BRITISH MASTITIS CONFERENCE 1990

MASTITIS TREATMENT - MAKING IT WORK

Jointly organised by:
GENUS ANIMAL HEALTH
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& CIBA-GEIGY AGROCHEMICALS

I N D E X

ABSTRACTS OF POSTERS

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

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The theme for the third British Mastitis Conference, "Mastitis Treatment - Making it Work" could not have been chosen at a more appropriate time. Within the next twelve months, farmers throughout the United Kingdom will be paid on the bulk cell count of their herd with bonuses payable on cell counts of less than 400,000 cells per millilitre and penalties for milk with more than 700,000 cells per millilitre.

Recent figures indicate that a third of all herds fall into the penalty category and farmers need to take action now if they are to avoid further losses after the implementation of the payment scheme.

With this information in mind and the feed back received from delegates at the Conference last year, the organisers decided that mastitis treatment should form the main part of the programme this year. As in the 1988 Conference, they also felt that there was a need to include an update on current mastitis research.

The organisers would like to thank Dr John Bramley, who is taking up the post of professor of Dairy Science at the University of Vermont in the USA, for the major input he has had in the organisation of the programmes for these Conferences. The programmes have been designed to be of interest to everyone involved in mastitis control and we hope that today's Conference will be no exception.

CELL COUNTS MEAN MONEY

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Introduction

Cell counts have always meant a loss of money but, come next year, they can mean a gain in money too. The EEC Health and Hygiene Directive requires milk to have a cell count of 400 thousand cells/ml or less in order to achieve the Step 2 quality standards. Not a particularly onerous requirement, but only two-thirds of British dairy herds achieve it at present.

To encourage the rest, the Milk Marketing Board (MMB) of England and Wales has introduced weekly bulk milk cell counting this month and a payment scheme which starts in a year. Similar schemes will operate in Scotland and Northern Ireland.

In this paper I aim to answer three questions:

Who will be affected?
Why will they be affected?
What should they do?

Before doing so, it might be helpful to contrast some of the features of bulk milk cell counts and total bacterial counts, because confusion between the two is still quite common - even with some MMB staff!

TABLE 1: Total bacteria counts (TBC) and cell counts (CC)

Feature	TBC	CC
Size (diameter)	<1 micron	5 microns
Are they essential?	No	Yes
Is a low level good?	Yes	Yes
Counts (thousands/ml):		
is zero possible?	Yes	No
how does a level of 101-200 rate?	High	Low
Main control measures	Hygiene	Hygiene and antibiotics
Influence on milk quality	Direct	Indirect
Start of payment scheme	1982	1991

Who will be affected?

The cell count payment scheme, which applies to all milk produced from 1 October 1991 onwards, is based on the three month rolling geometric mean (average) cell counts (Table 2).

TABLE 2: Cell count payment scheme

Band	Cell count (thousand/ml)	Payment adjustment (pence/litre)
1	400 and below	+0.2
2	401 to 700	nil
3	701 to 1,000	-0.2
4	over 1,000	-0.4

Because the October 1991 cell count payment is based on a three-month average, this means that the weekly cell counts from the beginning of next August will count towards the average.

The number of herds in the bonus band is likely to fluctuate considerably through the year. Although the figures in Table 3 are not totally applicable to the payment scheme, because the cell count categories are not identical to the payment bands nor, more importantly, are they based on three-month averages, nevertheless the table shows that there is a far lower proportion of herds in the future bonus band during the three month period July to September (56%) compared to the four month period December to March (73%).

TABLE 3: Dairy herds in England and Wales according to monthly cell count

	Month	Cell count category (thousand cells/ml) under 400	400-699	700-999	1,000 & over	Total herds tested
1989	Jun	20,742	8,336	1,997	873	31,948
	Jul	17,671	10,161	2,744	1,346	31,922
	Aug	16,767	10,498	2,939	1,534	31,738
	Sep	18,696	9,086	2,451	1,306	31,521
	Oct	21,106	7,439	1,989	997	31,531
	Nov	22,440	6,289	1,726	961	31,416
	Dec	22,833	5,650	1,540	871	30,894
1990	Jan	22,445	5,998	1,766	1,064	31,273
	Feb	22,621	5,973	1,608	987	31,189
	Mar	22,790	5,999	1,552	927	31,268
	Apr	22,252	6,710	1,666	855	31,483
	May	22,268	7,048	1,482	581	31,379
Averages		21,053	7,431	1,955	1,025	31,464
	%	66.9	23.6	6.2	3.3	100

To provide more direct examples of how herds may be affected by the cell count payment scheme, Table 4 lists the recent results

of six herds covering a range of cell counts.

TABLE 4: Effect of 3-month geometric average cell count

Cell count range	Example herd	Present annual average CC	Monthly CC Lowest	Monthly CC Highest	Geometric average CC over 400 Months	Geometric average CC over 400 No.
101-200	A	185	97 (Dec)	346 (Jul)	-	0
201-300	B	261	117 (Sep)	391 (Sep)	-	0
301-400	C	356	237 (Jan)	608 (May)	Jul-Oct	4
	D	367	270 (Apr)	502 (Jul)	Aug-Oct	3
401-500	E	442	307 (Jan)	632 (Oct)	Apr-Dec	9
>500	F	643	238 (Mar)	1269 (Sep)	Apr-Dec	9

(All cell counts are in thousands/ml)

Herds A and B, both of which have an annual average cell count below 300 thousand cells/ml, would have been in the bonus band throughout the last twelve months, although their individual monthly counts showed a good deal of fluctuation. Herds C and D, which both have annual averages around 350 thousand cells/ml at present, would have been out of the bonus band for four and three months respectively. Herd E, with an annual average only a little above 400 thousand cells/ml, would have missed out on nine of the twelve monthly bonus payments! Herd F would also have missed out in nine months, but would have suffered four penalty band deductions too.

It is of interest that the use of the geometric average as opposed to the arithmetic average (the true average) only reduced the months outside the bonus band in one herd for one month in this table - that was in herd F which had the highest cell count. In most instances the geometric average was very close to the arithmetic average, usually within 10 thousand cells/ml, so simple averaging of cell counts will usually tell you which band you will be in.

Why will they be affected?

The most obvious effect will be the loss of the bonus payment and, for some, an actual financial penalty. That is the easy part of the loss to calculate.

Assuming an annual production of 5,000 litres per cow, herd C which contains 80 cows would lose approximately £270 in bonus payments; herd D with 190 cows would lose £475; and herd E with 115 cows would lose a massive £860, despite having an annual average which is only just over the 400 thousand cells/ml limit at present. Herd F, with 62 cows, would lose over £150 a month in October and November when it was in penalty band 4 and would suffer an annual loss of £775.

These losses are direct financial penalties and the farmers

suffering them will soon become aware of them. However, all calculations of the financial losses due to subclinical mastitis in these herds indicate that, at a conservative estimate, they are three to five times higher than the cell count payment penalties.

Over the last twenty years very many farmers have reacted to the hidden losses due to subclinical mastitis by operating a mastitis control programme in their herds. That is the main reason for the halving of the national cell count over the past two decades. Some farmers remain unconvinced of these losses due to subclinical mastitis. No doubt they will react to the cell count payment schemes. Provided they also start a mastitis control programme, or tighten up the present one, and don't simply rely on culling high cell count cows, they will also reap the rewards of improved milk yields and quality through controlling subclinical mastitis.

What should they do?

Every herd is different in some respects and may need to have its mastitis control programme tailored to its individual needs. That is where the specialist adviser comes in, whether he or she is the farmers' own veterinary surgeon, an MMB mastitis technician, an ADAS adviser, or other trained technician.

That said, the outstanding simplicity of the five point control programme, developed twenty years ago from the work of the NIRD/Weybridge team, is that it can be applied in virtually every herd to advantage. So the farmer or herdsman with a cell count problem should first of all ensure, preferably with his veterinary surgeon or other adviser, that all elements of the five point programme are being properly applied in the herd.

A simple checklist would include:

1. Milking machine:
   Has this been tested in the past six months? Were the faults corrected? Is the regular monthly maintenance being carried out?
2. Teat disinfection:
   Is the dip or spray at the correct strength? Are all teats being covered? At every milking? All the year round?
3. Dry cow treatment:
   Are all cows being treated? As soon as they are dried off? Is the antibiotic effective? Ask your vet.
4. Clinical cases:
   Are cases being detected as early as possible? Is the treatment effective? Ask your vet. Do you keep a written record??
5. Cull chronic cows:
   Any cows with three or more cases in the last year? Seriously consider culling. Must have records!

Are all these actions being taken? If not, start the full

programme at once, but be aware that proper control can take several months, even up to a year, to achieve. Other checks you can carry out include:

6. Housing and bedding:
   Are the cubicle beds or lying areas dry? Is slurry removed twice a day?

7. Milking order:
   Milk the first calvers first as most of them should not be infected, and the known infected cows last.

8. Individual cow cell counts:
   Do these at least twice, preferably three times, at monthly intervals to find out how many cows have high cell counts.

9. Bacteriology:
   Sample 10-20 of the high cell count cows to determine the main mastitis bacteria in the herd. Different bacteria require emphasis on different parts of the control programme.

For the short-term only, in order to bring down the herd milk cell count rapidly, you can:

10. Withhold milk:
    After identifying the cows with regularly high cell counts, keep their milk out of the bulk tank, and either cull or treat them. Take good care to ensure that antibiotic residues do not get into the bulk milk. Remember this is a last resort - it is not the long term answer to high bulk milk cell counts nor is it a method of controlling mastitis in the herd.

Action guide

The table below is a guide on the action needed if you are to receive the cell count bonus, worth an average £10 a year for every cow in your herd, from next October.

The annual average cell count you have been receiving on your milk statement for the last 13 years is the best guide to the level of subclinical mastitis in your herd and to the effectiveness of your control measures. Continue to calculate this and aim to keep the annual average below 300 thousand cells/ml.

Time	Action (all cell counts are in thousands/ml)	
Now	Check your annual average cell count.	
	Over 400:	Arrange immediate on-farm investigation by your veterinary surgeon or other mastitis adviser.
	301-400:	If trend upwards, consult mastitis adviser. If trend downwards, use checklist to ensure all controls in operation and working effectively.

GM 7380 Action

Time	Action
	201-300: Check all control measures in operation.
	200 or less: Continue present control measures.
Nov-Dec	Average your weekly cell count for each month and compare this figure with the same month in 1989. If trend upwards: Discuss with mastitis adviser. If trend downwards: Continue present control measures.
Jan 1991	Receive first full 3-month geometric average cell count. Autumn calver low avgrag
Feb-June	Continue to check the trend of your monthly cell counts. If a) the trend is upwards or b) the geometric mean is over 300 or c) any weekly counts are over 400: Discuss immediately with mastitis adviser.
July	Last month before cell counts are used in the payment scheme. Immediate action required if any counts are over 400.
Aug-Oct	Immediate action required if monthly averages regularly over 300 or any counts are over 400.
November	First payment on cell count for milk sold in October.

Before payments start we hope to see over 90% of herds regularly in the bonus band. Aim to be with them!

Due to Change of testing type
400-500 no change
600-800 higher
200-300 may drop 20-40

} Charge for Coulter counter to Rhoplomatic testing

Autumn calving herd

low Jan normally water at two goes or higher late summer/autumn.

FARMER'S GUIDE TO ANTIBIOTICS

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Two main factors govern the effectiveness of an antibiotic in mastitis: its penetration to the site of infection and its antibacterial activity when it gets there.

Penetration of injected antibiotics into the healthy udder

When an antibiotic is injected systemically for the treatment of an infection other than mastitis, it passes readily from blood into tissues through pores in the walls of the capillary vessels. The capillaries in the udder, however, like those in the brain, have no pores and so drugs can pass from blood to milk only by crossing the intact capillary membrane for which they need to be lipid soluble; thus antibiotics enter the healthy udder at different rates according to their lipid-solubility. The level of an antibiotic attained in milk following systemic injection is also affected by the phenomenon on 'ion-trapping'. Normal milk is acid (pH 6.7) relative to blood (pH 7.4). Thus in the case of a basic antibiotic such as a macrolide the non-ionised, lipid-soluble form penetrates the relatively acid milk where it is converted to the ionised, lipid-insoluble form which is trapped in the udder so that the concentration in milk far exceeds that in blood. Conversely an acidic antibiotic such as a beta-lactam is largely excluded from the udder. Table 1 shows for a variety of types of antibiotic, the ability to penetrate the healthy udder expressed as the milk-plasma ratio. In general, acidic antibiotics have shorter milk-withholding times than basic ones and in the case of cephalexin nil. Whether milk fails the MMB test of course, depends not only on the concentration of the antibiotic, but on its activity against the test organism which differs greatly in its sensitivity to different antibiotics.

Table 1

Antibiotic	Chemical type	Lipid solubility	Milk: plasma ratio
Benzylpenicillin	acid	moderate	0.20
Ampicillin	acid	moderate	0.26
Streptomycin	base	low	0.50
Erythromycin	base	high	5.0
Penethamate	base	moderate	4.0
Chloramphenicol	neutral	high	1.0
Oxytetracycline	amphoteric	moderate	0.8

Penetration of injected antibiotics into the mastitic udder

Two changes take place in mastitis which alter the extent to which antibiotics penetrate the udder: firstly inflammation breaks down the intact cellular barrier between systemic circulation and udder so that drugs may penetrate irrespective of their lipid-solubility and secondly the pH of milk rises to that 'ion-trapping' no longer occurs. Thus drugs penetrate the inflamed udder much as they do any other inflamed tissue.

