USA companies is marketing electronic milk temperature measuring devices. The main objective of screening all cows daily is to detect fever. It is argued that most fevers detected with temperature sensing are associated with the beginning of mastitis and metritis. Some devices are capable of being computer linked.

Discussion

These recent developments are interesting and offer considerable promise. However the main objective of milk production is to produce a high quality product and it must be remembered that many producers already achieve consistently low TBC and cell count levels by good management. The increasing interest in individual cow cell counts will result in more data being available to assist in controlling mastitis, provided of course, the data is interpreted correctly.

Cowside tests have tended to be laborious and subjective. However, many herds from time to time have problems and the use of a cowside test such as CMT as an investigation tool should not be discounted. Similarly, the hand held conductivity meters may well play a useful role in identifying mastitis suspects.

Electronic equipment capable of being installed in conventional parlours and linked to a computer offers considerable benefits. A database on individual animals could be developed improving the usefulness of data collected. The Compton/Silsoe work takes us one step nearer that objective.

However, this range of cowside tests to detect sub-clinical mastitis whilst providing interesting data, presents a number of difficult questions. Should we treat sub-clinical infections? Should bacteriology be done on all positives? Should we discard milk from

suspect cows without knowing the degree of contamination? Do nothing? These and other questions are worthy of discussion.

The interest in cowside testing is increasing. It is vital that any new devices are reliable and supported by adequate research and development. It is desirable for a reference test method to be adopted against which new equipment would be assessed.

In the not too distant future we can expect to see cowside equipment which will identify the pathogens involved. Work is currently underway at Compton using molecular techniques to recognise specific pathogens. Ultimately perhaps it will be as simple as a paper dipstick.

Summary

The work described in the paper has considerable relevance to dairy farmers and veterinary surgeons. A number of devices are already available, and although most have limitations, if used appropriately they can contribute to mastitis control. Current developments here and overseas offer the promise of reliable early detection of sub-clinical mastitis which will allow prompt and more effective treatment. As a result mastitis should decline in importance as a disease and as a factor reducing milk quality.

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TACKLING MY MASTITIS PROBLEM

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This paper relates to an outbreak of mastitis in the mid-eighties which cost me in the region of £60,000. The experience certainly educated me in a disease which is still one of the biggest menaces in the dairy industry. It soon became clear that despite all my efforts, determination and knowledge, the "tackling of the problem" was based on advice received from specialists on milking machines and mastitis control.

I would like to start with Professor Keen's last words at last years British Mastitis Conference, when referring to drug resistance in bacteria, he said "I suppose Mother Nature will always be one step ahead". As a pharmacologist he is competing with nature; as a farmer I have to work with nature all of the time. If I do not, it reflects on my stock, my crops and ultimately my balance sheet.

Modern farming like so many things on this planet, has developed so fast in the last 30 years that we are only just beginning to see the disastrous consequences of some of our actions. My problem with mastitis illustrates this in the dairy industry. Up to 1983 my concern, like many of my colleagues, was to produce more, and ever more, milk. We started 3 times a day milking in 1979, and have done so ever since. In that year I updated my 8 x 16 herringbone parlour, replacing the existing vacuum pump with a bigger one, introducing a memory feeder, and installing automatic cluster removers, so allowing one man to do the milking.

My father and grandfather, certainly influenced my thinking where cows were concerned. I realised that mastitis was one of the main evils I should guard against, and always observed the following basic rules:-

- 1) Cows should come into a parlour clean, with no need for washing.
- 2) Draw foremilk before milking a cow.
- 3) Change liners frequently.
- 4) The vacuum controller should be cleaned once a week.
- 5) The pulsators should be cleaned once a month.

I would hope that the need for hot water circulations once a day, and cold rinses after other milkings, go without saying. In other words, milking is a mini operation, and hospital rules for cleanliness apply just as much in a milking parlour, as they do in your local hospital. Despite these basic rules, in the Spring of 1985, we faced the battle of our lives. Although mastitis awareness and treatment are always dealt with thoroughly, being with the same herd of milking cows everyday, a farmer is often blind to a developing problem.

After turning my cows out in the May of that year, the incidence of mastitis increased. Being rather shocked to have an increase in the disease at a time it should be decreasing, I sought help from ADAS. The expert made me aware of teat end damage. I

admit this was something that had not concerned me in the past. In the August of that year, the cows were turned onto a field that had been cropped for silage, (no slurry had been applied, just a normal amount of fertilizer), and time to allow the grass to grow. The milk went up, but so did the incidence of mastitis. There were approximately 17 cases in 170 animals, and one cow was drawing blood into the jar. The true enormity of the problem had manifest itself.

I had done everything I knew in my limited field of knowledge to stop new infections, but nothing seemed to work. From the beginning of September to November, it was in the hands of experts. All I could do was observe, listen and hopefully learn.

The first thing to be scrutinised was the parlour. Although it was declared satisfactory according to British Standard 5545, the pulsators, which were 5 years old, were showing signs of wear with 3 times a day milking, so they were replaced.

On the hygiene side; the amounts of water, circulation powder, hypochlorite and temperature of hot water was found to be more than adequate, except the hot circulation was done an hour after milking and this was considered too long a delay. The milking routine was passed as adequate. The experts agreed with washing only when necessary, drawing foremilk and teat dipping after milking. I did find there were conflicting views on this part of our practise. Washing cows, to some experts, should be done at every milking to stimulate milk let down. Drawing foremilk should be done carefully, as the operator could pass on infection. Teat dipping to some was a must, to others not so important. However we elected to carry on with the existing routine.

Despite the magnitude of the problem, which was caused by *Streptococcus agalactiae*, all the people involved with the case agree nothing was seriously wrong. However, I think we all knew, with the few alterations outlined above, the problem was not going to go away. Indeed by the end of September, the problem had grown. The 30 infected cows were in a separate yard, and they were milked last. Hot water was now circulated through the milking system twice a day. Cows well in calf, were turned dry, in an effort to save them from infection.

So, although there was supposedly nothing wrong with the parlour, I had sold about 20 cows, I had a stock of infected cows and winter was fast approaching. The experts started to look for any other problems. The cubicles filled with loose box manure, invoked horror. It was suggested that they may have contributed to the problem, although the cows were still out at grass! Certainly when they returned for the winter months, these cubicles would add more fuel to the problem.

Having put the matter totally in the hands of experts, I was just listening. I was beginning to see a conflict of opinion. The experts were not happy with two matters.

- 1) I was not treating the mastitis infected cows with any antibiotics.
- 2) The manufacturer of the liner I was using was not the same as the rest of the parlour.

The first point had not been adopted because

- a) the added expense
- b) the mastitis was not actually making the cows ill. My method was to turn the cows dry or the alternative to sell them, this seemed to be my only way out.
- c) If the mastitis was *S. agalactiae* then this is usually associated with the milking machine. I was beginning to realise, that the BS 5545 was clearly misguiding our belief that the parlour was satisfactory.

Furthermore, when I treated 8 infected cows night and morning, with a thorough 5 day programme of antibiotics all the cows cleared up, but within a week it returned to all 8.

Regarding the second matter. The liners I had been using were a type my grandfather, father and I had used for the last fifty years. My grandfather had always maintained, that the efficient milking of this liner, was of paramount importance to the cow. Indeed, if I was having so much trouble with mastitis, he could not believe that there was not something wildly wrong with my milking machine. As one of the pioneers in the use of it, he found mastitis to be rife amongst his 160 cow herd in the 1930s. He maintained "though it existed, mastitis was never a problem until the milking machine was invented". For those of you who doubt his theory, let me point out that he lived through the introduction of milking machines when there were no antibiotics, no milking machine technicians, no maintenance timetables, no exotic detergents or teat dips and no ADAS - just milking machines and a lot of mastitis!!

The question I asked myself all winter was, "who was right"? Instinct told me by now my parlour was far from right, and advice was telling me to be wary.

Just before the cows came in, an interesting event happened. A heifer lost the end off her front left teat. The damaged quarter ran freely with milk, making it exposed to the evils of the cubicles filled with loose box manure. The other 3 teats were undamaged and were milked with the milking machine. During that winter, the heifer not only came through free of mastitis in the injured quarter, but had mastitis in all of the other three quarters. My own experience was now telling me, though the cubicles were far from satisfactory, the milking time was clearly a haven for mastitis bacteria to establish themselves. The winter of 1985/6 was pursued with caution and we managed to keep the problem at bay, but I cannot say it was under control. When we turned-out, the milk went up, but again, so did the incidence of mastitis. We therefore called on the last expert, who initially was not treated with the respect he deserved. My knowledge on the subject of mastitis had increased a hundredfold. It was as though I had a large jigsaw before me. I had completed most of the scene, but there were still many pieces to put together and it was all blue sky. However things took an immediate change, different questions were being asked. When he first saw my parlour he could see little wrong, although he felt that the airflow might be too severe, the claw pieces could be improved on, as they were really too small. He knew that the liner we originally used could not be improved upon, but indicated that there was an alternative that was as good. Against the advice that I was given earlier in the episode, I installed a complete Surge update which included a big bore 3 inch pulse line and balance tank, plus liners, shells and claws and finally pulsators.

By now funds were short, so I could only install the update step by step, This had its advantages; we were able to see the individual benefits, of each improvement.

- a) The new claw piece liners and shells resulted in the teats being as dry when the units came off, as when they went on, resulting in no flowback.
- b) The 3 inch pulse line for pulsators provided a perfect rest phase.
- c) The new pulsators milked the cows out more thoroughly than I had ever seen before.
- d) The 2 inch wash line eliminated any vacuum fluctuation when units were going on.

Once this was installed the battle against *S. agalactiae* was reversed. We were now winning. There was only one fresh case of mastitis in 12 months and I am pleased to say we continue to win this never ending battle.

So, as farmers, these are the conclusions that my grandfather, father and I came to. The advent of the parlour in the 60s, was a tremendous breakthrough, with shorter milk and vacuum lines making the goal for constant vacuum easier to achieve. Milking machine manufacturers went further with their technology, some of the ideas were excellent. The automatic cluster remover eliminated the likelihood of overmilking. This improvement, like many others, was compatible with Mother Nature. However, the idea of quicker milking, altering pulsation ratios, introducing longer milking phases, thus reducing the rest phase and increasing pulses per minute, was a mistake. Stable vacuum is also of paramount importance, however, too efficient airflow in small bore tubing has brought about serious side effects, resulting in teat end damage. We overlooked the fact that we had designed an artificial calf, but for the sake of speed we had subsequently attached an alien to the cows teat. It must not be forgotten, that extracting milk from cows is a delicate operation, and Mother Nature should be treated with respect. Damaging teat tissue is unacceptable, as it leaves the cow wide open to all types of invading bacteria.

The drastic consequences of ruthless milking machine design, resulting in teat end damage has been camouflaged, by the biochemist with his arsenal of antibiotics to combat these bacteria. This has been a short term solution to a bad mistake on our part. To have used this method of curing a problem created by so called faster milking is ridiculous. The lactating tube of penicillin though often effective, is only an expensive cure, and indeed the last resort.

On reflection, the cow has been treated like a machine. She has been forced to produce. The senior veterinary partner in my local practice said "It is the fertilizer salesman and the corn representative that keep me in business". Indeed, a cow in her life is concerned about 3 things

- a) what she eats
- b) where she sleeps
- c) how she is milked

When considering mastitis, they all matter; but the top priority is how the cow is milked. I feel emphasis has been taken off the working of the machine itself and put on the milking routine, and the use of gadgets. These include:

- a) filters or detectors, for mastitis observation in the long milk tube - there only to contaminate
- b) hydraulic milking, with the use of balls in claws - soaking all teats in milk and producing a perfect environment for bacteria to establish a bridge head
- c) dipping clusters in chlorinated solution between cows - an unnecessary chore to prolong milking time
- d) dipping before milking! - Teat dips definitely have a place in the defence against mastitis, to keep teat tissue healthy, but I feel we expect too much of these products. If the teat is damaged during the operation of milking, we cannot expect the teat dip to defend the damaged tissue and it is a fallacy to say it assists in healing damage that a milking machine has created.

I am the first to recognize that I have no knowledge in the field of science, so I am placed in a difficult position; the frontline, everyday, milking cows. The routine we adopt, has to be simple, otherwise it will be vulnerable to breakdown by the human factor alone. We are not working under laboratory conditions where the environment is constant. The conditions we work under, as many of you will know, varies every day. However, I agree milking is an operation, so the parlour should be as clinically clean as our bovine friends will allow, and most important of all the machinery used to perform the milking operation, should function perfectly. Failure to achieve this makes all the other effort to avoid mastitis a complete and utter waste of time.

The patient, the cow in this case, should arrive in the operating theatre (the parlour), clean, with no need for washing. In my opinion foremilk should be drawn to allow the vigilant cowman to check in approximately 10 seconds for:-

- 1) a clean teat and udder
- 2) any irregularities that may have developed on the teats or udder such as warts, rashes or sores providing a means for bacteria to colonise and develop a bridgehead.
- 3) that the milk is healthy free from contamination. This is usually indicated by the fact the milk is powder blue in colour.

Clean the teat canal, opening the sphincter muscle, assuring the cowman it has not been damaged, and is therefore healthy and functioning normally. This action also stimulates the cow to realise it is time for her to once more let her milk down. The quality milk that we strive to produce in quantity also acts as a lubricant on the inside of the teat during milking.

To complete the needs of the cow outlined earlier; it is essential to have clean dry cubicles, that are big enough and comfortable for the cow to sleep in and also easy to get out of when the time comes, together with a balanced diet throughout the seasons.

These are areas that should never be ignored. However, if these two areas are perfect and the milking machine is anything but perfect, then your efforts with the first two will be in vain.

Remember the words of my grandfather, as I am sure this is the base from which we should all work in our pursuit against mastitis, "though it existed, mastitis was never a problem until the milking machine was invented".

TESTING MILKING INSTALLATIONS TO PREVENT PROBLEMS

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Machine Milking

The milking machine has been in use for almost a century and during that time it has progressed from a simple cow side bucket milker, to a sophisticated parlour system with assistance from varying degrees of automation.

