BRITISH MASTITIS CONFERENCE 1993

Jointly organised by:

GENUS ANIMAL HEALTH

AFRC INSTITUTE FOR ANIMAL HEALTH

and CIBA AGRICULTURE

Genus
ANIMAL HEALTH

INSTITUTE FOR ANIMAL HEALTH

ciba

I N D E X

INTRODUCTION

R D JAMES
Director of Animal Health
Ciba Agriculture
Whittlesford
Cambridge CB2 4QT

This year our theme for the sixth British Mastitis Conference is "Managing Mastitis on the Farm". This is in direct response to the delegates' feed-back from the 1992 Conference, when a greater emphasis on practical aspects of mastitis control was requested. The most popular requests were for the effect milking machines can have on mastitis, methods of mastitis control and therapy.

The papers to be given will address how the milking machine affects mastitis, two different approaches to mastitis control and a veterinary surgeon's view of treatment of difficult cases. A review will be given of the level of knowledge obtained at the Mastitis Quiz last year and this popular event will be repeated as part of the Poster Display throughout the day.

A dairy farmer will present his views on the controversial subject of the benefits of low cell counts and at a time of rapid change in the field of milk marketing, a paper on mastitis and how it could affect the milk price should make a lively finale to the 1993 Conference.

It is the organisers' intention that this Conference will give delegates a practical and up-to-date review of managing mastitis on the farm and they hope that you will feed back your views on the questionnaire provided, so that the next Conference can again be based on what delegates see as the most important topics.

HOW THE MILKING MACHINE AFFECTS MASTITIS

DESIGN OF MILKING MACHINES

Dr. Eddie O'Callaghan, Teagasc, Dairy Production Research and Development Division, Moorepark, Fermoy, Co. Cork, Ireland

Introduction

The primary purpose of machine milking is to harvest the maximum quantity of high quality milk without injury to the cow while expending minimum labour. Milking machine design is complex in that many of the operational parameters interact. These characteristics depend on the combined performance or interactions of the basic elements such as the vacuum pump, vacuum controller, the cluster, milk lift and pulsation system. The plumbing layout and the size of the milk and air pipelines can also affect the operational characteristics of a milking machine.

Installation and performance standards

Due to the international nature of the milking machine industry it is evident that there are important advantages in having common standards adopted internationally. The development of the ISO standards (1) in the 1980's was an important step towards this goal. However there remain some significant differences between countries in accepted standards in the construction and performance of milking machines. Table 1 shows the comparative standards for European (ISO) and US (Californian) milking equipment (2). Current US Standards, published as 3-A accepted practices (3) are lower than those in Table 1.

The ISO standards are presently being reviewed and revised. Common sense and practical perspective are needed in the formulation of new standards. More scientific evidence and greater emphasis on performance standards are needed in order to resolve the divergence in standards. In this paper I will briefly review the relevant criteria for design of milking machines and suggest new ideas for improving the practical performance of milking machines.

As a general principle national and international tests are useful only if they give improved milking, better physical, chemical and bacteriological quality of milk, or less mastitis.

Table 1. Some approximate comparative standards for European and US milking equipment.

	No. of milking units				
	4	8	12	16	20
Vacuum pump capacity (l/min)					
Europe:	470	760	1100	1225	1440
USA:	1200	2400	3600	4800	6000
Bore of main airline (mm)					
Europe:	31	38	38	50	50
USA:	50	75	100	100	150
Bore of milkline (mm)					
Europe:	31	38	50	50	50
USA:	50	2 x 50	2 x 64	2 x 75	2 x 75
Bore of pulsator airline (mm)					
Europe:	25	31	38	38	38
USA:	50	50	75	100	100
Effective reserve (l/min)					
Europe:	200	300	370	410	450
USA:	1000	2000	3000	4000	5000

Vacuum fluctuations

In all discussions on milking machine design the magnitude and type of vacuum fluctuations that are applied to the cow's teat are a major consideration. Three types of vacuum fluctuations affecting liner and pulsation vacuum have been identified (4).

(a) Irregular fluctuations occur in a large part of all the vacuum system resulting from changes in airflow during milking.

(b) Liquid flow fluctuations are caused by the movement and amount of milk in tubes and pipelines used for the dual purpose of providing vacuum at the teat and for transporting milk.

(c) Cyclic fluctuations are generated in the cluster mainly as a consequence of pulsation.

A fourth type of vacuum fluctuation occurs when the liner becomes unstable on the teat end and slips downwards allowing air to be drawn into the cluster. The rate of decline of vacuum during these liner slippages fluctuations is much faster than with other types of vacuum fluctuations.