Table 2 shows the time for which systemically-injected antibiotics maintain effective concentrations in normal and mastitic udders. In the healthy udder ion-trapping means that penethamate (which is merely a basic ester of benzylpenicillin which must be broken down to the parent compound to exert its antibacterial action) persists very much longer than the parent benzylpenicillin, but this difference is almost entirely abolished in mastitis. For the same reason erythromycin is not concentrated to the same degree in mastitic milk as in the healthy udder. On the other hand gentamicin which, like all aminoglycosides, is poorly lipid-soluble penetrates poorly into the normal udder, but well into the inflamed udder where the cellular barrier has been broken down by the inflammation.

Table 2

Time (hours) for which the concentration of an antibacterial in milk following systemic injection exceeds its MIC for a non-penicillinase-producing staphylococcus.

	Normal	Mastitis
Benzylpenicillin	6	8
Penethamate	24	10
Gentamicin	2	12
Erythromycin	12	7

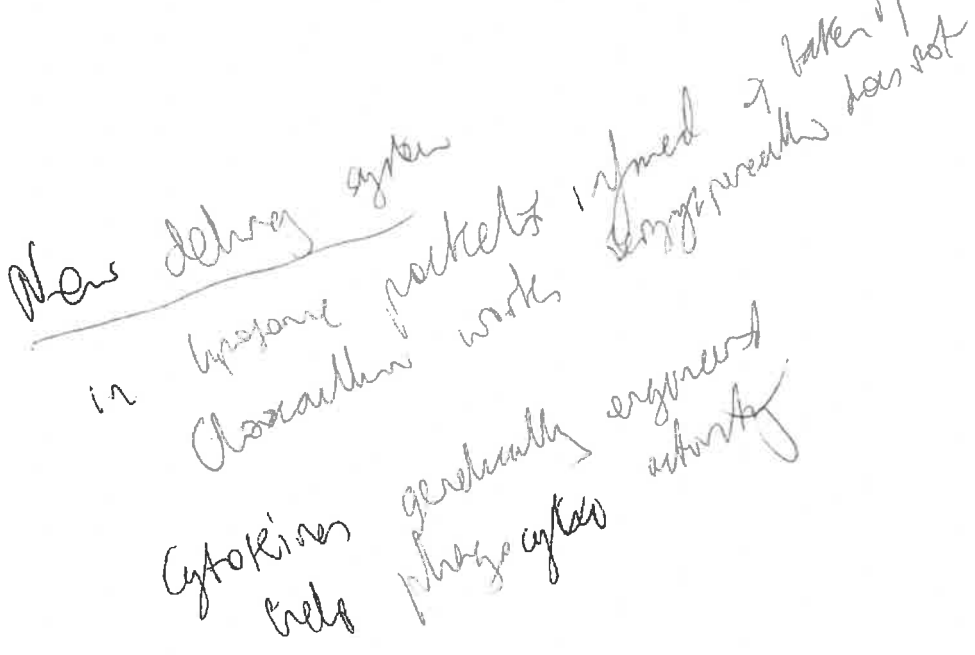
Adapted from Ziv (1980a)

Antibacterial activity of different antibiotics

Table 3 summarises the activity of the major antibiotics against the common mastitis organisms. Bacteria acquire resistance to antibiotics in various ways. Staphylococci (but not streptococci) and a number of Gram-negative organisms may secrete beta-lactamase enzymes which break down and thus inactivate the different beta-lactam antibiotics to differing extents. Drugs such as clavulanic acid are only weakly antibacterial, but have the property of binding irreversibly to the beta-lactamase enzymes of resistant bacteria thus protecting drugs such as ampicillin and amoxycillin, with which they are administered, from degradation by these enzymes. Of the antibiotics listed in Table 3, the beta-lactams and the aminoglycosides are bactericidal, that is they kill susceptible bacteria, whereas the others are bacteriostatic, that is they prevent bacterial multiplication, but rely upon host defences to eliminate the infection. Unfortunately bactericidal drugs act only against dividing bacteria so that under conditions where multiplication is inhibited (e.g. co-administration; in neutrophils) these agents may be ineffective.

Choice of antibiotics for treatment of severe mastitis by injection

In the treatment of severe, and especially toxic mastitis, local application of antibiotics by intramammary infusion (see below) is supplemented by systemic treatment by injection. The desirability of using the same antibiotic for systemic and intramammary treatment is obvious. As discussed above the efficacy of an antibiotic following systemic administration depends on both its ability to penetrate the mastitic udder and its antibacterial activity. Ziv (1980a) used a measure which combines these two factors, namely the length of time for which the concentration of an antibiotic in the udder exceeds its minimum inhibitory concentration for the organism in question, to assess the likely efficacy of a series of


New delivery system
in liposome rockets
Doxorubicin works
Cytokines
cells

antibiotics. He concluded that for E. coli infections the agent of choice was likely to be gentamicin, for S. aureus infections erythromycin and for streptococcal infections benzylpenicillin.

In severe, toxic, coliform mastitis Non-steroidal anti-inflammatory drugs (NSAID's) such as flunixin are used to counter the systemic effects of bacterial endotoxin. NSAID's inhibit formation of the prostaglandins on which the action of bacterial endotoxin depends.

Intramammary infusions of antibiotics

Intramammary infusion is the treatment of choice for mild and subacute mastitis and is used as an adjunct to systemic treatment in severe mastitis. The length of time for which the antibiotic persists in the udder (short-acting for milking cows and slow-release for dry cows) depends on the nature of the vehicle in which it is formulated and on the solubility of the salt of the antibiotic used (salts of benzylpenicillin in order of decreasing solubility and hence of increasing length of action are: sodium, procaine and benzathine). Antibiotics distribute within the udder by passive diffusion. Some, notably the aminoglycosides, become bound to udder tissue. There is evidence that antibiotics may be more effective when administered in a large volume of saline. Antibiotics do not pass from one quarter to another by diffusion through udder tissue, but may enter a non-infused quarter via the blood stream. In the inflamed udder tissue debris may block the alveolar ducts, preventing penetration of the antibiotic to the alveoli and necessitating systemic treatment to by-pass the obstruction. Alternatively oxytocin may be used in an attempt to expel material from the alveoli and thus clear the ducts. Corticosteroids may be added to intramammary infusions; whilst they symptomatically reduce cell counts there is no evidence that they affect the course of the infection.

Intracellular staphylococci

Unfortunately, although antibiotic administration may produce a clinical cure it does not always produce a bacteriological cure, i.e. the offending organism may still be present in the udder some weeks later. This is particularly the case with staphylococcal mastitis and is due, at least in part, to the ability of staphylococci, having been phagocytosed by neutrophils, to survive within the phagolysosome where they are protected from the action of antibiotics and thus set up a chronic infection. When the neutrophils degenerate these viable organisms are released within the udder to cause a recurrent episode of mastitis. Some antibiotics, particularly acidic ones (see Table 1) are unable to penetrate the phagolysosome. Rifampin is one which is able to penetrate to and kill sequestered staphylococci in vitro, but this result does not apparently translate to bovine mastitis. Another possible reason for the failure of antibiotics to kill intracellular bacteria is that such organisms are metabolically dormant and so not susceptible to the action of bactericidal agents (see above).

Table 3 The activity of the major antibiotics against the common mastitis organisms

Antibiotics	Type	Spectrum	Beta-lactamase stable	Susceptible bacteria
Benzylpenicillin	beta-lactam	Gram+	No	S. aureus (only if non-beta-lactamase), Strep. agalactiae, dysgalactiae, uberis, Actinomyces pyogenes
Cloxacillin	beta-lactam	Gram-	Yes	Beta-lactamase S. aureus only indication
Ampicillin, Amoxycillin	beta-lactam	Broad-spectrum	No	S. aureus and Escherichia coli only if non-beta-lactamase, Strep. agalactiae, dysgalactiae, uberis, A. pyogenes
Cephalosporins e.g. cefoperazone	beta-lactam	Broad-spectrum	Stable to staph enzyme, moderately to enzyme Gram-	S. aureus (including beta-lactamase), Strep. agalactiae, dysgalactiae, uberis, A. pyogenes, E. coli and Klebsiella pneumoniae (including some beta-lactamase)
Streptomycin	aminoglycoside	Gram- some Gram+ but not streps.	Yes	Some S. aureus, A. pyogenes, E. coli and Klebsiella pneumoniae
Oxytetracycline	tetracycline	Broad-spectrum but more active against Gram+ than Gram-	Yes	A proportion of S. aureus, Strep. agalactiae, Strep. uberis (not Strep. dysgalactiae) and A. pyogenes
Erythromycin	macrolide	Gram+	Yes	S. aureus, Strep. agalactiae, Strep. dysgalactiae, Strep. uberis, A. pyogenes

Further reading

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TACKLING THE MASTITIS CASE

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Approach to t/M of Clinical Mastitis

INTRODUCTION

Mastitis continues to be a great source of loss to the U.K. dairy industry and recent estimates put the cost of clinical mastitis to the U.K. dairy industry at about £40 million per year. This represents slightly less than half the total cost of mastitis (clinical and subclinical) which has been estimated at around £90 million per year (Booth, 1988). The Milk Marketing Board (England and Wales) has recorded an annual average incidence of 39 cases of clinical mastitis per 100 cows in a recent survey and with each case costing in excess of £40 (Beck and Dodd, 1988) the economic loss is significant even to the small dairy farmer. Ideally, we should be looking to prevent these clinical cases by effective mastitis control programmes, but even where control measures are excellent clinical cases will still occur and these require rapid, effective treatment.

WHAT IS A CLINICAL CASE?

A clinical case is one in which there are obvious signs of mastitis. These signs may be present in the milk as clots or a change in colour, in the udder as heat, pain, swelling or other change in consistency, and in the more severe cases in the cow as fever, reduced appetite and sometimes recumbency. The point at which an early case becomes clinically apparent is the source of much debate and a recent study (Torgerson *et al.*, 1990) has demonstrated that detection of clinical cases can be very dependent on the dairyman as they recorded recently detected clinical cases with cell counts varying from 300,000 to 760,000 cells/cmm. Other authors (Dohoo and Meek, 1982) have suggested that mastitis does not become clinically apparent until the quarter cell count reaches one million.

The main emphasis of this paper will be on the more routine clinical case, either the case with clinical signs restricted to clots in the milk or that in which, in addition to milk abnormalities, the systemic reaction is mild to moderate.

WHY TREAT?

Treatment of clinical cases of mastitis is carried out with certain motives in mind and the aims vary slightly with the severity of the clinical case involved. In the peracute case the saving of the cow's life is the immediate concern so that ultimate disposal can be premeditated. In the less severe cases treatment is instigated to enable the cow to regain good health, to enable the udder to regain its previously healthy status, to enable milk production to be re-established, to reduce cell count and to reduce the microbial contamination of the milk.

If a cow is due for culling either because of her mastitis history or for other reasons, then on economic and health grounds it may be advisable not to treat her as the longer she remains in the herd the greater the economic loss and the greater the health risk to the rest of the herd. On welfare grounds she should be treated but it should be inadvisable to put the udder health of the rest of the herd at risk.

THE APPROACH TO TREATMENT

There can only be one approach to treating any clinical case of mastitis and that is that it must be rapid and effective. Treatment must be instigated as soon as abnormality is detected and at an appropriate level i.e. systemic therapy if there is systemic illness and with products that are known to have the best chance, based on previous experience and knowledge, of producing a rapid clinical cure.

After these primary considerations, the individual choice of suitable preparations must consider other factors such as milk-out time, stage of lactation, and yield, age of cow, cost etc.etc.

WHAT SHOULD WE TREAT WITH?

In the 1990s we may be asked increasingly to consider treatments other than conventional antibiotic therapy for treatment of mastitis cases. The demand for herbal and homoeopathic treatments is increasing and veterinary surgeons should be aware of the possibilities and problems that lurk in these areas.

The values of the various antibiotics have been covered elsewhere but I would like to put them in context. Although it is only possible to generalise in terms of the pathogens involved in clinical cases of mastitis there are several features of the organisms involved that should be considered.

When pre-treatment samples from clinical mastitis cases are cultured for bacteriology organisms are usually cultured from 90% or more, the vast majority (≥70%) of samples yield cultures of *Staphylococcus aureus* and more than half of these are penicillin resistant. As it is not usually possible, except in certain circumstances, to identify the organism responsible when an initial clinical examination is made this basic fact must be borne in mind and an appropriate antibacterial used. The majority of other isolates from clinical cases of a mild to moderate nature are usually of *Streptococci*. These findings indicate that for the majority of cases an antibacterial which is effective against Gram-positive bacteria including those which are penicillin resistant is indicated. However if there is any suspicion that other bacteria may be involved, then a broad spectrum antibacterial should be used. The individual choice of therapy depends on previous experience of the mastitis flora in the herd, personal preference, pharmacokinetic advantages of a particular preparation or any other special requirements.

Intramammary therapy is always indicated except in occasional peracute cases. When it is used alone the antibacterial used should fulfil the criteria detailed above. The simpler the preparation then the easier it is to change therapy should the initial course be wholly or partly unsuccessful. When intramammary and systemic therapy are combined then the same antibacterial should be used by both routes whenever possible. It is important that dose levels by both routes are adequate to

reach the MIC required for the infecting organism, otherwise the therapy is wasted. With intramammary therapy the infected quarter(s) should be retubed after each milking in order to maintain high antibacterial levels within the udder.

