BRITISH MASTITIS CONFERENCE 1997

Progress in Mastitis Control

Jointly organised by:

GENUS

INSTITUTE FOR ANIMAL HEALTH

NOVARTIS ANIMAL HEALTH UK LTD

MILK DEVELOPMENT COUNCIL

INDEX

INTRODUCTION

Eric Hillerton
Institute for Animal Health

The British Mastitis Conference has reached it tenth anniversary. It seems over this last decade that British dairying has been in constant flux and even turmoil with no end to this in sight. Many aspects of mastitis may appear little changed, yet in previous Conferences many new ideas have been presented.

Considering progress made against mastitis seems opportune, ten years on, and is reviewed in papers on the current situation, developments in milking technology and on better understanding of husbandry. Up-to-the-minute information comes in the research reports, including two sponsored by the Milk Development Council. The Milk Development Council are a welcome addition to the organising group this year, allowing a more expansive, reflective and international approach. This is the aim of the final session, to learn what others are doing and thinking about mastitis. The Conference concludes with a welcome return by John Bramley. John, along with James Booth and Robert James, started these Conferences. Now he reviews, from the outside, just what has happened over the ten years of Conferences.

WHERE HAVE WE GOT TO?

PROGRESS IN MASTITIS CONTROL - AN EVOLVING PROBLEM

JAMES M BOOTH, 18 Hill View Road, Worcester WR2 4PJ

SUMMARY

Over the past decade the average mastitis cell count of milk sold off British dairy farms has been halved, chiefly due to the influence of cell count payment schemes, and in mid-1997 it stands at around 170 thousand cells/ml. However, clinical mastitis appears to have declined little, if at all, remaining at an annual rate of around 35-40 cases per 100 cows. The environmental bacteria *Escherichia coli* and *Streptococcus uberis* are now the main cause of clinical mastitis in many herds, although *Staphylococcus aureus* remains ubiquitous. There has been no information on the prevalence of subclinical mastitis for the past twenty years. Mastitis is estimated to cost British dairy farmers at least £80 million a year with further associated losses estimated at over £10 million.

INTRODUCTION

Fifty years ago the average British dairy herd contained 15 cows, had about 23 cases of clinical mastitis a year, and the main bacteria causing mastitis were *Streptococcus agalactiae* and *Staphylococcus aureus*. The average cell count was probably about 750 thousand cells/ml. Penicillin had just become available for animal use and it was thought by some that mastitis would become a thing of the past.

Thirty years ago the average herd had almost doubled to 29 cows, clinical mastitis stood at 39 cases a year and the main bacteria were the streptococci and the staphylococci, especially *Staph. aureus*. The average cell count was still about 750 thousand cells/ml. The five point plan for the control of mastitis had just been developed out of the research by Frank Dodd and his colleagues at the National Institute for Research in Dairying at Reading and the late Douglas Wilson at the Central Veterinary Laboratory at Weybridge. This programme was highly effective when applied efficiently and great progress was made in reducing and controlling mastitis.

Ten years ago, a year before the first British Mastitis Conference, the average herd had doubled again to 65 cows, it had 26 cases of clinical mastitis a year, but the environmental bacteria *Escherichia coli* and *Streptococcus uberis* were coming to the fore as significant causes of mastitis. The average cell count was down to 357 thousand cells/ml, EU cell count standards had been set and cell count payments were just three years away.

Today the average herd has 74 cows, it still has about 26 cases of clinical mastitis a year, and the main causes are *E. coli, Strep. uberis* and the highly resilient *Staph. aureus*. The average cell count is now around 170 thousand cells/ml and cell count bonuses are only available to herds below 150 thousand cells/ml.

Despite this evolving problem and the fact that mastitis has not been eradicated, except for *Strep. agalactiae* in most herds, very considerable progress in controlling mastitis has been made. This becomes obvious when we consider that without this progress the average herd

would be suffering well over 100 cases of mastitis a year and still have a cell count of around 750 thousand cells/ml, both about **four times** the current levels.

However, problems remain and some are little different from those highlighted at the first British Mastitis Conference in 1988.

CLINICAL MASTITIS

Minimal progress appears to have been made during the last decade in reducing clinical mastitis. No national data are available but the incidence in one large field trial in the mid 1980s was 37 cases per 100 cows per year (1). The most recent data from DAISY herds gave a figure of 33 cases per 100 cows (2). As these herds are probably rather better than average, the national average is probably still around 35-40 cases per 100 cows a year.

According to the figures from the VIDA Report of the Veterinary Laboratories Agency of the Ministry of Agriculture, Fisheries and Food (3) there has been a change in the relative proportions of bacteria causing mastitis over the past 20 years (Table 1). Although not all the samples examined came from cases of clinical mastitis, the table indicates a substantial increase in mastitis due to *E. coli* and, to a lesser extent, *Strep. uberis*, i.e. the environmental bacteria. The other main pathogens have declined, although the change is small in the case of staphylococci. There is no indication of an upsurge in mastitis due to the coagulase negative staphylococci, as is apparently the case in some other countries.

Table 1. Main mastitis isolates (VIDA) (3)

	Percentage of diagnoses		
Pathogen	1975	1995	Change from 1975
Staph. (not specified)	23	2	] -2
Staph. aureus	-	19	]
E. coli	21	28	+7
Strep. uberis	13	17	+4
Strep. dysgalactiae	10	7	-3
Strep. agalactiae	7	5	-2
A. pyogenes	6	3	-3

However, although Table 1 appears to indicate an increase in mastitis due to the environmental bacteria, this may be just a *relative* increase as the other pathogens are reduced. There are no recent British figures but the last survey 15 years ago indicated that, at least at that time, there had been no *actual* increase in the environmental bacteria (Table 2). The two tables are in agreement in that, following the huge reductions achieved in clinical mastitis incidence, the three main bacteria involved were *E. coli, Strep. uberis* and *Staph. aureus*.

Table 2. Incidence of clinical mastitis

Pathogen	Cases per 100 cows 1967 (4, 5)	1982 (6, 5)	Change from 1967
Staph. aureus	51	6	-45
Strep. dysgalactiae	27	4	-23
Strep. uberis	24	7	-17
Strep. agalactiae	4	1	-3
E. coli	7	7	0
Others	8	10	+2
No isolate	21	6	-15
Overall	135*	41	-94

* Includes mixed infections

Present day opinion from the field suggests that there has been an increase in clinical mastitis due to the environmental bacteria, but there are no national data available to prove or disprove this view.

SUBCLINICAL MASTITIS

Unlike many other countries, no new national data on the prevalence of subclinical mastitis in the British dairy herd have been available for 20 years.

Table 3 shows that the 1977 national survey recorded almost a halving of the prevalence of infected cows over the previous ten years, the main reduction being in *Staph. aureus*. However, it remained by far the most frequent cause of subclinical mastitis - and probably still is. As a matter of interest, 0.1% of quarters were infected with *E. coli* in the 1977 survey, indicating little change from the 0.2% of quarters found with coliform bacteria in 1967.

Table 3. Main pathogens causing subclinical mastitis

Pathogen	% cows infected 1967 (4)	1977 (7)	Change from 1967
Staph. aureus	39	21	-18
Strep. uberis	12	5	-7
Strep. dysgalactiae	9	4	-5
Strep. agalactiae	9	7	-2
Others	2	2	0
Overall*	57	32	-25

* Includes mixed infections

Since 1977 there has probably been a further reduction in most bacterial causes of subclinical mastitis, especially *Strep. agalactiae*, but the present situation is unknown.

CELL COUNTS

Herd milk cell counts have been recorded on a national basis from 1971 onwards, but this stopped in 1994 on the abolition of the Milk Marketing Boards. Attempts to collate cell count data from the numerous milk buyers have been unsuccessful over the past three years. From being one of the world leaders in this area the UK has now joined the underdeveloped countries in having no national statistics.

Progress in reducing the national cell count in England and Wales, which reflects the UK situation, is given in Table 4. This demonstrates substantial though irregular progress for the first 20 years, with a rapid reduction following the introduction of cell count payments in 1991.

Table 4. Cell counts in England and Wales (from 8 & 9)

Year	Mean cell count (thousand cells/ml)	% change over previous five years
1971	573	-
1976	467	-18
1981	465	0
1986	352	-24
1991	310	-12
1996	180*	-42

*Estimated

The distribution of herds according to their annual mean cell count in Table 5 shows a similar large improvement up to 1993, the last full year for which figures are available. There will certainly have been further improvements since then, with the proportion of herds below 200 thousand cells/ml now exceeding 50% and those above 400 thousand now probably less than 1%.

Table 5. Percentage distribution of herds according to annual mean cell count

Cell count (thousands/ml)	1979	1993	Change from 1979
<200	2	26	+24
200-399	35	47	+12
400+	63	27	-36

OTHER ASPECTS

There are two other aspects of mastitis in the British dairy herd which appear to have evolved little over the past decade. These are the incidence of summer mastitis and the resistance of mastitis bacteria to antibiotics.

Table 6 shows the results of annual surveys of summer mastitis carried out by the Milk Marketing Board's Mastitis Control Service. There is the suggestion of a reduction in both the percentage of herds affected and the number of cases per affected herd. However, the incidence of summer mastitis is notoriously variable ranging, in these surveys, from 59% down to 35% of herds affected, so the trend is not significant and summer mastitis remains a problem for many dairy farmers.

Table 6. Summer mastitis incidence

Years	% herds affected	Average cases per affected herd
1978-83 (10)	47	3.0
1984-88 (from 11)	45	2.7
1989-93 (from 11 & 12)	40	2.3

The resistance of mastitis bacteria, especially *Staph. aureus*, to penicillin and other antibiotics has been a consistent concern ever since penicillin became available for animal use 50 years ago. A survey in 1961 found that 71% of staphylococcal isolates from mastitis cases were resistant to penicillin (13). Another survey 25 years later in 1986 (14) produced the slightly lower figure of 58% and recent informal laboratory surveys indicate little change. In addition it is reassuring to be able to state that, to date, no staphylococci of bovine origin have been identified which are resistant to cloxacillin, despite its widespread use in both lactating and dry cow intramammary syringes for over 25 years.

Ten years ago the hope was expressed that it might become possible to make further progress in controlling mastitis through breeding and vaccination. Although considerable research has been carried out over the past decade, and both have been the subjects of papers given at previous British Mastitis Conferences, neither has yet contributed to progress. However, it does now seem that both may contribute during the next decade, although possibly to a more limited extent than we had originally envisaged.

COSTS

Mastitis has been estimated to be costing British dairy farmers over £80 million a year (15) based on the DAISY figure for clinical incidence of 33 cases per 100 cows and a conjectured 15% prevalence of subclinical mastitis. On top of these losses individual farmers will also suffer losses due to reduced cell count and bacteria count payments, as well as penalties for antibiotic residues. It has been estimated that annual cell count penalties amount to approximately £9 million and antibiotic residue penalties to £1 million, totalling £10 million, although some of these penalties will of course return to the pool for payment to all farmers supplying the milk buyer.

CONCLUSIONS

In examining the evolving nature of mastitis in the British dairy herd, especially over the past decade, the lack of recent data is a considerable obstacle. Objective records are essential to evaluate the true situation and to form the basis for future policy decisions.

There are some indications that current recommendations for reducing clinical mastitis are not effective as, despite the substantial reduction in the national cell count over the past decade, clinical incidence does not appear to have been reduced significantly. How much of this is due to the partial replacement of contagious bacteria by environmental bacteria is not known.

Subclinical mastitis has been reduced substantially since the 1960s although it is impossible to know what progress, if any, has been made over the past two decades. Despite the reduction in cell count in most herds, the new infection rate is completely unknown and it seems likely that *Staph. aureus* remains a major cause of subclinical mastitis.

It is concluded that, in order to make further progress in controlling mastitis in the UK, it is necessary to define the current mastitis situation, to monitor future changes in the evolving situation, and thus to be able to direct our limited resources for research to maximum effect. This requires action to:

1. Set up a pilot monitoring system to monitor the national incidence of clinical mastitis.

2. Carry out a survey to determine the current prevalence of subclinical mastitis and the main bacterial causes.

3. Collate the present cell count data to provide a readily accessible and on going monitor of progress.

REFERENCES

1. BOOTH J M & ROWLANDS G J (1990) Proceedings International Symposium on Bovine Mastitis, Indianapolis, USA, 336-341
2. ESSLEMONT R J & KOSSAIBATI M A (1996) Veterinary Record 139 486-490
3. VETERINARY LABORATORIES AGENCY (1979 & 1995) VIDA Veterinary Investigation Surveillance Reports
4. WILSON C D & KINGWILL R G (1975) IDF Document 85 422-438
5. BOOTH J M (1988) British Veterinary Journal 144 316-322
6. WILESMITH J W, FRANCIS P G & WILSON C D (1986) Veterinary Record 118 199-204
7. WILSON C D & RICHARDS M S (1980) Veterinary Record 106 431-435
8. BOOTH J M (1988) Veterinary Record 122 299-302
9. BOOTH J M (1997) Proceedings Regional Seminar on Milk Quality, Uruguay, Supplement 1-8
10. O'ROURKE D J, CHAMINGS R J & BOOTH J M (1984) Veterinary Record 115 62-63
11. BOUMAN M & BOOTH J M (1992) Veterinary Record 131 417-418
12. BERRY E A (1997) Personal communication

13. WILSON C D (1961) Veterinary Record 73 1019-1024
14. FRANCIS P G & CARROLL P J (1986) Veterinary Record 118 361-363
15. BOOTH J M (1997) Cattle Practice 5 (2) 61-65

A DIFFERENT MILKING MACHINE

J ERIC HILLERTON, Institute for Animal Health, Compton Laboratory, Newbury, Berkshire RG20 7NN

SUMMARY

The type of milking machine in use in Britain has changed in many features over the last years, but with no obvious direction. There is probably insufficient replacements of and improvement to milking plants to allow continued improvement in the overall standards of machine milking. Attention will have to be given to standards of installation, maintenance and use to satisfy milk assurance schemes. A form of certification may become necessary. The idea is presented that motives for selection and use of milking systems may not be good business decisions.

INTRODUCTION

Nothing is so fundamental to successful dairying than an efficient milking machine. However, no individual part of the dairy farm receives quite so little attention (1,2) having cost so much in the first place.

Ten years ago the statistics available (3) suggested that there were more than 38,000 milking installations in Great Britain. There are no current figures available but probably there are now fewer than 30,000 plants. Whilst many parlours have disappeared out of service, the greatest losses have been proportionally in hand milking and bucket or pipeline systems. Recently there has been the introduction of the first handful of automated milking systems (AMS) using robots.

Milking machines have a design life of 15-20 years and many are obviously used for this long. With 30,000 plants there are only some 800 new replacement installations each year, half of the level needed, and various levels of upgrade. Old machines in many cases are not replaced, like the farmers and their small farms they just fade away.

There are many, if not all, new milking machines being bought which are quite different to those they are replacing. They may appear to those buying them to be revolutionarily different but actually only reflect accumulated changes. Milking robots, one apparent extreme of change, only introduce automatic cluster attachment. The other features from feeding boxes to automatic teat spraying are integration of components which have been available elsewhere. Indeed in many features, especially the actual milking components, the AMS is quite conventional if not old fashioned (4).

NEW KINDS OF MACHINE

Pipeline systems are not high fashion in the UK, but elsewhere remain common, if not the most popular of new installations. In many countries tie-stall barns are the favoured way to keep cows and, although labourious to use, the milking units offer all of the features found in sophisticated parlours and are cheap.

At least three types of AMS are on the UK market. Despite proof being required of their flexibility, durability and longevity, some users are quite convinced of their success. However, in various countries many early devotees have replaced their AMS with a parlour.

Modern parlours are as variable as they are imaginative in size, sophistication and success. They may be very expensive as a new parlour requires not just a milking machine, it is usual to need a building too, with associated cow traffic systems. Despite these costs, this is the preferred route for most remaining committed to dairying.

Dairy farmers in the UK have long been devoted to herringbone parlours and their derivatives. New styles have polarised to extremes from rapid exit parallel set-ups with all sorts of data collection and milking control in stainless steel to bare-essential plants milking direct to line with no accessories.

MILKING MACHINE POLICY

Devotees of different systems have well defined ideas on the total superiority of their milking machine. There is no easy way of making valid comparisons given different motives for using or preferring quite different systems although early information from comparing different systems is showing some of the weak points of each type (5).

Whichever type of machine is chosen this is done with a particular objective in mind. The objectives applied vary greatly between the various major milk producing countries. In the UK there seems to be a fashion of trying to spend as little time in the milking parlour as possible. Plants are often sold expressly to achieve throughput, usually at the expense of milking routine and good husbandry. Fore-milking and/or teat cleaning are not common and less commonly done well.

It is not surprising that a limited time is desired in the milking parlour. Most are not pleasant places to be. They may be cold and wet in winter, hot and full of flies in summer and always grimy at best. However, this is not always typical internationally. The UK industry accepts extremely poor working conditions that would not be tolerated in many other countries and it is small wonder that the aim is to spend little time in such a parlour. This leads to a fairly absurd business decision of spending as little time as possible using the single most expensive facility on the farm. There may be practical restrictions on many farms on time available to milk from staffing levels etc. but these do not entirely excuse the tying up of major capital investment in a system which is relatively rarely used, perhaps only four out of each twenty four hours.

On this basis, which would be flatly rejected if presented honestly to any investment advisor, the dairy farmer is not investing in the correct system for the average farm. There may be better choices. These might include an AMS for a small herd (now probably less than 150 cows) with limited labour available. For larger herds a more US approach of investing in a parlour where size has been calculated to give the correct throughput in cows/hour or cows/man, but using it for perhaps twenty hours each day. This is achievable even with herds of 400-500 cows especially if milking three times daily. This means using more flexible recurrent costs instead of capital investment. This is a more flexible method and copes better with variations in output, input costs, investment strategy and milk price (6).