It is still the basic milking machine which provides the elements of such sophisticated systems. It usually responds to all demands made of it with little care or maintenance. However, it is difficult to assess how well the average system responds with the level of maintenance it gets. If one draws an analogy with a modern motor vehicle with extended service intervals, and similar daily usage, a milking machine should receive a minor service every 60 days and a major service every 120 days. Thus, an installation should have at least three major services per year. My experience is that one service per year is more usual, with a number of farmers opting for two and some who have no planned maintenance at all. It is difficult to obtain a true figure of farmers using regular maintenance procedures, as there are so many agencies claiming to supply those needs. Currently around 60% of milk producers in the SMMB area have at least a test and service once per year.

Farm staff "servicing" is usually limited to changes of liners and rubber tubes as and when necessary and checking pump oil levels either regularly, or when remembered. Considering that the milking system is one of the very few items of farm dairy equipment which is 'used' every day, on two or three occasions, it is some testament to reliability that it does not suffer from more problems.

Machine Testing

In this context I propound my own methods of machine examination. You may follow similar lines and arrive at a similar conclusion.

In order that the farmer can tell you of any real, or imagined problem which he regards as being significant, arrival at the farm at the arranged time is paramount. These discussions usually take place as you tour the milking place to establish the location of the components and system layout. Thereafter I prefer to conduct the test procedures without interruptions. However, should the farmer or dairyman wish to see the test being carried out, I have no objection; indeed I would be delighted if more people did so. It is more usual for the person concerned to keep popping in to see what is going on, or what conclusions have been reached.

The first part of my test involves a walk round the entire system noting any points which attracts my attention, such as condition of rubbers, clawpieces, pulsators and general impression of the condition of the unit. Detail such as original designed size, together with extensions and modifications after installation, are all important and can give an indication of possible problems. During this examination, belt tension, pulley sizes, alignment and

pump speed, are all checked as well as type of electrical supply, either three phase or single phase.

The actual mechanical test procedures follow on from this. The vacuum pump is left running while all the preparatory work, such as plugging teat cups and setting up for milking, is carried out and the operating vacuum level noted.

The vacuum pump is then switched off to permit direct connection of the flow meter to the pump, in order to assess its capacity. The pipe connections to the pump are also noted at this time for size and condition. The pump should be connected with a pipe of the full flange diameter, through to receiver or balance tank. All too frequently the flange diameter is reduced to a smaller pipe size, so effectively throttling air inflow. The pipe from the pump should be fitted using easy bends rather than elbows to all parts where vacuum supply is required. In addition, a full bore tee-piece should be fitted just above the pump inlet, with a full bore valve fitted to the branch, so that the pipe between the sanitary trap or interceptor bucket, can be brought back to atmospheric pressure quickly, in the event of the float cut off in the trap shutting off. The pump can then be switched off without it turning in reverse with possible damage to the pump.

The pump capacity is noted at 3 or 5 different vacuum levels, usually two above and two below the operating level, thus giving a better picture of the pump condition.

I prefer to use a larger pump driven at the lowest end of its speed range, rather than one driven at its maximum, or in some cases, just over its maximum, to provide the capacity required.

The second part of the test is a check on the capacity available when the vacuum pipeline alone is connected to the pump, this being carried out at the same vacuum levels as for the pump. Differences in readings here, from those at the pump, are due to pipeline losses. These might be due to leaking drain valves, faulty connections, badly corroded pipework, partially blocked pipework, or restricted/blocked filters. During this test all vacuum operated equipment is disconnected and subsequently reconnected and retested for each component (i.e. feeders, pulsators, regulator) to ascertain contribution of these to overall consumption.

The test is repeated yet again with all clusters, recording jars/meters connected as for milking, with teat cups plugged and clawpieces open in milking position. Differences in readings here will be due to cluster air bleeds, holed tubes and leakage in the milking system. Air bleeds on clusters are also checked individually for operation and consumption.

This is followed by connection of pulsators, feeders, gates and ACRs to check their effects and recovery rate after reinstatement of the vacuum controller.

The vacuum controller is checked for its performance, and its operational characteristics at the time. Should it be in dirty condition, it would then be cleaned and retested.

Following these checks, the individual pulsator checks on the clusters would be carried out to assess the pulsation effect being produced at the teat cups.

On completion of the test, minor remedial work is carried out (cleaning regulator, pulsators, air bleeds, drain valves) and the test results explained to the farmer and/or dairymen, with reference to the installation. The farmer will also be supplied with a written explanation of the condition of the installation and what remedial work is necessary to correct short-comings, or improve existing operation.

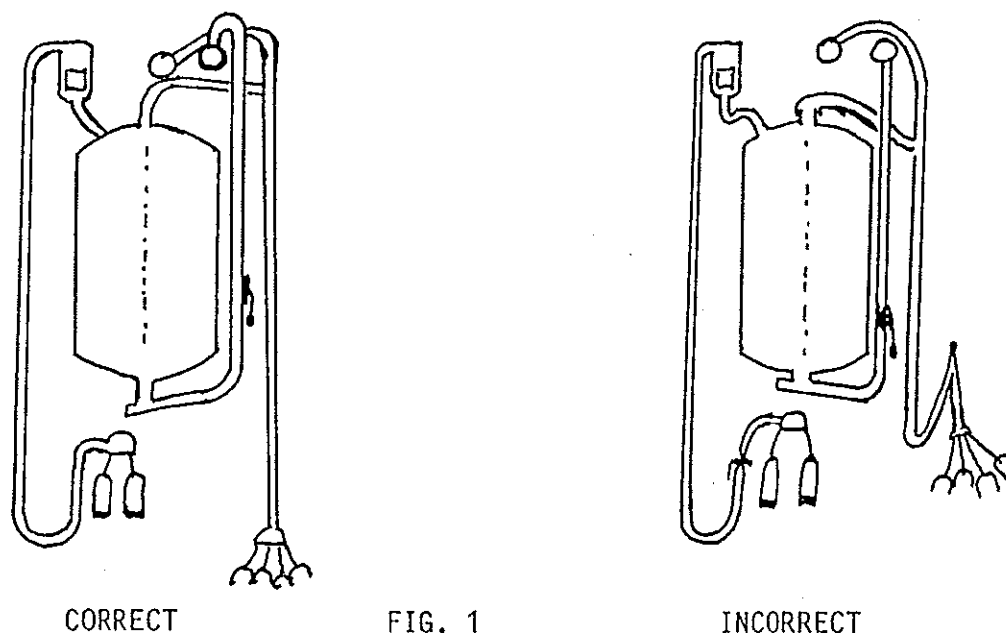
Interpretation

It is important to note that the test figures alone do not give a total picture of the condition of the installation. As indicated above, a milking parlour with five recording jars may have been extended to six, seven or even eight, by merely extending wash, milk and vacuum pipes to a position suitable for the extension, together with either a larger vacuum pump, or speeding up existing units. Often, little or no consideration is taken of the extra air flow that this will require.

Whilst a badly functioning milking machine, may predispose a herd to mastitis, it is essential to remember that unsatisfactory washing and disinfection of the clusters can act as a vehicle of bacterial transfer. It is necessary therefore to examine and understand the washing sequence and component parts. The air supply pipe to the recording jars during milking, is the water supply pipe during washing, delivering wash water to the jars through central nipples and to the teat cups through jetters.

The rubber tubes associated with this system are frequently in poor condition, with the tube to the central jar nipple bent over on itself restricting or completely cutting the vacuum supply to the jar. A walk down the parlour during washing may give evidence of poor wash water flow which increases if the attached tube is raised to give a slow loop. Ideally, the jetter tubes should be left free of any clamping except during milking time, otherwise they can become permanently flattened, so restricting or preventing passage of wash to teat cups. Poor flow rates through these tubes may also be due to insufficient capacity of the wash pipes for the number of milking points, or simply that the connection to the pipe is fitted such that it has dropped below the pick up point, so not allowing the wash solution to pass down to the jars or jetters.

The inflow of wash solution is frequently a problem in washing recording jars due to insufficient capacity in the pipe for the number of jars in use. However, even in situations where a plentiful supply of wash solution is available, it still fails to give a satisfactory wash. In this case it is almost certainly due to one or more of the following: too many jars being washed on the one system (i.e. milk transfer pipe overloaded); jar outlet connected into underside of milk transfer pipe, so that the wash solution has difficulty entering if wash water is already passing along the pipe; or finally, and most commonly, the filling of the pipe either because of the outflow from too many jars, or formation of slugs of wash, preventing vacuum from drawing wash through clusters and into jars (See Fig 1 below).



In my view the transfer tube from the jar outlet to milk transfer pipe should enter the top of the transfer pipe with the minimum of lift involved, and should then flow to the receiver without further rise, thus ensuring minimum obstruction of flow during washing.

The number of jars which should be connected into the transfer pipe depends on the diameter of the pipe. I recommend 3, or at maximum 4, with a 25 mm (1" glass) tube and 4, or at maximum 5, with a 32 mm (1¼"). So for 8 x 16 two wash/milk transfer systems would be installed with milk entry via a sweep tee-piece.

All pinch clips on rubber tubes should be left open when plant is not in use, so prolonging useful life and minimising flattening and restriction.

It is important that the flow in the pipes should be as smooth as possible, so utilising as much of the pipe bore as possible and minimising losses due to turbulence. Easy bends and easy tee-pieces (pitcher tee) should be used to change the direction, or link in additional pipes. There is little benefit from very large bore pipes if the pump flange is not of the same size, as the flow at the reducer will decrease the effective bore even further. If it is desired to fit large bore pipes for various facilities in the parlour, the use of a balance tank is particularly suitable. Several separate circuits/lines may be connected directly to this tank, from which the pipe of the pump flange diameter may be connected back to the vacuum pump without reducers, and hopefully a minimum number of bends.

To avoid the accumulation of foul smelling slippery mass, self emptying traps should only be fitted where drainage is provided for the residues. My preference is for an easily removed trap, which facilitates proper washing on a regular basis, together with checking for any signs of corrosion which may be the cause of considerable leakage.

The build up of dust and oily soot in the valves of vacuum controllers often results in wide fluctuations in vacuum levels. Although cleaning of these valves is essential, lubrication is not recommended. Dust usually adheres to the lubricating oil causing even more sticking of the valve. If carried out regularly, all that should be required is a wipe with a lint free cloth or paper towel. In more dusty atmospheres more frequent cleaning will be required. Should the valve be situated in a location where the air being drawn in is polluted by the vacuum pump exhaust, cleaning will certainly be required more frequently and perhaps necessitate use of white spirit to cope with oily contamination.

Conclusion

It is easy with a regularly used installation as complex as a modern milking system, for an operator to become complacent of its operational effectiveness. It always operates when the green button is pressed, it is only when nothing happens after the green button is pressed that panic takes over. Infrequently, the start is followed by a terrifying screech, a loud bang, and finally a deathly hush. No one checked the oil in the vacuum pump!

The person responsible for milking the cows should always be aware of the right noise. Any change in this may foretell of troubles to come. To help the milker to be more aware of what the system should be doing, regular use of the following routine will help:-

Mini test

- 1) Switch on vacuum pump with milk lines disconnected, check vacuum gauge reading, and strength of hiss at control valve.
- 2) Connect milking system with clawpieces closed, check vacuum gauge and strength of hiss at control valve.
- 3) Fit units to cows and check vacuum gauge, and strength of hiss at control valve.

IF:-

- a) the vacuum gauge is the same as (1) and strength of hiss at the control valve is slightly less than (1), you have a Reserve of Vacuum. Check again with feeder operating (if vacuum operated), if there is only a slight loss of vacuum, reserve is still available.
- b) the vacuum gauge is the same as (1), but the hiss is intermittent or absent, there is insufficient reserve to operate feeders, ACR or other vacuum operated equipment. Have the system checked immediately.

- c) the vacuum gauge is less than (1), with a strong hiss at control valve, then the valve is stuck open, due perhaps to a perished seal ring. Because a hiss is present, you cannot assume that all is well. Use of other vacuum equipment may not affect the strength of the hiss. In this situation the vacuum pump noise will also have a different tone.

As a matter of routine the milker should, just before fitting teat cups to the cow, insert a finger into a teat cup on opposite sides of the cluster with the claw open and feel pulsation. If there is a difference between the two, there is a problem. It may only be a holed pulse tube, but it is worth checking.

Finally, try to be present at your machine check and ask what is happening and what it means. This will help to understand the test sheet which is left with you. Comparison of the most recent test with the previous one, may help you foresee possible problems before they become serious.

THE ROLE OF HOMOEOPATHY

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What is homoeopathy?

For readers who have little or no knowledge of homoeopathic medicine, a brief description of its essentials is necessary to the proper understanding of the role of the remedies in treatment.

Homoeopathy is a branch of medicine which states that any substance which can cause symptoms of illness in man or animal can also be used in the treatment of any condition showing similar symptoms. The principle of likeness between disease condition and remedy is emphasised. If we imagine the illness and the provings of the remedy representing two clinical pictures we should endeavour as far as possible when treating to match one picture against the other. The closer the approximation of the two pictures (the likeness) the more likely we are to achieve satisfactory results in treatment. This is much easier to achieve in human than in veterinary medicine as subjective (mental) symptoms known only to the patient are difficult if not impossible to elicit in animals. Mental symptoms are extremely important in the treatment by homoeopathy in the human patient.

Observation of an animal's behaviour and how it reacts to any given situation, to other animals or people, to noise etc. will in some measure compensate for the lack of communication by speech. In certain circumstances it may be possible to imagine how the animal is feeling e.g. the one which may feel grief at the loss of a companion; the one subjected to forced separation from the owner as in quarantine kennels, or those suffering post-operative psychological trauma.

Fortunately the homoeopathic material medica contains remedies which are helpful in all these instances.

Nature of homoeopathic remedies

Homoeopathic remedies are obtained from all natural sources, e.g. plant and animal kingdoms and also minerals and their compounds with other chemicals. Homoeopathy is frequently referred to (quite erroneously) as herbal medicine. Nothing could be further from the truth as study of the previous remarks will show. While herbal medicine employs many plants successfully, it is unable to exploit the intrinsic merits of plants in the way that homoeopathic medicine is able to do.

Preparation of remedies

Preparation of homoeopathic remedies is a scientific procedure which is best left to a qualified pharmacist trained in the particular techniques. Homoeopathy is too important for remedies to be prepared in any way but the best obtainable. Briefly the system is based on a series of dilutions and succussions (of which more later) which is capable of rendering even a poisonous substance safe to use.