There are many advantages to taking sterile milk samples for bacteriology before treatment of any cases of mastitis so that the organism can be isolated and its *in-vitro* antibacterial sensitivity pattern determined. This may seem excessive but it is certainly worthwhile if there is a sudden increase in the number of cases occurring within a herd as it can establish the identity of the pathogen and enable more effective treatment and control to be carried out. If an outbreak is encountered then more than one case should be sampled or misleading information may ensue. Although *in-vitro* antibiotic sensitivity testing may not be 100% accurate it is often a good indicator of resistance rather than sensitivity. It is worth remembering that any case of mastitis may be the start of an outbreak, and they are all just the tip of the iceberg as regards udder health.

SHOULD WE USE ANY OTHER THERAPEUTIC AGENTS?

The use of therapeutic agents other than antibacterials in the conventional treatment of clinical mastitis mainly depends on the severity of the disease. In the peracute cases which are usually seen in the dry period or early lactation the use of fluids, preferably by the intravenous route, and a systemic antiinflammatory agent are recommended in addition to systemic antibacterial therapy to ensure that the systemic effects of the disease such as toxaemia are alleviated. Under these circumstances the use of corticosteroids is contraindicated and a nonsteroidal anti-inflammatory drug (NSAID), flunixin meglumine, has been shown to be beneficial in the treatment of such cases (Christie, 1987).

The use of antiinflammatory agents in intramammary preparations is a continued subject of debate as the use of a corticosteroid preparation has often been considered to be contraindicated but may be of some value in certain cases. At present there are no intramammary preparations that contain NSAIDs.

Other agents are probably of doubtful value except where there are specific indications.

WHAT IF TREATMENT DOESN'T WORK

Treatment failure is always disappointing but a careful evaluation of the case will improve the chances of success second time round. This is where initial bacteriology of mastitis cases is of value as the results will be available by the time failure of treatment is apparent! Resampling the case is usually of little value at this stage.

If treatment continues to produce poor or nil results then the diagnosis should be questioned. There could be an unusual pathogen involved and often a consideration of other mastitis cases in the herd can be of value in identifying the pathogen.

CONCLUSIONS

In tackling the mastitis case the major factors which lead to success and as accurate a diagnosis as possible, followed by prompt treatment at an appropriate level. However, it must be the long-term aim of every dairy farmer and veterinary surgeon in the country to reduce the incidence of clinical mastitis through improved mastitis control measures.

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SHOULD I STILL USE DRY COW TREATMENT?

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Treatment of cows at the end of lactation by intramammary antibiotic infusion was originally carried out as a prophylactic measure to reduce the incidence of 'summer mastitis' an acute form of mastitis usually associated with streptococci and Corynebacterium pyogenes in non lactating cows and heifers. The therapeutic use of antibiotic infusions and the improvement of specific preparations coincided with the development of a comprehensive mastitis control programme. Infusion of cows at the end of lactation could drastically reduce the duration of an intramammary infection by eliminating the organism at this time. Staphylococci generally recognised as the most difficult common mastitis pathogens to eliminate from the udder, are more responsive to intramammary therapy given to non-lactating, rather than lactating cows.

Comprehensive mastitis control measures based on good hygiene during milking and housing, post milking teat dip and dry cow therapy have greatly reduced the incidence of mastitis associated with Staphylococcus aureus, Streptococcus agalactiae and Streptococcus dysgalactiae, the 'contagious organisms'. The incidence of so-called environmental organisms, Staphylococcus uberis and Escherichia coli has not shown a similar reduction. These organisms are now the commonest isolates from mastitis cases showing an actual and relative increase in incidence (Epidemiology Unit, CVL, Weybridge). Is there a conflict between the therapeutic and prophylactic use of antibiotics during the dry period and does the use of dry cow therapy eliminate some types of infections only to leave the udder more susceptible to colonisation by other major or minor pathogens?

The fact that an udder quarter has already been infected, means by definition, that it is a susceptible quarter. In assessing the future role of antibiotic therapy we must consider the possible protective role of minor udder pathogens in preventing new intramammary infections with major pathogens. It has been suggested that the continued use of control measures may, in some way predispose the udder to infection with environmental organisms. Are the relatively low levels of coccal mastitis maintained if the control measures are discontinued? Can repetition of dry cow therapy be justified when an infection has not been eliminated by previous treatment?

In an attempt to assess the relevance of the basic mastitis control measures, we set up a trial to collect data from five established herds which had used teat-dipping and routine dry cow therapy on all cows for over four years and had previously had a problem with coliform mastitis, and one newly established heifer herd.

The herds were divided at random into two groups of similar parity. All cows in the full-treatment group received routine dry cow therapy and were teat dipped after every milking.

In the partial treatment group cows were not teat dipped and received selective dry-cow therapy based on the bacteriological examination of drying-off quarter milk samples; only those quarters found to be infected with a major pathogen received dry-cow therapy. The previous benefits from mastitis control measures can be judged from the fact that all the herds were free from S. agalactiae infection, had rolling mean herd milk cell counts about or below the national average and at the start of the trial had less than 15% of quarters infected with major pathogens. In the 2 years preceding the trial,

the herds had used between 1.7 and 6.3 tubes of lactation intramammary therapy/cow/year. Some of the results were predictable, whilst others are more difficult to explain. There were significantly more uninfected quarters in the full treatment group both at calving and drying off. Also according to expectation, there were significantly more Corynebacterium bovis infected quarters in the partial treatment group at the beginning and end of lactation. Significantly more quarters were infected with major pathogens at calving in the partial treatment group, but the difference was insignificant at drying off. The level of micrococcal infections was similar in the two groups at calving, but increased in the partial treatment group during lactation (Table 1). Inspite of the levels of infection being similar at drying off the use of selective dry cow therapy instead of whole herd treatment was associated with a near doubling of the number of quarters infected with major pathogens.

Table 1

The infection status of quarters at drying off and calving

Infection Status	Calving		Drying off	
	Full Treatment	Partial Treatment	Full Treatment	Partial Treatment
Uninfected	1424*	986*	1008+	747+
Infected with major pathogens	142*	268*	175	197
Infected with C. bovis	100*	319*	585+	715+
Infected with micrococci	115	125	104+	188+
Total	1781	1698	1872	1847

** Significant (P<0.05) difference between treatment groups

During the dry period (Table 2) nearly twice as many quarters in the partial treatment group developed clinical mastitis due to large and statistically significant differences (P 0.05) in the susceptibility of uninfected quarters and those infected with micrococci.

Quarters infected with a major pathogen or C. bovis at drying off had a similar susceptibility to the development of clinical mastitis in each treatment group. Within the full treatment group clinical mastitis associated with a new major pathogen infection was more likely to occur in quarters which had had a major pathogen or C. bovis infection at drying off emphasising the need to keep herd mastitis levels low. Infection with micrococci at drying off did not seem to predispose quarters to develop clinical mastitis associated with new major infections in this full treatment group, but it was associated with a significant increase in clinical mastitis in the partial treatment group. Using only selective dry cow therapy therefore increased greatly the incidence of clinical mastitis in previously uninfected quarters or micrococcal infected quarters. It could be postulated that the 'resistance status' of uninfected and micrococcal infected quarters is similar and low, whereas infection with C. bovis, or a major pathogen at drying off, offer similar resistance. This thesis is supported by the rates of new major pathogen infections in quarters of different infection status presented in Table 3.

Table 2

The rates° of clinical mastitis associated with new infections in the dry and periparturient periods in quarters of different infection status at drying off

Infection status	Rates°	
	Full treatment group	Partial treatment group
Uninfected	1.7*	4.8*
Infected with major pathogens	6.9+	6.1
Infected with C. bovis	3.6+	2.5+
Infected with micrococci	1.9*	9*+
Total	2.8*	4.5*

° Cases of mastitis/100 quarters
* Significant (P<0.05) difference between treatment groups
+ Significant (P<0.05) difference between infected and uninfected quarters in same treatment group

Table 3

The rates of new major pathogen infection during the dry and periparturient periods in quarters of different infection status at drying off

Infection Status at Drying Off	Rates°	
	Full treatment group	Partial treatment group
Uninfected	6.6*	14.9*
Infected with major pathogens	14.3+	18.3
Infected with C. bovis	10.2+	13.4
Infected with micrococci	7.7*	22.9*+
Total	8.5*	15.5*

° New infections/100 quarters
* Significant (P<0.05) difference between treatment groups
+ Significant (P<0.05) difference between infected and uninfected quarters in the same treatment group

In both treatment groups quarters infected with a major pathogen at calving (Table 4) were nearly three times more likely to develop clinical mastitis in the 3 months after calving than were uninfected quarters. This similarity between treatment groups is to be expected as 70% of these cases were associated with the same pathogen as was found at calving and were likely to be persisting infections which had failed treatment. The with-holding of dry cow therapy from uninfected quarters or those with minor pathogens did not reduce the rate of clinical mastitis during the first 3 months of lactation.

Table 4

The rates of clinical mastitis during the first 3 months of lactation in quarters of different infection status at calving

Infection Status at Calving	Rates°	
	Full treatment group	Partial treatment group
Uninfected	5.9	6.6
Infected with major pathogens	19.7+	17.2+
Infected with C. bovis	8.0*	2.2*+
Infected with micrococci	7.8	4.0
Total	7.2	7.2

° Cases of mastitis/100 quarters
* Significant (P<0.05) difference between treatment groups
\+ Significant (P<0.01) difference between infected and uninfected quarters in the same treatment group

E. coli and S. uberis were the commonest major pathogens causing clinical mastitis (Table 5). A much higher proportion of these clinical cases occurred in the partial treatment group during the dry period or at calving (Table 6) when the only difference was the absence of 'blanket' dry cow therapy.

Table 5

Incidence of clinical mastitis caused by major pathogens in two treatment groups

	Full treatment		Partial treatment	
	No.	Rate*	No.	Rate*
S. uberis	58	15.9	99	27.3
Coliform	56	15.3	40	11.0
Other major pathogens including mixed infections	70	19.1	72	19.8

* Cases/100 cows/year

Table 6

The stage of lactation cycle at which S. uberis and coliform mastitis cases occurred in two treatment groups

	S. uberis mastitis cases		Coliform mastitis cases	
	Full treatment	Partial treatment	Full treatment	Partial treatment
Dry period	5	23	0	1
Calving	14	21	6	6
Lactation - infected at calving*	13	23	3	2
Lactation - new infection	26	32	47	31
No. of calvings	467	458	467	458

* These clinical mastitis cases occurred in quarters subclinically infected with the same pathogen at calving

In addition, 23 clinical cases of S. uberis mastitis occurred during lactation as a result of infections already present at calving in the partial treatment group compared with 13 in the full treatment group. The number of clinical cases of mastitis associated with S. uberis infections acquired during lactation was similar (26 full, 32 partial treatment) further emphasising that the major difference causing the high incidence of S. uberis mastitis was the absence of dry cow therapy.

The 'blanket' use of dry-cow therapy is desirable as a prophylaxis to prevent new streptococcal and C. pyogenes infections. It has been suggested that this continued prophylactic use of antibiotics may increase the cows' susceptibility to new infections. Our data does not support this contention. Indeed the presence of untreated micrococcal infections not only failed to protect quarters in the dry period, but seemed to actually increase the susceptibility of these quarters to mastitis. The beneficial use of dry cow therapy to prevent summer mastitis has been well documented. In the case of staphylococcal mastitis the therapeutic effect of intramammary antibiotics is so much greater than treatments given during lactation, that the dry period would be the preferred period for attempting to eliminate staphylococcal intramammary infections. Table 7 shows that a 60% cure rate can be obtained for infections first treated at the end of lactation. The expected benefit from attempting to treat persisting infections is greatly reduced as the cure rates drop dramatically to 24% if one previous dry period treatment (DPT) has been given and to only 10% after more than one DPT.

Clearly protracted use of dry cow therapy is not a substitute for the practice of culling cows with chronic staphylococcal mastitis.

Table 7

The response of staphylococcal infections treated during or at the end of lactation in relation to previous courses of cloxacillin therapy

Therapy given	No. previous courses of therapy given							Total No. Treatments
	At end of lactation			During lactation				
	0	1	>1	0	1	2	>2	
During lactation				46%	21%	17%	12%	897
		or		25%	15%	13%	7%	227
End of lactation				61%	46%	24%		1124
	60%	24%	10%					

HOW TO AVOID ANTIBIOTICS IN MILK

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The concern over antibiotics in milk is strongly motivated by both economics and the desire to protect the consumer from the perceived effect of residues.

It is well documented that the potential risk to human health from antibiotic residues in milk, is negligible, but effective control is necessary to protect the reputation of milk as a healthy food as well as to prevent economic loss in the dairy manufacturing industry.

I will refer to the residues in milk from antibiotics used in the dairy cow and not to the other possible causes of milk failure. Lactating cow intramammary preparations are responsible for 50% of failures and dry cow preparations for about 25%. Injectables also play a significant part and must also be strictly monitored.

Since 1965, milk from dairy farms in England and Wales has been regularly tested for antibiotic using a test organism. The test will theoretically detect any substance capable of inhibiting the growth of the organism and is not exclusive to antibiotics. In practice the test is a reliable measure of antibiotic contamination, and failures resulting from non antibiotic causes are not a problem.

The sensitivity of the test has increased from 0.02 iu/ml in 1965, to 0.01 in 1986 and 0.005 in October 1990 in line with the other main milk producing countries in Europe.

In 1961 as many as 11% of herds were positive for antibiotics; this had dropped to 0.6% by 1985 and is currently about 0.3%. In July this year, the number of failures stood at 337 herds per month, a drop of 17% from a year ago.