Anyone planning to invest in a new milking system must consider

- the best system to use and to present as an image for hygienic food production
- achieving an acceptable working environment
- making the wisest business decision irrespective of trends

Answers and decisions will depend on where and how far this industry has to go to secure its market for raw milk.

THE NEW MILKING MACHINE

The concept of the new milking machines, its components and operation are probably significantly better than ever, if still far from good enough. Systems are now designed to help to minimise mastitis and ensure milk quality. They rarely seem to operate that way despite increasing understanding of what can be achieved and templates provided by standards for installation and maintenance (7). The problems seem to start at installation (5) and they certainly continue and are exacerbated during use (1,2). The milking machine is commonly abused and neglected. This is to the detriment of the cows, the milk and the success of the farm. It cannot be long before milking machine operation is required to be improved significantly. The neglect bourne out of familiarity and complacency has no place in farm assurance schemes. Perhaps a MOT equivalent, i.e. an enforceable test and compliance in place of testing services has a future. It is probable that it would be cost effective, but will require a much higher level of skill and expertise to ensure the standards than currently available.

The milking machine has been estimated to contribute to 7% mastitis cases when operating properly (8). When faulty in any of many ways, which most machines are, it may be related to all mastitis on the farm and certainly be involved in any major health problems.

ASSESSING GOOD MILKING

New styles of milking machine all claim to milk better including machine operation, cow responses and operator performance. Guidelines on how these aspects may be assessed are available (9) and being developed further. Measurements are relative and subjective so far. A frequent response to comments on cow distress in the parlour is "You should have seen them with the old machine!" Less bad is no more acceptable.

There is an internationally recognised need to be able to assess good milking. The Milk Development Council have funded a study in this area which will compliment work in the US and elsewhere. Results will be available at the end of the year (10). It is already clear that the pressures in the more fashionable milking systems leave too little time to assess cow behaviour and teat responses. Even if what is acceptable was known. Washing down the parlour between batches of cows and getting out of the pit to fetch each batch of cows and load them quickly overtake good husbandry.

Milking performance and cow reactions may be better with new milking systems, but it is more than likely that they are not good enough for efficient dairying and successful promotion of the correct image of milk.

CONCLUSION

It is becoming rapidly necessary to build machine standards and good operation into farm assurance schemes. To have any effect the requirements will have to bite and this could hurt virtually all in dairying.

REFERENCES

1. NICHOLLS B W (1995) Proceedings of the British Mastitis Conference pp 24-34
2. BERRY E A & M SCRIVENS (1997) Proceedings of the British Mastitis Conference (this volume)
3. UK DAIRY FACTS AND FIGURES (1994) England & Wales Residuary Milk Marketing Board, Thames Ditton
4. HILLERTON J E (1997) Computing and Electronics in Agriculture 17 41-51
5. OHNSTAD I (1997) Proceedings of the British Mastitis Conference (this volume)
6. SMITH J F, ARMSTRONG D V, GAMROTH M J & MARTIN J G (1997) Journal of Dairy Science 80 1866-1871
7. BS ISO 5545, 6690 (1996) Milking machine installation - construction and performance. Milking machine installation - mechanical tests, British Standards Institute, London
8. RABBOLD K (1983) Milchgewinnug. In: Die Milch Hrsg. Ed. H O Gravert, Ulmer Verlag, Stuttgart
9. IDF BULLETIN 321 (1997) In: Guidelines for evaluation of the milking process, International Dairy Federation, Brussels p 31-35
10. HILLERTON J E, BAINES J R & OHNSTAD I (1998) American Society for Agricultural Engineering Meeting, St. Louis, Mass. (to be presented)

HUSBANDRY, MANAGEMENT AND MASTITIS

W ROBERT WARD, University of Liverpool, Leahurst, Neston, South Wirral, L64 7TE

SUMMARY

There is relatively little information available on the effects of changes in husbandry and management on mastitis. Several studies have indicated the importance of motivating farm staff because even well known techniques are poorly adopted and applied. Changes in management, in response to advice, can significantly affect milk output and milk quality. It is important to find effective ways of demonstrating good practice and ensuring compliance with the mechanisms of husbandry and management.

INTRODUCTION

It is helpful to distinguish between husbandry and management, both the terms themselves and their application to individual cows and the herd. Husbandry, sometimes referred to as stockmanship, implies all of the day-to-day activities such as milking the cows, detecting cases of mastitis, moving cows, cleaning floors, bedding cubicles or straw-yards, and so on. Management implies longer-term strategic procedures and decisions such as genetic selection, modifying or replacing buildings, tracks or equipment. On many small farms, one person (the farmer) carries out all these jobs, whereas on large farms there will be division of labour between managers and stockmen.

The application of financial incentives, e.g. payment for low cell counts or penalties for residues, is based on the assumption that farmers will respond, and most do so. Many advisers, however, have been disappointed at the lack of application of the five point mastitis control plan for control of mastitis by some farmers. A better understanding of the complex interactions between the characteristics of stockmen and managers and the incidence of mastitis would be helpful in analysing why recognised procedures are sometimes not followed, and how advisers could be more effective.

PROGRESS IN APPLICATION OF HUSBANDRY AND MANAGEMENT TECHNIQUES

There is limited information on the effect of changes in husbandry and management on the incidence of mastitis in British dairy herds. There have been no major studies of the incidence or prevalence of mastitis since 1979-82 (1). A study into lameness in 15 British dairy herds (2) has shown strong relationships between the farmer's knowledge, training and awareness, and the prevalence of lameness. It is likely that similar associations exist for other conditions including mastitis.

The incidence of mastitis varies greatly between farms. Kossaibati and Esslemont (3) found, in 90 British herds believed to have reliable records, that the number of cases of mastitis in 1990 - 1992 varied from 2 to 215 cases per 100 cows per year. The upper quartile of herds recorded 29 cases, while the lowest quartile had 7. They reviewed previous studies estimating the incidence of mastitis and suggest that the incidence of clinical and sub-clinical mastitis has

decreased over the past 25 years. They suggest that the best herds should be studied in order to shed light on the crucial factors that lead to success, and speculate that the best herds might comprise first class ownership with clear objectives, employing highly competent stockmen. It is tempting to speculate that this dramatic difference in incidence of mastitis between herds in Britain, and the gradual decline over the years, are related to the awareness and enthusiasm of farmers, but there is little practical evidence to show this.

One study (4) referred to encouragement to apply a modified five point mastitis control plan in a British experimental farm, and reported a large decline in sub-clinical mastitis and a smaller reduction in clinical cases.

The effect of quality of management and enthusiasm for the task has been identified by Seabrook (5). He showed in Britain that stockmen affected the milk production of dairy cows. When a new stockman took over, yield sometimes changed dramatically. When a stockman was upset, yields were affected. Unfortunately he did not report on the influence of stockmen on mastitis.

Further support for the effect of enthusiasm and interest comes from Hillerton *et al.* (6) who found a significant reduction in mastitis between winters in control group herds in Britain. They concluded that this was probably caused by better supervision of mastitis control, showing that even good herdsmen have scope for significant improvement.

Johnson (7) discussed the motivation of farmers and herdsmen in mastitis control in Britain, and advised that veterinary surgeons must first motivate themselves, and find out about the cell counts, usage of antibiotic tubes, and milking routines. As methods of stimulating clients' interest the use of practice meetings, newsletters, and individual advice were proposed.

This type of programme has been studied in California (8,9,10) where a system of appraising management practices was developed and applied on 50 dairy farms. It was found, mainly by interview, that many farmers accepted that good husbandry practices were not adopted. For example, only on 17% of farms were all teats dipped, 30% dried teats with paper towels, and 34% kept records of mastitis treatments. All these procedures were significantly associated with at least one measure of udder health or production. On the other hand, 96% of farms used dry cow therapy, 92% stimulated milk let-down, and 91% checked for abnormal milk. Farms with corrals sloping away from lanes and sited less than 150 metres (500 feet) from the parlour were also associated with better udder health or production.

A further study (11) showed that many changes in management practices in 24 dairy herds in Maryland, USA, over two years, were made in response to advice. Peters *et al.* (12) showed in the same project that these herds had greater increases in milk yield and reduction in cell counts than control herds. An additional report on the same project (13), found that some farms did not adopt recommended practices because of family disputes, emphasising the importance of including all the people involved in running a farm.

Cassell *et al.* (11) evaluated 36 management practices relating to mastitis. Those affecting the most farms (with number out of 24 regarded as deficient initially and number still deficient at the end) included:

Fore-milking (12 -> 7)
Thorough drying of udder and teats (12 -> 1)
Flush clusters after *Staph. aureus* infected cows (11 -> 6)
Identify *Staph. aureus* infected cows (14 -> 7)
Repair air leaks (14 -> 1)
Correct pulsation ratio and rate (13 -> 0)
Regular maintenance (11 -> 8)
Sample clinical cases, dry and fresh-calved cows (24 -> 10)
Use antibiotics recommended from sensitivity tests (19 -> 0)
Cull *Staph. aureus* infected cows (13 -> 2)

In this study (11) it was found that the mean rate of clinical mastitis from June to December decreased from 14.9 per 100 cows a month to 8.8. The proportion of quarters infected with major pathogens decreased from 22.4 to 15.8%. The proportion with positive California Mastitis Test (CMT) fell from 38.1% to 25.6%.

The application of Total Quality Management (TQM) on dairy farms in six states in USA has been studied (14). TQM involves setting goals for producing a high quality product, and defining systems to achieve these outcomes. While most farmers produced TQM protocols, few produced treatment protocols, and the authors conclude that this inability was because of the busy schedules of veterinarians and producers.

Crist *et al.* (15) measured the effectiveness of educational efforts in Ohio, USA, on 13 dairymen given intensive effort, 12 given intermediate effort, and 11 controls. Over three years, herdsmen receiving education showed a significant increase in teat dipping and dry cow therapy, and a significant reduction in prevalence of infection. The Wisconsin Mastitis Test (WMT) scores were significantly lower in herds practising teat dipping and dry cow therapy. The authors comment on the difficulty in getting dairymen to participate: 150 farms with a high WMT score were asked, but only 43 agreed, seven of whom left the project.

Finally in a study conducted by interview (16,17) the attitudes, management practices and herd performance of 102 dairy farmers in Ontario, Canada were determined. It was concluded that the farmers were not very different from other occupations studied, and that the socio-psychological variables of farmers (e.g. level of formal education, amount of continuing education, or satisfaction with farming) were as important or more so than the management variables (e.g. days from calving to first service, policy for genetic improvement, or culling policy). It was found that record-keeping was generally weak. The study did not report on mastitis, but milk yield and butterfat were among their performance indicators.

CONCLUSIONS

Despite the lack of published research information in Britain, it is clear from North American studies that husbandry and management can be important risk factors in the incidence of clinical and sub-clinical mastitis, and in the production of high quality milk. Simple methods of assessment of husbandry and management have been published, and it would be helpful if standard methods could be agreed and used in future studies.

Since farming methods differ considerably between geographical areas, it is important that research should be carried out locally. Demonstration farms, where 'real' farmers can show their neighbours the benefits of better husbandry and management, are likely to be strong influences.

A better understanding of the ways in which stockmen and managers influence mastitis and milk production should lead to more effective implementation of good practices.

The current popularity in Britain of farm assurance schemes means that there may soon be a system of incentives for production of high quality milk. It is, therefore, vital to understand what procedures are needed, and how best to encourage compliance by stockmen and managers.

REFERENCES

1. WILESMITH J W, FRANCIS P G & WILSON C D (1986) Veterinary Record 118 199-204
2. MILL J M & WARD W R (1994) Veterinary Record 134 162-164
3. KOSSAIBATI M A & ESSLEMONT R J (1995) Wastage in dairy herds. Report No. 4. DAISY, Department of Agriculture, Earley Gate, University of Reading, Reading, pp 84-87
4. HILLERTON J E, BRAMLEY A J, STAKER R T & McKINNON C H (1995) Journal of Dairy Research 62 39-50
5. SEABROOK M F (1984) Veterinary Record 115 84-87
6. HILLERTON J E, SHEARN M F H, TEVERSON R M, LANGRIDGE S & BOOTH J M (1993) Journal of Dairy Research 60 31-41
7. JOHNSON N J (1993) Cattle Practice 1 149-152
8. GOODGER W J, RUPPANNER R, SLENNING B D & KUSHMAN J E (1984) Journal of Dairy Science 67 675-685
9. GOODGER W J, GALLAND J C & CHRISTIANSEN V E (1988a) Journal of Dairy Science 71 2535-2542
10. GOODGER W J, REPP S & GALLAND J C (1988b) Preventive Veterinary Medicine 6 109-126
11. CASSEL E K, VOUGH L R, VARNER M A, EICKELBERGER R C, MANSPEAKER J E, STEWART L E, DOUGLASS L W & PETERS R R (1994) Journal of Dairy Science 77 2461-2476
12. PETERS R R, CASSEL E K, VARNER M A, DOUGLASS L W, VOUGH L R, MANSPEAKER J E, STEWART L E, WYSONG J W & EICKELBERGER R C (1994b) Journal of Dairy Science 77 2450-2460
13. PETERS R R, CASSEL E K, VARNER M A, EICKELBERGER R C, VOUGH L R, MANSPEAKER J E, STEWART L E & WYSONG J W (1994a) Journal of Dairy Science 77 2438-2449
14. SISCHO W M, HUTCHINSON L J, GILSON W, RENEAU J, SEARS P, TIMMS L & WAILES W (1996) World Buiatrics Congress, Edinburgh July 1996. British Cattle Veterinary Association 1 333-335
15. CRIST W L, HEIDER L E, SEARS P M, BARR H L, KOWALSKI J J & SCHMIDT G H (1982) Journal of Dairy Science 65 828-834

16. BIGRAS-POULIN M, MEEK A H, BLACKBURN D J & MARTIN S W (1985a) Preventive Veterinary Medicine 3 227-240
17. BIGRAS-POULIN M, MEEK A H & MARTIN S W (1985b) Preventive Veterinary Medicine 3 241-250

RESEARCH REPORTS

INVESTIGATING NON-ANTIBIOTIC MASTITIS THERAPY

CHRISTOPHER H KNIGHT, JULIE L FITZPATRICK[1], DAVID N LOGUE[2], DAVID J PLATT[3], STUART ROBERTSON AND HELEN E TERNENT[2], [1] Hannah Research Institute, Glasgow University Veterinary School, [2] Scottish Agricultural College, [3]Glasgow University Bacteriology Department

SUMMARY

A topically applied liniment and the hormone oxytocin were investigated as possible non-antibiotic mastitis therapies. The experiment involved repeated single-quarter exposure of dairy cows to low-virulence *Staphylococcus aureus* followed by treatment according to a replicated Latin square design, which included a negative control of massage alone and a positive control of intramammary antibiotic. Bacterial clearance was assessed over three weeks, and a further two weeks were allowed before the next infusion. Bacterial clearance was significantly improved over the negative control by oxytocin, but not by liniment.

INTRODUCTION

Mastitis is a major cause of impaired animal health and welfare. There is a need to achieve prevention where possible and rapid cure where not, whilst avoiding excessive use of antibiotics which have detrimental effects on product quality. Several putative non-antibiotic mastitis therapies are in use, topical liniments being popular in Britain and oxytocin in the USA, and it is believed (but not proven) that these may act by enhancing milk removal and hence 'flushing' of pathogen. Uddermint (Teisen) is the most widely used liniment in the UK and is evidently popular with farmers, but its efficacy is not established. Oxytocin is deserving of re-examination, since a recent study of clinical mastitis in commercial dairy herds found oxytocin to be as effective in achieving cure as intramammary antibiotic (1). The aim of this study was to establish the efficacy of these two non-antibiotic mastitis therapies against challenge with *Staphylococcus aureus*.

METHODS

The model employed repeated single-quarter intramammary infusion of low-virulence strain of *Staph. aureus* at a dose of $5x10^4$ organisms (2). Each infusion was made after an afternoon milking and treatment commenced at the following milking, approximately 16 hours later. Repeat infusions in the same cow were done 5 weeks apart, utilising a previously uninfused quarter each time. Eight Friesian Holstein cows from the Hannah Research Institute herd (32 quarters) were used in a duplicated Latin square design with four treatments, such that each cow received each treatment and the order of giving treatments was randomised across cows. The treatments were:

Negative control (C); 1 minute manual massage of the infused quarter at every milking for three weeks after infusion.

Topical liniment (U); 1 minute massage of the infused quarter with 10ml of Uddermint (Teisen Products Ltd, Redditch, B96 6RP, UK) at every milking for 3 weeks after infusion.

Oxytocin (O); 100 i.u. of synthetic oxytocin (Oxytocin-S, Intervet UK Ltd, Cambridge CB4 4FP, UK) by i.m. injection before each milking for 1 week after infusion.

Positive control (S); a single course of intramammary antibiotic (Synulox LC, Pfizer Ltd, Sandwich CT13 9NJ, UK) into the infused quarter according to label instructions.

Milk samples for bacteriology were drawn aseptically from the infused quarter and from its diagonally-opposed pair immediately before a morning milking 1 week before infusion (screen), the morning before infusion (pre-infusion), the morning after infusion (post-infusion) and then three times weekly for three weeks. Positive *Staph. aureus* isolation was counted as 1, negative as 0. A sum of the three values for each post-infusion week (i.e. commencing with the sample after the post-infusion sample) was calculated (*Staph. aureus* score) and statistical analysis performed using a three-factor analysis of variance investigating treatment, infusion cycle number and week post-infusion. Days to first positive *Staph. aureus* isolation were also calculated. Other measurements were made but only bacteriology will be reported here.