To prepare a potentised remedy, a measured drop of a solution called mother tincture (Ø) derived from plant or biological material is added to 99 drops of a water/alcohol mixture and the resultant dilution subjected to a mechanical shock which is called succussion. This process, which is essential to the preparation, imparts energy to the medium which is rendered stable. One drop to 99 parts water/alcohol mixture is represented by 1c on the centesimal scale. Preparations are also made on the decimal scale and marketed as 1x (on the continent as 1d). Repeated dilutions and succussions yield higher potencies releasing more energy in the process. It will be appreciated therefore that homoeopathy is a system of medicine which concerns itself with energy and not with material doses of a drug.

After a dilution of 3c has been reached which represents 1/1,000,000 all poisonous or harmful effects of any substance are lost and only the curative properties remain.

Selection of potencies

Once the simillimum or 'most likely' remedy has been selected, the question of which potency to use arises. As a general rule, in the author's experience, the higher potencies which are more energised than the lower should be employed in acute infections or conditions while the lower should be reserved for chronic conditions with or without pathological changes being present. It will be found occasionally that there are exceptions to this point of view and indeed many practitioners especially on the continent rely mostly on lower potencies for general use.

The potencies mentioned under each remedy in the text covering the various diseases are a guide only. Higher potencies than those mentioned will necessitate professional advice.

Care of remedies

The delicate nature of the remedies which is inherent in the preparation renders them subject to contamination by strong-smelling substances, e.g. camphor, disinfectants etc. and also by strong sunlight. It is essential therefore that they be kept away from such influences and stored in a cool dry place away from strong light. The use of amber glass bottles is helpful in this connection for storage of tablets.

Nosodes and oral vaccines

A nosode (from the Greek NOSOS meaning disease) is a disease product obtained from any part of the system during illness and thereafter potentised e.g. cat flu nosode prepared from respiratory secretions of affected cats. In specific, i.e. bacterial, viral and protozoal disease the causative organism may or may not be present in the material and the efficacy of the nosode in no way depends on the organism being present. The response of the tissues to invasion by bacteria or viruses results in the formation of substances which are in effect the basis of the nosode.

An oral vaccine is prepared from the actual organism which causes a disease and may derive from filtrates containing only the exotoxins of the bacteria or from emulsions containing both bacteria and their toxins. These filtrates and emulsions are then potentised and become oral vaccines.

Nosodes and oral vaccines can be used therapeutically or prophylactically.

When we employ nosodes therapeutically we may use them for the condition from which the nosode was derived e.g. cat flu nosode in the treatment of a viral rhinotracheitis. This may be termed isopathic, i.e. treatment with a substance taken from an animal suffering from the same disease; or we may employ the nosode in any condition, the symptoms of which resemble the symptom-complex of the particular nosode e.g. the use of the nosode *Psorinum* in the treatment of the particular form of skin disease which appears in the provings of that nosode. This method may be termed homoeopathic i.e. treatment with a substance taken from an animal suffering from a similar disease. In this connection it must be remembered that many nosodes have been proved in their own right, i.e. each has its own particular drug picture. Many veterinary nosodes have been developed but no provings exist for them and they are used almost entirely in the treatment of prevention of the associated diseases.

Autonosodes. This particular type of nosode is prepared from material provided by the patient alone, e.g. pus from a chronic sinus or fistula and after potentisation used for the treatment of the same patient. Many examples of this could be quoted, but I think it is sufficient to explain the theory. Autonosodes are usually employed in refractory cases where well indicated remedies have failed to produce the desired response and frequently they produce striking results.

Oral Vaccines. As with nosodes, oral vaccines may be used both therapeutically and prophylactically. If the condition is caused wholly by bacterial or viral invasion, the use of the oral vaccine is frequently attended by spectacular success, but this is less likely when there is an underlying chronic condition complicating an acute infection. Here we may need the help of constitutional and other remedies.

Bowel Nosodes. The bowel nosodes are usually included under the heading of oral vaccines as the potentised vaccines are prepared from cultures of the organisms themselves.

Homoeopathic mastitis control on a herd basis

When advising a farmer in this connection, we employ the various potentised agents in drinking water. These agents are termed mixed mastitis nosodes and include the common bacterial pathogens associated with mastitis e.g. *Streptococcus*, *Staphylococcus*, *E. coli*, *Pasteurella* and *Corynebacteria*. A suspension of these nosodes is dissolved in 500 ml sterile water and 5-10 ml added to the drinking water twice per week for 4 weeks. This can be reduced to once per week after another month. This regime is variable according to the history of mastitis in any particular herd. In practice it may be that a reduced dosage would suffice.

A variation on this approach is to employ certain remedies which have a selective action on the physiology of the mammary gland. These can be employed in the same way as the mastitis nosodes and in the more chronic cases could be alternated with them.

In-calf heifers which are at risk from summer mastitis should be treated separately with *C. pyogenes* nosode in 3c potency starting at the beginning of May. Twice monthly treatment through the summer should help to reduce the incidence of this particular infection.

SOME ALTERNATIVES TO ANTIBIOTICS

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As the title suggests, my remit is limited and I have been asked to confine myself to treatments which are applied to the outside of the udder.

In preparing this paper, I wrote to 12 organisations asking for information on likely materials, only 2 firms replied. However, because of the nature of the subject I may have missed someone and I apologise in advance for any product that I have not included.

Introduction

To encourage the production of milk of high bacteriological quality the MMB introduced bonus payments for milk with a low TBC. From October 1st 1991 an additional bonus will be available for milk with a low somatic cell count. A major factor influencing the level of both the TBC and cell count of milk is the presence or absence of mastitis. If milk from an infected cow finds its way into the bulk tank it will not only lower the bacteriological quality of the whole, but will also contravene the MMB's terms and conditions of sale.

Current research is exploring new approaches to the control and treatment of mastitis such as vaccines and genetic engineering. It will, however, be some time before the results of this research are translated into everyday use. In recent years the main method of treating mastitis has been by the use of antibiotics and for many farmers this will continue to be their first choice.

For some time there have been standards and controls on the presence of antibiotics in milk. These have become increasingly more strict in both the level of antibiotic allowed and in the penalties on those who infringe the standards. In addition, greater public interest and awareness of health matters, together with the increase in production of organic milk and milk products, have all combined to make the production of an effective non-antibiotic treatment for mastitis most desirable.

Alternatives

There are several options currently in use. These include:-

- Cold water treatment
- Uddermint (sold without any medicinal claims)
- Golden Udder
- Homoeopathy - dealt with by another speaker

Cold water treatment

This is a practice which is now used by some organic milk producers and like most treatments, works best when the infection is detected at an early stage.

After milking, the infected quarter is hand stripped then hosed with cold water at approximately 2-3 bar (equivalent to good mains pressure) for several minutes. The aim being for the cold water to massage the udder and stimulate the flow of blood to the site of infection. The udder is then dried and the diseased quarter again hand stripped.

This procedure is repeated after each milking until the milk returns to normal.

With more severe infections the treatment should be carried out much more frequently.

Uddermint (Manufactured by Teisen Products Ltd)

This material is sold without any medicinal claims and therefore does not need or have a product licence. Its main ingredient is Cai-Pan Japanese peppermint oil. When applied to the skin the oil has a deep warming effect and has been used as a treatment for arthritis and rheumatism. Pure Cai-Pan peppermint oil has been used in Denmark and Poland to treat cows with mastitis. In both these countries 10-20 drops of the pure oil were massaged into the udder 2 or 3 times a day for several days.

Uddermint was launched in the UK in 1987 and is, to quote the manufacturers, "a natural liniment cream with 35% pure Cai-Pan peppermint oil as its main active ingredient". The cream is massaged into the udder (when required) after milking and is normally applied twice a day.

Information provided by the manufacturers gives details of a short study of mastitis control carried out by a student at Walford Agricultural College near Shrewsbury.

The study was carried out between October 1987 and February 1988. During this time there were 34 cases of mastitis, 10 of which were treated with Uddermint. Of the 10, only one failed to respond. It was felt that the response was due to the cream stimulating blood flow to the udder.

With mild cases the cream was rubbed into the quarter for approximately one minute after each milking for 2-3 days and up to 4 times a day for more severe cases. The report does not indicate how the cows were allocated to the Uddermint treatment or how a cure was measured.

An article in the Dairy Farmer dated September 1987 reports the comments of several farmers who were using Uddermint. The farmers interviewed all felt that Uddermint had helped in the treatment of mastitic cows. Other farmers interviewed by the manufacturers made similar comments, but not all were prepared to use Uddermint exclusively.

The same article also included a report from a study in Poland where Cai-Pan peppermint oil was used for treating mastitis in 5 herds. This report indicated a cure rate of nearly 60% for the Cai-Pan oil.

Golden Udder (Marketed by Shep-Fair Products Ltd)

Introduction

This product is an aqueous gel of plant origin containing sulphur BP 10% W/W and salicylic acid BP 1.5% W/W as active ingredients giving it anti-microbial properties. Golden Udder was licensed as a mastitis treatment when licensing was first introduced. It is applied to the outside of the affected quarter and rubbed in for approximately 1 minute after each milking for a minimum of 3 days. Treatment can be extended if the cure is not complete.

Initial studies

These studies were carried out in the laboratory where Golden Udder was checked for and found to have bactericidal properties. It was then checked to see if it would kill mastitis pathogens in milk. The results were positive, but the speed with which organisms were killed depended on the dilution used and the type of organism. The final part of the study examined whether there were any inhibitory effects after treatment had ended. No inhibition was found when tested on cheese and yogurt starter cultures, confirming the manufacturers claim that there was no need to withhold milk after the end of treatment.

Clinical trials

Six dairy herds in West Wales were involved. The farms chosen all had a significant level of mastitis in the previous year, were willing to cooperate and agreed to keep the necessary mastitis record. All farms were visited at the start of the trial to demonstrate how to use Golden Udder, to explain how to keep the necessary records and how to take and handle the milk samples. All samples were sent to ADAS at Trawsgoed and examined in the microbiology laboratory. A special mastitis recording chart was given to each farm to record treatment details and any symptoms seen. The symptoms were divided into 3 severity groups:-

1. Changes in the milk, but without palpable changes to the udder
2. Changes in the milk, together with hardness or swelling in the udder
3. As for "2" above coupled with the cow being ill

On each farm alternate cases were treated with Golden Udder or the antibiotic normally used by the farmer on the advice of his vet. If a cow failed to respond to either treatment, the farmer was free to change to whatever form of treatment he considered suitable.

Results

In all, 159 clinical quarters were treated, 86 with golden udder and 73 with intra-mammary antibiotics. The difference between the two treatments was due to the correct course of treatment not being given, or to more than one quarter being affected at the same time.

Most mastitis occurred, as to be expected, during the winter housing period (111) compared with during the period when the cows were at grass (48). As had been noted elsewhere, clinical mastitis was recorded more frequently in the rear quarters (99) than in the front quarters (59).

Details of clinical cases are given in Table 1.

Table 1 Response of clinical symptoms to treatment

Outcome	Golden Udder		Intra-mammary Antibiotic	
	Cases	%	Cases	%
Cured	70	81	62	85
Not cured	16	19	11	15
Total	86	100	73	100

These results indicate that although there were more cases treated with Golden Udder than antibiotic, the proportion for which clinical symptoms were cured was similar for both treatments. There was no statistical difference between treatments.

Details of cases according to the farmers observation of symptoms are given in Table 2.

Table 2 The effect of severity of symptoms on the response of those symptoms to treatment

Type of symptoms	Golden Udder		Intra-mammary Antibiotic	
	Cases	%	Cases	%
Clots in milk				
Cured	36	77	19	86
Not cured	11	25	3	14
Clots in milk and swelling				
Cured	29	85	41	89
Not cured	5	15	5	11
Clots, swelling and cow ill				
Cured	2	100	2	40
Not cured	0		3	60

In cases where there were clots in the milk only, treatment with antibiotics was rather more effective than with Golden Udder, but this difference was nowhere near statistically significant. Where there were clots and swelling or hardness of the udder, cure rates were almost identical. Only 7 cows were recorded as being ill with mastitis and of the 2 treated with Golden Udder, both responded successfully. Golden Udder therefore proved equally effective to antibiotics in the treatment of each severity group.

Milk samples taken from mastitic cows prior to treatment were examined bacteriologically on 120 occasions. The main organisms found in the samples that were bacteriologically positive were *Staphylococcus aureus* and *Streptococcus uberis*. The results are summarised in Table 3.

Table 3 The effect of treatment on the outcome of infections - bacteriological results

Bacteriological result-post treatment	Golden Udder		Intra-mammary Antibiotics	
	Cases	%	Cases	%
Negative	25	50	23	56
Positive	25	50	18	44
Total	50	100	41	100

Negative bacteriological results were obtained in 50% of the cases treated with Golden Udder and 56% of those treated with antibiotic. The difference between the treatments was very small and not statistically significant.

The quantity of milk discarded varied considerably due to the variation between the yields of different animals and to the stage of lactation at which the infection occurred. The average difference between the 2 treatments was 30 litres which represented a saving of 29% in milk discarded when using the Golden Udder treatment. Many more cases would be needed to verify the size difference between treatments.

Conclusions

On the evidence of this trial, Golden Udder gave results which did not differ significantly from those obtained using intra-mammary antibiotics. It appears to offer a valid alternative in the treatment of clinical mastitis.

Practical advantages and disadvantages of alternative therapies

Advantages

1. No need to withhold milk after treatment has finished
2. Reduced risk of failing inhibitory substances test
3. Being able to treat suspect cases without commitment to a long period of withholding milk

Disadvantages

1. It takes longer to apply the treatment
2. Less likelihood of milk samples being tested for causative organisms
3. A possible reduction in veterinary involvement in udder health

To sum up, there are alternatives to antibiotics on the market which offer certain advantages. It is up to the individual to decide if and how they would fit in with their farming system.

IS TREATMENT NECESSARY?

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Why do we treat clinical mastitis? A simple answer is because we believe that in doing so it will result in an overall benefit to the cow or herd (i.e. health, welfare gain) and/or benefit to the dairy farmer (limit financial loss). For the majority of mild clinical cases, detected and treated by the herdsman, the objectives of therapy are to resolve clinical signs to rapidly repermit milk sales, to limit udder damage and to prevent spread of infection.