It is important to look at a few statistics which help to explain the economic implications of antibiotic contamination of milk. Many farmers assume that antibiotic can be diluted below the detection limit. In 1983 Wishart calculated that milk from one cow treated with 200 mg of Penicillin, has the potential of contaminating all the milk from 8,000 cows! More practically, the milk of a cow in mid lactation, producing 20 litres per day, or 10 litres per milking, which is treated in one quarter with a tube containing 1,000,000 units of Penicillin, could contain 20 iu/ml at the first milking after treatment which would be sufficient to cause 20,000 litres of milk from 1,000 cows, or two tanker loads to fail the test at 0.01 iu/ml.

The potential for residues to appear in milk is enormous, considering that many millions of tubes are used every year in the UK. The fact that the antibiotic failure rate is as low as it is, suggests that most milkers are taking all the necessary precautions.

In 1981, the two main reasons for failure were:

1. Poor, or non existent record keeping (32%)

2. Not withholding milk for the full period (32%)

The reasons suggested for test failure in recent years are:

		1984/85	1989
1.	Not withholding milk for the full period	16.5	15.6*
2.	Accidental transfer of milk	16.7	14.5*
3.	Prolonged excretion of antibiotic	8.2	6.5
4.	Contamination of jars	7.2	11.2*
5.	Early calving/short dry period	7.3	9.5*
6.	Accidentally milked dry cows	6.2	4.5
7.	Poor records (or none) of treatment	3.0	2.0
8.	Treated cows not identified	4.0	4.8*
9.	Mechanical failure (leaking valves)	4.5	6.7*
10.	Recently purchased cows	3.1	4.0
11.	Lack of advice of withdrawal times	2.0	1.5
12.	Milking through jars	1.7	2.6
13.	Use of dry cow tubes during lactation	0.8	0.7
14.	Withheld milk from treated quarter only	2.5	1.5
15.	Other	4.4	4.6
16.	No cause identified	11.9	9.5*

* = points discussed below

Apart from the marked improvement in general record keeping, it is interesting to note that the reasons given for antibiotic failure have not changed very much.

1. Not withholding milk for the full period: In most cases the milker has gambled, although lack of attention to a revised withholding period may often be the cause.

2. Accidental transfer of milk: This may occur in the busy parlour, but strict attention to the marking of antibiotic treated cows should prevent this happening. Financial penalties can be avoided if the Milk Marketing Board is informed before the consignment of milk is collected.

3. Prolonged excretion of antibiotic has always been a contentious issue and is very difficult to verify. The manufacturer's withholding period for each preparation is very carefully calculated and provides a wide safety margin.

4. Contamination of jars: This is grossly underestimated by most herdsmen and it is essential to rinse any jar which may have held milk with containing antibiotic.

5. Early calving/short dry period: This in some cases can account for nearly 10% failures, and is most easily avoided by never running dry cows which have been tubed with the milkers. Positive marking is also essential. Financial penalties can be avoided by having suspect milk tested before allowing it into the bulk tank.

6. Mechanical failure such as leaking valves has been known to allow up to 400 mls of milk to be accidentally drawn into the system.

7. 'Unknown cause' of antibiotic failure still accounts for nearly 10% of cases and is very perplexing and worrying. Several small, apparently insignificant factors may contribute to some of these cases and it is then always essential to review the procedure for ensuring that residues cannot pass into the bulk supply.

Summary of measures necessary to ensure that bulk milk is residue free:

1. All cows must be accurately identifiable.

2. All treated cows MUST be clearly marked with a red mark for 'DANGER'

3. All medicines used MUST, by law, be recorded and withdrawal times in hours, not number of milkings must be followed.

4. Cows which calve prematurely or which have short dry periods should be sampled and tested before the milk is allowed to leave the farm.

5. When contaminated milk is known to have been accidentally transferred the Board's regional office should be informed before the milk is collected.

6. NEVER gamble with stated withdrawal periods. They are there to protect you and the customer.

7. Ask your veterinary surgeon to clearly state the withholding periods for the medicines which he prescribes and supplies. This is particularly important for one off treatments which he or she administers.

Conclusion

Economics more than public health considerations dictate that milk should be sold to the consumer free of antibiotic residues. At least 0.3% of farms in the UK will fail the test for residues this year. The main cause would appear to be the accidental transfer of milk from an unmarked treated cow. No one can afford even one failure and the consumer must continue to believe in the wholesomeness of milk.

A well tried routine is the only way to ensure that the benefit of any therapy is not overshadowed by severe financial penalties.

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SUMMER MASTITIS: EPIDEMIOLOGY, AETIOLOGY, PREVENTION AND THERAPY

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Introduction

Summer mastitis is an acute bacterial infection of the non-lactating mammary gland from which Actinomyces (formerly Corynebacterium) pyogenes, Peptococcus indolicus and sometimes other pathogenic bacteria are isolated (6). The inflamed quarter is usually lost for milk production. There is strong circumstantial evidence for the role of the sheep head fly (Hydrotaea irritans) in the transmission of the disease (1).

Epidemiology

A good definition of summer mastitis is required for the epidemiology. Although the definitions vary, one common point is made: summer mastitis is an acute mastitis found in the summer months in pastured, non-lactating cattle, i.e., dry cows, heifers and calves. The bacteria isolated are usually Actinomyces pyogenes, Peptococcus indolicus and sometimes other anaerobic bacteria (6). On top of this definition there are extensions and limitations. In the United Kingdom, newly calved cattle from which A. pyogenes is isolated alone, or in combination with other pathogens (5) are also included. In Sweden (8) it is limited to "inflammatory reactions developed in the udder of female cattle in the period from the onset of puberty to a fortnight after first calving". Therefore they refer to heifer mastitis. Dry cows are excluded in this definition. Some countries (U.K., Sweden and Denmark) also include in summer mastitis cases of mastitis caused by A. pyogenes in dry or newly calved cattle in the wintertime (1,8), which can be a problem (7).

There is strong circumstantial evidence (1,16) that the fly, Hydrotaea irritans, is the transmitter of summer mastitis, or that this fly plays at least an important role in the transmission. On the other hand, in Japan, Hamana (4) describes a clinical picture which resembles highly summer mastitis; the same bacteria were isolated, it was also seen mainly in the summer in heifers and the use of insecticides or intramammary infusions of long acting antibiotics had a good preventive effect. In Japan non-blood-sucking flies and other insects are considered to play a major role in the transmission of the pathogens. Mention has been made of Stomoxys calcitrans, but not Hydrotaea irritans which is not found in Japan (4).

The rest of this paper on summer mastitis will be limited to non-lactating, pastured cattle during the summer months.

Vegetation (trees and bushes) is important, but summer mastitis is also seen in open areas without trees and bushes. Summer mastitis occurs primarily on sandy soils (7.2%), less on marshy soil (5.3%) and hardly ever on clay, (13). Summer mastitis may occur year after year on some farms, but is, in any particular year, unpredictable. Similarly, comparison with neighbouring pastures is also impossible (14).

In the Netherlands the peak is usually between mid July and mid August but it seems to be a little later in Ireland (2) and the U.K. (5).

The influence of pregnancy is questionable. There seems to be no influence in the Netherlands (14), but in the U.K. and Ireland (2,5) there seems to be a negative influence, (more summer mastitis in pregnant cattle).

Cows with a high milk yield at drying off are more susceptible to summer mastitis. If there is a calf, heifer or cow which sucks other cattle, the incidence of summer mastitis rises dramatically in a herd.

There is a difference in susceptibility between various breeds (18). There are also heritable differences. Daughters of bulls with a high peak milk flow, are more susceptible, as are daughters of easy milking cows. This is probably the reason why heifers from certain farms contract more summer mastitis than heifers from other farms, at the same time, in the same pasture (14).

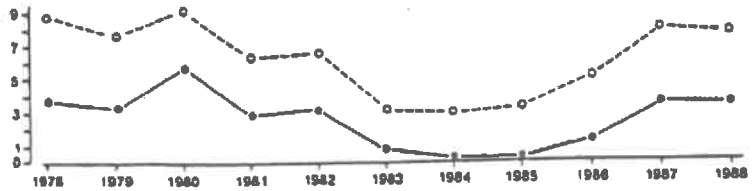
Front quarters are affected twice as much as hind quarters (5,14), whereas mastitis in the lactating cow is more likely to develop in a hind quarter. In about 90% of summer mastitis cases just one quarter is inflamed (5,11,14). The incidence of summer mastitis varies between years as can be seen in Table 1.

Table 1: Summer mastitis in heifers on 198* farms in Overijssel, The Netherlands

Year	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989
No. heifers (1-2 year)	3,290	3,706	3,959	3,852	3,989	3,957	3,912	3,780	3,468	3,611	3,676
% summer mastitis	9.5	10.0	3.4	4.1	1.1	0.2	0.2	0.2	2.16	1.52	1.55

*the number of farms decreased to 185 in 1989.

Reichmuth (10) saw the same tendency in Schleswig-Holstein in North Germany (Fig 1).



● % of heifers with summer mastitis in all questionnaired farms

○ % of heifers with summer mastitis only in the questionnaired herds with summer mastitis

The highest incidence is seen in dry cows in The Netherlands (Table 2).

Table 2: Incidence rate of summer mastitis on 815 farms in 1989 in Overijssel, The Netherlands.

	Number	% summer mastitis
Dairy cows	8,885	0.42 (2.1)*
Heifers (1-2 year)	3,676	1.55
Calves (below 1 year)	3,652	0.08

* of all dairy cows only 20% are dry during the summer time and thus at risk.

Aetiology

Actinomyces pyogenes and Peptococcus indolicus are, in combination with anaerobic bacteria, considered to be the most important bacteria in summer mastitis (5,18). However in The Netherlands it was shown to be possible to experimentally produce a clinical picture resembling summer mastitis in heifers using combinations of bacteria without A. pyogenes (19). In challenge experiments in the U.K. (6) it turned out to be very easy to infect a dry quarter with A. pyogenes or P. indolicus. However, teat end damage was found to be necessary before experimental contamination of the teat with A. pyogenes resulted in cases of severe clinical mastitis (12). In Germany, it is considered that a subclinical A. pyogenes mastitis in heifers can become clinical after stress caused by a heavy fly load (18). Wounds on the teats are very attractive for the sheep head fly (Hydrotaea irritans).

There is strong circumstantial evidence that Hydrotaea irritans is a transmitter of summer mastitis (1,16). Recently (1989) successful experimental transmission using Hydrotaea irritans was achieved in Sweden by Chirico c.s. (Thomas, personal communication).

It is now possible to culture the fly, Hydrotaea irritans (9) and there is much more known about the conditions which attract the fly to cattle (17). This knowledge has been used to develop attractive traps for studying the behaviour and the density of the fly at various times during the day and season. In the Netherlands this year, we started a Hydrotaea irritans counting program. Flies will be counted every two weeks in several places in summer mastitis areas. The results of these counts will be compared with the summer mastitis incidence in the same areas. If there is a relationship, we intend to develop the fly traps so that predictable monitoring of the flies can be carried out using this method - an early warning system.

Prevention

The loss caused by summer mastitis is large and has been estimated to be as high as £17 million in a bad year in the U.K. The minimal yearly loss in The Netherlands is £1.5 million. Every summer mastitis case will cost the farmer about £400. It is not possible to predict the yearly summer mastitis incidence in the country, or on a farm. Over the last few years the incidence rate has been about 2% (Table 1). This means an average loss of £800 per 100 heifers. A preventive treatment with two insecticidal ear-tags will cost in the Netherlands, roughly £4 per heifer and for 100 heifers £400. But despite the use of ear-tags, outbreaks of summer mastitis still occur (Table 4). In risk areas most farmers usually carry out preventive measures, although this is possibly not economically justified.

Prevention may consist of:

1. Zootechnical precautions

Examples of this include keeping the animals in the stall, as Hydrotaea irritans is only found out of doors, or using pastures where summer mastitis is rare, e.g. on clay soils. Dry cows are also held indoors more, partially to prevent fat build up from a too large food availability.

2. Protection of the udder by using long acting antibiotics

Forty years ago, Pearson (15) obtained good results with long acting antibiotics. Pearson pointed out that it was remarkable how often cases of mild infection occur in dry cows. This may possibly be a predisposing factor for the occurrence of summer mastitis, but this could not be confirmed in a later experiment in The Netherlands (12).

Table 3: Comparison of summer mastitis in weeks after intramammary treatment of 2901 heifers with 250 mg cephalonium (13).

Weeks after treatment	1	2	3	4	5	6	7	8	>8	date unknown
Cases of mastitis										
Treated	-	-	-	1	1	3	6	6	17	
Control	3	9	1	5	6	15	7	9	21	16

Beimgraben (18) also obtained good results with long acting antibiotics. He recorded the first cases at least 44 days after treatment. Hamana (4) recorded the first cases at 68 days and 75 days after treatment. This is markedly longer than the findings of Sol (Table 3, 13) and Hillerton (5). Edmonds and Welch (5) found that one treatment with a long acting antibiotic was insufficient to cover the dry period completely. Treatment with long acting antibiotics is too labour intensive and difficult for calves and heifers, but is superior for dry cows and even preferable to ear-tags. However, there should be a second administration 3 to 4 weeks after drying off, but plenty of time before calving.

3. Protecting the teat from H. irritans

ODOURS: the use of strong smelling odours to repel flies is an old method which is now declining. One of the best known is Stockholm tar. The results are doubtful (13) and its application is problematical, particularly with rather wild heifers. Another method is to introduce a billy-goat into the herd of heifers. This has been done by some farmers in the Netherlands, but again the results are doubtful.