RESULTS

The scoring system was devised to enable quantification of bacterial presence. By definition, the weekly *Staph. aureus* score must be a value between zero (0/3 samples positive) and three (3/3 positive). Data are given in Table 1. It will be seen that approximately half of all samples from C and U quarters were positive for *Staph. aureus*, with no significant difference between the two. Oxytocin reduced the score to half of the control value, a significant improvement. Synulox was also effective, with only one-sixth of all samples being positive for *Staph. aureus*. Individual analyses by both paired and unpaired *t* tests confirmed that O and S had significantly lower counts than C and U, and there were no significant differences within these pairings.

Table 1. Bacteriology results for quarters infused with low virulence *Staph. aureus* and massaged (Control), massaged with Uddermint liniment, treated with systemic oxytocin or treated with intramammary antibiotic (Synulox). Within a row, values with different letters are significantly different.

	Control	Uddermint	Oxytocin	Synulox	Statistical analysis
Staph. aureus score	1.54±0.27 a	1.42±0.27 a	0.79±0.2 b	0.50±0.2 b	p=0.005
Days to first positive	5.0±1.3 a	9.1±2.4 a c	14.9±2.0 b c	22.9±2.5 b	p=0.02

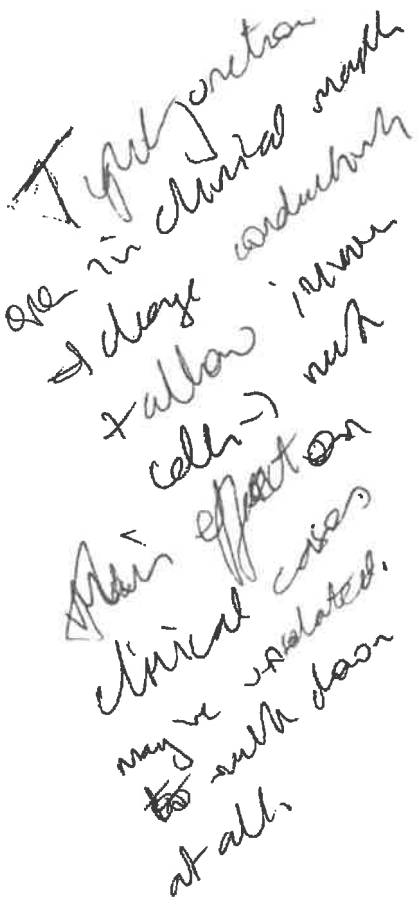
Days to first positive isolation were also analysed (Table 1). All three treatments delayed the appearance of *Staph. aureus,* but in the case of U this effect was not significant. O and S were both significantly better than C but not different from each other. There was no significant difference in *Staph. aureus* score between weeks one, two and three post-infusion. There was a tendency for the score to increase with time in both O and S, but separate analysis of these two groups still revealed no significant week effect.

With the exception of one individual sample, all screen and pre-infusion samples of previously uninfused quarters were negative for *Staph. aureus*. Treatment was initiated at a common time after infusion; 68% of samples taken at this time (post-infusion) were positive for *Staph. aureus*. There was a tendency for quarters with a positive post-infusion sample to have a higher total *Staph. aureus* score (sum of all 3 weeks), but this difference was not significant. The proportion of positive and negative post-infusion samples was not significantly different between treatment groups. These observations indicate that the single-quarter infusion model functioned effectively.

DISCUSSION

Bacterial clearance following intramammary challenge is a rigorous test of product efficacy. This is particularly true of *Staph. aureus*, since this is arguably the most difficult of all mastitis pathogens to clear from the udder. On the basis of a positive post-infusion sample followed by no further positives, total clearance was observed in only 3/32 quarters (less than 10%), reinforcing the tenacity of this particular microorganism. However, of 220 milk samples which followed a positive post-infusion isolation, 122 were negative and 98 were positive which indicates that a single-sample regime could have yielded a clearance rate of over 50% It is very evident that such a regime would be inadequate. The strain of *Staph. aureus* used was originally isolated from a case of subclinical mastitis and therefore represents a naturally occurring pathogen. Although the data are not reported here, clinical signs were observed in 81% of infusions. These ranged from only restlessness through clots in milk to, in only one case, outright illness and pyrexia. With this one exception all signs were very mild and resolved without additional therapy.

Although total clearance was seldom achieved, there were quantifiable differences in bacterial presence in response to the different treatments. Control quarters returned a positive *Staph. aureus* isolation in approximately 50% of all samples, a situation which was not improved by liniment treatment. To our knowledge this is the first time that bacterial clearance has been assessed in response to treatment with any topical liniment. The negative nature of the data does not necessarily negate a use for such treatment in mild clinical mastitis; prior to the advent of antibiotics vigorous massage to remove as much milk as possible would have been the only available treatment, and any product which encourages the dairyman to do this may be beneficial. It is also of note that the time to first isolation of *Staph. aureus* was increased by the liniment, albeit not significantly, which may suggest a potential benefit as an early therapy to be replaced by antibiotic if the problem does not quickly resolve. Nevertheless, it is evident that this particular product (Uddermint) does not have any bacteriostatic or therapeutic properties against *Staph. aureus* other than those (untested) conveyed by the massage.



Oxytocin is anecdotally reported to be in widespread use in some parts of the USA as an early-onset mastitis therapy. While it has been compared with antibiotic treatment of naturally occurring mastitis in commercial American herds (1) this present study gives a more rigorous comparison. The data obtained in this study show that oxytocin does have potential as a non-antibiotic mastitis therapy. It reduced the *Staph. aureus* score to half of that of the negative control, still higher than but not significantly different to the positive control of antibiotic, and increased the time to first isolation by a factor of three. This is in broad agreement with the American trial, which showed that both clinical and bacteriological cure rates were improved to the same extent by oxytocin and each of two proven antibiotics. It is important to reiterate that neither oxytocin nor antibiotic totally prevented the establishment of *Staph. aureus* in the majority of our infused quarters, with the result that the cows became chronically but sub-clinically mastitic.

Given that oxytocin is efficacious, how might it be acting? The most obvious explanation is that, by stimulating the milk ejection reflex, it increases removal of pathogen from the udder. There is an alternative possibility. The doses of oxytocin used in commercial practice (and here) are considerably in excess of what is needed to cause milk ejection, and are sufficient to cause a partial opening of the tight junctions between neighbouring secretory cells. This will allow equilibration of plasma and milk, with a resultant shift in ionic balance, and may also aid recruitment of immune cells with a consequent enhancement of the intramammary immune response. This hypothesis is presently being tested. It is worth noting that endogenous oxytocin release is increased during experimentally induced mastitis (3) and tight-junctions become leaky during mastitis; the latter is probably dependent on the former and the whole may be part of an immunological defence mechanism designed to protect against the invading organism. However, whilst leaky tight junctions may be good in the short term for defence against disease, in the longer term milk yield will be compromised if tight junctions remain leaky. Large doses of oxytocin given over extended periods (more than a few days) are likely to reduce milk yield, so care will be needed in its application and we suggest that further research is needed to identify the most appropriate way(s) of using oxytcin.

ACKNOWLEDGEMENTS

This study was supported by the Milk Development Council. We thank Biological Resources staff at HRI for care of the animals and laboratory staff at HRI, SAC and Glasgow Bacteriology for skilled technical assistance. The oxytocin used was a gift from Intervet UK Ltd. and Synulox a gift from Pfizer Animal Health.

REFERENCES

1. GUTERBOCK W M, VAN EENENNAAM A L, ANDERSON R J, GARDNER I A, CULLOR J S & HOLMBERG C A (1993) Journal of Dairy Science 76 3437-3444
2. PLATT D J, CANDISH D, PYETT H E, SMITH I W, SOMERVILLE, GUNN J & LOGUE D N (1994) Proceedings of the British Mastitis Conference 1994 p 69
3. GOREWIT R C (1993) Journal of Dairy Science 76 722-727

THE DURATION OF *STREPTOCOCCUS UBERIS* INTRA-MAMMARY INFECTIONS

CATHERINE WATT, The University of Oxford/Institute for Animal Health

SUMMARY

Analyses of frequent milk samples have shown the rate and level of infection by *Streptococcus uberis* in herds at the Institute for Animal Health (IAH) and Ohio Agricultural Research and Development Centre (OARDC). Statistical methods were used to estimate the probable persistence of infection, the rate of recovery and hence the duration of infection. At OARDC infections resulting from clinical mastitis persisted for an average of 1.2 months and sub-clinical infections for 3.3 months. OARDC treat persistent sub-clinical infections with antibiotic therapy. At IAH, in the absence of antibiotic therapy in lactation, sub-clinical infections persisted for an average of 8.5 months. The rate of recovery was high initially, then lower, increasing again later in lactation probably due to use of dry cow therapy at both institutes. These results illustrate the possible positive effects of antibiotic therapy on *Strep. uberis* infections.

INTRODUCTION

Mastitis in the UK has been much reduced by the application of control measures such as improved hygiene, including teat dipping, and dry-cow antibiotic therapy. *Streptococcus uberis* has been controlled less well than many other pathogens, and is responsible for an increasing proportion of sub-clinical and clinical infections. This failure to control *Strep. uberis* has been widely attributed to the growth of this species in the environment, although the exact nature of the interaction between infected cows and the environment is not clear (1).

Until recently, the bulk of mastitis research was directed at refining and comparing control practices. While this direct approach has been fruitful, further progress requires more detailed understanding of the microbiology, immunology and epidemiology of the various mastitis-causing pathogens and their bovine host. Some studies on *Strep. uberis* are reported.

METHODS

Herds

The majority of *Strep. uberis* infections are sub-clinical for some or all of their duration, yet information about the duration of these infections is scarce. The data presented here come from the Institute for Animal Health (IAH) in Compton and the Ohio Agricultural Research and Development Centre (OARDC) in Wooster.

Both herds practise post-milking teat dipping and dry-cow antibiotic therapy. At the IAH, clinical cases of mastitis only are treated with antibiotic therapy, whereas at the OARDC some sub-clinical cases are treated.

Sample Collection

In early February of 1995, 96 and 97, herd tests were carried out at IAH. These involved the aseptic collection of quarter milk samples from all cows in milk. Bacteriological examination of the milk samples revealed that a number of cows had quarters infected with *Strep. uberis*. Infected quarters were then sampled monthly (while the cows were in milk) for the period of the study (until July 1997). The identity of *Strep. uberis* isolated from quarter samples was confirmed by use of a 16S rRNA probe (2), and isolates were stored for later analysis.

Since 1983, an intensive sampling program has been in place at the OADRC (3). Individual quarter samples are collected from all quarters of all cows at 3, 7, 14 and 30 days after calving, at 30 day intervals for the remainder of the lactation, and at 14, 7 and 0 days prior to drying off. Samples are also collected prior to and 7 and 14 days after antibiotic treatment for clinical mastitis. In addition, when a sub-clinical infection is identified, more frequent samples were taken in order to follow the fate of the infection. Bacteria are identified using standard biochemical techniques. A database is maintained in which the infection status of each quarter of each cow is summarised monthly.

Data Analysis

The data were analysed using survival analysis techniques, which are useful for the analysis of the time between two events (4). While in the case of 'survival' those events are birth and death, in this case we are interested in the time between infection and recovery. In particular, it is important to know what affects the rate of recovery. In many infections the likelihood of recovery changes with time since the start of infection. For an infection with an average duration of 13 days, with only a small amount of variation around the mean (Fig. 1a) the rate of recovery will initially be very low, increasing dramatically as the time approaches the average duration (Fig. 1b). If, however, the rate of recovery for an infection is constant with time since infection, then the duration of infection will vary, giving an exponential distribution (Fig. 2).

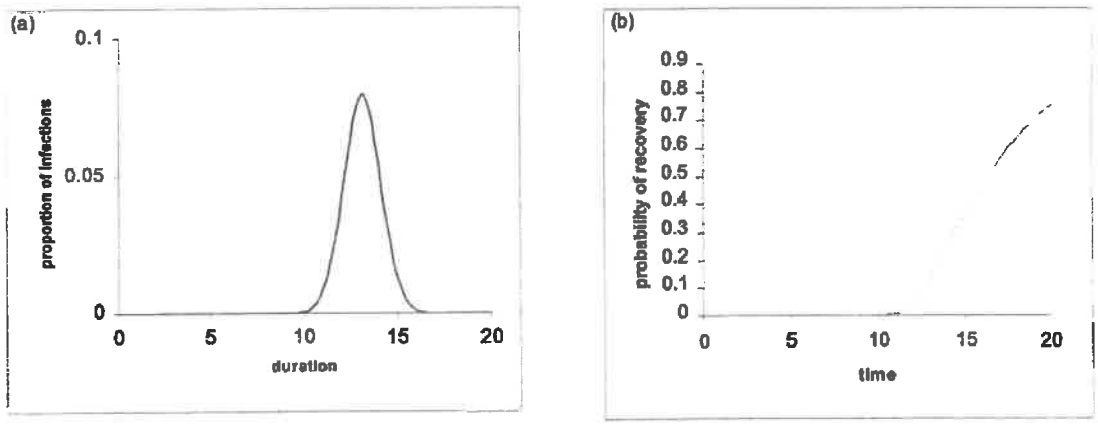


Figure 1. (a) Frequency distribution of duration of infection for an infection where the duration shows a normal distribution, with a mean of 13 days, (b) rate of recovery as a function of time since start of infection for the same infection.

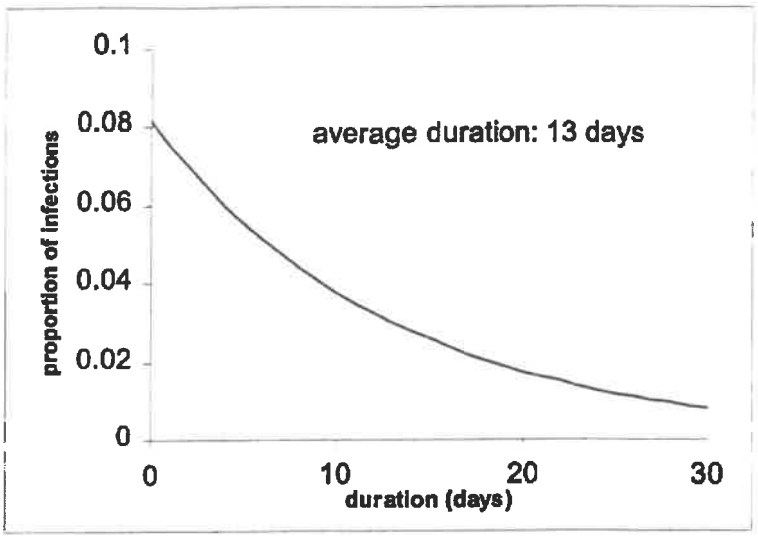


Figure 2. Frequency distribution of duration of infection for an infection where the rate of recovery is constant, with a mean duration of 13 days.

In the case of the OARDC herd, it is known (to within at most a month) when each infection started. In the case of the IAH data, however, infections were detected at the herd tests, and it is not known when they began. Data of this type are referred to as being *left-censored*. Because the IAH data are left censored, and the OARDC data are not, the time since detection for the IAH data, and the time since the start of infection for the OARDC data are considered.

In some cases the infections under consideration did not recover before the end of the study, or before the cow left the herd. These infections give rise to *right-censored* data. Right censored data are still informative, indicating a minimum duration.

Survivorship curves

The Kaplan-Meier method was used to estimate the probability of an infection persisting as a function of time since infection (in the case of OARDC) or detection (in the case of IAH). This method allows the inclusion of right censored data, and the effect of various factors to be compared. Categorical factors were compared using the log-rank test, and continuous variables using the Cox proportional hazards model. Factors considered in this analysis were (i) whether the infection was clinical or not, (ii) the parity of the cow, and (iii) the stage of lactation. Because the IAH data set was small, it was not possible to analyse the effects of these factors in this data set.

By assuming a constant rate of recovery, the survivorship curves can be used to estimate the rate of recovery, and hence the average duration.

Recovery rates

To explore the effect of the time since start of infection on the rate of recovery, the rate was estimated directly for each month after infection. This involved dividing the number of infections which recovered in a given month by the maximum number which could have recovered (i.e. all those still infected at the start of the month, and which did not leave the data set as a result of right censoring during that month). The rates were then compared using a chi

squared test, comparing rates for 3 month intervals. Since for the IAH data set the time since the start of infection was not known, this analysis was only performed for the Ohio data set.

RESULTS

Twenty three infected quarters were included in the IAH data set, none of which were clinical infections. The OARDC data set, comprised 210 quarter infections, of which 67 were clinical and 143 sub-clinical.

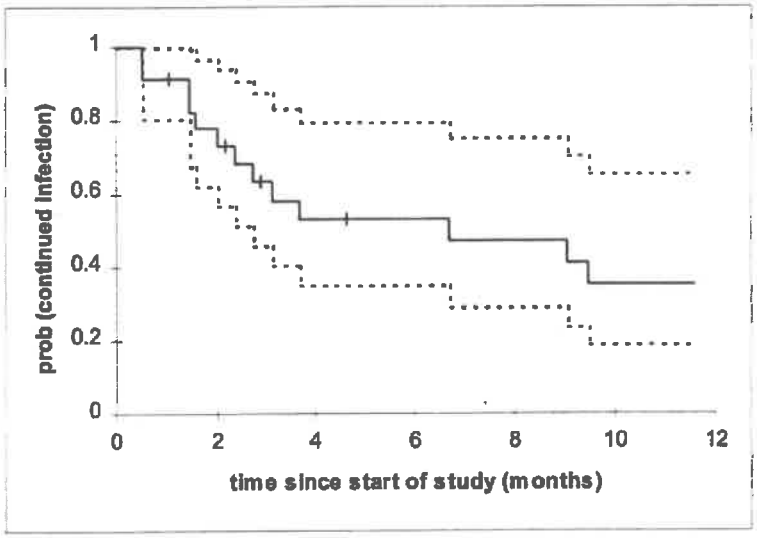


Figure 3. Kaplan-Meier curve for the IAH data set, showing the estimated probability of continued infection as a function of time since detection. The dotted lines represent 95% confidence intervals.