On recognition of a clinical case it is the herdsman who initially must decide whether to treat, when to treat, with what and whether to seek veterinary advice. The outcome of therapy of clinical mastitis results from a complex interaction of microbiology, immunology, pathology and pharmacology. An informed decision would require knowledge of the mastitis agent involved, its susceptibility to antibiotic and the history of infection. The likelihood of spontaneous recovery must then be weighed against the prospects of successful therapy and the additional costs incurred and benefits arising. It is unlikely that this information will be at the herdsman's disposal at the time he makes his decision!

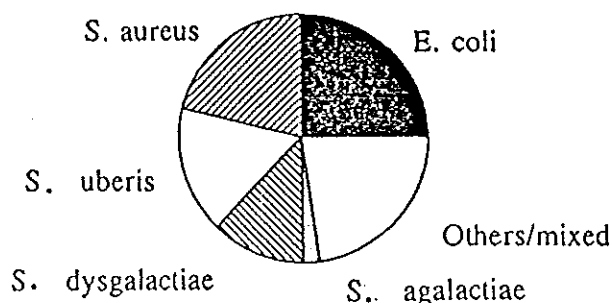
All herdsman will however be aware that once started, antibiotic therapy necessitates that milk be discarded for a recommended withholding time and that failure to comply runs the risk of severe financial penalties.

In this paper, factors influencing 'the treatment decision' are explored and future developments for lactational therapy in mastitis control are considered.

Mastitis aetiology

Mastitis commonly results from infection caused by any of a variety of micro-organisms. The relative incidence of pathogens isolated from clinical mastitis in a cohort of British dairy herds over three years (1) is summarized in Figure 1. The presenting clinical signs of these different infections are often similar, and the causative organism can only be reliably identified by microbiological examination of the milk. Thus, it is usual to commence therapy with a broad-spectrum antibiotic or combination when clinical signs are first noticed. Alternatively, antibiotic preparations which are only effective against gram-positive organisms may be used (since many gram-negative infections will self-cure - see below).

FIGURE 1 - Pathogens from clinical mastitis cases
in 300 British dairy herds, 1980-1982



Efficacy of treatment and non treatment

There is an extensive literature on antibiotic therapy of clinical mastitis during lactation, including information on efficacy of different products and treatment regimens. Comparison of results is questionable because of numerous variables in different experiments including aetiology, duration of infection, animal variation and herd management. Also, the criteria used in assessing the clinical and, particularly, the bacteriological responses vary considerably. In an attempt to evaluate the reported efficacy of various antibiotic treatment, Le Louedec (2) reviewed the literature and calculated average cure rates for each antibiotic involved. Reports were selected according to rigorous criteria. Findings for single antibiotics and penicillin-streptomycin combinations, are summarized in Table 1. It is not possible to draw firm conclusions about the relative effectiveness of different products. However, the data consistently show a greater bacteriological cure rate in the treatment of *Streptococcus agalactiae* infections than those due to *Staphylococcus aureus*.

Table 1 Efficacy of treatment with different antibiotics

Antibiotic	For mastitis caused by:					
	<i>S. aureus</i>			<i>S. agalactiae</i>		
	Mean % cure	Range	(No. of reports)	Mean % cure	Range	(No. of reports)
Penicillin	32	0-87	(12)	84	50-100	(11)
Cloxacillin	41	21-84	(14)	92	40-100	(8)
Neomycin	27	25-36	(2)	27	27	(1)
Tetracycline	54	17-96	(8)			
Chloramphenicol	33	20-48	(7)	75	61-84	(2)
Erythromycin	63	51-76	(2)			
Spiramycin	70	45-82	(2)			
Rifamycin-SV	66	65-66	(2)	75	75	(1)
Penicillin + Streptomycin	39	21-78	(5)	91	91	(1)

Table shows average bacteriological cure rates reported for treatment of mastitis during lactation (from (2)).

The efficacy of treatment of infections due to different pathogens is further illustrated in Table 2. Of 270 cases of mastitis in the USA given undefined antibiotic treatments, all showed clinical improvement but only about a third of infections were eliminated (3). In a larger British study, intramammary cloxacillin produced a bacteriological cure in 61% of treated quarters (4).

Table 2 Efficacy of treatment of mastitis caused by different pathogens

Pathogen	No. quarters treated	Treatment	% cure	
			Clinical	Bacteriological
<i>S. aureus</i>	121	ND ¹	100	24.8
<i>S. agalactiae</i>	31	ND	100	51.6
Other Streps.	111	ND	100	36.0
Coliforms	7	ND	100	71.4
All	270	ND	100	33.7
			from (3)	
<i>S. aureus</i>	1141	Clox ²	ND	26
<i>S. agalactiae</i>	762	Clox	ND	85
<i>S. dysgalactiae</i>	75	Clox	ND	90
<i>S. uberis</i>	797	Clox	ND	77
Coliforms	218	Clox ³	ND	90
All	2993	Clox	ND	61
			from (4)	

¹ Undefined antibiotic(s)

² Sodium cloxacillin 0.2g, 3 x 24 h

³ Coliforms insensitive to cloxacillin, therefore equivalent to no treatment

ND = Not disclosed

In infections caused by certain pathogens, bacteriological self-cure following a clinical episode may contribute substantially to the apparent efficacy of the treatment given. The high cure rate (90%) of coliform infections in quarters treated with cloxacillin (Table 2) is significant in this respect since coliforms are insensitive to this antibiotic. This apparent cure rate may reflect the high spontaneous recovery rate which occurs in coliform infections once the udder defence mechanisms are stimulated.

The effect of not treating mild clinical mastitis was examined in a single herd by Chamings (5). A spontaneous clinical cure occurred in 87% of cases (mostly *S. aureus* infections) with a 20% bacteriological cure apparent under the assessment criteria used. The average duration of clinical signs in untreated mastitis cases was 3.2 days (Table 3). In the more severe cases that received antibiotic therapy, bacteriological cure rates were only slightly improved (28%) over untreated mild cases. However, the rate of spontaneous elimination in more severe cases may be low.

Table 3 Efficacy of non-treatment

Pathogen	No. quarters	Treatment	% Cure	
			Clinical	Bacteriological
<i>S. aureus</i> (mild cases)	56	No	ND	20
<i>S. uberis</i>	16	No	ND	19
All (includes other pathogens)	83	No	87 ¹	ND
<i>S. aureus</i> (more severe cases)	53	Yes	ND	28

Summarized from (5)

¹ Average time for clinical cure with no treatment = 3.2 days

ND = not disclosed

Only about 30-40% of new infections arising during a lactation develop clinical signs. Spontaneous recoveries of infections which remain subclinical may equal the number of infections eliminated by antibiotic therapy. However, such self-cures often follow long periods of infection during which milk yield will have been suppressed and irreversible pathological changes may have resulted.

Taking the incidence of various pathogens as causal agents of clinical mastitis in England and Wales (1), the overall bacteriological self-cure rate that might result in the absence of antibiotic treatment has been estimated (Table 4).

Table 4 Rate of spontaneous elimination of infection that may be expected to occur if clinical mastitis is not treated

Pathogens initially isolated	Percentage of clinical mastitis cases ¹ (A)	Expected bacteriological self-cure rate (B)	Overall percentage of clinical cases that should bacteriologically self-cure (A X B)
<i>E. coli</i>	20.2	70% ²	14.1)
All other pathogens	63.1	10% ³	6.3) 37.1
None	16.7	100%	16.7)

¹ From Figure 1 and (1)

² From (6)

³ Will vary according to pathogen, conservative estimate

Economics of therapy during lactation

It is a simple matter to estimate the cost of treatment of clinical mastitis: the value of discarded milk, antibiotic costs and veterinary fees. It is less easy to estimate the financial benefit which results or rather, the loss that would be incurred if treatment was not given during lactation. For cases of severe peracute mastitis, therapy may be aimed at reducing fatalities and thereby salvaging the carcass-value of the cow rather than restoring full production. However, for the majority of mild clinical cases, restoration of normal milk production by elimination of infection is the goal. The financial losses due to persisting infection will include decreases in milk yield and quality, increases in the level of herd infection and an increased culling rate of chronically affected animals.

The predominant source of loss due to mastitis is the depression of yield associated with continuing subclinical infection. Estimates of the loss vary from 9% to 45% per infected quarter.

For illustration, let us assume that a cow has mild clinical mastitis which results in a depression of quarter yield by 40% and total yield by 10%. Let us also assume that this yield loss may be fully restored on elimination of infection and that treatment is given using 3 intramammary infusions at 12 h intervals followed by a 72 h discard time i.e. total treatment + withholding time = 4 days.

Since clinical cure rates for mild mastitis appear similar (>80%) with or without treatment and clinical signs persisted on average 3.2 days in untreated quarters (Table 3), in our example, additional milk loss due to treatment, $4 - 3.2 = 0.8$ day's milk.

Thus if treatment is successful and yield loss (10%) is restored this milk discarded would be recouped in 8 days.

The improvement of bacteriological cure rate due to therapy (i.e. after correcting for spontaneous cure), given a typical pattern of pathogens may be calculated from Tables 2 and 4:

Net bacteriological cure rate due to therapy	=	Apparent antibiotic cure rate	-	Spontaneous recovery rate
	=	61%	-	37%
	=	24%		

Thus in our example with a 24% improvement in bacteriological cure rate truly due to treatment, milk discarded would be recouped not in 8 days but in 32 days.

The purpose of this example is simply to illustrate the economic importance of short duration lactating cow treatments with minimal milk discard time. No account was taken of the cost of treatment itself and the assumption was made that yield losses could be fully and rapidly restored on successful elimination of infection - which is questionable.

However, this highly visible cost of treatment in terms of milk discarded should not be allowed to obscure the other less obvious and longer term benefits of antibiotic treatment.

Milk quality considerations

The predominant influence on bulk milk somatic cell count (SCC) is the level of subclinical infection within a herd. Appropriate treatment of clinical cases will have a positive influence in reducing duration of infections and decreasing lateral spread but will be limited since only about one third of infections appear with clinical signs. Dry cow therapy combined with appropriate hygiene measures will have a greater impact. Nevertheless it is notable that the incidence of clinical mastitis due to *S. aureus* increased following a trial cessation of antibiotic treatment during lactation (5).

SCC has direct financial implications. EEC Directive 85/397 sets an upper limit of 400,000 cells per ml for milk for intracommunity trade. In implementing step 2 of this directive, quality payments based on SCC levels are currently being introduced into national schemes. In England and Wales from October milk with an SCC of less than 400,000 per ml earns an extra 0.2p/litre (or £1100 per year for a 100 cow herd). Further proposals under consideration would extend the principles of this directive to all community trade in milk - which could ultimately result in milk with cell counts in excess of 400,000 being classified as unmarketable.

Total bacterial counts (TBC) may be adversely affected by high bacterial numbers in milk from infected quarters. Sporadic high values are unlikely to affect TBC quality bands but there is a genuine risk of producers losing out on the premium as a result of using non-antibiotic preparations to treat clinical mastitis and thereby allowing large numbers of bacteria to regularly enter the bulk tank (7). Recent U.K. MAFF microbiological sampling returns show an increase in the incidence of *S. agalactiae* mastitis which may be indicative of inappropriate therapy and a reduction in the use of dry cow therapy (7).

Other adverse changes in milk composition as a result of mastitis (e.g. decreases in fat and lactose and increases in non-casein proteins and sodium) are also well recognized. These may be fully reversed on successful elimination of infection.

There is no doubt that these various aspects of milk quality will have a growing significance on the future profitability of dairy farms in the EC and, indeed, on the saleability of milk produced. Any farmer who may be tempted by apparent short term gains arising from non-antibiotic alternative treatments and reductions in the use of dry cow therapy should consider that he may well be storing up problems for the future.

Future trends in mastitis therapy

The goals of treatment during lactation are to attain the maximum clinical and bacteriological cure consistent with a minimum treatment and milk discard time and with a rapid return to normal production of good quality saleable milk. Consequently, in response to some or all of these aims, developments are occurring in a number of areas:

a. Products with improved efficacy and reduced withdrawal time.

- New antibiotics
- Microbicidal proteins - which are specific, rapidly bactericidal and have no oral toxicity e.g. lysostaphin, Nisin (8)
- Cytokine immunomodulators (e.g. interleukins, interferon) which may enhance natural defence mechanisms and/or augment conventional therapy

b. Products with questionable efficacy but zero withdrawal time

- Homoeopathy
- Ointments and "stripping out"

c. Vaccination

Our understanding of mechanisms of immunity in the udder and of bacterial virulence is progressing and holds out hope for future mastitis vaccine development. To be of commercial value a vaccine will need to be multivalent. The potential for reduction in average duration of intramammary infection via vaccination approaches exceeds that of improvements in therapy, since all infections, not just clinical cases will be affected. Furthermore, any increase in self-cure rates will also lead to an apparent increase in antibiotic efficacy in vaccinated herds.

Conclusion - is treatment necessary?

On animal welfare grounds alone all cases of clinical mastitis should receive appropriate therapy. Notwithstanding these welfare considerations, a review of data on the effectiveness of antibiotic therapy of mastitis leads to the conclusion that improvements in cure rates are unlikely to economically justify any increase in the duration of lactational therapy. Nevertheless, high spontaneous clinical recovery rates in the absence of therapy and the often limited success of antibiotic treatment in effecting bacteriological cures should not be interpreted as a reason for abandoning treatment of mild clinical cases. Reduction in bacterial numbers in infected quarters as a result of antibiotic treatment helps to reduce spread of infection and to improve the bacteriological quality of bulk milk, both of which are instrumental in maintaining premium milk quality.

Short duration therapy with appropriate antibiotic preparations thus remains the recommended approach for treating clinical cases during lactation, with the proviso that all cows continue to receive dry cow therapy.

Is treatment necessary? The short answer is "Yes" - in order to safeguard the health and welfare of the dairy cow and the dairy enterprise.

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TEAT DIPPING BEFORE MILKING - UK RESULTS

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Introduction

The adoption of post-milking teat disinfection by the majority of farmers in the UK has resulted in a significant fall in the incidence of mastitis caused by those pathogens known to be transmitted at or shortly after milking (1). A similar reduction in the incidence of mastitis caused by environmental pathogens has not, however, occurred.