TEAT SEALING: Danish reports (18) indicate positive results with "sealing" of the teats. This has also been done in Holland with plasters (11) without good results.

INSECTICIDES: these are toxic and often repellant. Insecticides may be applied in several ways, e.g.:

a) dust bags: good results have been achieved with this method in the Netherlands (15), Japan (4) and Germany (18). The animals must walk under the dust bag frequently. For this reason the system is suitable for cows that are milked in the stall and can be forced to walk under the bag on leaving.

b) sprays: a large variety have long been available and used with success in preventing summer mastitis (18). Their method of working and efficacy supports the hypothesis that flies play an important role in the occurrence of summer mastitis. The results obtained with sprays are good, given that they are applied sufficiently early, frequently and accurately (18). However, results can often be disappointing (3,13). Yeoman and Warren (15) also obtained poor results on dry cows which were regularly treated.

c) "the anti-summer mastitis drinking box": animals using this system in the Netherlands (13) are automatically sprayed during drinking.

d) pour-ons: they are effective from 4 to 6 weeks and usually contain synthetic pyrethroids. They appear to be successful in reducing summer mastitis. Pour-ons and sprays are the most appropriate treatments for preventing summer mastitis in calves, as they are usually given additional concentrates daily and are easy to treat at this time. The same applies to milk cows where pour-ons are a potentially attractive method for controlling flies.

e) ear-tags: the incidence of summer mastitis decreases when ear-tags are used and the results are comparable to the effects obtained with long acting antibiotics (13). Since 1982, ear-tags with different active compounds have become available in Holland. The efficacy appears to be high, but the incidence of summer mastitis has been very low since 1982 (see Table 1). Occasionally a severe outbreak has been observed despite the use of ear-tags. The infections reported in ear-tagged heifers in the summer are often caused by bacteria which are not typical for summer mastitis (13). Yeoman and Warren (15) also found that, when using insecticides, a change occurred in the type of infection, in that significantly far fewer C. pyogenes could be isolated. This change was not seen when antibiotics had been used. After using ear-tags there also seems to be relatively more infections in the rear udder, although this is not significant. Usually summer mastitis cases are more common in the fore udder. This may indicate an exogenous influence in summer mastitis and an endogenous one in other udder infections during the summer.

Table 4: Method of prevention and percentage of summer mastitis on 356 farms in Overijssel, the Netherlands, in 1989.

	Number of heifers	Number of farms	% summer mastitis
No prevention	754	42	2.39
Ear-tags	5,176	244	1.31
Pour-ons	746	39	1.34
Keeping inside	350	22	0.00
Sprays	149	7	2.01
"Anti summer mastitis drinking box"	38	2	0.00
Total	7,213	356	1.37

Table 4 shows that only 11.8% of the farmers did not carry out preventive measures.

One marked difference between use of long acting antibiotics and ear-tags is the latency of the first summer mastitis cases after treatment. With long acting antibiotics, no cases are seen until 3 to 4 weeks later, whereas with ear-tags these may occur very soon after treatment.

It is also possible to utilize ear-tags as a neck band. If prevention of summer mastitis is the underlying reason for application of ear-tags, then it is recommended to use two tags per animal (13).

Furthermore, it is essential to wash the hands thoroughly after insertion, as several farmers have complained of headaches following treatment of animals. Although resistance has not yet been recorded in the Netherlands it has become a problem in the U.S.A., particularly in areas where cattle flies have more than one cycle annually. To prevent the build up of resistance, it is recommended to remove ear-tags when animals are brought into the stall at the end of the season.

Approximately 200-250,000 heifers are treated with ear-tags annually, in the Netherlands. There appears to be very little difference between the efficacy of the different ear-tags available on the market.

Therapy of summer mastitis

Despite therapy many cases of summer mastitis result in loss of the quarter as far as milk production is concerned. The best results that have been obtained so far are those of Heidrich and Fiebiger in 1964 and 1965 (11). The therapy was carried out on 52 dry and lactating cows in which bacteriological examination confirmed the presence of a A. pyogenes infection. Therapy consisted of intramammary treatment with proteolytic enzymes (fibrolease and desoxy-ribonuclease) and antibiotics plus, in severe cases, intramuscular or intravenous injection of antibiotics. The udder tissues of 48% of the animals recovered completely and 27% partially. The quarter was completely lost in only 25% of the cases. Complete recovery could also be obtained with 4-6 week old infections in 35% of the quarters.

Saes (11) used 20 cc intramammary injection of dimethylsulphoxide to which the proteolytic enzyme trypsin was added to treat 30 milk cows in which a A. pyogenes mastitis had been confirmed. Occasionally, additional treatment with intramuscular injection of antibiotic was carried out. Complete recovery was found only in 3 cases.

Buscher (18) made use of the foam injection Ubrocelan^{R} (Boehringer) with 63 summer mastitis heifers. Injection was carried out every 24 hrs and quarters were stripped frequently during the last half of the inter-injection period. Complete functional recovery of the udder tissue was found in 29 of the 63 animals.

Therapy trials were carried out in the Netherlands on 29 and 19 acute mastitis cases in pastured animals in 1984 and 1985, respectively. The standard therapy in 1984 consisted of an intramuscular injection of 20% Spiramycin (Suanovil 20^{R}, Rhone Poulenc) on the first and third day. An intramammary treatment with an injector containing a spiromycin combination (Mammivert^{R}, Rhone Poulenc) was also applied on 5 consecutive days. In 1985 the standard therapy consisted of an intramuscular injection of 6 million IU Na-pen G and 10 million IU procain benzyl penicillin on the first day. An intramammary treatment with an injector containing 200 mg cloxacilline and 75 mg ampicilline (Ampliclox^{R}, Beecham) was applied on the first 5 consecutive evenings.

It appeared that the hardness of the infected quarters was a very important factor for the prognosis. Usually the quarter was lost if the udder was hard and swollen and in all but one case A. pyogenes and/or obligate

anaerobic bacteria were isolated. On the other hand, in the soft, swollen quarter group, only one udder did not heal completely. In this case, a S. uberis infection was involved. In the soft swollen group no A. pyogenes and/or obligate anaerobic bacteria were isolated. Very little difference was found between the two treatments except that there appears to be a slight tendency that spiromycin has better effect if the infection belongs to the soft quarter group.

The clinical aspects of the udder and the general condition of the heifer are the most important indicators for the prognosis. When the heifer has a systemic illness and/or the secretion has a distinct odour and/or the udder is hard and swollen (independent of the causative bacteria) the quarter will be usually lost, irrespective of therapy.

When the udder is soft, oedematose and swollen, the prognosis is usually good. Farmers generally refer to these cases as being summer mastitis infections which are discovered very early. This is almost always incorrect as bacteriological investigation usually reveals species which are atypical for summer mastitis, such as S. uberis and other streptococci and a staphylococci.

Application of a therapy is important in preventing the animal from becoming or remaining sick. In all cases where therapy was applied, the animals become clinically healthy although, in most cases, the udder tissue is no longer functional. It is necessary to bring the sick animal into the stall for efficient therapy. This also ensures that a source of the infection is removed from the pasture. Excision or incision of the teat is also possible. If the teat has been removed, it is, however, essential not to replace these animals in the pasture as secretion containing A. pyogenes can continue to leak from the udder, thus causing a large infection source.

It is necessary to inspect the remainder of the herd thoroughly, as in 35% of the cases, a new summer mastitis case will either occur directly within a few days of the first case (18).

If the teat remains swollen for several weeks and the sick quarter contains hard lumps, then the prognosis is usually poor.

Diagnosis and prognosis is more difficult in cows. According to Weitz (15) there is little difference between a summer mastitis udder infection and one caused by S. dysgalactiae as far as the clinical picture is concerned, but the prognosis for a S. dysgalactiae case is better. For this reason it is important to carry out bacteriological tests.

Cows often become seriously ill and can also die from summer mastitis infection. They often lie, as they will not, or cannot, stand. Infected heifers are readily identifiable as they usually stay isolated from the herd and walk rather stiffly. It is rare that heifers cannot stand. Calves are usually the least sick.

The best therapy is to treat the animal intramuscularly with antibiotics, strip the quarter frequently during the day and use an injector on the sick quarter in the evening. The therapy must be continued for at least 5 days.

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PRE-MILKING TEAT DISINFECTION

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Introduction

Post-milking teat disinfection has been shown to reduce new intramammary infections during lactation by 50% Mastitis Field Experiment 3(1). In that 30 herd experiment (MFE3) there was found to be wide variation in the rate of new intramammary infections between the herds. Differences in herd management systems probably contributed to this variation (2). Since the widespread introduction of post-milking teat disinfection, infections from Staphylococcus aureus and Streptococcus agalactiae in particular have been dramatically reduced, Figure 1. However, the pathogens termed environmental, Streptococcus uberis and Escherichia coli have become the major problem in some herds during the housing period (3,4,5). It should be understood that in many herds, although the greater percentage of clinical mastitis is due to the environmental pathogens, it is due to the fact that the other pathogens have been reduced by post milking teat disinfection and improved milking machines.

Studies in the U.S.A. into use of a disinfectant teat dip before milking, in addition to the usual post-milking disinfection, appear to reduce the rate of new intramammary infections caused by the environmental pathogens by up to 50%. This could amount to an annual reduction in the U.K. of perhaps 250,000 cases of mastitis costing some £12.5 million. It is claimed that pre-milking teat disinfection is being applied in some 18% of dairy herds in the U.S.A.

Udder Preparation

Preparing the udder before milking has several functions, primarily it is to clean the teats in order to produce milk with minimal faecal contamination. Secondly it is to stimulate the cow to let down her milk. To obtain the first, cleaning the teats would suffice, the latter involves the massage of the udder as part of a preparation routine. As herd sizes have grown, time has been saved by reducing udder preparation until at present udder preparation is uncommon. Stimulation relies on the action of the milking machine, or as a conditional reflex to the milking routine.

The object of cleaning is to reduce the bacterial population on the teats, resulting in a lower total bacteria count (TBC) in the milk (6). Using cows free from intramammary infection, McKinnon et al (7) examined bacterial contamination of milk from cows on three different bedding conditions, a) minimal straw, b) minimal sawdust and c) well strawed. He reported that contamination of the bulk milk was least where teats were washed and dried, compared with no preparation. However, he does state that there were wide variations within the treatment groups.

In 1983, a within herd experiment on eight commercial herds compared udder washing and drying with no preparation. McKinnon et al (8) examined the contamination of the milk from cows on both treatments in all eight herds. During the winter housing period, washing and drying of the teats prior to milking showed a significant reduction in bacterial contamination of the milk. The difference of 40% between the treatments was half that found in the earlier short term trial (7). During the summer months no difference was found between washed and the no preparation groups.

In 1982 the MMB (9) carried out a survey of udder washing methods and their influence on TBC and average annual somatic cell counts. This survey of 707 herds indicated that at least during the summer months (June to October), herds dry wiping the teats had the lowest TBC and equal lowest average cell count. The poorest results were from herds using a bucket and cloth wash system.

TABLE 1

M.M.B. Survey 1982 June - October (9)
Method of udder preparation on TBC and Annual Cell Counts

Udder Preparation Method	Average TBC (X 1,000)	Annual Av:Cell Count (X 1,000)
Dry Wipe only	14.3	405
No preparation	26.7	404
Wash all/running water/dry	18.9	490
Wash dirty udders/dry	19.9	424
Wash dirty udders/no dry	20.4	463
Wash all/water/No dry	23.2	495
Wash dirty udders/bucket/cloth	28.5	558
Wash all/bucket/cloth	39.2	682

notes: not all herds were using a disinfectant in the udderwash.
TBC result - average of the month prior to visit.

Pre-Milking Disinfection

F.H. Dodd and colleagues (10) reported on the results of swabs taken from the teats of cows on the first mastitis field experiment (MFE1). The results showed that the application of a hygiene routine using disinfectants, including post-milking disinfection, compared to no hygiene, reduced the bacterial contamination of the cows teats prior to the following milking. The herds on the hygiene routine showed a 50% reduction in new lactation infections (11). From this it may be accepted that the level of exposure influences the rate of new infection, this would be influenced by udder preparation.

Pre-milking disinfection means the application of a specially formulated disinfectant, to each teat after the udder preparation sequence. A minimum of 20s contact time has to be allowed before dry wiping and attaching the milking units. The final dry wipe is necessary to remove any droplets of the disinfectant and so minimise the residue effect in the milk.

To date most of the work on pre-milking teat disinfection has been carried out in the U.S.A. and the results given in this paper are based on that work. In this country, work has recently been carried out by the Institute for Animal Health (Compton) on several herds in the South of England. The preliminary results have encouraged further work for the coming winter months involving at least 20 herds.

Pankey et al (12), have carried out trials with pre-milking teat disinfection as a method of controlling the environmental pathogens. By reducing the numbers of pathogens on teats immediately prior to milking the chance of a new intramammary infection is reduced.

In the trial, Pankey used four herds (52 to 115 cows) and two different disinfectants (Tables 2 and 3); an iodophor at two concentrations (0.25% and 0.1% available I_2)and a mixture of an iodophor at 0.55% available I_2 with linear dodecyl benzene sulfonic acid, 1.9%. The study lasted for 12 months on three herds and 6 months on the fourth. The pre-dip treatment was applied to half the cows, in each herd. The results showed a reduction in new infections in the order of 50%. Post-milking disinfection was carried out using the same disinfectant as pre-milking.