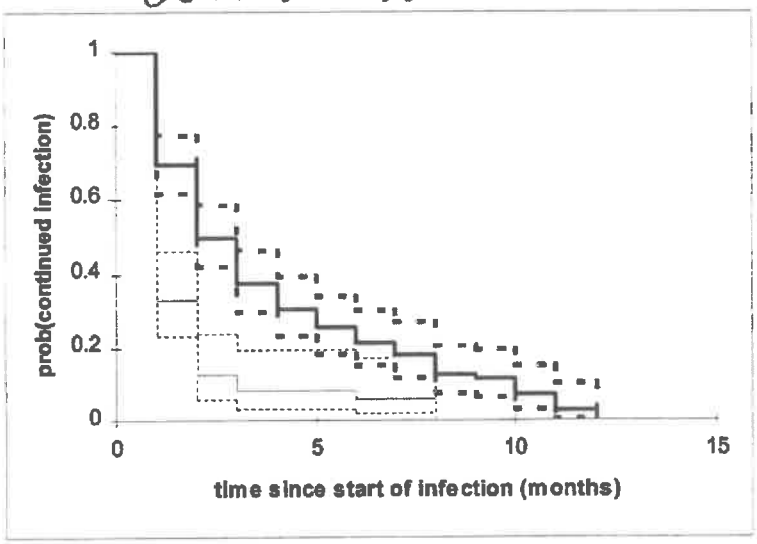


Figure 4. Kaplan-Meier curves for the OARDC data set, showing the estimated probability of continued infection as a function of time since infection. The heavy lines represent sub-clinical infections, the light ones clinical infections. The dotted lines represent 95% confidence intervals. The curves for sub-clinical and clinical infections are significantly different ($p<0.001$).

Figures 3 and 4 show the estimated probability of recovery as a function of time for the IAH and OADRC herds, respectively. The estimated average duration for sub-clinical infections at IAH was 8.5 months, and at OADRC was 1.2 months for clinical infections, 3.3 months for sub-clinical. The survival curves for clinical and sub-clinical infections at OADRC were significantly different ($p<0.001$). Neither parity nor stage of lactation significantly affected the survival curves.

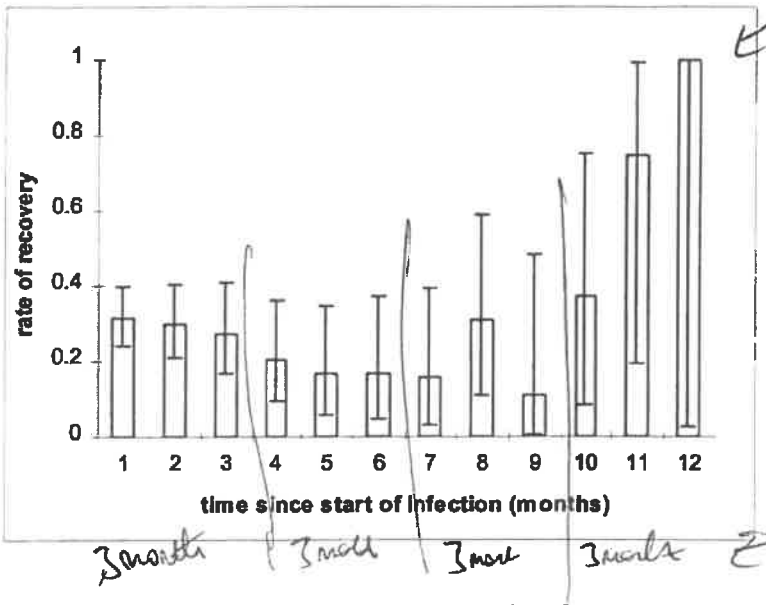


Figure 5. Estimated rate of recovery with time since infection for the sub-clinical infections in the OARDC herd. Error bars are exact binomial confidence intervals. The rates differed significantly between the 3-month intervals ($p < 0.025$).

Figure 5 shows the estimated rate of recovery with time since infection for the sub-clinical infections in the OARDC herd. The error bars are exact binomial confidence intervals, calculated using the statistical package, EpiInfo. The rates differed significantly between the 3-month intervals ($p < 0.025$).

DISCUSSION

It is clear from this analysis that *Strep. uberis* infections can last a long time. In both data sets there were infections which lasted for 12 months. It is unsurprising that clinical infections recover more quickly than sub-clinical ones (Fig. 4), since they are routinely treated with antibiotics. The difference in average duration of sub-clinical infections at OARDC and IAH may be due in part to the treatment of sub-clinical infections at OARDC.

Figures 3 and 4 suggest that the distribution of durations is close to exponential, but the comparison of recovery rates (Fig. 5) shows that the rate of recovery is not, in fact, constant. The recovery rate is initially high, falling over the first 6 months, and then rising again over the next six months. The initial fall may be due to variation in the virulence characteristics of the bacteria, or in the effectiveness of the immune response of the cow. Those infections which are more easily cleared, whether due to pathogen or host factors, would recover soon

after infection, leaving the more persistent infections, which recover more slowly, leading to the observed reduction in recovery rate.

It is likely that the increase in recovery rate in the second six months after infection is a result of dry-cow therapy. Most *Strep. uberis* infections start early in lactation, or during the dry period (3). Therefore, in the majority of cases, dry-cow therapy will be applied 10-12 months after the beginning of the infection, leading to an increase in recovery rate at this stage.

This analysis has provided useful estimates for the duration of infections, although the evidence that the rate of recovery changes suggests that estimating a constant rate of recovery not entirely appropriate. The apparent impact of dry-cow therapy on recovery rates suggests that this remains a useful tool in preventing long-lived sub-clinical infections.

Future research will refine this work. The potential of DNA fingerprinting of bacterial isolates to distinguish between long-lived infections and re-infection is being explored. More detailed information about the treatment of infections in the OADRC herd will be used to improve the estimate of the rate of recovery of untreated vs. treated infections.

Improved understanding of the range of durations of infections will allow the development of mathematical models of *Strep. uberis* infections, which will be used to investigate the possible role of environmental sources of bacteria, and the likely impact of vaccines.

ACKNOWLEDGEMENTS

Data from OARDC were kindly provided by K.L. Smith and P.S. Schoenberger. I would also like to thank the farm staff at the IAH for their co-operation, and C. Donnelly for statistical advice. This work is part of a PhD project funded by the IAH and supervised by M.E.J. Woolhouse (University of Oxford) and J.E. Hillerton (IAH).

REFERENCES

1. BRAMLEY A J (1984) *Streptococcus uberis* udder infection - a major barrier to reducing mastitis incidence. British Veterinary Journal 140 328-335
2. BENTLEY R W, LEIGH J A & COLLINS M D (1993) Development and use of species-specific oligonucleotide probes for differentiation of *Streptococcus uberis*. Journal of Clinical Microbiology 31 57-60
3. SMITH K L, TODHUNTER D A & SCHOENBERGER P S (1985) Environmental mastitis: cause, prevalence and prevention. Journal of Dairy Science 68 1531-1553
4. PARMAR M K B & MACHIN D (1995) Survival Analysis: a practical approach. Chichester: Wiley.

COMPLIANCE OF NEW MILKING INSTALLATIONS WITH RELEVANT STANDARDS AND STATIC TEST PERFORMANCE

IAN OHNSTAD, ADAS

SUMMARY

Preliminary results are reported from one part of a Milk Development Council project comparing new milking installations. Twenty new milking parlours were visited within six months of installation to assess compliance with the relevant standards on installation and performance. No parlour met all of the requirements. Deviations varied from vacuum gauge faults to sealed test points. Static performance tests revealed incorrect working vacuum levels, serious faults in pulsation systems and large air leaks. Rubberware was very frequently poor. Some of the faults could seriously affect udder health.

INTRODUCTION

Capital investment on dairy farms has always required economic justification. With falling milk prices, this is now more important than ever. The decision to purchase new milking equipment must therefore be made after careful consideration of all relevant information.

Many developments in milking technology are now based on the technical recommendations of the International Standards Organisation (ISO) and the British Standards Institute (BSI) which are founded in research from around the world. This work has led, over 20 years, to lighter clusters, use of ACR systems, liners to minimise strip yields and reduce vacuum in the mouthpiece chamber, and effective pulsation. These developments have been made to improve milking performance, udder health and product quality in developed dairy industries.

At the same time, an alternative milking strategy has been developed using a liner designed to minimise liner slip. To ensure satisfactory milking, a heavier clawpiece is required. ACRs are not used as the issue of over milking is not considered of importance.

Any dairy farmer considering investing in new milking equipment may receive conflicting advice. To provide independent, practical information regarding the two milking philosophies and compliance with BS 5545 and ISO 5707, the usual measure of quality, the Institute for Animal Health, working in conjunction with ADAS and the SAC, has carried out a study sponsored by the Milk Development Council. Preliminary results on the compliance of the installation with British and international standards for installation and operation are reported along with other observations.

OUTLINE OF WORK

Twenty farms were selected. They had all invested in new milking equipment in the previous 12 months. Eleven farms were using large capacity clusters from a variety of manufacturers and nine farms were using the Dairymaster cluster. Initial visits were made

to the farms during the winter of 1996-7 when static tests were performed to at least BS5545:1988 and observations made relating to milking performance and teat condition. The initial visits were followed up on a sample of fourteen of the farms after six months to monitor any changes.

During the initial visit, details of the installations were recorded and compliance with BS ISO 5707 (1996) noted. Results from the static test were compared with the requirements of BS ISO 5707 (1996). This paper reports the results of installation compliance and the static test.

INSTALLATION COMPLIANCE

Installation compliance for the 20 farms is summarised in Table 1.

A major problem was identified with vacuum gauges. BS ISO 5707 (1996) clearly states that the gauge should be mounted where it can be read during milking and register within 1 kPa of the working vacuum level. Despite this, 6 of the farms had vacuum gauges which could not be observed during milking or were not reflecting the actual working vacuum.

The provision of automatic drain valves on all airlines is a necessary requirement yet one plant had no drain valves fitted while a second plant did not have drain valves fitted at the lowest point on the line. These points were quickly addressed by the manufacturer when the deficiency was pointed out.

All the plants tested were direct to pipeline. BS ISO 5707 (1996) requires that the milk line has an even and continuous fall towards the receiver with a minimum fall of 2mm per metre. One installation had a high point in the centre of the milk line with the milk line mainly supported by string!

BS ISO 5707 (1996) requires additional test points to be installed on new milking installations. As a number of the tested plants were ordered and installed before BS5545:1988 had been superseded, provision of test points was considered in terms of BS5545:1988. Despite this, four plants were inadequately provided with test points to assess air flow and vacuum level and one plant which had all necessary test points fitted had the points glued closed.

STATIC TEST RESULTS

A summary of the static test results for the 20 farms can be found in Table 2.

Working vacuum level was greater than the level recommended by the manufacturer on four of the plants tested at the first visit. One installation was operating at 2 kPa below the level recommended by the manufacturer. The working vacuum level recorded during the second visit had been adjusted at two of the units but remained incorrect on the remaining two. In addition a plant which had been operating at the recommended vacuum level during the first visit, was working 2 kPa higher on the second visit.

Pulsation rates and ratios were compared to the manufacturers recommendations. In addition, note was taken of pulsation line sizes, fitting of restrictors and vacuum line layout to estimate whether liner movement would be acceptable. During the first round of visits, 7 of the farms had unsatisfactory pulsation characteristics ranging from incorrectly adjusted rates and ratios and relays not operating, to inadequate vacuum line capacity for the number of installed units.

To comply fully with BS ISO 5707 (1996), the total air leakage through the regulator must be less than 35 l/min or 5% of the manual reserve, which ever is the greater. The vacuum regulator leakage exceeded these figures on two of the plants at the time of the first visit. One of these regulators had been rectified by the time of the second visit but the second unit had deteriorated further.

Air admissions into the vacuum system and milk system were measured where possible. Leakage into the airlines must not exceed 5% of the pump capacity while the maximum permissible leakage into the milk system is 10 l/min plus 2 l/min for every milking point. Twelve of the farms visited had leakage which exceeded the maximum permissible with the result that effective vacuum reserve was seriously compromised at two of the units.

Air bleeds were found to be either blocked or excessive at 4 of the parlours during the first round of visits. This number did not change between visits, although different units were affected.

Rubberware (long milk tubes, long pulse tubes and short pulse tubes) was found to be damaged or worn on 12 of the units at the first instance. On one farm 25% of short pulse tubes were holed at the first visit. This had increased to 38% by the time of the second visit. By the time of the second visit the majority of units were showing some rubberware which was damaged or worn (in need of replacement) with only two units recorded with completely satisfactory rubber condition.

CONCLUSION

Although the participating parlours were tested according to BS ISO 6690 (1996), due consideration was taken of the fact that many of the installations were ordered and installed before the publication of the new BS ISO standard. Most manufacturers had been aware of and aspired to the updated standard which is actually a minimum requirement and not a high level standard.

Despite this, it was not possible to report a single plant fully complying to the new requirements. Some of the faults reported were relatively minor, and unlikely to affect udder health or milking performance significantly to any great extent while a significant number of the faults identified would be likely to have serious effects on cow welfare.

ACKNOWLEDGEMENTS

This work was sponsored by the Milk Development Council and involves ADAS, Institute for Animal Health and the Scottish Agricultural College at Auchincruive. The co-operation of twenty dairy farmers is warmly acknowledged.

Table 1. Compliance with ISO/BSI standards on installation as determined on an initial visit to all plants then a second visit to selected farms after reporting on the plant

FACTOR		FARM																			
		A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T
Vacuum Gauge	Visit 1	✓	✓	✓	✓	X	X	X	X	X	✓	X	✓	✓	✓	✓	✓	✓	✓	✓	✓
	Visit 2	✓	✓	✓	-	-	X	X	-	X	X	-	-	✓	-	✓	-	-	✓	-	-
Satisfactory	Visit 1	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	X	✓	✓	✓	X	✓
Vacuum Line Drainage	Visit 2	✓	✓	✓	-	-	✓	✓	-	✓	✓	-	-	✓	-	✓	-	-	✓	-	-
Milk Line Fall	Visit 1	✓	✓	✓	✓	✓	✓	✓	✓	✓	X	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
	Visit 2	✓	✓	✓	-	-	✓	✓	-	✓	✓	-	-	✓	-	✓	-	-	✓	-	-
Provision of Test Points	Visit 1	✓	✓	✓	✓	✓	✓	✓	✓	X	X	✓	✓	✓	✓	X	✓	✓	X	✓	✓
	Visit 2	✓	✓	✓	-	-	✓	✓	-	X	✓	-	-	✓	-	✓	-	-	X	-	-

✓ = at or above standard X = below standard - = not tested

Table 2. Evaluation of performance in static tests on an initial visit to all farms then on selective farms after a report on the parlour performance

FACTOR		FARM																			
		A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T
Vacuum Level	Visit 1	X	✓	✓	✓	✓	✓	✓	✓	✓	X	✓	✓	X	✓	✓	✓	✓	X	✓	X
	Visit 2	✓	✓	✓	-	-	✓	X	-	✓	✓	-	-	X	-	✓	-	-	X	-	-
Pulsation	Visit 1	X	X	X	X	X	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	X	✓	X
	Visit 2	✓	X	✓	-	-	✓	✓	-	✓	✓	-	-	✓	-	-	X	-	X	-	-
Vacuum Regulator	Visit 1	✓	✓	✓	✓	✓	✓	X	✓	✓	✓	✓	✓	X	✓	✓	✓	✓	✓	✓	✓
	Visit 2	✓	✓	X	-	-	✓	✓	-	✓	✓	-	-	X	-	✓	-	-	✓	-	-
Excessive Vacuum Leakage	Visit 1	✓	X	X	✓	X	X	X	✓	✓	X	✓	✓	✓	X	X	✓	X	X	X	X
	Visit 2	✓	X	✓	-	-	X	✓	-	✓	✓	-	-	✓	-	✓	-	-	✓	-	-
Air Bleeds	Visit 1	✓	X	✓	✓	✓	✓	✓	X	✓	✓	X	✓	✓	✓	✓	✓	✓	✓	X	✓
	Visit 2	X	X	✓	-	-	✓	✓	-	X	✓	-	-	X	-	✓	-	-	✓	-	-
Rubberware	Visit 1	✓	✓	✓	X	X	X	✓	X	✓	✓	X	X	X	X	X	X	X	✓	X	✓
	Visit 2	X	X	X	-	-	X	X	-	X	✓	-	-	X	-	✓	-	-	X	-	-

✓ = at or above standard X = below standard - = not tested

WHERE ARE WE GOING?

VACCINES FOR BOVINE MASTITIS: FACT AND FICTION

ROBERT J YANCEY Jr, Central Research, Pfizer Inc., Groton, CT, USA

SUMMARY

At least one *Staphylococcus aureus* bacterin has been commercially available for a number of years, however no large, independent field trials have been published in refereed journals to support the efficacy of this vaccine. Experimental vaccines for *Staph. aureus* composed of pseudocapsule-enriched bacterins supplemented with α- and/or β-toxoids appear promising, but none of these have been commercialized. Recently the Institute for Animal Health at Compton reported that with a live strain or a bacterin of *Streptococcus uberis* protection against homologous strain challenges could be obtained. Nonetheless, these materials would not protect against heterologous strain challenges. Additional studies by that group using a small number of dairy cows have shown that a vaccine containing the plasminogen activator of *Strep. uberis* could provide protection against experimental challenge with a heterologous strain. At present, there exists only one highly effective vaccine for mastitis, the core-antigen vaccine for coliform mastitis. All of the commercially available vaccines for this disease are killed preparations (bacterins) of rough mutants of *Escherichia coli* strain J5 or *Salmonella* spp. Little success has been reported with vaccination against other mastitis pathogens.