In the USA, an increasing number of dairy herds have adopted the practice of pre-milking teat disinfection to combat environmental mastitis. Studies on which these recommendations have been based were reviewed by Martin Shearn at the last British Mastitis Conference (2).

In 1983, McKinnon *et al* (3) showed that, during the winter months, washing and drying of teats prior to milking resulted in a significant reduction in bacterial contamination. Previous work with post-milking disinfection (4,5) had shown that a reduction in bacterial contamination of teats is associated with a reduction in new intramammary infections. Good udder preparation might, therefore, be expected to result in fewer intramammary infections.

More recently, Pankey *et al* (6) have performed a trial in the USA to investigate the use of pre-milking teat disinfectants to reduce the incidence of mastitis. Studies on four farms recorded a reduction in new intramammary infections of 40 to 50 per cent in groups practising pre-milking teat disinfection.

Based on this work, a number of products are now commercially available in America with recommendations for pre-milking teat disinfection. A recent survey (7) shows that in areas of the United States up to 57 per cent of dairy farmers have adopted pre-milking disinfection in their hygiene routine.

Consequently, it was decided to investigate the practice of pre-milking teat disinfection in UK herds, under normal management conditions.

The objectives of the study were (i) to determine whether pre-milking teat disinfection has a significant effect on environmental mastitis, (ii) to ascertain the effects of pre-milking teat disinfection on chemical residues in milk, and (iii) to determine whether the practice is an acceptable addition to the normal milking routine.

Results of a preliminary investigation 1989/90

During the winter housing period of 1989/90, a split herd study involving 310 Friesian cows from three commercial herds was performed in Southern England.

The disinfectant used for pre-milking teat disinfection treatment was a specially formulated iodophor, containing 0.25 per cent w/w available iodine. This concentration was chosen since it had been shown by Pankey and colleagues (6) to be effective in US trials.

The teats of animals in all herds were disinfected post-milking with Iosan Superdip (Ciba-Geigy plc), in order to minimise variables.

Herds were selected on the basis of the level of clinical mastitis during the previous winter period from a survey performed by the Milk Marketing Board. Willing co-operation of all milking staff and management was essential, as was a good standard of milking routine and the ability to collect individual jar samples. Cows were paired by lactation number, stage of lactation and known history of clinical mastitis, and then members of a pair randomly allocated to one or other treatment group. Treatment groups were identified with coloured tail tapes. Cows calving after the start of the trial were allocated to a treatment group in the same way. Within each herd, cows were housed, fed and milked together.

Udder preparation of cows allocated to the control group consisted of dry wiping with individual paper towels. Only grossly contaminated teats were washed with running water and then well dried with individual paper towels.

Animals allocated to the pre-milking teat disinfection group were prepared in the same way, after which their teats were dipped in the iodophor disinfectant. A contact time of at least 30 seconds was allowed, after which the teats were wiped with individual paper towels.

New clinical infections (defined as an abnormal quarter or changes in the milk) were recorded, sampled aseptically, and bacterial pathogens identified (8). Herd tests, in which all lactating cows were sampled aseptically at the beginning and the end of the study period, or when they joined the study at calving, were performed in order to identify new intramammary infections arising during the trial.

Teat condition was recorded prior to and at regular intervals throughout the trial period.

Unannounced farm visits were made throughout the trial in order to check that the experimental routine was being adhered to and that clinical cases were being recorded and sampled correctly.

Residue studies showed that iodine levels in milk were not significantly raised by the additional use of the pre-milking iodophor disinfectant.

Milking times were estimated to increase by ten to twenty minutes per 100 cow herd, which was considered by herdsmen to be acceptable.

Teat skin condition improved in both groups during the study period.

The resulting effect of pre-milking teat disinfection on the number of new infections is presented in Table 1, where data from all three farms have been combined. When the data is presented in this way, pre-milking teat disinfection would appear to reduce the incidence of new intramammary infections by 39 per cent.

Table 1 Preliminary investigation of the effect of pre-milking dipping on environmental mastitis

Group	No. quarters at risk	No. infected quarters	
		Clinical mastitis	Total new infections
Control	588	21	28
Pre-milking teat disinfectant	580	14	17

However, if the three farms are viewed separately, it can be seen that the effect between farms was very variable (Figures 1 & 2).

It was decided, therefore, that it would be necessary to collect data from a large number of farms in order to show whether a reduction in environmental mastitis could be attributed to pre-milking teat disinfection.

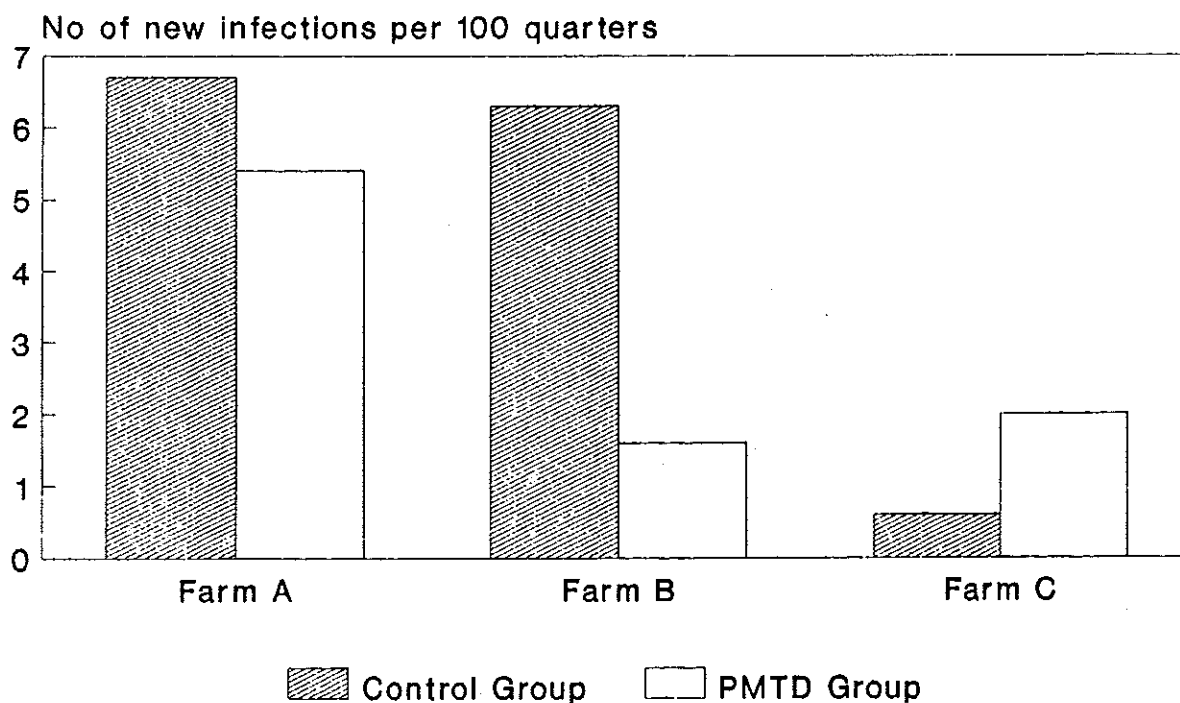


Fig. 1 Effect of pre-milking teat disinfection on incidence of environmental mastitis

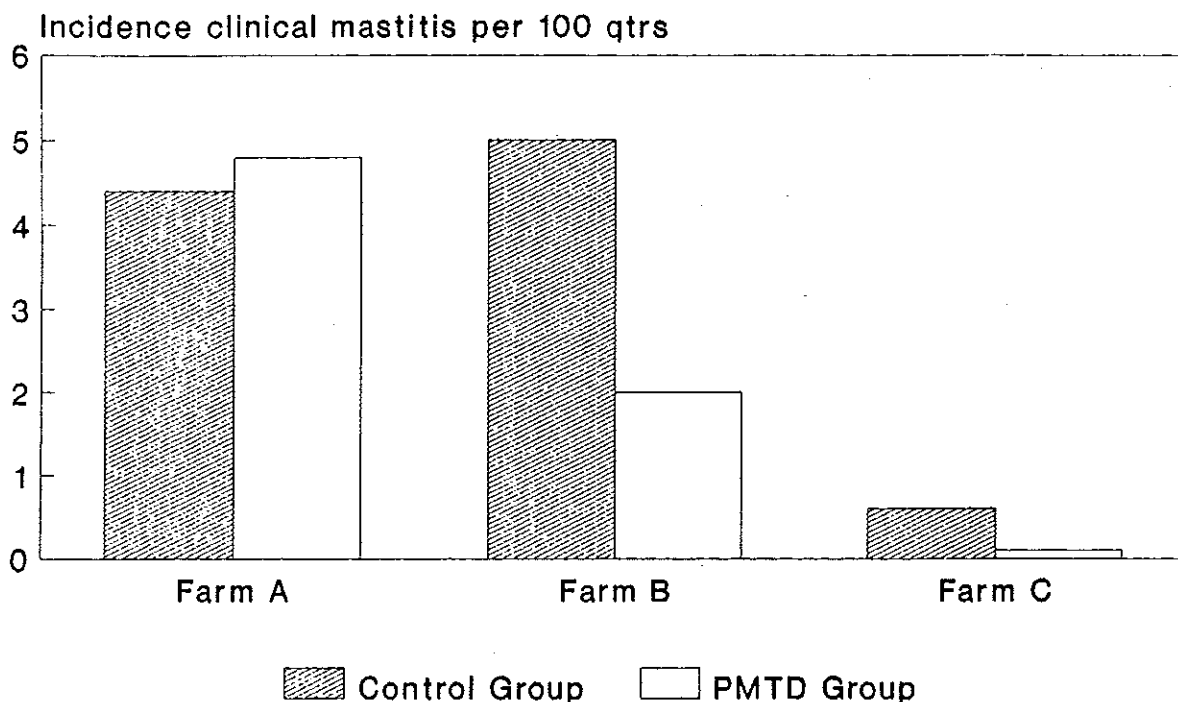


Fig. 2 Effect of pre-milking teat disinfection on incidence of environmental mastitis

Results of a large scale farm trial 1990/91

A paired-herd study was performed during the winter housing period of 1990/91. This design gave a true representation of pre-milking teat disinfection under commercial conditions, and allowed a large number of farms to be monitored. Ensuring compliance with a split herd design would not have been possible on so many farms.

Twenty-two herds were selected for the trial on the basis of their previous histories of environmental mastitis. These were paired on the following criteria (in descending order of priority):- incidence of clinical mastitis in the previous year; herd size; calving pattern; type of housing. Members of each pair were then allocated to either the control or the pre-milking teat disinfection group, dependent upon the herdsman's ability and willingness to adopt pre-milking teat disinfection into his usual routine. Udder preparation was as described in the preliminary study. Results for one pair of farms were excluded for non-compliance with the protocol, or poor recording of clinical cases. Efficacy was measured as a reduction in clinical cases in those herds practising pre-milking teat disinfection, when compared to their paired controls.

Figure 3 shows the pattern of infection recorded in the 18 herds during the 24 week trial period. As expected, the majority of clinical cases (approximately two-thirds) were identified as being caused by environmental pathogens. 12 per cent were due to "common" mastitis pathogens (ie. *Staphylococcus aureus*, and *Streptococcus dysgalactiae*), 3 per cent due to minor pathogens, and in 18 per cent of cases, no pathogens were isolated.

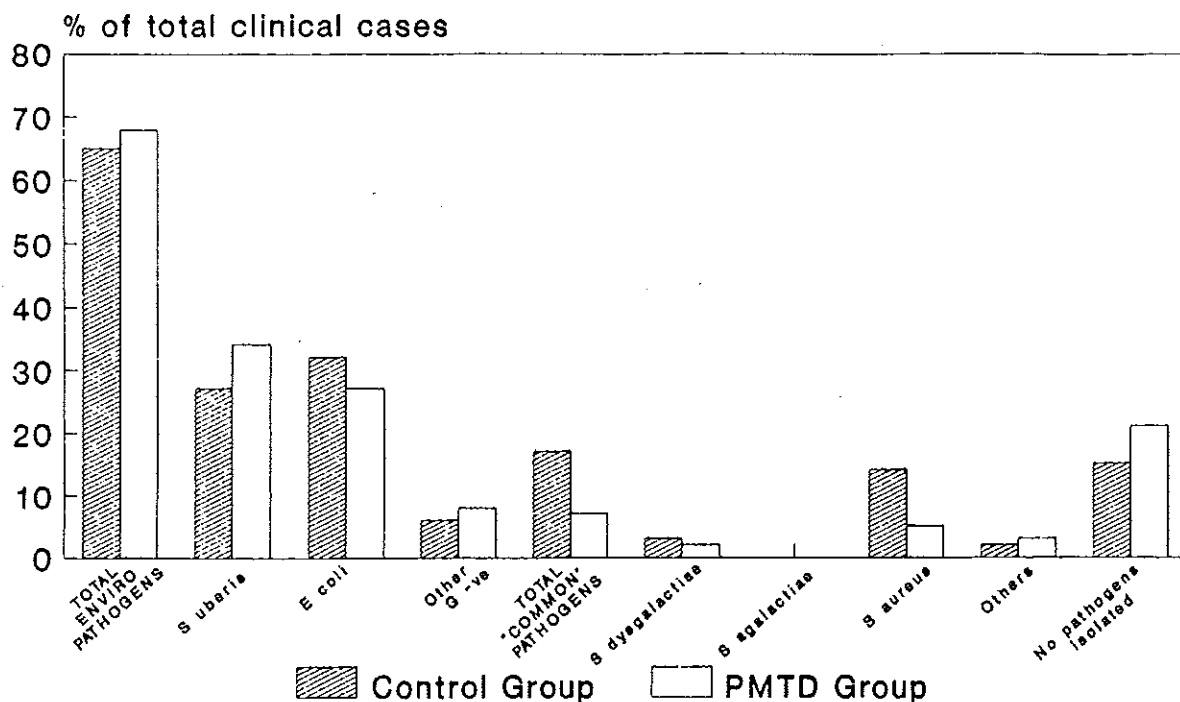


Fig. 3 Pattern of infection in trial herds

A comparison of environmental mastitis in the two groups is shown in Figure 4. It is clear from this pooled data that there is no advantage in pre-milking teat dipping, as the incidence of clinical cases was higher in the group in which it was practised. Variables between farms may have masked a beneficial effect (in both groups the standard deviation is approximately equal to the mean), so pairs of farms were then examined individually (Figure 5).