TABLE 2

New intramammary infections in 12 months
pre-milking disinfectant 0.25% available Iodine (two herds)(from ref. 12)

Treatment	No. Qtrs at risk	New Infections Staph	Aesculin +ve Streptococci	E. coli	Total Infections Major	(Env)
Pre-dip	243	0	13	5	18	18
Control	204	5	23	11	39	34

% Infections per quarter

	Total major infections Quarters	Reduction	Environmental* Quarters	Reduction
Predip	7.4	61.3	6.0	54.2
Control	19.1		13.1	

* environmental pathogens = Streptococcus uberis and E. coli

TABLE 3

New intramammary infections in 12 months
with a pre-milking disinfectant (two herds)(from ref. 12)

Treatment	No. Qtrs at risk	New infections Staph	Str. agalactiae	Aesculin +ve Streptococci	E. coli	Total Infections Major	(Env)
[1] Pre-dip	137	3	0	2	7	12	9
Control	137	4	0	6	12	22	18
[2] Pre-dip	239	3	0	3	9	15	12
Control	212	2	3	2	18	25	20

[1] 0.9% linear dodecyl benzene sulfonic acid + 0.55% iodophor
[2] 0.1% iodophor

% Infections per quarter (two herds)

	Total major infections Quarters	Reduction	Environmental* Quarters	Reduction
Predip	7.2	46.3	5.6	48.6
Control	13.4		10.9	

* environmental pathogens = Streptococcus uberis and E. coli

Disinfectant

In the U.S.A. the disinfectants used in pre-milking disinfection include Iodophors and Hypochlorites. A problem of using a concentrated disinfectant immediately before milking, is the possible increase of that disinfectant entering the milk either directly or from absorption through the skin and subsequently into the milk. Evidence will be required to show that an increase is minimal and will not affect the overall levels of the chemical in the milk.

An aspect of application which needs to be carefully assessed is the use a sprayer. Spraying is not the most efficient way of disinfecting teats added to the fact that disinfectant is released into the atmosphere (13). The combined use as a pre-milking applicator will further increase atmospheric contamination. Any possible increased risk of inhalation by the operator will need to be measured.

Iodine in the milk associated with post-milking disinfection has been considered by various workers (14,15,16). In 1974, Dodd et al (16) measured the iodine levels in the bulk milk of 30 herds, prior to and after the introduction of iodophor teat dips, Figure 2. A wide variation in the total iodine levels was found between the herds from the dietary intake. Samples of the bulk milk were taken 14 to 21 days after commencing teat dipping. The iodine levels rose from a mean of 26 μg/100 ml (range 9 - 49 μg/100 ml) to 38 μg/100 ml (range 16 - 86 μg/100 ml). The increase varied considerably, with three herds showing a lower level of iodine in milk after the introduction of the disinfectant dip. After using the iodophor dip, 23 of the herds were still below the starting level of the highest herd.

Although little data is available, it is likely that the changes in iodine levels in milk following the introduction of pre-milking teat disinfection will also vary considerably from herd to herd.

Total Bacterial Count

As indicated in Table 1, the method of udder preparation can influence the bulk milk TBC. Furthermore, if the teats are thoroughly disinfected and wiped off with a clean paper towel prior to milking, then the levels of the TBC in the bulk milk will fall. This will only be true if the standard of cleanliness of the teat after the final wipe is better than the previous preparation routine. Galton et al (6) showed that pre-milking teat disinfection with a teat "dip", reduced the TBC and that drying of the teats further reduced bacterial counts, including coliforms.

The lowering of the TBC will have a beneficial effect in herds which are in quality bands other than Band A. Those in band A will gain an additional "weapon" against a lowering a high standards.

Further evidence of the reduction in TBC's is required to substantiate these findings in particular with trials on the disinfectant pre-dip finally developed.

Discussion

Good udder preparation before milking reduces bacterial contamination. However it does not eliminate all pathogens from the teat and can still allow contamination from hands to pass from cow to cow. The main reason for pre-milking teat disinfection is to further lower the level of mastitis pathogens on the teat, in particular the environmental bacteria. In addition, other pathogens may be reduced thus enhancing mastitis control overall. It has been shown in the U.S.A. that pre-dipping works, however, it must be stressed that under experimental conditions, routines are usually carried out to a higher standard. The results of the I.A.H. trial, carried out under commercial conditions, are encouraging and new trials are planned. The additional work routine will add to the time taken to milk the herd, but the returns in those herds with an environmental mastitis problem, will outweigh the extra time spent, provided the routine is carried out properly.

Carrying out any routine which is below the required standard will not give the best results, and if results are not up to expectations lead to the routine be dropped altogether.

Good teat skin condition is paramount in keeping bacterial levels lower (17) and makes for easier cleaning before milking. A build up of contamination on the teat skin leads to peeling of the epidermal layer and in extreme cases open lesions.

Further research over the winter 1990-91 should see a significant advance in recommending a routine including a pre-milking teat disinfectant dip.

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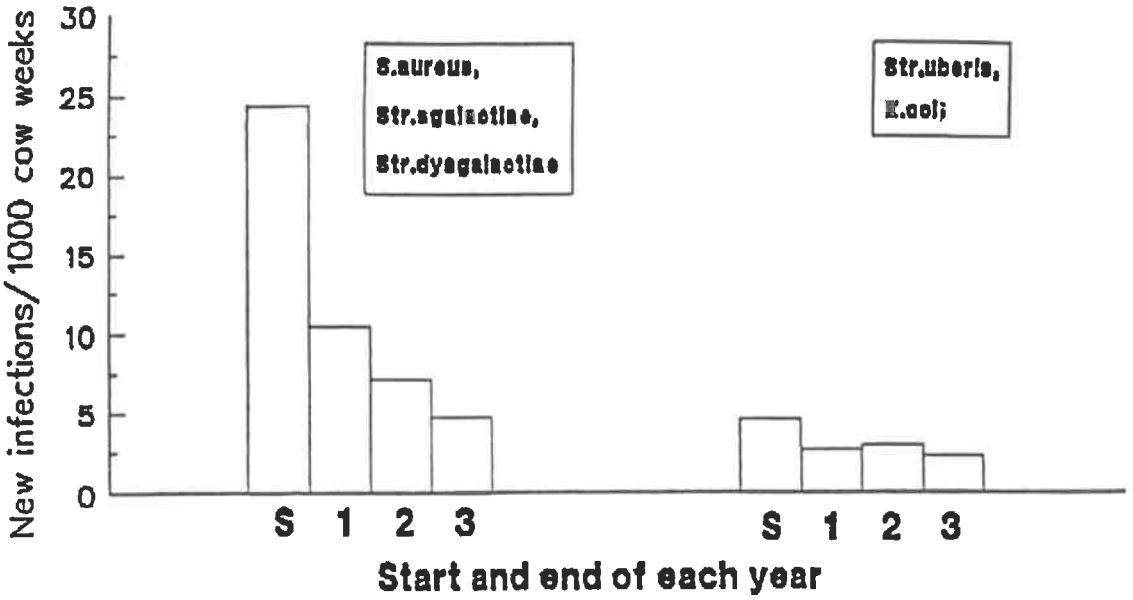
Figure 1

Mean levels of infection in cows in milk in 30 herds at he beginning of the experiment and after 1, 2 & 3 years. MFE3



Incidence of clinical mastitis

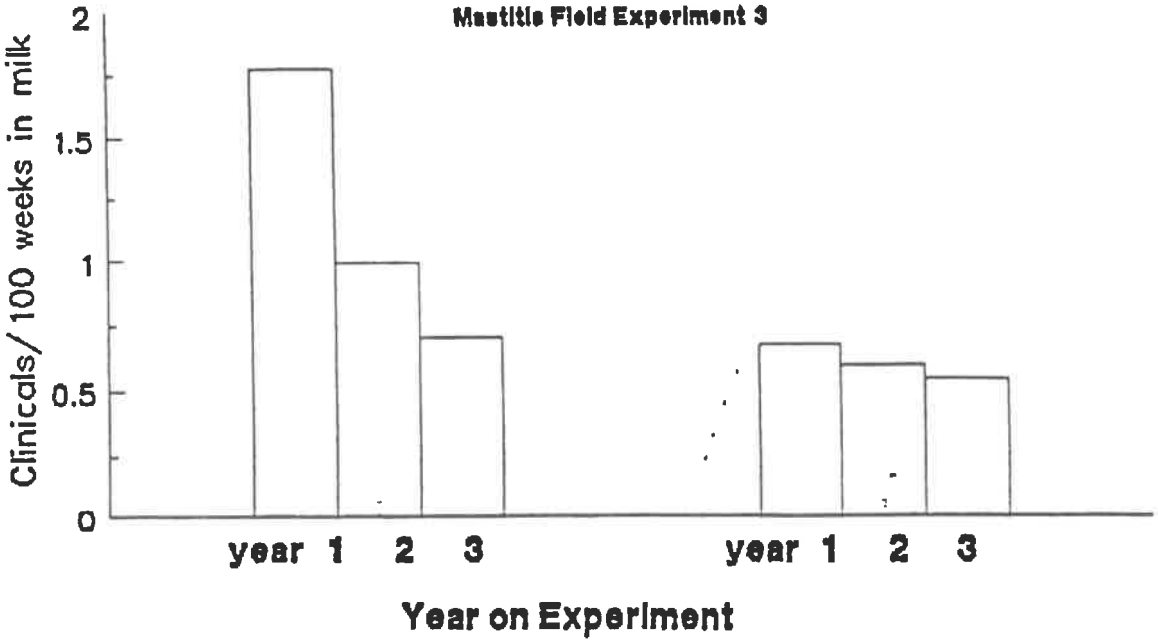
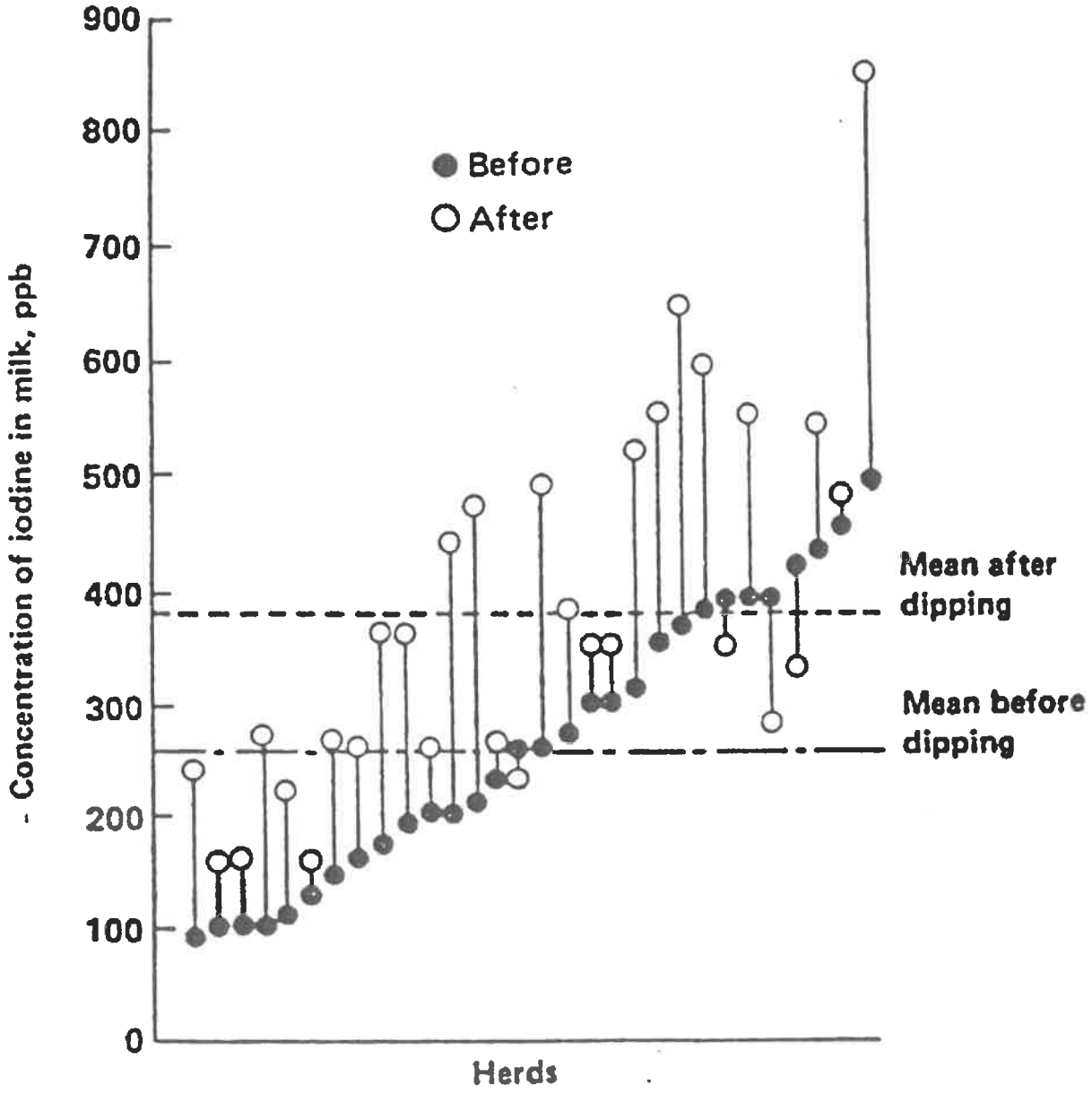


Figure 2

Mean iodine levels in milk of 30 herds
on winter rations in England and Wales
before and after using iodophor teat-dip



Herd variation in iodine level from dietary sources

MASTITIS IN SHEEP

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Introduction

Ovine mastitis was first systematically investigated in the U.K. by Leyshon in 1929 who described all the major causes of the disease which we recognise today and estimated the incidence (in the Eastern counties of England) to be 6%. Since then, there have been few investigations of the disease - most reports concentrating on estimations of prevalence by post-weaning (Gibson and Hendy, 1976; Hendy, Pugh, Harris and Davis, 1981; Watson and Buswell, 1984) or abattoir (Herrtage, Saunders and Terlecki, 1970; Madel, 1981) examinations. Unlike bovine mastitis, there is a paucity of information on ovine mastitis which means that many important facets of epidemiology and pathogenesis are not understood and adequate control measures are often not available.