INTRODUCTION

There are a number of problems uniquely associated with vaccination of dairy cows for mastitis (1). One of these is that mastitis, an inflammation of the mammary gland, is usually an immune response of the gland to invading microorganisms i.e. the disease is the immune response. Therefore, specific enhancement of the immune response through vaccination might also exacerbate the disease. Another of these problems is that the number of mastitis pathogens is large and the types heterogeneous. Watts (2) estimated that there are more than 135 agents of bovine mastitis. The majority of these infections, however, are caused by staphylococci, streptococci, and Gram negative bacilli, which are categorized as either contagious pathogens (e.g. *Staphylococcus aureus*, *Streptococcus agalactiae*, and *Streptococcus dysgalactiae*) or environmental pathogens (e.g. the coliforms and *Streptococcus uberis*). Vaccine efforts for bovine mastitis have concentrated mainly on the contagious and the environmental pathogens. In addition to these problems, the success of a mastitis vaccine is difficult to define. Success could be based on reduction in severity or frequency of clinical signs, prevention of new intramammary infections (IMI), or elimination of existing IMI (spontaneous cure rates). These and other issues will be discussed.

VACCINES FOR CONTAGIOUS PATHOGENS

While *Staph. aureus* bacterins are commercially available, efficacy has not been proven in large, independent field trials, the data from which have been published in peer-reviewed journals. One such commercial bacterin composed of a lysate of five isolates of different bacteriophage types was found to increase significantly the number of spontaneous cures in a small, experimental challenge trial (3), although the rate of clinical mastitis and new IMI were unaffected through three lactations (Table 1). In addition, the somatic cell count (SCC) was

significantly lower for the vaccinated group compared to the control group, although milk production was not affected. Although not published in a peer-reviewed journal (4), Pankey *et al.* also found this same bacterin to be effective at increasing spontaneous cure rates compared to unvaccinated controls in a field trial in three herds in New Zealand (Table 2). Again, there was no difference in new IMI. The results of these studies suggest that while bacterins may not be efficacious when disease or new IMI is the primary criterion, vaccination with certain bacterins may provide a management tool to reduce the frequency of subclinical infections within a herd.

Table 1. Efficacy of *Staph. aureus* bacterins in an experimental challenge model (3)

Treatment	Number			
	Cows	Quarters	New IMI	Spontaneous Cures (%)
Bacterin	10	40	33	24 (73)*
Protein A	10	40	29	24 (83)*
Unvaccinated	10	37	30	14 (47)

* $p \leq 0.025$ compared to unvaccinated controls

Vaccines against *Staph. aureus* based on conjugates of the type 5 capsular polysaccharide or surface proteins such as the fibronectin-binding proteins and protein A (Table 1) have also been the subject of active investigation (1,3,5,6). However, none of these experimental vaccines has been shown to provide protection in field trials in published reports.

Inclusion of staphylococcal toxoids and pseudocapsular materials in *Staph. aureus* vaccines have been a common strategy for a number of years. Watson and his colleagues in Australia were the first to demonstrate the importance of inducing opsonic IgG_2 in protection against *Staph. aureus*. Watson showed that a vaccine composed of killed *Staph. aureus,* cultured under conditions to simulate *in vivo* growth and induce a pseudocapsule, combined with staphylococcal toxoids and a mineral oil/dextran sulfate adjuvant, provided statistically significant protection from clinical mastitis in dairy heifers experimentally challenged with different strains of *Staph. aureus* (7). Nickerson *et al.* (8), confirmed that Watson's vaccine was effective in reducing new IMI in experimental challenge studies. When this vaccine was tested in a blinded trial involving 582 cows from five commercial Australian dairies, the incidence of clinical mastitis and new IMI due to *Staph. aureus* were significantly reduced (9; Table 2). In a more recent, larger field trial (1819 cows in 7 herds), Watson *et al.* (10; Table 2) found that although the number of clinical and subclinical mastitis cases due to *Staph. aureus* were lower in the vaccinated groups (45 cases in vaccinates, 67 in controls), these differences were not statistically significant across all herds. In the herd with the highest prevalence of staphylococcal mastitis, however, vaccination did significantly reduce the incidence of clinical and subclinical mastitis due to *Staph. aureus*.

Table 2. Efficacy of various *Staph. aureus* vaccines in field trials

Study (reference)	Number of cows/herds	Statistically significant ($p \leq .05$) reduction in:			
		Mastitis	New IMI	Exiting IMI	SCC
Pankey (4)	201/3	N	N	Y	NR
Watson (9)	582/5	Y	Y	NR	NR
Watson (10)	1819/7	N (Y)*	NR	NR	NR
Nordhaug (11)	108/16	N	N	NR	N
Giraudo (12)	30/1	Y	Y	NR	N
Calzolari (13)	164/2	Y	Y	NR	Y

N = no; Y = yes; NR = not reported
* Significant reduction in the high prevalence herd

In another recent placebo-controlled, multicenter field trial involving 108 heifers, a vaccine was used which contained killed whole cells with pseudocapsule and the α- and β-toxoids, in a mineral oil adjuvant (11; Table 2). While the incidence of clinical and subclinical *Staph. aureus* mastitis was numerically lower in the vaccinated group, 8.6% vs. 16.0% for the control group, these differences were not statistically significant. The rates of *Staph. aureus* IMI was also similar among groups.

A "combination vaccine" was recently evaluated in three commercial dairies in Argentina with a high incidence of staphylococcal mastitis (12,13; Table 2). This vaccine was composed of two different strains of *Staph. aureus*, a crude capsular extract of *Staph. aureus*, and one strain each of *Strep. uberis* and *Streptococcus agalactiae*, in an aluminum hydroxide adjuvant. The vaccine provided statistically significant protection from clinical and subclinical mastitis due to *Staph. aureus* in both heifers and cows compared to adjuvant-vaccinated controls. In addition, there was a significant reduction in IMI due to *Staph. aureus*. There was no impact on IMI or mastitis due to Streptococcus spp provided by this vaccine. It will be interesting to determine whether this vaccine will be effective against *Staph. aureus* in larger field trials. It is not obvious why this bacterin should be more effective than the pseudocapsule/toxoid-containing bacterins discussed above. It may be simply that these pseudocapsule-containing vaccines are more effective statistically, in herds with a high prevalence of *Staph. aureus* infection. It might also be that selection of the strains of *Staph. aureus* or the adjuvants used with these bacterins were important variables in these studies. Nonetheless, it appears that *Staph. aureus* vaccines require more work before they are ready for large scale application.

For *Strep. agalactiae*, vaccine research has been less prodigious. Immunization of cows with formalin-killed *Strep. agalactiae* cells was not protective (1,12). A French group (13) has evaluated a surface antigen, the X-protein, as a potential immunogen (1, 14). This antigen may be coupled with the group B polysaccharide to enhance protective response to that immunogen. No efficacy data have been published yet with those antigens as vaccines in cows.

Stop when
produce
PamA which
breaks down protein
of milk + then use
products for growth
⇒ vaccinate against
PamA + prevent
growth of Strep.

No success has been reported for vaccination against any of the other contagious agents such as *Streptococcus dysgalactiae* or *Mycoplasma* spp.

VACCINES FOR ENVIRONMENTAL MASTITIS PATHOGENS

With *Strep. uberis*, previous exposure does provide resistance to infection, at least with the homologous strain. Nonetheless, simple bacterins fail to provide protection in the field against this important environmental pathogen (12,13).

Studies at the Institute for Animal Health at Compton, UK, have shown that both killed, and especially live, vaccines provide protection against experimental challenge, however, protection occurred against challenge only with the homologous strain (15-17). The investigators concluded that protection did not correlate with the presence of either milk or serum opsonizing antibody and that the abundance of neutrophils in the milk (increased SCC) did not reduce the number of *Strep. uberis* in a quarter as it does for coliform mastitis. Leigh (17) hypothesized that interference with bacterial colonization or growth within the gland may have provided the protection from clinical mastitis seen in these studies. To further evaluate this hypothesis, the Compton group has investigated the role of the plasminogen activator, PauA, as a protective antigen for *Strep. uberis*-induced mastitis. Leigh recently reported on challenge-infection experiments in a small number of lactating cows using 100 μg partially purified PauA as the antigen (Table 3). PauA was administered with either incomplete Freund's adjuvant (IFA) or an experimental adjuvant containing an oil-in-water emulsion (SB62). Cows were vaccinated subcutaneously (SC) either five times (IFA) or two times (SB62) and challenged after a few weeks with virulent *Strep. uberis* in two of four quarters of each cow. In the SB62 adjuvanted group, significant protection from clinical mastitis was obtained and the majority of quarters cleared the infections. Since the PauA was isolated from a different strain than the challenge strain, the partially purified PauA provided heterologous strain protection in this trial. It was recently determined that the *pauA* genes from two strains of *Strep. uberis*, one from the USA and one from the UK, had over 99% sequence identity, suggesting that this antigen is highly conserved across strains (18). Whether highly purified PauA will provide protection in natural infection trials remains to be determined.

Table 3. Effect of vaccination with partially purified PauA on *Strep. uberis*-induced clinical mastitis in mid-lactation dairy cows (17)

Group	Adjuvant	# Cows	Quarters with:		Percent
			Clinical mastitis	Spontaneous cures	Protection
Control	None	5	9/9	0/9	0
PauA	IFA[1]	4	5/8	3/8	37.5
PauA	SB62[2]	4	3/8	5/8	62.5*

* $p \leq .05$ based on reduction of clinical mastitis compared to controls
# = number
[1] IFA = incomplete Freund's adjuvant, 100 μg PauA, five SC immunizations
[2] SB62 = experimental adjuvant, 100 μg PauA, two SC immunizations

One of the major advances in the control of environmental mastitis in the USA has been the introduction of core antigen vaccines for clinical coliform mastitis (CCM). The core antigen vaccines are bacterins composed of *Escherichia coli* or Salmonella spp strains which have lesions in their ability to produce complete lipopolysaccharide (LPS), resulting in rough (R) mutants. The most studied of these strains is *E. coli* J5, a genetically stable Rc mutant of *E. coli* O111:B4, and Re-17, a rough mutant of *Salmonella typhimurium*. These organisms have an exposed LPS core region which induces antibody that is cross reactive with most gram-negative bacteria, especially during periods of active growth. Core antigen bacterins provide protection against the multiple serotypes of *E. coli* as well as other genera and species of gram-negative bacteria which cause CCM. Several field studies (19-25; Table 4) have shown these core antigen bacterins to be effective in reducing the number of cases of CCM in herds. The mean percentage reduction of CCM from five field trials using J5 bacterins and one trial using a *Salm. typhimurium* Re-17 bacterin was 55% (Table 4). If the two-dose Salmonella bacterin study was eliminated from this analysis, the mean reduction provided by J5 bacterins was 65%. A partial budget analysis of vaccinating cows against CCM with the J5 vaccine (26) concluded that when >1% of cow lactations resulted in CCM, increased profits of $57/cow lactation could be obtained. This resulted in a 1700% return on investment from the J5 vaccination program as used in this model.

Table 4. Efficacy of core antigen vaccines at reducing clinical coliform mastitis (CCM) in field trials

Study (reference)	Vaccine type	Group	# of Doses	# of Cows	# of cases of CCM	Incidence (%)	Reduction (%)
Gonzáles (19)	J5*	Control	0	227	29	12.8	80
		Vaccinates	3	233	6	2.6	
Cullor (20)	J5	Control	3	229	25	10.9	70
		Vaccinates	3	212	7	3.3	
Cullor (21)	J5	Control	3	421	48	11.4	65
		Vaccinates	3	424	17	4.0	
Hogan (22,23)	J5	Control	0	112	9	8.0	78
		Vaccinates	3	113	2	1.8	
Gonzáles (24)	J5	Control	0	60	27	45.0	72
		Vaccinates	3	180	23	12.8	
McClure (25)	Re-17**	Control	0	646	90	13.9	41
		Vaccinates	2	646	53	8.2	
Mean	**Core**	**Control**	1	283	38	13.4	55
		Vaccinates	2.8	301	18	6.0	

* *E. coli* strain J5
** *Salm. typhimurium* strain Re-17
# = number

In contrast to the results of field trials, challenge trials have not been as successful in demonstrating the efficacy of J5. Hill (27) found that J5 vaccination did not protect cows from a virulent strain of *E. coli*. Others have since demonstrated that challenge models can be used to demonstrate differences between vaccinated and unvaccinated animals, but the differences between groups are usually small (28,29). It was also found in one study that changes in management practices can minimize differences afforded by J5 vaccination (22). Additionally, a recent study showed that vaccination of cows during lactation can cause a significant, short term reduction in milk production (30). Most of the core antigen vaccines, however, recommend vaccinating cows during the dry period. While the core antigen vaccines are not a magic-bullet for CCM problems, they do provide the dairy producer with a valuable tool.

CONCLUSION

While the problems associated with effective vaccination of dairy cows for mastitis are significant, there has been some progress towards this end within the last decade. The greatest progress has been in vaccines for environmental mastitis. Within the next decade, additional efficacious vaccines for several of the most common agents for bovine mastitis are likely. A review written at that time then can be more fact than fiction.

REFERENCES

1. YANCEY R J (1993) Journal of Dairy Science 76 2418-2436
2. WATTS J L (1988) Veterinary Microbiology 16 41-66
3 PANKEY J W, BODDIE N T, WATTS J L & NICKERSON S C (1985) Journal of Dairy Science 68 726-731
4. PANKEY J W, DURIS G, MURRAY G & TWOMEY A (1983) Dairy Research Report 1983, Hill Farm Research Station, Homer, LA. pp 151-161
5. GILBERT F B, POUTREL B & SUTRA L (1994) Vaccine 12 369-374
6. MAMO W, JONSSON P, FLOCK J-I, LINDBERG M, MÜLLER H-P, WADSTROM T & NELSON L (1994) Vaccine 12 988-992
7. WATSON D L (1992) Research in Veterinary Science 53 346-353
8. NICKERSON S C, OWENS W E & BODDIE R L (1993) Journal of Dairy Science 76 1290-1297
9. WATSON D L & SCHWARTZKOFF C L (1990) *In:* International Symposium on Bovine Mastitis, National Mastitis Council, Arlington, VA, pp 73-76
10. WATSON D L, McCOLL M L & DAVIES H I (1996) Australian Veterinary Journal 74 447-450
11. NORDHAUG M L, NESSE L L, NORCROSS N L & GUDDING R (1994) Journal of Dairy Science 77 1267-1275
12. GIRAUDO J E, CALZOLARI A, RAMPONE H, RAMPONE A, GIRAUDO A T, BOGNI C, LARRIESTRA A & NAGEL R (1997) Journal of Dairy Science 80 845-853
13. CALZOLARI A, GIRAUDO J E, RAMPONE H, ORDIERNO L, GIRAUDO A T, FRIGERIO C, BETTERA S, RASPARANTI C, HERNÁNDEZ J, WEHBE M, MATTEA M, FERRARI M, LARRIESTRA A & NAGEL R (1997) Journal of Dairy Science 80 854-858
14. RAINARD P, SARRADIN P & POUTREL B (1995) *In*: 3rd IDF International Mastitis Seminar, M. Lachmann Printers Ltd, Haifa, Israel S-1 82-83

15. FINCH J M, HILL A W, FEILD T R & LEIGH JA (1994) Infection and Immunity 62 3599-3603
16. FINCH J M, WINTER A, WALTON A & LEIGH J A (1997) Vaccine 10 1138-1143
17. LEIGH J A (1997) *in,* Udder Heath Management for Environmental Streptococci, National Mastitis Council, Arlington, VA, pp 59-74
18. ROSEY E L, YANCEY R J & LEIGH J A (1997), Unpublished data.
19. GONZÁLEZ R N, CULLOR J S, JASPER D E, FARVER T E, BUSHNELL R B & OLIVER M N (1989) Canadian Journal of Veterinary Research 53 301-305
20. CULLOR J S (1991) Veterinary Medicine 86 836-844
21. CULLOR J S (1993) *In*: 1993 Coliform Mastitis Symposium, Veterinary Learning Systems, Washington State University
22. HOGAN J S, SMITH K L, TODHUNTER D A & SCHOENBERGER P S (1992) Journal of Dairy Science 75 78-84
23. HOGAN J S, SMITH K L, TODHUNTER D A & SCHOENBERGER P S (1990) *In:* International Symposium on Bovine Mastitis, National Mastitis Council, Arlington, VA, pp 200-204
24. GONZÁLEZ R N, WILSON D J & SEARS P M (1995) *In*: 3rd IDF International Mastitis Seminar, M. Lachmann Printers Ltd, Haifa, Israel S-4 64-68
25. McCLURE A M, CHRISTOPHER E E, WOLFF W A, FALES W H, KRAUSE G F & MIRAMONTI J (1994) Journal of Dairy Science 77 2272-2280
26. DeGRAVES F J & FETROW J (1991) Journal of the American Veterinary Medical Association 199 451-455
27. HILL A W (1991) Veterinary Research Communications 15 7-16
28. HOGAN J S, WEISS W P, TODHUNTER D A, SMITH K L & SCHOENBERGER P S (1992) Journal of Dairy Science 75 415-422
29. HOGAN J S, WEISS W P, SMITH K L, TODHUNTER D A & SCHOENBERGER P S (1995) Journal of Dairy Science 78 285-290
30. MUSSER J M B & ANDERSON K (1996) Journal of the American Veterinary Medical Association 209 1291-1293

Noran far G-tve [illegible] lachan
[illegible] Vroos cozge [illegible]
Shoel
Zuhrdelay
oran yeni ve
[illegible] [illegible]
Stat. t/h [illegible]

THE SCANDINAVIAN WAY OF TREATING COWS WITH ACUTE CILINICAL MASTITIS

PER JONSSON, DVM, PhD, University of Agricultural Sciences, College of Veterinary Medicine, Department of Veterinary Microbiology, Box 7073, S-750 07 Uppsala, Sweden

SUMMARY

The distribution of antibiotics in the udder after intramammary treatment is poor. After parenteral administration, however, the concentration of antibiotics is higher at the infection site. According to restrictions for use of drugs, all cases of acute mastitis in Scandinavia are treated by a veterinarian by the parenteral route for 3-5 days or longer depending on the bacteria involved. The practising veterinarian usually take a milk sample which is examined in the "home laboratory" on SELMA-plates or other blood agar plates. A cow-side test (LIMAST) can also be used at the visit to the farm. This test gives within 15 minutes the answer gram negative or gram positive infection. The antibiotic treatment is therefore always directed and against the mastitis causing microorganism. Narrow spectrum antibiotics are used in most cases. The Scandinavian antibiotic policy is probably the reasons for the good situation with low resistance to antibiotics.