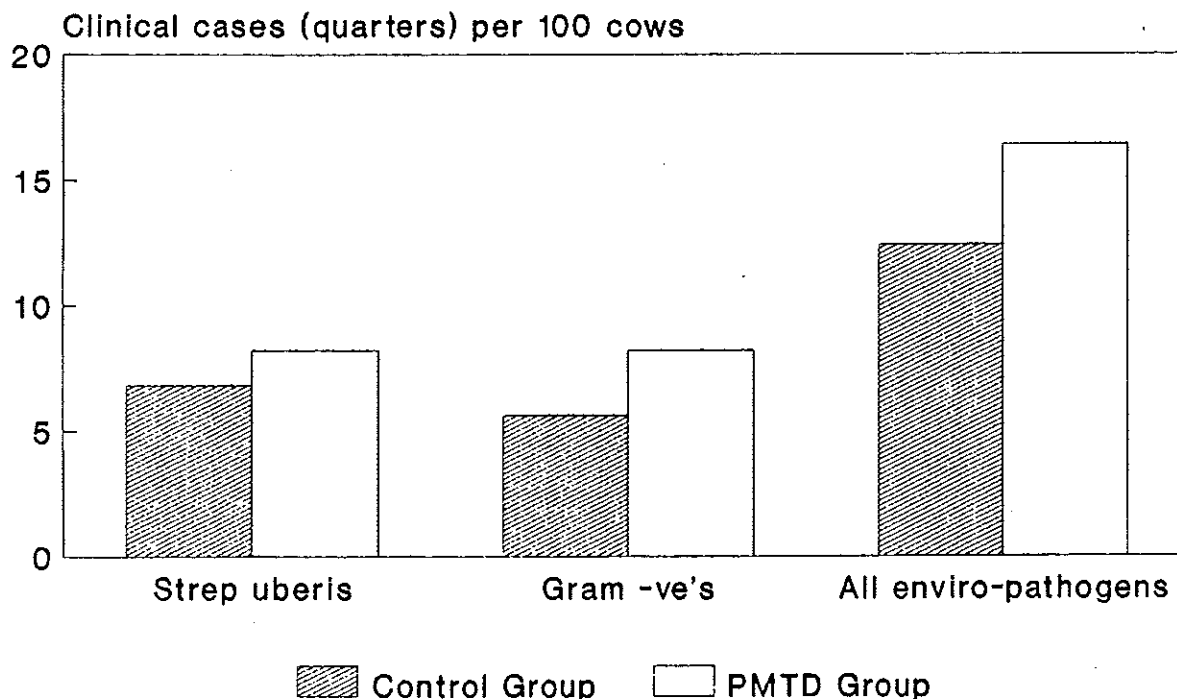


Fig. 4 Incidence of environmental mastitis in control and pre-milking teat disinfection groups (Nov-May 1990/91)

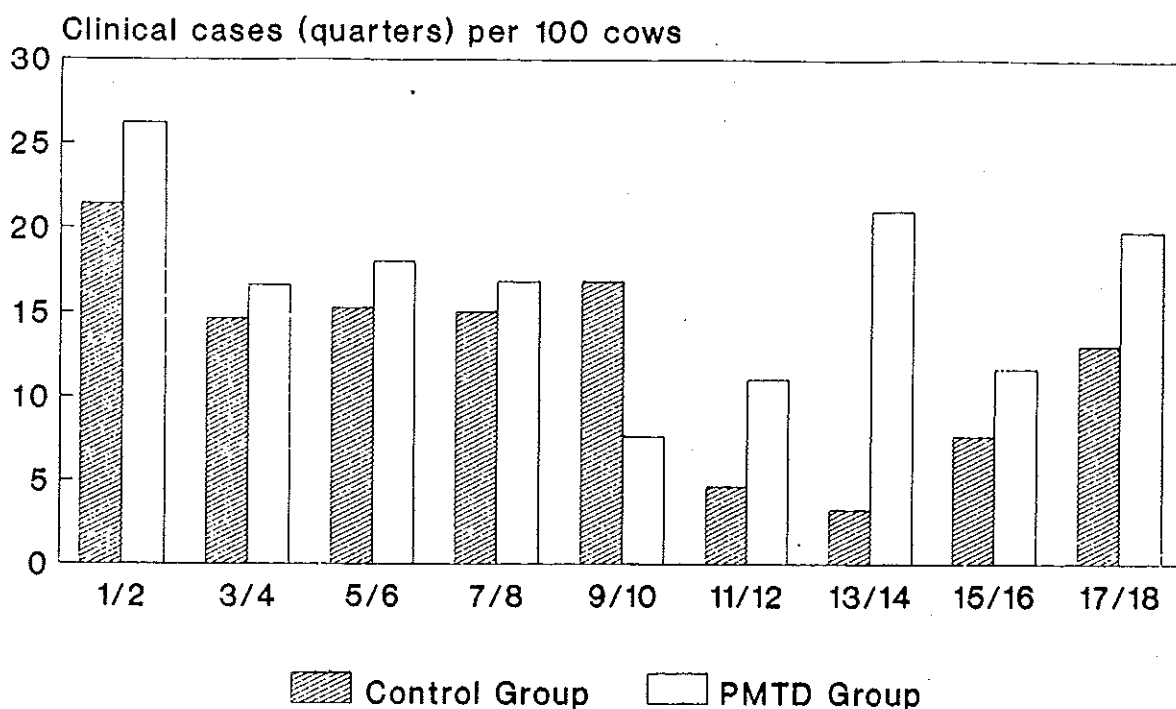


Fig. 5 Incidence of environmental mastitis in paired farms (Nov-May 1990/91)

In only one pair of herds did pre-milking teat disinfection have an apparently beneficial effect. In all others, more clinical cases were recorded in herds practising pre-milking teat disinfection than their paired controls.

It was further considered that a predominance of either *Streptococcus uberis* or *Escherichia coli* on certain farms may have masked a possible effect against one or other pathogen. Incidence of each organism was therefore compared within each pair (Figures 6 & 7). It can be seen again that there is no reduction in clinical cases over control herds.

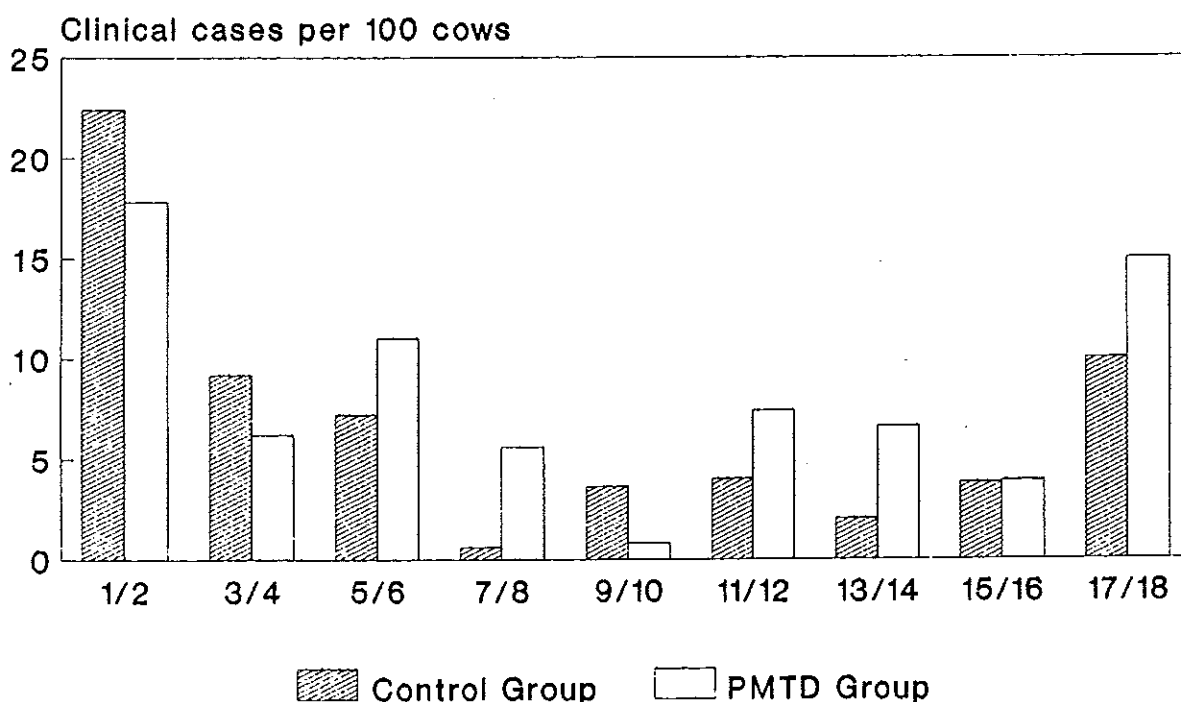


Fig. 6 Incidence of *S. uberis* infections in control and pre-milking teat disinfection groups (Nov-May 1990/91)

These results indicate either (i) that pre-milking teat disinfection does not reduce the incidence of environmental mastitis, or (ii) that the pairing of farms was inappropriate, introducing too many variables to be a reliable control.

In order to overcome the latter, the incidence of clinical mastitis in the two groups was compared with that in the previous year.

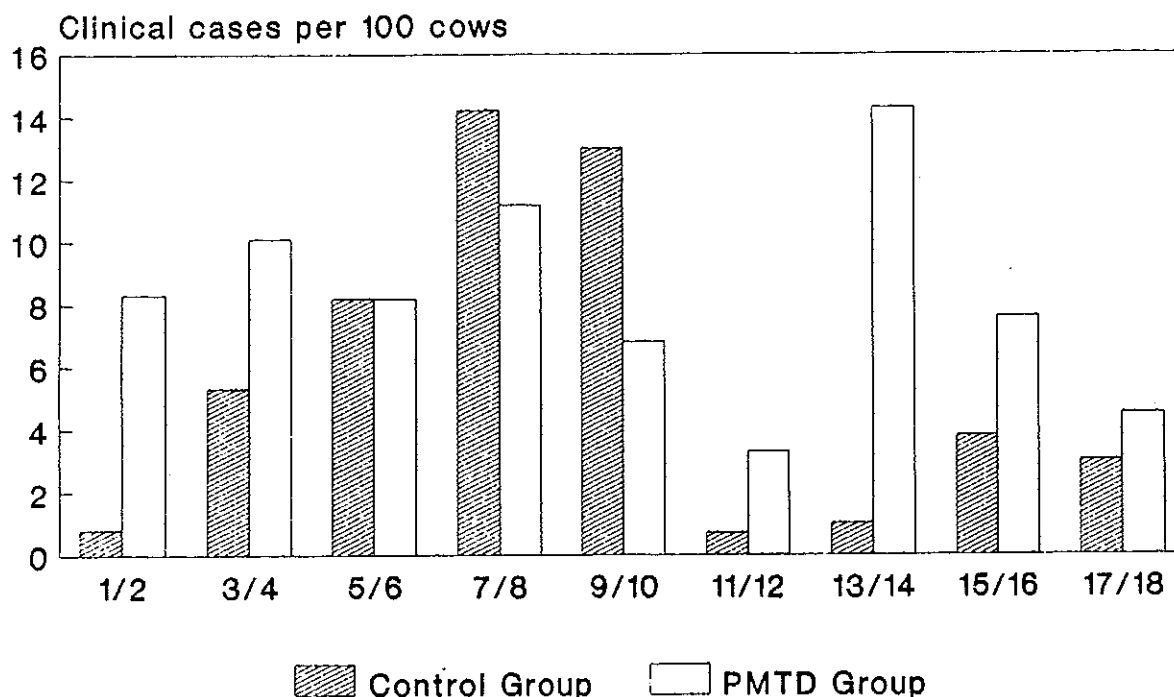


Fig. 7 Incidence of gram-ve infections in control and pre-milking teat disinfection groups (Nov-May 1990/91)

In the pre-milking teat disinfection group, five of the nine herds saw an apparent benefit from the introduction of pre-milking teat disinfection (Figure 8). In these herds, the reduction in clinical cases during the same 24 week period ranged from 11 to 45 per cent. If this data had been taken alone, it might be concluded that the majority of farms using pre-milking teat disinfection would benefit from a resulting reduction in clinical cases of mastitis.

However, if the same comparison is made in the control group (Figure 9), it can be seen that a greater number of herds saw a reduction in mastitis over the previous season than in the pre-milking teat disinfection group (seven of the nine). In this group, reductions in clinical cases ranged from 27 to 59 per cent.

The overall conclusion from these studies must therefore be that pre-milking teat disinfection does not result in a reduction in environmental mastitis. The reduction in clinical cases following the first season of use might be explained by either the herdsman's increased attention to detail when being monitored, or the change to a superior post-milking teat disinfectant, or to a lower incidence of environmental mastitis in the season of use. Whichever data set is taken as a control, a reduction in environmental mastitis as a result of pre-milking teat disinfection was not proven.