The classification of complex diseases such as mastitis are often arbitrary and artificial. However, certain differentiations need to be made if the condition is to be understood. Subclinical and clinical mastitis will, therefore, be considered separately although it is likely that they are closely linked epidemiologically. Mastitis in dairy sheep will be considered separately.

CLINICAL MASTITIS

Incidence

Because sheep, unlike dairy cattle, are not examined regularly, there is no simple way to estimate the incidence of mastitis. Estimates based on the number of clinical cases detected by the shepherd are inadequate, as only ewes with severe systemic signs of infection are likely to be detected. The most accurate and easy to perform estimation of incidence is the examination of ewes' udders after weaning - a practice which is normally performed by farmers as part of the annual pre-tupping selection of cull ewes.

This is made possible by the fact that even "mild" mastitis in sheep results in permanent, palpable damage to the mammary gland - usually in the form of one or more abscesses with thick, fibrous capsules. Such lesions often preclude lactation in subsequent seasons and so ewes are often culled as a consequence. In a survey of over 30,000 lowland ewes in the South of England the incidence of mastitis, based on post-weaning examination, was 3.7% and 5.5% in 1986 and 1987 respectively, with more than 10% of ewes affected in 10% of flocks. Interestingly, this was more than twice the number of affected animals which had been detected by the shepherd during lactation (Jones, Lanyon and Watkins, unpublished data). The significance of this will be discussed later.

In a smaller survey of mastitis in hill ewes in 1988, again based on post-weaning examination, the incidence of mastitis was less than 1% (Watkins and Jones, unpublished data).

Aetiology

The relative importance of bacteria causing mastitis in sheep was determined in a 3-year survey conducted at the Royal Veterinary College, the results of the first year were reported by Jones (1985). In each year, two bacteria predominated, namely *Pasteurella haemolytica* and *Staphylococcus aureus* (see Fig. 1). Whilst the latter has been well documented as a cause of mastitis in sheep, goats and cattle, the importance of *P.haemolytica* was

a new finding. This organism is an important cause of mastitis in Northern Europe, Western U.S.A. and New Zealand, which adopt husbandry practices similar to our own. However, it is of lesser importance in parts of the world where sheep are kept largely for milk production and in British dairy sheep.

Other bacteria which have been isolated from ewes with mastitis include *Escherichia coli*, *Streptococcus* and *Bacillus* spp and coagulase-negative staphylococci (C-NS).

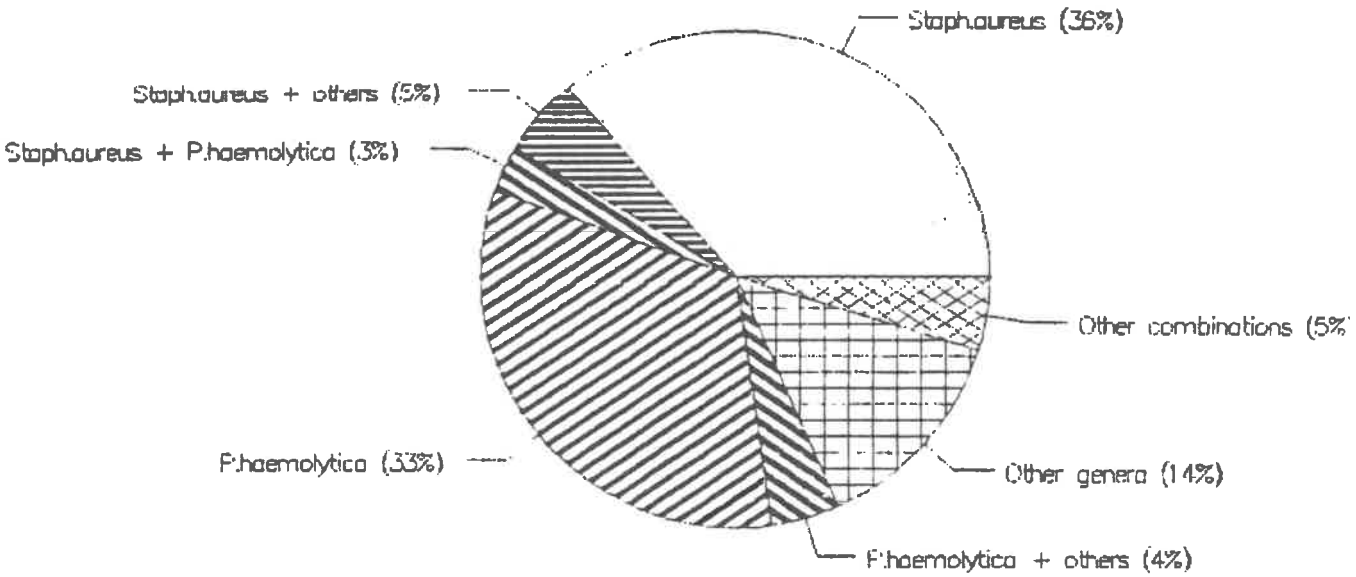

Fig.1. Bacteria isolated from the milk lowland ewes with clinical mastitis.

Epidemiology

It has become customary to divide ovine mastitis into an acute form, which is often severe and is seen during lactation and a chronic form in whcih there are no systemic clinical signs and which is usually seen after weaning. However, if ewes are examined on the day of weaning and then again three weeks later, very few cases of mastitis are found and it is now clear that chronic (or mistakenly termed "post-weaning" mastitis) is merely a sequel of acute mastitis which commenced during lactation and which may not have been detected. Acute and chronic mastitis are often, therefore, manifestations of the same disease incident, merely detected at different stages in the production cycle.

Most cases of mastitis caused by *P. haemolytica* or *Staph. aureus* occur in the third and fourth week after lambing whilst mastitis due to environmental organisms occurs mainly in the first week after lambing, when ewes are still in the lambing sheds.

The epidemiology of mastitis caused by *Staph. aureus* and environmental organisms has been extensively studied for dairy cattle and much of what is known for them also applies to sheep. For example, *Staph. aureus* is widely present on the skin of sheep and particularly infects teats, sores, cuts or orf lesions which increase the likelihood of bacteria penetrating the teat

duct. In the RVC survey, there was a positive association between the presence of teat sores and mastitis caused by *Staph. aureus.*

The epidemiology of mastitis caused by *P. haemolytica* has warranted special attention by our group, as it is only an important cause of mastitis in sheep. T biotype stains are rarely isolated from ewes with mastitis but the distribution of serotypes of the A biotype is similar to that documented for ovine pneumonic pasteurellosis (Fraser, Gilmour, Laird and Donachie, 1982) and carriage in the upper respiratory tract of lambs (Shreeve and Thompson, 1970). This similarity has led to the hypothesis that the upper airways and oral cavity of lambs are the source of mammary infection of ewes. In addition, there is epidemiological evidence that if lambs are removed and ewes milked by hand or machine, as happens in British sheep dairying and many overseas husbandry systems, *P. haemolytica* rarely causes mastitis. Furthermore, isolates of *P. haemolytica* taken at random from the nasal cavity of lambs and inoculated into the mammary gland of ewes always cause mastitis, even in small doses (Watkins, 1990). However, *P. haemolytica* often colonises teat sores and cuts and may also cause subclinical mastitis. The importance of these features to the epidemiology of *P.haemolytica* mastitis is not clear.

There is no obvious equivalent in sheep to summer mastitis of cattle. New intra-mammary infections rarely occur in the dry period, most of those detected at that time are the irreversible pathological sequels to mastitis that commenced during lactation. The ability of bacteria to survive in the dry gland and the fate of intra-mammary infections at weaning has only been studied for *P. haemolytica* (Watkins, 1990). An A9 *P. haemolytica* isolate which causes severe, acute mastitis when inoculated in small numbers into the lactating gland produced only mild, transitory mastitis when inoculated three weeks after weaning and the orgnaism did not survive for more than xx days in the dry gland. Isolates of *P. haemolytica* from ewes with subclinical mastitis produce similar infection when inoculated experimentally into lactating mammary glands. These infections, however, are promptly eliminated at weaning. The inability of *P. haemolytica* to survive throughout the dry period is important in understanding the epidemiology of infection. A knowledge of the ability of other mastitis pathogens, particularly *Staph. aureus* and C-NS, to survive in the gland during the long dry period in sheep is required to understand the epidemiology of these infections.

SUBCLINICAL MASTITIS

Definition and diagnoses

Subclinical mastitis may be defined as the presence of inflammation within the mammary gland, usually due to infection, in the absence of clinical signs. There have been few studies of the incidence and epidemiology of subclinical mastitis in sheep and widely recognised diagnostic criteria have not been defined. In our laboratory, milk samples are considered positive if there is an increased somatic cell count (SCC) and the presence of more than 10 bacterial colonies in a 0.01ml sample in milk. The upper limit of normality of the SCC (as measured by the Coulter Counter) of ewes' milk is 1 x 10^6 (El Masannat,1987; Fthenakis, 1988) and Whiteside and California mastitis test scores of greater than "+" are diagnostic of increased SCC (El Masannat, 1987).

Incidence and aetiology

The incidence of subclinical mastitis is usually higher than that of the clinical form of the disease and in some flocks may be as great as 40% (El

Masannat, 1987). In survey of 358 lowland ewes distributed amongst 7 flocks the incidence of SCM during the lactation period was 11.7% of ewes and xx% of mammary glands (Watkins, Burriel and Jones, unpublished data). In that survey, the major bacterial isolates were various streptococci species, C-NS, *P. haemolytica* and *Staph. aureus*, in order of descending importance. Previous studies have found C-NS to be the most common cause of SCM (El Masannat, 1987) and the pathogenesis of infection caused by *Staph. simulans* has been studied experimentally (Fthenakis and Jones, 1990).

Physiological and economic consequences of SCM

In dairy cows SCM results in smaller yields of poorer quality milk. In ewes this would be manifested by reduced growth rates of lambs sucking affected glands, especially in early life when the major determinant of growth rate is the amount of milk consumed. Experimental production of SCM by inoculation of a coagulase-negative staphylococci isolate results in reduced yield but no significant changes in the composition of milk. This, in turn, leads to reduced growth rates of lambs and prolonged weaning ages, especially if ewes are infected early in lactation.

Epidemiology

The prevalence of SCM in the flocks surveyed by our group remains the same throughout lactation but increases with the age of ewe. Many of the bacteria which cause SCM are also also isolated from ewes with clinical mastitis. The role of subclinical infection in the epidemiology of clinical mastitis, especially of that caused by *P. haemolytica* is also not known but requires further investigation.

Acknowledgements

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VACCINATING AGAINST MASTITIS

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Mastitis and its aetiology

Very significant progress has been made during the last 20 years in the control of bovine mastitis (Fig 1). However, the

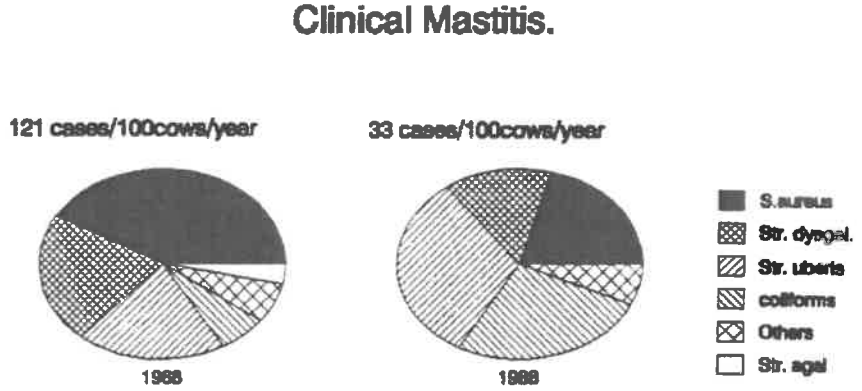


Fig 1.

disease remains economically important to dairy farmers worldwide. Much of the progress can be attributed to improved management, together with a general acceptance by farmers of post-milking teat disinfection and blanket intramammary antibiotic therapy at drying off. It is apparent that these control measures have very effectively reduced the incidence of mastitis caused by the contagious organisms, namely Staphylococcus aureus, Streptococcus dysgalactiae and Streptococcus agalactiae where existing intramammary infections in the herd are the major source of new infections. In contrast, clinical mastitis caused by Streptococcus uberis and coliform organisms, where the environment is the major source of new infections, is not well controlled. Nevertheless, it is generally agreed that diligence in milking parlour hygiene and housing management can help reduce new infections associated with environmental organisms by reducing teat end bacterial challenge.

The present position is therefore, a much reduced incidence of disease, with a movement in the aetiology, away from contagious organisms toward the less easily controlled environmental ones. This situation is also true in the rest of Europe and North America where animal need to be housed for part of the year. In contrast, in areas where animals graze pasture all year, mastitis caused by S. aureus often remains the predominant problem.