INTRODUCTION

Studies by Funke more than 25 years ago showed that the distribution of various antibiotics in the inflamed udder after intramammary administration is poor. After parenteral administration, however, the concentration of the antibiotic used is higher at the site of infection (1). These findings, in combination with additional scientific knowledge and practical experience, have led to the situation that almost all cases of acute clinical mastitis in the Scandinavian countries are treated with antibiotics administered by the parenteral route for 3-5 days or longer depending on the bacteria detected (2,3,4). Intramammary treatment is often used complementarily.

A veterinarian treats all cases of acute mastitis in Scandinavia because of the restrictions on use of drugs. This may be one of the reasons for relatively minor problems with antibiotic resistance in Scandinavia. Most *Staphylococcus aureus* strains (94%) and all "mastitis streptococci" are *in vitro* sensitive to penicillin in Sweden (5). About 30% of coagulase-negative Staphylococci are penicillinase producers. The situation is about the same in the other Scandinavian countries.

Milk sampling, with improved bacteriological diagnostic possibilities in the field, has given the practitioner better opportunities to select a drug with its effect directed against the mastitis causing microorganism and a treatment regime that is suitable for the individual cow. The importance must be stressed of taking into consideration parameters such as clinical investigation of the udder quarter, case history (including previous cell count

registrations of the diseased cow), stage of lactation, age etc. when treating a cow with acute mastitis.

RAPID TESTS FOR AETIOLOGICAL DIAGNOSIS

Mastitis may arise as a consequence of trauma or physiological disturbance without microbial involvement but is usually a consequence of udder infection with one or more species of microorganism. Most often bacteria are implicated, but fungi, algae and even multicellular parasites can be responsible for mastitis. Only some bacterial species are responsible for most bovine mastitis cases. In the Scandinavian countries infections caused by Staphylococci, Streptococci and coliform bacteria, mainly *Escherichia coli*, amount to about 90% of the clinical cases, where bacteria can be isolated.

Bacteriological examination of milk samples of cows with acute clinical mastitis and identification of the aetiological microorganism have been used for many decades in countries with control methods against bovine mastitis. This gives necessary information to aid choice of therapeutic agent, time of treatment and prognosis.

In Scandinavia, central and regional laboratories with excellent facilities to examine milk samples are spread over each country. However, sending of a milk sample to these laboratories by post, means that a bacteriological diagnosis will not be obtained earlier than 2-3 days, or longer, after the sample is taken. This is too long to be able to use the result for treatment of the individual cow.

For this reason tests (systems) have been developed which make it possible for the practising veterinarian to perform the bacteriological examination or to get a preliminary aetiological diagnosis in the field.

THE SELMA®-PLATE

SELMA® is a petri dish divided into three fields containing different agar media: bovine blood agar containing esculine, staphylococcus medium and gram-negative medium. This combination of selective substrates enables veterinarians who are not specialised in bacteriology to differ between the most common mastitis pathogens.

The plates are incubated in a small table-incubator at 37°C for 18-24 hours. If no growth is seen the plate is incubated for another 24 hours.

Staph. aureus of bovine origin grows with a clear and a diffuse zone of heamolysis on the blood agar field and gives a yellow colour on the staphylococcal field. Coagulase-negative staphylococci are most often lacking the clear zone of haemolysis and never produce a diffuse zone on blood agar. Many strains of coagulase-negative staphylococci are not changing the staphylococcal medium into yellow.

Streptococcal species grows only on the blood agar field. It is not easy to differ between the various species of streptococci by this method but *Streptococcus agalactiae* are most often ß-haemolytic (clear zone), *Streptococcus dysgalactiae*, α-haemolytic (diffuse, green zone) and *Streptococcus uberis*, non-haemolytic.

Coliform bacteria grow on the blood agar field and on the coliform medium. *Escherichia coli* and Klebsiella species grow with purple colonies on the coliform medium.

The staphylococcal field can be used to test staphylococcal strains for the production of β-lactamase (penicillinase). With the use of a Nitrocephin disc which is placed on staphylococcal colonies and then incubated for 15-30 minutes at 37°C.

Bacteriological examination of milk samples in a "home laboratory" by using the SELMA® plate enables the practising veterinarian to obtain an aetiological diagnosis within 24 hours of the milk sample was taken. This makes it possible to change therapy, if this was initially wrong, or continue with the proper treatment if the cow shows no signs of recovery within the first day of treatment. The SELMA® plate is commercially available and today is used in Denmark, Finland and Sweden. In Denmark most practising veterinarians use a different "home laboratory" system.

LIMAST-TEST

The LIMAST test is a rapid Limulus test, which is modified for use as a cow-side test (6). By the use of this LIMAST test it is possible to differ between Gram negative and Gram positive bacteria in a milk sample within 15 minutes. The test is based on the fact that endotoxin (lipopolysacharide) which is part of the cell wall of Gram negative bacteria coagulates the amoebocytes of the horse-shoe crab (*Limulus polyphemus*). The Limulus test is an extremely sensitive method, which detects picograms of endotoxin. The LIMAST test is a modified substrate method of the Limulus test, which after 15 minutes incubation at 37°C can detect the presence of gram negative bacteria (*E. coli*), by a colour change to yellow.

To perform the test a milk sample is taken *lege artis* in a plastic tube. A 0.2 ml aliquot is transferred to an ampoule with diluting suspension. From this ampoule another 0.2 ml is transferred to the test ampoule with Limulus lysate. This ampoule is incubated at 37-40°C (water bath or bucket) for 15 minutes. If the contents of the ampoule turn yellow then the milk sample contains Gram negative bacteria and when the colour is unchanged, the mastitis is caused by Gram positive bacteria.

The LIMAST test is commercially available in the Scandinavian countries.

By the use of LIMAST test and the SELMA® plate in combination it is possible with LIMAST to obtain information whether the mastitis is caused by Gram positive or Gram negative bacteria. If Gram positive the penicillin can be used in most cases. In cases of mastitis caused by gram negative bacteria, antibiotics such as Trimethoprim/Sulpha, Enrofloxacin (used in Sweden) or other broad-spectrum antibiotics effective against gram-negative bacteria are the drugs of choice. In these cases of mastitis supportive treatment such as frequent milkings, rehydration therapy etc. is important and antibiotic treatment probably of minor importance (7).

The day after the milk sample is taken the SELMA® plate is examined. Now it is possible to confirm the diagnosis obtained by the LIMAST test and to decide definite treatment regime (type of drug, time of treatment, dosage etc.).

TREATMENT

There are slight differences in treatment regimes in the various countries in Scandinavia. The Swedish regime is discussed.

In all the three Scandinavian countries the aim is to direct the treatment against the mastitis-causing microorganism, which means that only one single component antibiotic with efficacy against the pathogen causing the mastitis. Cocktails containing several antibiotics in combination are not used, except the combination penicillin/dihydrostreptomycin, which is still available but seldom used. This Scandinavian way of mastitis treatment is based on efficacious, ecological and economical considerations.

It is regarded as important that clinical cases of mastitis are treated intramuscularly or intravenously. Both field trials and treatment trials on experimentally infected cows shows that the parenteral treatment should be given over more than one day (8,9).

In general it is recommended that streptococcal infections should be treated over at least 3 days and *Staph. aureus* infections over at least 5 days. In cases with severe clinical signs the length of treatment should be longer. The Swedish recommendations for treatment of the most common mastitis pathogens are presented in Table 1. Again, it is important to emphasise that all cases should also be treated individually according to clinical signs, stage of lactation, value of the cow etc.

Penicillin is the first choice because of its high activity against streptococci and staphylococci. The highest concentrations in the udder are obtained by potassium benzyl-penicillin given twice a day. Spiramycin is the second choice and first choice on staphylococcal strains producing penicillinase.

Broad-spectrum antibiotics such as Oxytetracycline and Trimethoprim/Sulpha are third choice drugs. These drugs have no advantages and in general higher MIC-values, compared with penicillin and Spiramycin.

For cases caused by coliform bacteria Enrofloxacin and Trimethoprim/Sulpha are the most commonly used drugs in Sweden today with which it is possible to obtain high enough concentrations in the milk.

An extensive clinical trial was carried out in Sweden 1982 in order to evaluate the effect of parenteral treatment with penicillin on the elimination of udder pathogens in bovine udder quarters with clinical mastitis. The cure rate (clinical recovery and udder quarter free from bacteria 1 week and 8 weeks after treatment) was, after a 5 days parenteral penicillin treatment, 63%. This is much higher than reported from intramammary treatment in other countries (10). The cure rates for streptococci and coliform bacteria were high (71% and 82% respectively) in this trial (9).

The spontaneous recovery rate for mastitis caused by Gram positive bacteria is estimated to be about 20-25% (10) but for *E. coli* as high as 70-80% (10,7). Especially, in peracute cases of mastitis caused by *E. coli* it is important to use supportive therapy in form of intravenous liquid therapy, frequent milkings after administration of oxytocin and administration of anti-inflammatory drugs.

FAILURE MECHANISMS IN MASTITIS TREATMENT

Though *Staph. aureus* strains are sensitive to ß-lactam drugs *in vitro*, they may be difficult to eliminate *in vivo*. Thus, *Staph. aureus* strains phagocytosed by bovine neutrophils are less susceptible to ß-lactam drugs (11,12). Moreover, the distribution of these drugs in mastitic udder quarters are often uneven (12,1).

Reasons for failure in antibiotic treatment of mastitic infections include too low a dose or too long dose intervals, pharmacokinetic problems in keeping adequate levels of antibiotic over a required period of time, diffusion barriers during intramammary treatment, problems in delivery of antibiotic across the blood/milk barrier during parenteral treatment, binding of the antibiotic to milk proteins, L-forms of bacteria, bacterial cells in the stationary phase (non-multiplying) being insensitive to antibiotics.

Although it is not possible to eliminate totally either clinical or subclinical cases of mastitis the Scandinavian countries i.e. Denmark, Norway and Sweden as well as Finland have a fairly good udder health (Table 2). This is probably due to extensive prophylactic control measurements in combination with effective treatment of clinical and subclinical mastitis.

REFERENCES

1. FUNKE H (1961) Acta Veterinaria Scandinavica 2 Suppl 1
2. EKMAN T, FRANKLIN A, HALLÉN-SANDGREN C & JONSSON P (1995) Svensk Vet Tidn 47 59-61
3. EKMAN T, FRANKLIN A, HALLÉN-SANDGREN C & JONSSON P (1995) Svensk Vet Tidn 47 665-669
4. EKMAN T, FRANKLIN A, HALLÉN-SANDGREN C & JONSSON P (1995) Svensk Vet Tidn 47 713-718
5. NILSSON L, FRANKLIN A & FUNKE H (1997) Proceedings Posters Society for Veterinary Epidemiology and Preventive Medicine Annual Conference Chester England
6. JONSSON P & FRANKLIN A (1995) Svensk Vet Tidn 47 317-319
7. KATHOLM J (1989) Dansk Vet Tidskr 72 171-177
8. FRANKLIN A, HOLMBERG O, HORN AF RANTZIEN M & ÅSTRÖM G (1984) American Journal of Veterinary Research 45 1398-1402
9. FUNKE H (1982) Symposium on Mastitis 21-23 April 1982 at Novo Industri A/S Copenhagen
10. BRAMLEY A J & DODD F H (1984) Journal of Dairy Research 51 481
11. CRAVEN N & ANDERSON J C (1980) Journal of Veterinary Pharmacology and Therapeutics 3 221-226
12. CRAVEN N & ANDERSON J C (1982) Journal of Comparative Pathology 92 579-588

Table 1. Recommended antibiotic therapy in Sweden

Mastitis pathogen	First choice	Second choice	Length of treatment (days)
Staph. aureus	K-benzyl penicillin (6-10 mg/kg x 2/day) or procaine penicillin (20-30 mg/kg/day)	Spiramycin (8 mg/kg/day)	5
Coagulase-neg. staph.			3-5
Step. dysgalactiae			3
Strep. uberis			4-5
Other streptococci			3
Actinomyces pyogenes			5-10
E coli	Trimethoprim/Sulpha (3/17 mg/kg x 2 day) Enrophloxacin (2,5 mg/kg/day)	Dihydrostreptomycin (25 mg/kg x 2 day)	3
Klebsiella spp.			3-5
Other gram neg. spp.			

Table 2. Swedish policy for the use of antibiotics in treatment of acute mastitis

1. All use of antibiotics must be based on science and practical experience.

2. A goal in the veterinary practice should be to avoid treatment with antibiotics - if possible.

3. Treatment with antibiotics should always be based on the expected effect and prognosis. The cow's age, production, udder changes and anamnesis must be taken into consideration. Antibiotics should not be used in the following cases:

   - *E. coli* mastitis when the general signs of the cow are not severe (these cases should be treated by frequent milking, oxytocin, fluid and anti-inflammatory drugs)
   - relapsing mastitis if the signs are mild to moderate
   - chronic mastitis if the signs are mild to moderate

4. The veterinarian should always clinically examine the udder and the cow before treatment.

5. A milk sample for bacteriological examination should always be taken.

6. The therapy should be directed to the actual agent. The use of broad-spectrum antibiotics must be limited and antibiotics inducing resistance must be avoided and only be used on strict indications.

7. Antibiotics should be given in correct doses and for long enough to kill the microorganisms involved in the mastitis. The treatment should be interrupted when new information indicates use of an ineffective antibiotic.

PROGRESS IN MASTITIS CONTROL

A JOHN BRAMLEY, Department of Animal and Food Sciences, University of Vermont, USA

SUMMARY

Control of bovine mastitis continues to require daily attention to the "5-point plan" supplemented by measures such as pre milking teat dipping, improved machine milking and housing management. Over the course of the last 25 years these measures have been extremely effective in mastitis control. Coliform mastitis vaccines recently introduced into the USA have been widely adopted and are likely to be followed by staphylococcal and streptococcal vaccines over the next few years. These will be useful supplements to the existing control measures but will not replace them.

Public concern over issues such as antibiotic usage and food safety will continue, perhaps intensify. While non-antibiotic approaches to therapy have merit under certain circumstances identifying suitable cases on clinical grounds is difficult. Currently non-antibiotic techniques are limited to homeopathic remedies, external rubs and more frequent milking. In the future recombinantly produced cytokines and BST may also play a role in mastitis therapy.

INTRODUCTION

The tenth anniversary of the British Mastitis Conference is an opportunity to review progress since 1987 and present some future perspective. In 1984 a review of progress in mastitis control identified some key areas in which further progress was required (1). Among these areas were:

Short term strategies for improved mastitis control
"Careful and conscientious application of current control methods will still be needed"

"Discarding current methods because of their imperfections will result in a return to the immediate post war years in which reliance was on the treatment of clinical cases in lactation. That system failed then and would do so again"

"Most progress will be made by attention to reducing exposure to pathogens by attention to housing, the mechanics of milking and hygiene"

Long term strategies for improved mastitis control
Manipulation of the bovine mammary immune system
Genetic modification of host susceptibility
Identification key events and virulence factors in pathogenesis for their use in vaccines
Automated detection of mastitis to facilitate early treatment

Major barriers to better control
Control of exposure to environmental pathogens
Poor response of staphylococcal infections to therapy
Control of peracute coliform infections

CONTROL OF EXPOSURE TO PATHOGENS

Two areas of progress can be identified; one of which is more controversial in the UK than in the USA. Dipping of teats in disinfectant followed by wiping with a paper towel before cluster application was the subject of research trials in the USA and in the UK. The USA data indicate that "predipping" can reduce rates of environmental mastitis by about 50% (2). Shearn *et al.* (3) in a large trial were unable to show efficacy. Field experiments show marked variation between herds in effectiveness. This herd variation is not surprising since the effectiveness of predipping in a particular herd is likely to depend upon the functioning of the milking equipment, level of teat cleanliness, type of housing, and the predominant pathogens.

The control of exposure of the teat surface to pathogens outside the milking parlor remains a substantial challenge. The adage has become that "clean, dry and comfortable" should be the goal in terms of bedding systems. It has been clearly established that housing systems using organic bedding materials (e.g. sawdust, shavings, straw etc.) harbour higher counts of mastitis pathogens than do inorganic bedding materials. The inorganic bedding material that conveys the lowest risk factor for environmental mastitis is washed sand and this is being used in many herds to good effect.

MACHINE MILKING AND MASTITIS

Machine milking can directly increase rates of udder infection by two mechanisms, both of which were identified in the 1970s. These are:

a) the "impact mechanism" in which vacuum fluctuations generate rapid movement of contaminated milk back towards the teat end

b) the "no pulsation" effect in which inadequate liner collapse leads to increased growth of bacteria at the teat end.

Over recent years research on machine milking and mastitis has declined worldwide for several reasons. Firstly as milking equipment has been replaced newer equipment has generally provided improved vacuum stability beneath the teat reducing the probability of infections associated with the "impact mechanism". The increased use of static and dynamic machine testing has reduced pulsation failures and ensures that milking equipment provides adequate liner collapse time eliminating the "no-pulsation" effect. The widespread installation of automatic cluster removers reduces overmilking and usually prevents clusters being removed under vacuum. With properly functioning equipment an increased frequency of milking generally leads to a fall in the incidence of mastitis and in levels of milk somatic cell count.