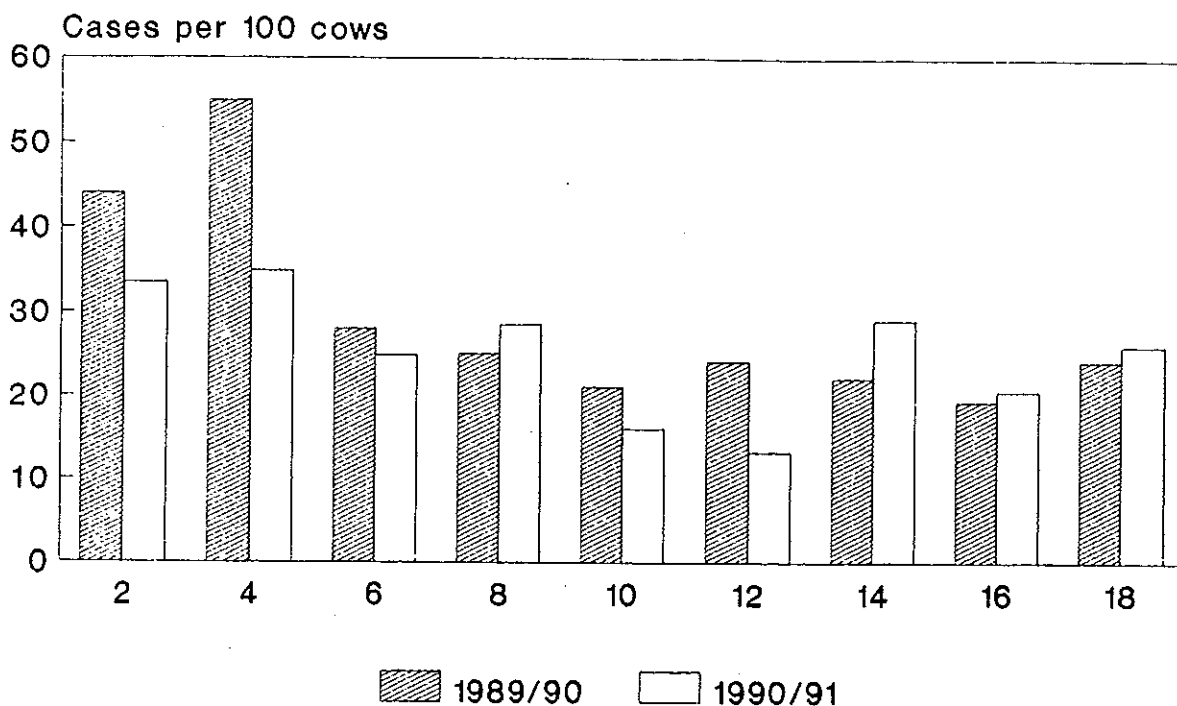


Fig. 8 Incidence of clinical mastitis (infected cows) for equivalent periods 1989/90 and 1990/91 - pre-milking teat disinfection herds

Suggested reasons for lack of effect

The question now arises, why was there no effect in UK herds, when studies in America seem to have reported major improvements?

Firstly, the published studies of the Cornell and Vermont University groups should be studied more closely. Galton and colleagues (9,10,11) conducted a series of studies in which methods of teat preparation were compared. All methods of preparation showed a significant reduction in bacterial counts compared to no preparation (9,10). Pre-milking teat disinfection was proven superior to wiping with a dry towel, but was not significantly better than washing with running water containing a disinfectant udder wash, followed by drying. In the last study (11), the relationship between method of preparation and incidence of new intramammary infections was investigated. A contact time of 15 to 20 seconds was allowed before wiping off the pre-milking dip with individual paper towels. Pre-milking dipping proved to be the best method of preparation, but clinical cases were too few to make a valid comparison between groups.

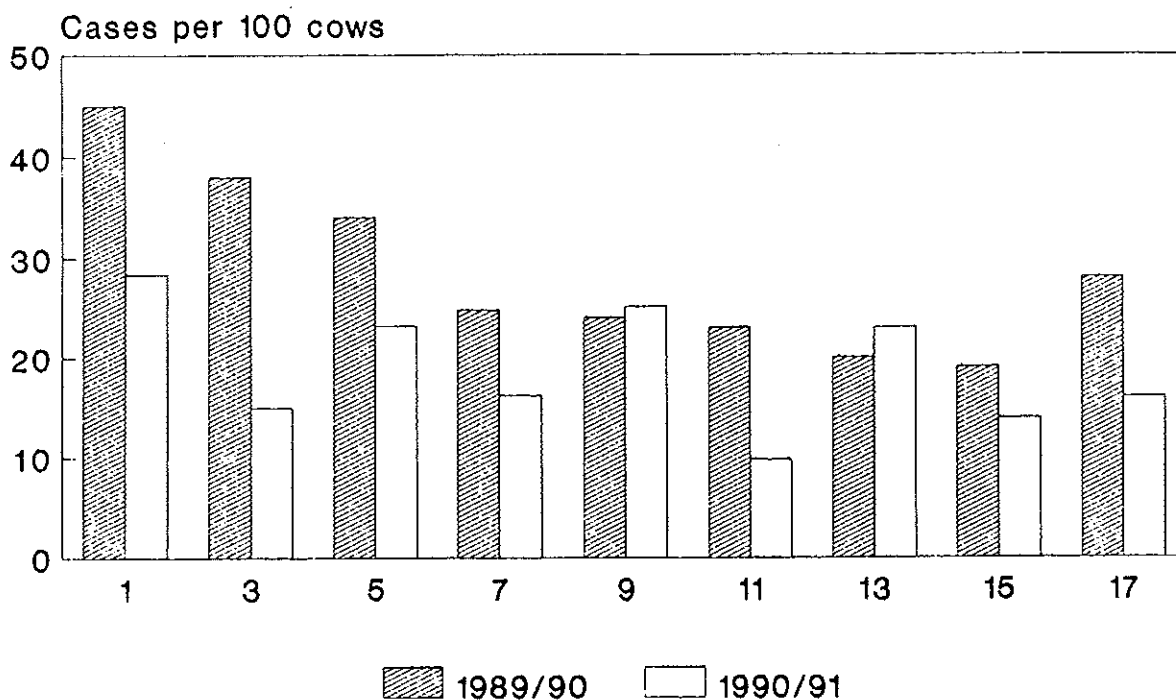


Fig. 9 Incidence of clinical mastitis (infected cows) for equivalent periods 1989/90 and 1990/91 - control herds

The study by Pankey and colleagues (6) went one step further, investigating pre-milking teat disinfection on commercial farms. Some 308 cows in four herds were involved, of which approximately half were prepared for milking by pre-milking teat disinfection. The incidence of new intramammary infections was reduced by approximately 50 per cent in pre-milking dipped cows. The incidence of clinical cases, however, was not reported in this paper, but a personal communication with the author has indicated that there was little, if any, difference between the two groups.

Additionally, a small study has recently been performed at the Institute for Animal Health (personal communication; Teverson, Shearn and Hillerton), in which teats of fifteen cows were dipped in a suspension of *E. coli* and *S. uberis* and allowed to dry. Each teat was dipped in one of four solutions (water, a commercial teat dip diluted to containing either 0.25 or 0.5 per cent av. iodine and the trial product), and after a contact time of thirty seconds, dried with individual paper towels, and swabbed to determine bacterial contamination. Pre-milking teat disinfection had no advantage over dipping with distilled water. This would appear to be due to an inadequate contact time, rather than an insufficient level of free iodine. The bacterial activity of commercial post-milking teat disinfectants containing 0.5 per cent available iodine has been proven in numerous studies, but when allowed a contact time of only thirty seconds, a similarly poor effect was achieved. If the contact times were to be significantly prolonged, however, the increased time in the herdsman's milking routine would become unacceptable.

The following conclusions have been drawn from these studies:

- i) Pre-milking teat disinfection is a good method of preparing teats prior to milking. However, with a limited contact time, the reduction in bacterial contamination is most likely to be a result of careful preparation rather than bacterial kill.

The attention to detail required in the routine is most likely to be the factor resulting in reduced challenge.

- ii) Pre-milking teat disinfection is reported to reduce the incidence of new intramammary infections, presumably as a result of a reduced bacterial challenge immediately prior to milking. It does not, however, reduce the incidence of clinical mastitis. This suggests that environmental mastitis is not influenced by the bacterial contamination of teats prior to milking, but that infection is more likely to occur during the period between milkings. This agrees with the work of Bramley *et al* (12), who found that although pre-milking disinfection led to a reduction in exposure to *E. coli* at milking, it did not result in a significant reduction in *E. coli* infections. They postulated that the combination of high levels of *E. coli* and fluctuating vacuum levels, which cause a high rate of impacts, would result in an increased incidence of environmental mastitis. Under these circumstances, pre-milking teat disinfection would be expected to reduce the incidence of clinical cases. If, however, the milking machine is well maintained, pre-milking teat disinfection might offer no advantage at all. The farms in our studies were selected on the basis of good husbandry and attention to detail, in order to remove as many variables as possible and ensure compliance with the protocol. This may explain why no positive effect has been recorded.

The commercial benefit expected from pre-milking teat disinfection is a reduction in clinical cases of mastitis. This was not seen in studies conducted on 18 farms, involving more than 2,000 cattle in the UK. Benefits may be achievable on individual farms, but success cannot be predicted or assured. Attention to other areas, such as good machine maintenance and cleanliness of housing, are likely to be of greater benefit than adopting pre-milking teat disinfection.

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THE EFFECTS OF MILKING FREQUENCY ON MASTITIS

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The convention in UK dairying is to milk cows twice daily from calving for 305 days. A few producers milk thrice daily for part or all of the lactation and at least one has been reported recently to have milked a herd four times daily. It is rare for cows to be milked less frequently and then usually as a means of reducing yield before drying off or for quota management.

Many variations in milking frequency, or different lengths of inter-milking interval, have been investigated experimentally or tried commercially over the last few decades. Usually these have been for socio-economics reasons or for production management. These motives appear to be of increasing importance and, with the possibility of automated milking systems being available at about the turn of the century, support the need to consider various strategies of milking frequency.

Considerable research has taken place into the production and economic consequences of a reduced frequency of milking, once daily or by omitting one or two milkings per week. Similarly there is renewed interest in more frequent milking, which may be important for the success of automated systems. Much less consideration has been given to the effects on mastitis and milk quality. The research that has taken place on increased milking frequency has usually measured the effects as changes in the California Mastitis Test or Wisconsin Mastitis Test score, with one exception (1). At Compton part of the programme involves investigating the effects of four times daily milking on milk cell count, the rate of new infections, the incidence of clinical mastitis and the response of sub-clinically infected quarters. There is a broad history of research on less frequent milking.

Aetiology of infection and frequency

The rate of infection depends on the extent of exposure to pathogens, the rate of invasion of the gland and the probability of the invading bacteria establishing in the udder. The relative importance of these factors varies with the level of disease in the herd, the type of husbandry employed and the pathogens involved.

INVASION

Exposure of the uninfected mammary gland to bacteria causing contagious mastitis is related to the amount of teat contamination and the level of disease in the herd; the condition of teat skin and orifice condition allowing external colonisation; the design and efficiency of the milking system e.g. preventing impacts and ensuring good pulsation; and the efficacy of post-milking teat disinfection (Table 1). Invasion of contagious bacteria occurs mostly at milking time so it could be considered likely to increase with milking

Table 1 Factors affecting invasion mechanisms

<u>Increased invasion</u>	<u>Decreased invasion</u>
<i>Contagious mastitis</i>	
poor teat condition	good teat condition
teat contamination	teat disinfection
poor pulsation	good pulsation
impacts	protection
more frequent milking	less frequent milking
<i>Environmental mastitis</i>	
long milking interval	short milking interval
poor teat disinfection	good teat disinfection
less frequent milking	more frequent milking

frequency. Conversely invasion of the udder by environmental bacteria, particularly *Escherichia coli*, is mostly during the inter-milking interval (2) so less frequent milking might result in a higher invasion rate from longer exposure times (Table 1).

ESTABLISHMENT

The success of establishment of bacteria in the mammary gland is likely to vary according to the type, timing and frequency of invasion. Contagious pathogens can enter the gland directly by the impact mechanism or reverse pressure gradients and more slowly by colonisation of the teat duct and gradual diffusion. Either way the likelihood of establishment will vary with the rate of removal, the 'flushing' of bacteria from the gland during milking. Clinical mastitis might also be influenced by easier diagnosis and treatment with frequent milking.

With *E.coli* the onset of disease is so quick after invasion that the length of the milking interval can be very important. Three times daily milking is probably not frequent enough for effective flushing and there will be little difference between twice and once daily milking. So little is known of the invasion mechanism of *Streptococcus uberis* that the consequences of different frequencies of milking cannot be predicted (Table 2).

Experimental and trial findings

TWICE DAILY MILKING

Clinical mastitis is a rare event. The national average incidence is 30-40 cases/ 100 cows/year or approximately one case in the lifetime of each cow. A clinical case of mastitis occurs approximately every 8500 quarter milkings. Of course, the incidence of sub-clinical mastitis is much higher with over 5% of all quarters infected. Comparisons in all work are made to the relative incidence of infection in twice daily milked quarters.

ONCE DAILY MILKING AND OMITTED MILKINGS

With contagious mastitis, the rate of infection is significantly higher if the milking after experimental inoculation of the teat duct is omitted (3). The inoculated quarter secretes bacteria within 12 h of exposure but clinical signs and an elevated cell count take more than 24 h to develop on twice daily milking but occur much more quickly when milkings are omitted. There is a higher rate of infection in un milked quarters compared to milked quarters of the same udder with external exposure (4). Experimental inoculation into the teat sinus before milking produces a much lower rate of infection than inoculation after milking suggesting that 'flushing out' of the bacteria prevents establishment (5). Subsequently the flushing has been shown to be the most likely mechanism (6, Table 2).

Table 2 Effect of once daily or omitted milkings on mastitis

<u>Experiment</u>	<u>Effect</u>	<u>Ref</u>
teat duct inoculation with <i>S.aureus</i> and effect of omitting the next milking	3 times more infections when next milking was omitted	3
teat dip with <i>S.aureus</i> and <i>S.uberis</i> on milked and un milked quarters of the same udder	only un milked quarters became infected	4
inoculate the teat sinus before or after milking with <i>S.aureus</i>	fewer infections when inoculation was before milking	5
teat dip with a mixture of bacteria on milked and un milked quarters of the same udder	more infections in un milked quarters and not related to leakage, cell count etc.	6

With once daily milking, there will tend to be fewer machine-related invasions of the gland by contagious pathogens but longer for successful establishment of bacteria to occur. There will also be a longer time for contamination and invasion by environmental bacteria to occur and presumably less time when the teat is covered with active disinfectant. When teats are exposed to *E.coli* the rate of infection increases with the length of time the teat ends are contaminated (2) and invasion does not appear to be related to the process of milking. The limit to the rate of clinical mastitis is not the rate of invasion but the likelihood of successful establishment which is reduced most readily by flushing. Less frequent milking reduces this and so leads to a higher incidence of mastitis by contagious pathogens. The longer time interval also predisposes to a higher incidence of infection by *E.coli*.