There is no doubt that a better understanding of the immunological response of the bovine mammary gland, and type of antigen required, together with the route and timing of its administration continues to make the drive towards an immunological control of mastitis a rational concept. For the reasons outlined above, immunological control of mastitis caused by S. aureus, S. uberis and E. coli is now receiving most attention worldwide.

The concept and requirements of mastitis vaccination

The passage of pathogenic bacteria through the streak canal usually results in clinical or subclinical mastitis. To prevent the disease occurring or reduce the severity or duration, several defence mechanisms can be identified which may be of value either individually or together. These include the prevention of the initial bacterial multiplication in the milk by such mechanisms as antibody mediated complement inhibition or neutralisation of a physiologically important surface component; inhibition of bacterial attachment to epithelial cells; or killing of bacteria after initial multiplication with or without the involvement of leucocytes. A vaccination regimen to induce or enhance some of these mechanisms is required.

Studies on the pathogenesis of different forms of mastitis in ruminants (1,2,3 & 4), have concluded that phagocytosis of bacteria by leucocytes in the mammary gland is the most important natural defence mechanism for bacterial elimination. Normal milk from a healthy bovine udder contains very few phagocytic polymorphonuclear leucocytes (PMN) (5). The system depends on the infiltration of PMN during the inflammatory reaction which follows bacterial infection and subsequent multiplication. The speed with which this occurs affects the ultimate severity and duration of infection.

Another factor crucial for success is that the fluid within the udder is opsonic, ie. the correct type of immunoglobulins are available to coat the bacteria to allow them to be engulfed by the PMN. Ideally pre-inflammatory opsonic activity should be present in the milk, rather than serum derived immunoglobulin which arrives after infection. Opsonic activity can be very specific, with the immunoglobulin only promoting the uptake of the type of bacterium which induced it. Although both IgG2 and IgM have been implicated as important in this process (6 & 7), IgG2 appears to be the important isotype induced by specific vaccine regimes.

What has been advocated is a rapid and efficient inflammatory reaction as a response to infection. Unless this system is tuned to be quick and efficient, a "mastitis is being induced to prevent a mastitis". This principal is not without merit; the replacement of a severe, prolonged clinical episode or a chronic infection with a short, effective inflammatory response which kills all invading bacteria would be welcomed.

Another requirement might be to induce specific immunoglobulins which target bacterial toxins thus reducing their local or systemic effects, since these are often the most important pathogenic determinants of bacteria.

Most people however take the view that if bacterial numbers can be kept to a minimum the toxin problem will be overcome.

Since this is a research update, I shall now review, by bacterial species, some of the more recent techniques and preparations which have been used in vaccination regimes to try to control mastitis in ruminants.

Staphylococcus aureus mastitis

The pathogenesis of this disease in cattle is varied and complex. Very infrequently the bacteria show uncontrolled growth which results in severe, toxin induced, necrosis and finally gangrene. More commonly, PMN infiltration together with natural IgM in non-immune animals, NOT IgG2, (6) controls bacterial growth, but the final outcome is often a chronic infection which is refractory to antibiotic therapy. This insidious form of the disease, which is thought to be maintained in the udder by survival within PMN (8), constitutes the biggest problem with S. aureus mastitis in cattle not only because of milk loss but as a source of new infection to the herd.

Attempts to vaccinate against S. aureus have concentrated on using potential surface virulence factors as antigens, with the express intention of improving phagocytic function and intracellular killing.

Pankey et al (9), found that after vaccination with Protein A during the dry period and regular dipping of the teat in a culture of staphylococci during the subsequent lactation the rate of new infections were the same as for an unvaccinated control group. However, over three lactations infections in the vaccinated animals showed a spontaneous cure rate of 83% compared with a 47% rate in the control animals.

It is now known that S. aureus growing in vivo produce an antiphagocytic capsule around their cell wall (10). It is antibody to this material which is required for effective opsonisation in vivo. Sheep immunised with live organisms developed high titres of IgG2-antibody directed to the capsule, whereas animals given a conventional vaccine of killed in vitro grown bacteria failed to raise antibody (11). Organisms grown in vitro in a nutrient medium supplemented with 10% milk elaborated the "in vivo" capsule. Formalin killed organisms grown by this method together with a toxoided beta haemolysin were used by Watson (12) as an effective systemic vaccine in sheep. Following experimental challenge he found that the incidence of acute gangrenous mastitis was significantly lower in the vaccinated animals. Data from a similar trial in cattle is awaited with interest.

Guidry and his colleagues chose to use a mutant strain of S. aureus (13) which produced exceptionally large quantities of an easily isolated, loosely-attached, capsule. When used as a vaccine the capsular material greatly increased antibody titres which were cross reactive to a large proportion of encapsulated S. aureus isolated from cases of bovine mastitis. Further data from this group is also awaited.

Coliform mastitis

Escherichia coli is responsible for about 80% of the infections of cattle in this category. The pathogenesis of the disease can vary from a mild, rapidly resolving condition to a very severe peracute situation where the cow dies within 36h of infection. The promptness and magnitude of the PMN migration into the gland, which is most efficient in late lactation, appears to be the main factor controlling severity and duration (2). Opsonic activity is rarely a limiting factor; with the exception of a few capsular strains, naturally occurring IgM in milk is present in sufficient concentrations to act as a bacterial opsonin (7). The local tissue damage in the more severe cases which results in permanent agalactia of the infected quarter, systemic complications or even death are caused either directly by bacterial endotoxin (Lipopolysaccharide, LPS) or by the endogenous mediators induced by LPS in mammary tissue which are themselves responsible for pathophysiological effects (14).

Rainard (15), showed an enhanced recruitment of PMN following parenteral and local vaccination with killed E. coli. However, the most successful coliform vaccine to date has been targeted at inactivating LPS and is based on the parenteral administration of the common core portion of LPS produced by rough mutants of E. coli.. This portion of the LPS has been shown to induce IgG antibodies which inactivate endotoxins from all coliform organisms. Although the rational for this parenterally administered vaccine was to reduced endotoxaemia in infected animals; the results of a recent field trial in California (16) showed that vaccinated animals had a much reduced rate of new intramammary infections associated with coliform organisms (Table 1). Interestingly, an identical vaccination regime failed.

Table 1

Relationship between vaccination with E. coli J5 vaccine and clinical coliform mastitis in dairy cows (from Ref 16)

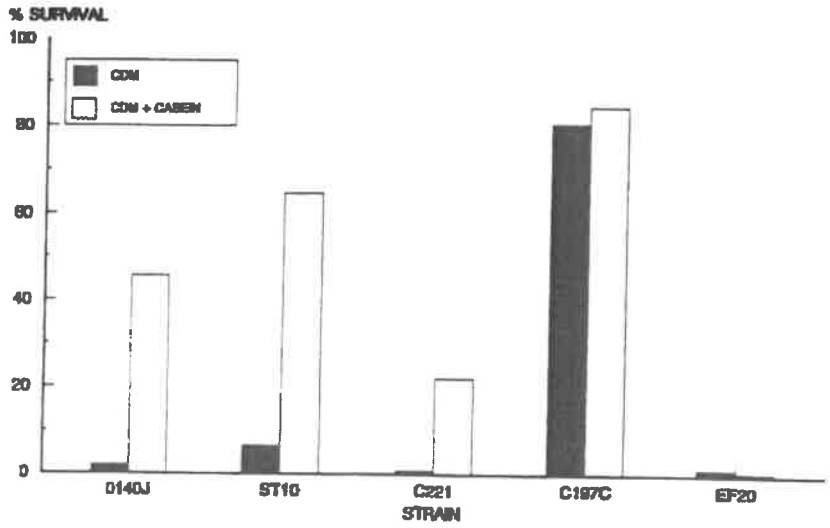
Cows	Coliform mastitis	No coliform mastitis	Total
Vaccinated	6	227	223
Unvaccinated	29	198	227
Total	35	425	460

to modify the infection rate following experimental challenge, (17 & 18). This vaccine, with minor modifications is being widely used in California, with over 50,000 animals already vaccinated (19).

Streptococcus uberis mastitis

One of the major differences which separates this bacterium from other mastitis causing organisms is its apparent resistance to opsonisation. PMN in the presence of normal milk or serum are not able to kill most strains of S. uberis. A recent finding (20 & Fig. 2) that bacteria grown in the presence of casein are even

Fig. 2. Effect of casein supplementation on the phagocytic resistance of five strains of S.uberis.



more resistant to phagocytosis and killing by PMN than when grown in a chemically defined medium again suggests that in vivo (intramammary) grown cells are elaborating an antiphagocytic factor. Clearly an understanding of the factors which control this resistance mechanism will be vital in the development of immune control of this disease. Early data (21) has shown that previous intramammary infection gives significant protection against subsequent challenge (Table 2). Although protection

Table 2

Protective effect of a previous intramammary infection with the same strain of S. uberis.

	No. ¼ challenged	% ¼ becoming clinical
Primary challenge	23	87
Secondary challenge	34	32

induced by previous infection is not a new finding, it does indicates that local antigenic challenge beneficially modifies the pathogenesis of the disease and certainly holds out hope for future vaccine research.

Conclusions

A better understanding of mechanisms of immunity and bacterial virulence is helping to explain the reason for the failure of many previous attempts to vaccinate ruminants against mastitis. More importantly it is leading researchers in directions which will undoubtedly result in the ability to control most of the major pathogens by vaccination.

Future work is likely to aim at identifying and isolating antiphagocytic "in vivo" antigens. With the correct vaccination regimen these antigens will induce the production of local opsonic IgG2 antibodies, together with an accelerated inflammatory response (PMN infiltration into the mammary gland) following infection. This type of inflammation (mastitis) should efficiently eliminate all bacteria and may not be noticed by the milker.

At the moment however, the coliform vaccine is the only one being used on a commercial scale and this is limited to an area of Northern California. If its ability to control coliform mastitis in the field is equal to that seen in the early trial its use is likely to become more widespread.

The most acceptable vaccine will need to be multi-valent for coliform, S. aureus and S. uberis mastitis, and if effective could control 75% of the disease in this country.

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MONITORING OF CLINICAL MASTITIS

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This poster features the new "Milk Cheque Mastitis Monitor" recently introduced by ADAS.

The service provides the producer with monthly forms to record clinical mastitis incidents and treatments, together with cell count and TBC results. The data is analysed by ADAS and the producer receives the detailed results of the analysis in the form of monthly and annual reports. These reports provide the producer with all the information needed to help reduce mastitis and improve profitability.

The poster will show completed examples of the clinical mastitis recording form and of the analyses of the monthly and annual output reports.

MASTITIS IN SHEEP AND GOATS

and

THE SAFETY AND QUALITY OF SHEEP AND GOATS' MILK

M. LANYON

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This post summarises the survey work carried out at the College over the past five years, studying the incidence, causes and time of occurrence of mastitis in commercial meat producing flocks. Our current work on dairy sheep and goats is described, with particular reference to the bacteriological, cellular and compositional quality and safety of milk from these dairy farms.

AUTOMATIC MASTITIS DETECTION

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AFRC Engineering have developed an in-line conductivity sensor which works on an inductive principle. There are no electrodes to contact milk, hence no problems with fouling and cleaning. The major technical limitations of electrode-free systems are overcome.

The sensor has been tested at the Milking and Mastitis Centre in infection trials to develop sensitivity, reliability and design algorithms for accurate mastitis detection. Mastitis can be detected two milkings before clinical signs appear and signals provided via a parlour computer to the cowman.

DNA FINGERPRINTING OF S. uberis

J.A. LEIGH and A.W. HILL

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Streptococcus uberis remains an important and inadequately controlled cause of mastitis in the dairy cow, resulting in both clinical and subclinical disease in the lactating gland. The lack of a reliable method of distinguishing between isolates within a herd hampers epidemiological studies.

A simple and reproducible method of typing based upon the restriction fragment size of chromosomal DNA was developed to compare isolates of Streptococcus uberis obtained from the bovine mammary gland. The endonuclease found to give the most useful restriction patterns was Hind III, although seven other endonucleases (Bgl I, EcoR I, Not I, Pst I, Sfi I, Sma I, Xba I) were tested in the system. An image analyser was used to produce a densitometric scan and graphic display of the restriction patterns. Such a system may allow large scale data storage for future computer-aided comparison.

ANTIGEN UPTAKE AND PRESENTATION IN THE BOVINE MAMMARY GLAND

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The long term requirement of a multicomponent vaccine against mastitis in cattle requires knowledge of the mechanisms of antigen uptake, its subsequent presentation and the ensuing nature and persistence of the immune response.

Using an immunoperoxidase technique, we have identified histologically, the antigens ovalbumin, (a soluble protein antigen), and Streptococcus uberis, (a formalin-killed particulate antigen), following their infusion into separate quarters of the dry mammary gland. Comparisons of uptake of the antigens and their distribution throughout the gland can be made.

Local antibody and cellular immune responses have been shown following immunization with antigen directly into the mammary gland. In order for such responses to occur, antigen has to be presented to T cells on the surface of antigen-presenting cells in association with class II glycoproteins. Our aim is to study the ability of cells expressing class II glycoproteins, both in the mammary tissue and mammary secretions, to present the antigens ovalbumin and S. uberis to responder cells. Initial studies involved the development of antigen-presenting assay using peripheral blood from naive cows. Antigen-specific lymphocyte transformation has been shown to occur although the response varies considerably between cows. By replacing blood antigen-presenting cells in this assay with mammary gland-derived cells at different stages of lactation, we propose to elucidate some of the mechanisms involved in the initiation of immune responses in the udder and hence increase our understanding of the ways in which mammary immunity may be manipulated.

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