Finally, the introduction of premilking teat disinfection reduces bacterial challenge at milking time and thus also reduces milking machine effects.

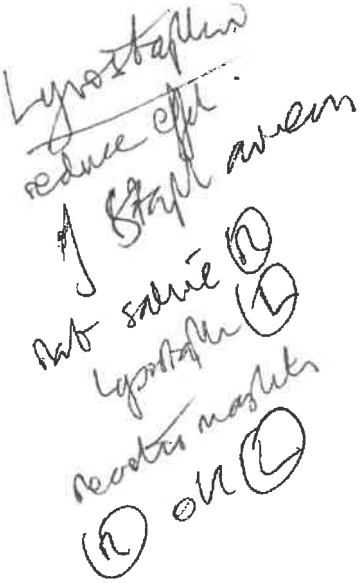
The major focus for research has been to measure the loads applied by the liner to the teat surface and identify their biological implications for stimulation of milk ejection, milk removal, teat condition and mastitis (4). These objectives have proved challenging and progress has been rather limited. Direct measurement of loads has not been achieved although indirect measurements have given some data on upper desirable limits. The hypothesis is that excessive loads applied by the liner to the teat surface will cause teat damage and facilitate bacterial invasion. The load applied to the teat by the liner is related to the characteristics of the liner and the rates and magnitude of pressure changes. Some techniques have been employed to measure the biological response of the teat to machine milking, most notably by the cutimeter, in which teat thickness readings before and after milking are compared (5). The repeatability and relevance of cutimeter readings to mastitis and the infection processes remain uncertain.

PATHOGENESIS OF MASTITIS

There has been substantial progress in our knowledge of the pathogenesis of mastitis. This progress has principally arisen from the application of techniques from molecular genetics and cell culture. Site-directed mutagenesis was applied to knock out virulence genes of *Staphylococcus aureus* and these mutants used to study pathogenesis and staphylococcal/host cell interactions (6,7). This work identifies the role of staphylococcal proteases and host plasminogen activators in enhancing bacterial growth in milk and aiding dissemination of the bacteria through the various tissues of the bovine mammary gland. In a series of experiments Leigh has also revealed that streptococci, particularly *Streptococcus uberis*, utilize plasmin to facilitate their growth in the mammary gland (8,9). Antibodies, which neutralize the *Strep. uberis* plasminogen activator, increase resistance to *Strep. uberis* experimental infection. This work holds some real promise of allowing the development of vaccines, which will offer broad protection against several mastitis pathogens simultaneously.

Mammary epithelial cells in culture have been used to investigate interactions between mastitis bacteria and host cell surfaces (10,11). These studies demonstrate that adhesion of bacteria to cells can occur in vitro and may be followed by invasion of the epithelial cells. These adhesion processes appear less specific and of lower intensity than those occurring in the enteric and urinogenital tracts. Specific adhesin or receptor molecules mediating these reactions have not been identified. Furthermore, evidence is still lacking that these adhesion and invasion occur frequently *in vivo*. Thomas *et al* (12) found no evidence of adhesion of *Strep. uberis* following experimental infection of the bovine mammary gland and extensive studies of the pathogenesis of *Staph. aureus* mastitis also failed to indicate that adhesion is a critical stage in pathogenesis (13).

A substantial amount of work has addressed the development of staphylococcal vaccines, particularly targeting capsular or surface polysaccharides as antigens. Some preliminary studies indicate that this approach can stimulate opsonic activity and increase host resistance (14).



HOST SUSCEPTIBILITY

Mastitis susceptibility shows a low level of heritability. This indicates that most of the variation in a population is associated with non-genetic factors. Whilst eradicating or reducing mastitis by selective breeding is attractive it is probably unrealistic. Most of the genetic factors influencing a cow's susceptibility to intramammary infections is related to factors related to the entry of bacteria into the mammary gland. For example, UK data showed that when the teat surface is experimentally contaminated with pathogenic bacteria very strong determinants of susceptibility are teat duct length and milk flow rate (15). Over the last 10 years information has been provided to farmers on the somatic cell counts of daughters of sires so that some aspects of inherited susceptibility can be accounted for (16). However, selection for higher yielding, faster milking cows may increase mastitis susceptibility (17). This clearly presents a substantial dilemma if selective breeding programme to reduce mastitis susceptibility are to be practical. The techniques of molecular genetics and animal science now allow genes to be introduced into the cow from other species to modify mammary gland function. This is termed transgenics and the most well known example with respect to ruminants is the production of pharmaceutical compounds in sheep milk. A current project in Vermont is to increase resistance of staphylococcal mastitis by expressing the gene for lysostaphin, a bacterial peptide which can kill *Staph. aureus*, in the mammary gland.

In an early British Mastitis Conference Dr Larry Smith presented data on research related to the effects of dietary selenium and vitamin E on mastitis susceptibility. Deficiencies of selenium and vitamin E impair immune function and result in increased rates of intramammary infection in both lactating and dry cattle (18). Work on other nutritional influences on mastitis susceptibility is in progress (19). Nutritional aspects may be important factors influencing both infection rate and severity in some herds.

Although various mastitis vaccines have been developed none have really stood the test of time in terms of widespread or long-term use. Gonzalez reported that a vaccine based upon the *Escherichia coli* J5 mutant protected against coliform mastitis (20). Field trials demonstrate a reduction in clinical coliform mastitis and a substantially lower incidence of the severe or peracute coliform infections. It is now widely applied in USA dairy herds.

HEIFER INFECTIONS

Infections in the late dry period or in heifers represent a significantly greater proportion of mastitis infections today than ten years ago. This is not because infection rates have increased but because of improved control of other infections.

The majority of preparturient infections in heifers are associated with coagulase negative staphylococci (21). These infections are usually mild or inapparent and their economic impact uncertain. A large proportion of the more severe preparturient infections in heifers is due to streptococci, particularly *Strep. uberis*. These will have a greater impact on milk yield and quality. Intramammary antibiotic administered prior to calving can reduce the incidence of

these infections at calving but risks introducing infection during antibiotic administration. In a series of studies in the USA and UK *Staph. aureus* infection of heifers was unusual. However, some herds in the southern USA have had significant outbreaks in heifers that may be associated with biting flies. Presumably these infections could be reduced by adequate fly control. Although prepartum dipping with conventional teat dips has little impact on these infections, Timms *et al.* (22) recently reported promising results from using a barrier teat dip to control infections at calving.

THERAPY

The major therapeutic limitation to improved mastitis control is the poor response of *Staph. aureus* infections, particularly clinical cases, to antibiotics (1). There are various reasons for the poor response including pathology, short duration of therapy in the lactating cow and antibiotic resistance. While new antibiotics have been developed or applied to mastitis including cephalosporins, semi-synthetic penicillins, these have not substantially improved the situation. It remains the case that a staphylococcal intramammary infection is most likely to be eliminated by dry period therapy.

There has been increasing interest in non-antibiotic approaches to treatment stemming from the economic costs of using antibiotics in lactation and the growing public concerns about antibiotics in the food supply. Many farmers, and some veterinarians, in the USA and the UK have expressed interest in alternative medicine approaches such as acupuncture and homeopathy (23). Some limited data are available but most material is anecdotal in nature. There is a need for clear, objective data and advice on this subject.

The use of frequent milking, whilst not a new technique, has had a resurgence in interest. Typically this now involves using oxytocin to improve the efficiency of milk removal. This approach works well for some forms of mastitis, particularly Gram negative infections, in which antibiotics play a small or negligible role in the elimination of infection. Frequent milking is of limited efficacy against infections due to staphylococci and streptococci unless supported by antibiotics. Consequently for optimum value this approach need to be correlated with some rapid diagnostic or culture system.

There has been a substantial amount of research and commercial interest in using various immunomodulators and cytokines, developed through biotechnology, to treat or stimulate resistance to mastitis (24,25). These compounds have a range of biological effects on inflammation, cellular immunology, and milk secretion. They have not yet developed to the stage where they have demonstrated efficacy or economy in mastitis therapy.

The approval and use of recombinant bovine somatropin (rBST) or growth hormone in dairy cattle has not been without controversy. There is some evidence that rBST indirectly increases clinical mastitis incidence because it increases milk yield (17). However, there is growing evidence that administration of rBST may speed the recovery from clinical mastitis and reduce milk yield losses (26). The recommendation that rBST be used as an adjunct to assist mastitis therapy can be anticipated.

REFERENCES

1. BRAMLEY A J & DODD F H (1984) Journal of Dairy Research 51 481-512
2. PANKEY J W, WILDMAN E E, DRECHSLER P A & HOGAN J S (1987) Journal of Dairy Science 70 867-872
3. SHEARN M F H, LANGRIDGE S, TEVERSON R M, BOOTH J M & HILLERTON J E (1992) Veterinary Record 131 488-489
4. BUTLER M C & HILLERTON J E 1989 Journal of Agricultural Engineering Research 44 77-86
5. HAMANN J, MEIN G A & WETZEL S (1993) Journal of Dairy Science 76 1040-1046
6. BRAMLEY A J, PATEL A H, O'REILLY M, FOSTER R & FOSTER T J (1989) Infection and Immunity 57 2489-2494
7. ZAVIZION B, BRAMLEY A J, POLITIS I, GILMORE J, TURNER J D, PATEL A H & FOSTER T J (1995) Journal of Dairy Science 78 277-284 1995
8. LEIGH J A (1993) FEMS Microbiology Letters 114 67-72
9. LEIGH J A (1994) FEMS Microbiology Letters 118 153-158
10. ALMEIDA R A, MATTHEWS K R, CIFRIAN E, GUIDRY A J & OLIVER S P (1996) Journal of Dairy Science 79 1021-1026
11. CIFRIAN E, GUIDRY A J, O'BRIEN C N & MARQUARDT W W (1995) Research in Veterinary Science 58 20-25
12. THOMAS L H, HAIDER W, HILL A W & COOK R S (1994) American Journal of Veterinary Research 55 1723-1728
13. ANDERSON J C (1982) Veterinary Record 110 372-376
14. WATSON D L, McCOLL M L & DAVIES H I (1996) Australian Veterinary Journal 74 447-450
15. GRINDAL R J, WALTON A W & HILLERTON J E (1991) Journal of Dairy Research 58 383-388
16. DEKKERS J C M, MALLARD B A & LESLIE K E (1994) Journal of Dairy Science 77 616-618
17. WHITE T C, MADSEN K S, HINTZ R L, SORBET R H, COLLIER R J, HARD D L, HARTNELL G F, SAMUELS W A, DE KERCHOVE G, ADRIAENS F, CRAVEN N, BAUMAN D E, BERTRAND G, BRUNEAU P, GRAVERT G O, HEAD H, HUBER J T, LAMB R C, PALMER C, PELL A N, PHIPPS R, WELLER R, PIVA G, RIJPKEMA Y, SKARDA J, VEDEAU F & WOLLNY C (1994) Journal of Dairy Science 77 2249-2260
18. ERSKINE R J, EBERHART R J, GRASSO P J & SCHOLZ R W (1989) American Journal of Veterinary Research 50 2093-2100
19. HARMON R J & TORRE P M (1994) Proceedings Annual Meeting National Mastitis Council Arlington VA pp 54-65
20. GONZALEZ R N, CULLOR J S, JASPER D E, FARVER T B, BUSHNELL R B & OLIVER M N (1989) Canadian journal of Veterinary Research 53 301-305
21. PANKEY J W, DRECHSLER P A & WILDMAN E E (1991) Journal of Dairy Science 74 1550-1552

22. TIMMS L L, STEFFENS A A & ALLEN L (1997) Journal of Dairy Science 80 Suppl p 225
23. DAY C (1991) New Farmer & Grower 21-23
24. PIGHETTI G M & L M SORDILLO (1995) Journal of Dairy Science 78 528-537
25. SORDILLO L M & L A BABIUK (1991) Veterinary Microbiology 28 189-198
26. HOEBEN D & BURVENICH C (1997) JOURNAL of Dairy Science 80 Suppl p225

ABSTRACTS OF POSTERS

BACTERIAL PATHOGENS IN BOVINE MASTITIS: A HISTORICAL PERSPECTIVE

T O JONES, Veterinary Investigation Centre, Sutton Bonnington

More than 200 species of pathogen can cause bovine mastitis. However, the great majority of cases in the United Kingdom are caused by a few species of bacteria. Changes which have occurred in the bacteriology of clinical mastitis in the United Kingdom this century are illustrated and explanations advanced for some of the changes.

General advice on the control of mastitis sometimes has to be modified because some of the causes change with time. The national herd at present is what would have been called a low cell count herd, and rather uncommon, a generation ago. It is important to define the national mastitis problem in our new situation, to ensure correct action.

PULSE TREATMENT OF CHRONIC *STAPHYLOCOCCUS AUREUS* INTRAMAMMARY INFECTION WITH CLAVULANIC ACID POTENTIATED AMOXYCILLIN WITH PREDNISOLONE

ANDREW BIGGS, The Vale Veterinary Laboratory, Tiverton

A herd with an elevated bulk milk cell count and an exceptionally high incidence of *Staphylococcus aureus* infected cows was selected for evaluation of pulse treatment of chronic *Staph. aureus* intramammary infection with *Clavulanic Acid Potentiated Amoxycillin with Prednisolone* (Synulox, Pfizer Animal Health). The principle was to maintain drug levels beyond the expected life of the neutrophil and improve cure rates. Individual quarters were sampled twice with an interval of one week to identify quarters persistently infected with *Staph. aureus* and to obtain individual quarter cell counts. One Synulox intramammary tube was infused into "treatment quarters" every 12 hours on three occasions. The standard 48 hours withhold was observed with the first two tubing sequences and then a 60 hours (OK at 6th milking) withhold was observed on the final sequence. This introduced a safety margin to avoid a bulk tank failure due to the high proportion of quarters under treatment in the herd.

The quarters treated were sampled at 7, 14 and 21 days after the last treatment to assess the cell count response and bacteria present.

MANAGING MASTITIS USING DECISION TREE ANALYSIS

J L FITZPATRICK, C NAISMITH & S J REID, Department of Clinical Veterinary Studies, University of Glasgow Veterinary School, Bearsden Road, Glasgow

Mastitis is a disease of major economic importance in the UK. While the adverse effect of clinical mastitis on both the profitability of dairy farming and the welfare of dairy cows is obvious, the effects of subclinical mastitis are far more difficult to recognise. Mastitis is a complex, multifactorial disease which can result from infection with a wide range of bacteria. A considerable amount of data relating to mastitis, such as bulk and individual cow somatic cell counts and antibiotic tube usage, is already available to the farmer and his veterinary surgeon. Making decisions about managing cows with mastitis is, therefore, complicated and computer-aided decision support tools may help to simplify the options available.

Decision free analysis is one such tool which can be developed for use on farms. An example for managing subclinical mastitis will be presented.

MASTITIS ORGANISMS AND HIGH SOMATIC CELL COUNTS

D N LOGUE, J GUNN, S CHAPLIN, H E TERNENT, J E OFFER & I SMITH, The Scottish Agricultural College, SAC Veterinary Services, SAC Auchincruive, Ayr, Scotland, UK KA6 5 HW

At SAC a pilot study has shown that the introduction of a herd specific programme will reduce the BTSCC more quickly than general advice (1). Study of the bacteriology of milk samples from this and other related studies since 1991 have yielded a number of interesting findings.

Examination of the general mastitis samples sent to SAC Veterinary Services VI Centres from 1991-95 shows that the predominant recoveries have been *Staphlococcus aureus* (17 to 28% depending on the year) and *Escherichia coli* (22 to 27%). Some 71% of *Streptococcus agalactiae* recoveries (6 to 12% overall) came from submissions of two or more samples and 22% from submissions with more than ten milk samples. Comparison of the Ayr pilot study, SAC herd investigations in Aberdeenshire (1974-90) (1,2) and VIDA data for multiple sample submissions (1991-95) shows that while *Strep. agalactiae* and *Staph. aureus* were the predominant subclinical pathogens throughout, there were differences in prevalence between populations. With the increased interest in BTSCC reduction in 1994/5 almost one third (32%) of the 589 herds who directly sought advice from SAC around that time carried out some bacteriological investigation(s). Good management data were available for 177 of these herds ("in-depth" herds). Comparison of the relative proportions of the five major isolates in these "in-depth" herds with the overall VIDA results from submissions of six or more samples showed that the relative proportions of *Staph. aureus* and *Strep. agalactiae* were quite similar for both but quite different from the pilot study. The last mentioned samples were solely from high SCC herds (BTSCC regularly $>400,000$ cells/ml).

Herds undertaking bacteriology were much more likely to test individual cows for cell count regularly ($p< 0.01$) and as a consequence had a lower BTSCC. There were highly significant ($p <0.001$) differences in the isolation frequencies of the different pathogens from herd tests (≥ 6 per submission) compared with samples from clinical cases (> 6 samples per submission). Although the proportion of *Staph. aureus* isolates was essentially unaffected by milk recording, such herds had proportionally more environmental isolates particularly *Streptococcus. uberis* ($p<0.05$). Possibly related to this last finding herds with a parlour milking system, rather than a byre, had a significantly ($p<0.05$) lower number of *Strep. agalactiae* isolates and greater numbers of *E. coli*. Few were using pre-milking teat dipping, but post-milking teat dipping (PMTD) was associated with a higher number of *E. coli* and, to a lesser extent, *Streptococcus dysgalactiae*. The use of DCT was associated with a significantly lower proportion of *Strep. agalactiae* isolates ($p<0.05$) and, a higher rate for *E. coli* though the numbers for the latter in non DCT herds were small for statistical purposes. *Strep. agalactiae* was significantly ($p>0.05$) more common in those herds that did not have an annual milking machine check while *E. coli* was less frequently isolated from these herds (*Staph. aureus* was also relatively more common but not significantly so). There was no strong evidence that the type of building, bedding or presence of floor slats affected the proportion of isolates. However, buying-in cattle was associated with a significantly higher number of *Staph. aureus* but lower number of *E. coli* ($p<0.05$).