THRICE DAILY MILKING

Most investigations of the effect of thrice daily milking on udder health have been asides to production trials and have usually measured the response by the CMT or the WMT. Even then the results have been contradictory. Some studies have reported lower scores; some the same; and some higher scores than found with twice daily milking. The only experimental infection study showed that there was no difference in the rate of new infections between cows milked thrice daily and those milked twice daily (1). Most of these infections

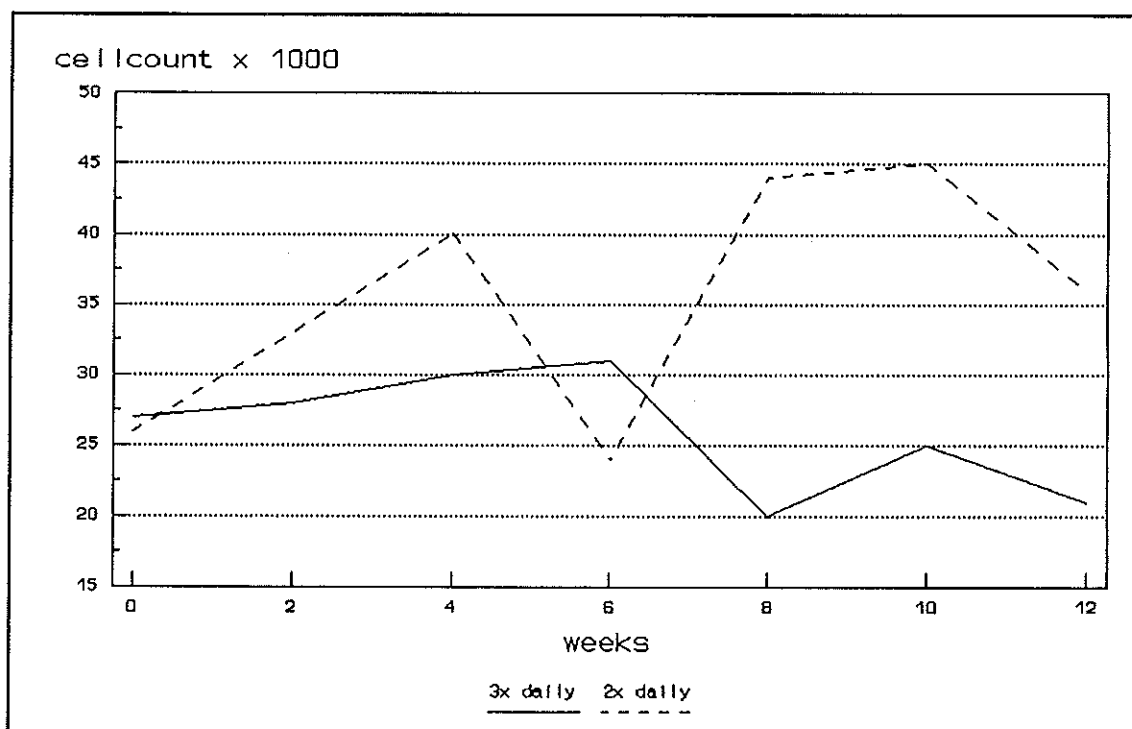


Fig.1 Effect of 3x daily milking on milk cell count.
(after ref. 1)

were by minor pathogens especially *Corynebacterium bovis* which rarely causes clinical mastitis. Few cases of clinical mastitis were caused by the challenge bacteria which were dipped on to the teat end. There were 7 cases in total in the twice daily milked group and one in the thrice daily milked group. Although not a statistically significant difference, this does suggest a benefit from more frequent milking over the 12 week trial.

The trend over this trial was for the milk cell count to be lower over the 12 weeks for the thrice daily milked cows when it would normally have tended to increase as yield decreased with the length of the lactation (Fig. 1).

FOUR TIMES DAILY MILKING

There is even less evidence for the effects of four or more times daily milking on udder health than for other frequencies yet this is needed to predict the consequences for automated milking systems which may milk 4-5 times daily (7). Very frequent milking will not be sustained for a whole lactation, only when it is profitable to harvest milk, but this has been shown to last well into mid-lactation (8). A series of experiments are being undertaken to estimate the consequences of four times daily milking for udder health.

Experiment 1

The rate of new infection (regular secretion of bacteria in the milk and/or a persistently elevated cell count) and clinical mastitis (infections requiring antibiotic therapy) have been determined in response to inoculation of the teat duct with *Streptococcus agalactiae* or *S.dysgalactiae* in cows milked twice or four times daily, in a cross-over designed experiment, at equal intervals.

Two infections included both pathogens; all of the others were caused by *S.agalactiae* only. Table 3 shows that for both parts of the experiment the same proportion of quarters samples was bacteriologically positive for both milking frequencies but that almost 3 times as many quarters suffered a clinical infection when exposed during twice daily milking than when milked four times daily (Table 3). This proportion is significantly different ($P < 0.05$). In this experiment there was a constant and high level of exposure to bacteria. The rate of invasion under both treatments appeared constant because the same proportions of quarters became bacteriologically positive at some stage but the extent of establishment differed due to either more flushing or more mobilisation of cellular defences. It appears that a higher frequency of milking could be protective against clinical mastitis.

Table 3 Effect of 2x versus 4x milking on mastitis.

Milking frequency	No. of quarters	Clinical mastitis	Bacteria +ve		Total
			2/3 samples	1/3	

Part 1

2x daily	34	8	5	1	14
4x daily	36	2	4	7	13

Part 2

2x daily	33	7	4	0	11
4x daily	34	3	1	1	5

Overall

2x daily	67	15	9	1	25
4x daily	70	5	5	8	18

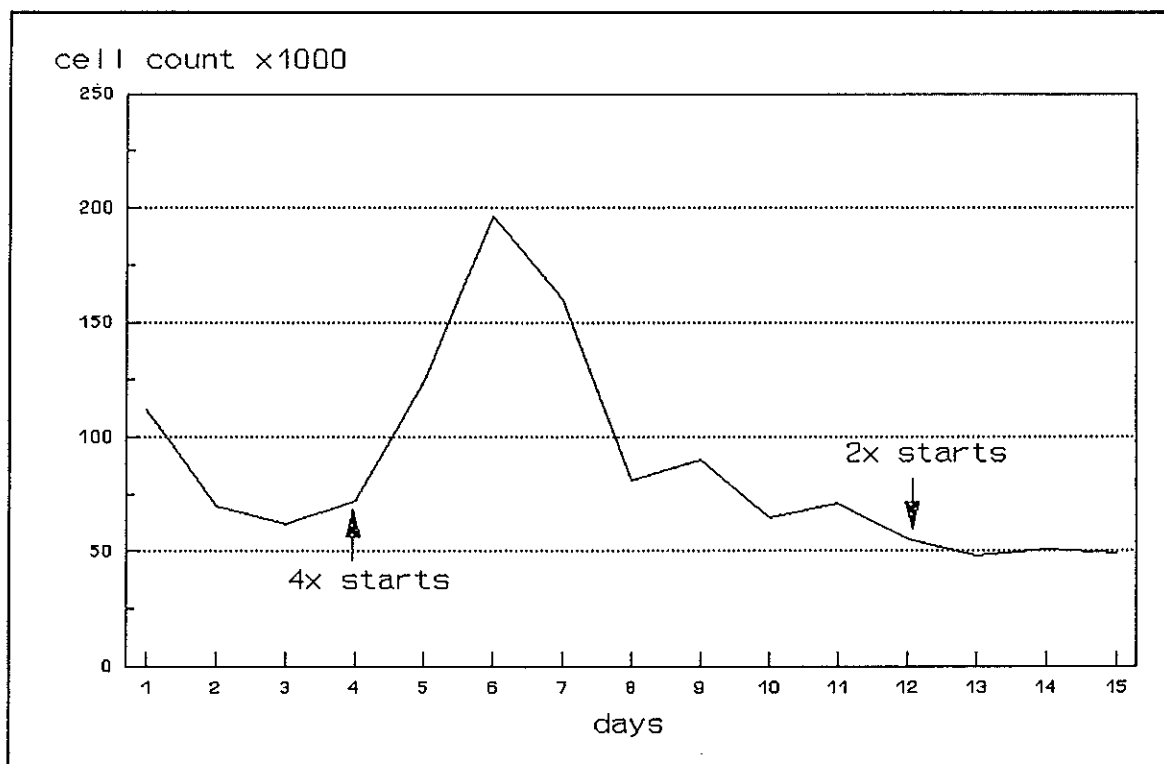


Fig. 2 Effect of 2x versus 4 x daily milking on milk cell count.

One of the initial responses to four times daily milking is a transitory increase in milk cell count but the longer term response is a small reduction, as for thrice daily milking. This is shown in Fig. 2, where on the change from milking with a 12 h interval to a 6 h interval the cell count increased 3 fold. It had returned to 'normal' within 4 days. The response was even quicker, within 2 days, when 6 times daily milking was employed (9). Lower cell counts with more frequent milking have also been reported from Dutch studies (7). The rapid control of cell count is important for milk quality and immune defences where cells must not be secreted unnecessarily. It is also important to know if there can be a therapeutic lowering of cell count in high cell count quarters such as occur with chronic *Staphylococcus aureus* infections.

Experiment 2

Ten cows, having 11 quarters in total with a chronic *Staphylococcus aureus* infection, were milked for 7 days every 12 h and then every 6 h for 7 days. Finally they were milked every 12 h for another 7 days. The effect of these milking frequencies on bacterial recovery and milk cell count was determined.

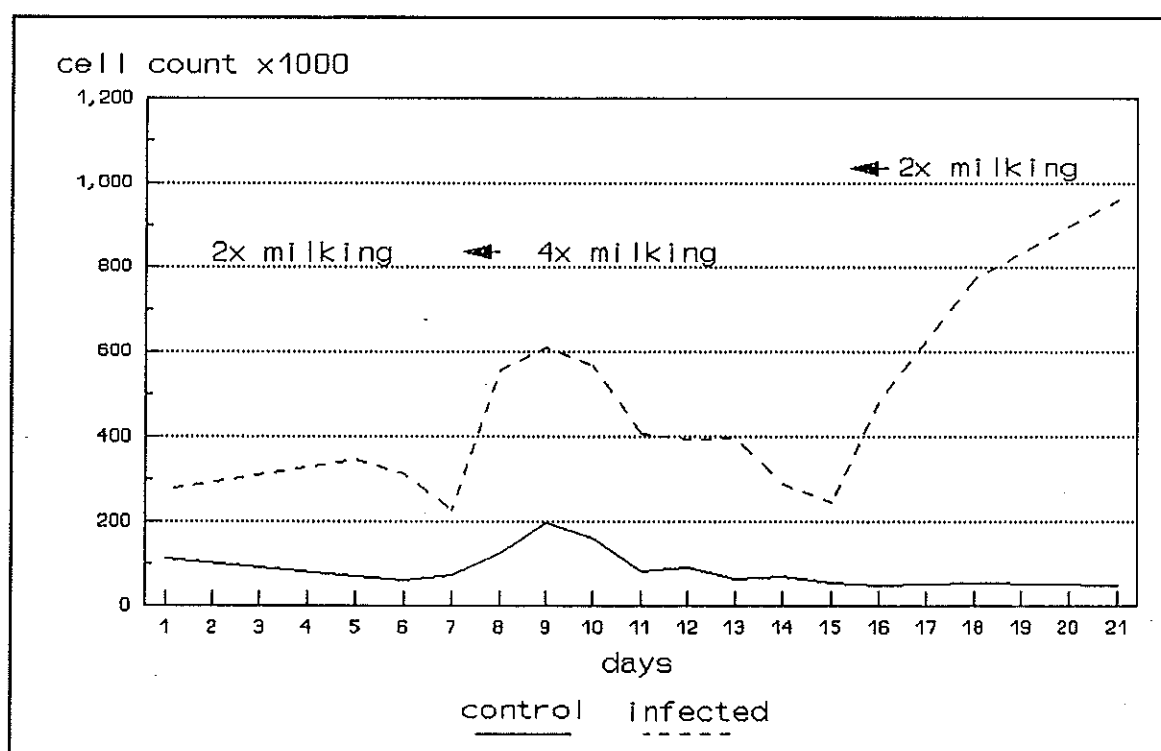


Fig. 3 Effect of changing milking frequency on chronic mastitis.

All quarters continued to secrete bacteria in regular samples taken through the 3 weeks. In the initial 7 days of milking, with a 12 h interval, there was a reduction in the cell count. The cell count however rose sharply when the interval was reduced to 6 h with the lower level being regained in about 4 days (Fig. 3). There was no obvious effect on the clinical status with more frequent milking. When 12 h milking intervals were resumed the cell count rose rapidly and 10 of the 11 quarters were found to have clots sufficient to warrant antibiotic therapy. The infections had become clinical mastitis.

There is no obvious explanation for this change of status unless it is considered that such changes of husbandry 'stress' the animal. Simple changes in husbandry such as grazing paddock or group structure are known to change the activity of leucocytes (J.Fitzpatrick, pers. comm.) and may be enough to upset the equilibrium which occurs in sub-clinical mastitis. This experiment certainly suggests that there may be many effects of the frequency of milking on mastitis still to be considered.

As yet there is virtually no information on the response to infection by environmental pathogens with more or less frequent milking and no significant data on long-term natural infection rates with any pathogen.

The only other benefit ascribed to frequent milking is that milking infected quarters 5-8 times daily can be as effective as antibiotic therapy in resolving clinical signs (10).

SUMMARY

Generally, the literature is consistent in reporting that less frequent milking results in more mastitis even though the best machine operation is used. This seems to result from less flushing of bacteria from the gland. Chronic mastitis may become acute, as in the change from four times to twice daily milking, when an equilibrium is disturbed. The effects on mastitis caused by environmental pathogens may be similar because there is more opportunity for invasion and establishment during the longer milking interval.

More frequent milking with more frequent opening of the teat duct may result in more invasions but these will be reduced by good practice including teat disinfection and the duct may recover more quickly following a shorter machine-on time. The single most important factor for clinical mastitis would appear to be flushing of bacteria from the gland, the time-honoured treatment!

There is obviously a need to investigate the consequences of milking frequently in long-term natural exposure trials. The effects of changing frequency on chronic infections may be an extremely useful research tool. So far the research suggests that more frequent milking is better for mastitis and the cow, if not for the milker.

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