In summary these data generally confirm that regular ICSCC testing, leading to targeted sampling and bacteriology of specific cows to identify the major pathogens acting in a herd, aids in the development of a control strategy for the two major cow-side pathogens *Staph. aureus* and *Strep. agalactiae*. However, success in controlling these pathogens may be associated with an increased vulnerability to infection with the environmental organisms. Such strategies must take account of this and recommend scrupulous environmental cleanliness to reduce the level of challenge of these environmental organisms.

ACKNOWLEDGEMENTS

We gratefully acknowledge the financial and practical assistance of the three Milk Marketing Boards in Scotland and subsequently the Milk Buyers in Scotland. We also gratefully acknowledge the EC for financial support, the many farmers and their veterinarians, and the SAC Veterinary Services staff without whom this study would not have been possible. The Scottish Agricultural College receives funding from the SOAEFD.

REFERENCES

1. GUNN J (1995) A study of mastitis and high somatic cell counts in Scottish dairy herds. PhD Thesis Glasgow University 1995.
2. LOGUE D N, GUNN J & FENLON D (1995) In: *Quality Milk from Grass. Proceedings of the British Grassland Society Winter Meeting 5th and 6th December 1994*, pp 23-34

ISLE OF MAN CELL COUNT PROJECT - MAY 1995- MAY 1997

H TERNENT, B WILSON* & D N LOGUE, SAC Veterinary Services, Auchincruive Ayr and *Isle of Man Creameries, Isle of Man

At the end of 1994 one third of the herds on the Island were in excess of the EC threshold for SCC of 400,000 cells/ml. A project was established to encourage a reduction in this figure. Study of monthly profiles has shown that during peak output most farms need to aim for a BTSCC of less than 200,000 cells/ml to avoid a penalty at critical times in the year. In May 1997 the Islands average BTCC was 186,000 cell/ml. Over the two years there was an increased awareness that sub-clinical mastitis was the underlying cause of high SCC figures and an understanding of the main principles of its control. This was reinforced by increasing penalties in co-ordination with advisory inputs. Virtually all farms made some changes in their management or improvements to their farm and their rate of progress can be linked to the extent of change (Table 1).

Table 1. Effect of management changes on BTSCC

Number of Management Changes	Average BTSCC (000) - 5 month period Jan-May			Index of Progress
	1995	1996	1997	
>3	379	217	193	+49
<3	287	174	182	+37
None	288	191	194	+33

Greatest progress was made by those who recorded individual cow SCC figures (ICSCC) especially in the first year. Herds were divided into those which undertook a herd profile (5 or more samples for bacteriology) or sent in fewer than five samples (usually one or two individual samples). Those doing a herd profile made the most progress (Table 2).

Table 2. Value of bacteriological herd tests in control of BTSCC 1995-1997

Categories of bacteriology submissions	Average BTSCC (000) - 5 month period Jan-May			Index of Progress
	1995	1996	1997	
Individual cows	304	217	216	+29
Full herd profile	425	243	196	+54

Analysis of data from the past two years has highlighted a number of financial benefits linked to the improvements in somatic cell count levels. These include:

Increased yields (both in milk and cheese);
Reduction in mastitis-linked costs, i.e. vet bills and lost milk;
Increase in farms receiving bonus payments (50% increase!);
Improved efficiency arising from better record-keeping.

Overall it is estimated that these improvements in yield and quality were worth as much as £50 per cow per year!

In summary these results show that it is possible to reduce the BTSCC of a population of herds quite dramatically by the correct balance of advice, aid and penalties and premium payments for milk quality.

ACKNOWLEDGEMENTS

Funding from IOM Department of Agriculture, Fisheries and Forestry; farmers and veterinarians on the Isle of Man; staff at Isle of Man Creameries, Knockaloe and NMR are gratefully acknowledged. SAC receives funding from SOAEFD.

EFFECT OF POLYMORPHONUCLEAR LEUCOCYTE CONTENT OF MILK ON DAIRY PRODUCT QUALITY

S M COONEY[1], D TIERNAN[2] & A L KELLY[1], [1]Department of Food Technology, University College Cork, [2]Department of Zoology, University College Dublin

Cheese quality is greatly affected by increases in milk somatic cell count (SCC) which can arise due to mastitis or seasonal variations in the milk supply, such as in late lactation. Specifically, increased SCC are associated with problems such as a decreased yield of cheese and undesirable changes in cheese textural and organoleptic properties. The importance of one specific type of somatic cell, polymorphonuclear leucocytes (PMN) (also known as neutrophils), the cell associated with mastitis and inflammation, on the quality and yield of cheese was examined. Preliminary research had shown that proteolytic enzymes associated with PMN were highly active against caseins in milk and may play a degrading role during cheese manufacture and ripening lowering yield and reducing quality. Thus, it was to be assessed whether PMN content or total SCC were a better predictor of the quality of cheese made from different milks, as it has been shown that bulk milks of similar SCC may have different levels of PMNs. An immunoassay specific for PMNs was developed and used to measure their levels in milk. The products examined were Swiss and Cheddar cheese. Preliminary data suggest that elevated PMN levels in milk are associated with decreased protein content and elevated moisture content of Swiss cheese, and altered levels of proteolysis during ripening. This would be consistent with an increased level of PMN-derived proteases in milk being incorporated into the cheese. Proteolysis in Cheddar cheese during ripening was similarly affected by high milk PMN content. Overall, elevated levels of PMN in milk appear to influence the yield and quality of cheese particularly proteolysis during ripening. Further studies are ongoing to elucidate the role of PMN and their enzymes in influencing cheese composition, ripening and flavour.

IDENTITY OF INHIBITORY SUBSTANCES IN MILKS FROM INDIVIDUAL COWS

PAUL NEAVES[1] & PETER HOPWOOD[2], [1]Williams & Neaves, The Food Microbiologists, 'Moleview', 28 Randalls Road, Leatherhead, Surrey KT22 7TQ, [2]Millwood Products, The Mill, Priston, Bath, Avon BA2 9EQ

The Delvotest Cow Test® is used increasingly on farms to confirm the absence of antibiotics in milk from individual cows, especially milk from freshly-calved animals following treatment with dry cow therapy. However, occasional reports of positive Delvotest® results, sometimes for prolonged periods after calving, have led some veterinary pharmaceutical companies to suggest the possibility of 'false-positive' reactions. To clarify the reliability of the Delvotest Cow Test®, milks that had given positive results in on-farm tests were examined by an independent laboratory (the former Milk Marketing Board, Microbiology Laboratory) to identify the inhibitor present.

Fourteen samples were collected from 6 farms that had experienced positive results using the Delvotest Cow Test®. Twelve of these were composite samples from individual cows; 2 were bulk tank milks. All samples were examined using a microbial inhibitor test, (Delvotest P®) and 3 immune-receptor tests (the Delvo X-Press β-L®, the SNAP test for β-lactams and the Lactek β-lactam test). The immune-receptor tests differ in the range of β-lactams they detect; the SNAP and Delvo X-Press® tests detect both penicillins and cephalosporins whilst the LacTek test detects penicillins only. A sample that passes the Lactek test but fails the SNAP and/or Delvo X-Press® tests therefore contains a cephalosporin. To confirm the presence of a β-lactam, all positive samples were re-tested after treatment with penicillinase (penase); to confirm the presence of a 'natural inhibitor', all positive samples were re-tested after a heat-treatment of 82°C for 5 min.

Beta-lactams were detected in all test samples. Positive, individual cow samples were associated with the use of two dry cow therapies (Cepravin Dry Cow™ which contains the cephalosporin, cephalonium and Kloxerate Plus DC™ which contains the penicillins, cloxacillin and ampicillin). 'Natural inhibitors' were not detected in any sample. All individual cow samples associated with Cepravin™ contained a cephalosporin but Cepravin™ was not responsible for either of the bulk tank failures.

The Delvotest Cow Test® gave no 'false-positive' reactions; i.e. all positive, on-farm Delvotest Cow Test® results were confirmed as 'true-positives'. However, the farmers' records clearly indicated that milk from individual cows is sometimes tested before the end of the withdrawal period, i.e. before the milk is fit for inclusion in the bulk tank.

The results confirm the validity of Delvotest Cow Test® when used to examine composite milks from individual animals on commercial farms in the UK.

RELATIONSHIP BETWEEN ANTIMICROBIAL SUBSTANCES AND SOMATIC CELL COUNT IN INDIVIDUAL COW MILKS

J ERIC HILLERTON[1], BEN I HALLEY[2] & PAUL NEAVES [3] [1]Institute for Animal Health, Compton, Newbury, Berkshire, RG20 7NN, UK, [2]University of Vermont, Burlington, Vermont, USA, [3] Williams & Neaves, The Food Microbiologists, 'Moleview', 28, Randalls Road, Leatherhead, Surrey, KT22 7TQ, UK

The Delvotest P® (Delvotest Cow Test®) is the UK 'Industry Standard' test for the detection of antibiotics in bulk, raw milk but is used increasingly on farms to test milk from individual cows. Following a preliminary study of milks that had given positive reactions in on-farm tests, a more extensive investigation was undertaken to determine whether any relationship existed between a positive Delvotest P® reaction and high somatic cell count.

In a 3-phase investigation, 443 milk samples from cows in herds at the Institute for Animal Health were examined for somatic cell count and for Delvotest P® reaction. As far as the authors could establish, the only antibiotic therapy administered to any animal was Cepravin Dry Cow™. To confirm the presence of a 'natural inhibitor', all positive samples were re-tested after a heat-treatment of 82°C for 5 minutes; to confirm the presence of a ß-lactam, all samples positive after heat-treatment were again re-tested after treatment with penicillinase (penase).

In Phase 1, 134 individual quarter samples were collected from mid-lactation cows with somatic cell counts in the range <400,000/ml to >4,000,000/ml. No sample gave a positive Delvotest P® reaction whilst controls containing penicillin or cephalonium tested positive.

In Phase 2, 144 individual quarter samples were collected from cows 4-6 days post-calving. Sixteen samples gave positive Delvotest P® reactions of which 13 had a somatic cell count >4,000,000/ml. 'Natural inhibitors' were responsible for the positive reaction in only 1 sample (somatic cell count >4,000,000/ml). Of the remaining 15 positive samples, 7 contained a ß-lactam; in view of the antibiotic history of the experimental animals, the presence of Cepravin™ seemed likely. The remaining 8 positive samples, contained inhibitors which were resistant to both penase and heat-treatment. All of these samples had somatic cell counts >4,000,000/ml but the inhibitors could not be identified. It was evident from the cell counts and farm observations that lactation had been improperly started by a period of incomplete milking.

Phase 3 of the investigation studied the rate of disappearance of positive Delvotest P® reactions by collecting a 'time-series' of individual quarter and composite (jar) samples from the day of calving for up to 10 days. In individual quarter samples, positive reactions persisted for up to 7 days post-calving and were due to both ß-lactams and 'natural inhibitors'. In composite samples, positive reactions persisted for up to 4 days post-calving. All 'composite sample' positives except one were due to a ß-lactam antibiotic.

The results showed that positive Delvotest P® reactions may occur in individual quarter samples from freshly-calved animals due to inhibitory substances other than antibiotics; however, such positive reactions were infrequent and strongly associated with a high somatic cell count (>4,000,000/ml). Incomplete milking in newly calved cows may be a risk factor associated with high SCC and positive reactions. There was no evidence of 'natural inhibitors' in composite milks collected from individual cows more than 4 days after calving.

SURVEY OF GENUS MILKING MACHINE TESTS

ELIZABETH BERRY & MARK SCRIVENS, Genus Ltd, Westmere Drive, Crewe CW1 1ZD

One of the recommendations in the 5 point mastitis control plan is a regular milking machine test. The milking machine has important roles in the welfare of the cow, the hygienic quality of the milk and financial losses due to inefficiency.

A survey has been made comprising 2500 static milking machine tests in England and Wales carried out from November 1996 to February 1997. All units were tested to BS 5545 by Genus Milking Machine technicians.

Parlours comprised 74% herringbone type, 25% abreast type and 1% rotary type. Average herd size was 100 cows and 50% of units were represented in the 10 to 16 unit range. Seventy five percent of units were tested annually and 25% biannually. The average running time, including milking and washing, is 5 hours a day or 1800 hours a year. The average machine should be tested every 800 to 1000 working hours, which would equate to two tests a year.

Seventy five percent of units tested twice a year failed and 70% of units tested once a year failed to meet BS 5545. This indicates that faults on machines are often not rectified from one test to another, irrespective of test frequency interval.

Plants failed for a variety of reasons, in decreasing order of frequency - air pipeline leakage, milk system leakage, lack of effective reserve, regulator problems, incorrect vacuum level and accuracy of a farm vacuum gauge. Forty seven percent of claws were bung types which are more likely to be unable to cope with a modern dairy cow yield.

There is a need for certification on the pass/fail status of the milking machine.

TREATMENT OF THE MILKING CLUSTER

MARTIN F H SHEARN

Treatment of the milking cluster between cows is not part of the 5-point mastitis control routine. Dipping the cluster in a bucket of water containing hypochlorite disinfectant was recommended many years ago, as a means of controlling the spread of mastitis pathogens from cow to cow. However, the rapid reverse movement of milk through vacuum fluctuations brought infected milk from the long milk tube (LMT) to the teat removing any advantage. Running water through the LMT and cluster was an 'advance' as it flushed out the LMT in addition to the clawpiece and liners but again did not remove all pathogens.

The treatment of the cluster and LMT with water at 85°C significantly reduced the number of bacteria in almost all tests, resulting in only 3% bacteria. In the 1970's automatic equipment from Israel flushed the LMT and cluster with cold and hot water, finishing with an air blast. This equipment was expensive and did not always clean heavily contaminated liners physically or of pathogens.

The more recent attempt to clean the equipment between cows alternates air and water blasts through the liner completing the cycle in 50-60 seconds. This latest system only washes the teat cup liner. A trial at IAH showed that all the teatcup liners were still contaminated with bacteria after treatment and that the numbers of pathogens were high (1).

There appears little real value in such systems. Results from the National Institute for Research in Dairying studies (1958 to 1965) showed that cluster treatment had no real advantage in controlling the spread of disease in the long term (2). Post-milking teat disinfection was the most important action in controlling the spread at milking time. Better attention to keeping the cows clean outside of the parlour and good udder preparation were much more successful in reducing contamination of the cluster and liners.

REFERENCES

1. SHEARN M F H, MORGAN J M & HILLERTON J E (1994) Veterinary Record 134 450
2. DODD F H & NEAVE F K (1970) National Institute for Research in Dairying Biennial Reviews Paper 3559

EARLY TREATMENT OF SUBCLINICAL INFECTIONS: A SHORT TRIAL

RAY WILLIAMS, The George Veterinary Hospital, High Street, Malmesbury

The regular monitoring of individual cow somatic cell counts (SCCs) allows both farmer and veterinarian an early indication of new infections as SCCs rise (<200,000 cells/ml). By using individual quarter cell counts, the source of elevated SCCs can be identified, sampled and treated. The value of early detection and treatment in 1st and 2nd lactation animals may lead to a lowering of herd exposure levels and hopefully a lower rate of chronic infections that otherwise last until and possibly through the drying off routine. Early results indicate a good response level in such a treatment regime in a small trial group where 11 out of 14 infections responded to treatment. The value of using such trial data in drug response trials will be discussed.

BRITISH MASTITIS CONFERENCE 1997

POSTER:

BUILDING DESIGN TO PROVIDE A HEALTHY ENVIRONMENT FOR THE DAIRY COW

I J Loynes BSc (Hons), Senior Lecturer in Farm Buildings and Environmental Control Systems at Harper Adams Agricultural College, Newport, Shropshire

The majority of the dairy herds in the United Kingdom are winter housed in some form of cubicle/kennel accommodation or are grouped in louse yards. A very small proportion are housed in 'traditional' cowsheds. The cows indoor climate is bound to influence the amount of exposure of the teat end to bacteria. The physical characteristics of the housing will also influence the likelihood of injury to the teat end. Therefore, good design of facilities and appropriate management of the environment is essential if the risk of infection is to be kept at a minimum.

Bacteria thrive in the sort of damp, humid environment found in cow housing systems. Good natural ventilation is essential to aid the removal of airborne bacteria and to keep humidity levels as low as possible. However, the relative humidity within a building will normally follow that of the outside, or be slightly higher due to deposition of faeces and urine. Therefore, we cannot reduce internal relative humidity levels without complete control of the environment, a solution that would be too expensive.

Passageways and concrete areas regularly accessed by stock should be designed to make the job of scraping slurry easy. For example, regular slurry removal from passageways is essential to reduce the amount of slurry that will be transferred from the cows feet to a cubicle bed. Automatic scraper systems should be installed so that the passageways are kept as 'clean' and 'dry' as possible.

Loose yards and cubicle houses should be designed with ample space for all the stock so that accidental injury of teats, ie by trampling or kicking, can be minimised. Cubicles should have a brisket board and a head rail fitted to position the cow correctly. Ample forward space will assist the cow to rise and lower with less risk of injury. Without these aids the cow can lie too far into the cubicle and may then foul the heel, thereby increasing the risk of a teat being contaminated with faeces. Cubicle bases should preferably be concrete, to avoid deformation, and should be laid with a fall of at least 75mm from front to back, to assist drainage. Cubicle divisions should separate each cow without increasing the risk of injury to the occupant or their neighbour, eg uprights in the division at the heel end should be avoided as they can restrict the cow too much and hence cause injury.

Another area where the teat can be exposed to a risk of contamination, is in and around the milking parlour. The collecting yard and milking parlour should therefore be designed with this in mind and all surfaces made to be easily cleaned. All floor surfaces should give the cows confidence, so that they do not slip, and where water is used, for washing etc., they should be laid to fall to a drainage system such that all contaminants are quickly removed from the [1]proximity of the cows.

end