A series of experiments carried out at the National Institute in Dairying, Reading (5-7) showed that high rates of new intramammary infection occurred with large irregular and large cyclic fluctuations. Experiments carried out at Moorepark (8-13) to extend the work at the NIRD suggested that generalized vacuum fluctuations *per se* did not increase the rate

of new infections but that the rate could be greatly increased by liner slip i.e. air admission between the teats and the liners.

Simulated liner slips have been shown to increase new infection rates at Beltsville (14), at Ruakura (15) and at Moorepark (16). It is now widely accepted that only the localized abrupt fluctuations that occur mainly during liner slippages as distinct from irregular or cyclic fluctuations can lead to infection. In many experiments in which irregular or cyclic vacuum fluctuations were the directly observed variable, liner slip may have been the operating but unnoticed factor. In a recent bacterial challenge experiment using a Biopuls cluster high rates of new infection occurred in quarters not protected by non-return flaps whereas no infections occurred in quarters protected by flaps. The flaps reduced the rate of change of vacuum at the teat end during simulated liner slips in the claw. Thus the rate of change of vacuum or rate of airflow towards the teat end are critical factors affecting new infection rate. The exact mode of bacterial penetration through the streak canal when liner slip occurs has not been established.

Design strategies

Three strategies have been taken by design engineers to minimise vacuum fluctuations

(a) design milking equipment in which the number of abrupt fluctuations are minimal when considerable liquid and cyclic fluctuations occur

(b) design milking equipment which prevents abrupt fluctuations from occurring at the teat end during milking e.g. Hydraflow cluster, Biopuls cluster, and

(c) provide equipment in which liquid and cyclic vacuum fluctuations are minimized. One method of achieving the latter is to install large-bore lowline milk and air pipelines, large capacity claws and wide-bore long and short milk tubes. Usually the large-bore milklines have been accompanied by large bore airlines and large capacity vacuum pumps. A large vacuum reserve is required in these systems due to high air intake during cluster changing or cluster fall-off. In some large bore installations the air intake can exceed 1000 l/min if a cluster falls off. The recommended vacuum reserves given in the ISO standards generally apply to plants with considerable liquid flow and cyclic fluctuations with maximum air intake during cluster changing or fall-off of about 300 litres/min.

Considerable effort has been expended by design engineers to shield the teat end from abrupt vacuum fluctuations. Deflector plates or shields at the base of the liner have been used. The Ambic Hydraflow reduces the effect of teat end impacts and cross infection. With non-return systems hydraulic milking can occur, this may lead to teat end vacuum levels greater than the system vacuum level. Grindal (17) reported faster milking, reduced lipolysis and more accurate sensing of milk flow with a valved (ball) claw.

In milking plants having air/milk separation in the claw the liquid flow type fluctuation can be minimized. Also, the vacuum applied to the teat end can be easily adjusted. Generally these systems have two vacuum pumps and two vacuum controllers. The differential in the vacuum levels in the system is used for moving milk through the system. In many such systems very fast milking rates are obtained compared to conventional highline or midiline

milking plants. Generally this is due to higher teat end vacuum levels. In the literature there is no clear relationship between vacuum level and the incidence of mastitis (18). However an increase in teat orifice erosion and hyperkeratosis has been noted (19) when vacuum levels greater than 60 kPa were used. An interaction between cluster stability, vacuum level and strip yields may be responsible for the conflicting evidence on the relationship between vacuum level and the incidence of mastitis.

It is often difficult for a design engineer to decide on the optimum levels of vacuum that should be applied to the teat end during milking. The general tendency in the future will be towards lower vacuum levels at the teat end to minimize teat damage and cow discomfort.

In Ireland 2 stalls/unit midiline plants with milklift of 1.2 m - 1.4m are common. The receiver vessels are usually located in front of the milkers pit. Generally cluster removers are not used in these plants. While considerable liquid flow and cyclic fluctuations exist in these plants emphasis is placed on cluster and liner design in order to minimize liner slippage and give good milking characteristics. Typical plumbing arrangements are shown in Figure 1 and Figure 2 for pipeline and recorder plants used in Ireland. Test points are installed for the measurement of pump capacity, vacuum reserve and general airflow measurements. The minimum vacuum reserves are given in Table 2. For recorder plants it is possible to milk through the jars by placing the milk transfer pipeline about 250 mm below the top of the jar with the receiver vessel at the front of the milkers pit (Figure 3). In this mode of operation the recorder plant will function as a direct to-line pipeline plant if required with a vacuum supply to the recorder jar from the vacuum/wash pipeline and also from the milk transfer pipeline.

Table 2. Minimum effective reserves (l/min) for different plants used in Ireland.

	No. of units									
	2	3	4	5	6	7	8	9	10	
Pipeline and recorder plants	175	200	225	250	275	300	325	350	375	
No. of units	11	12	13	14	15	16	17	18	19	20
Pipeline and recorder plants	390	405	420	435	450	465	480	495	510	525

Pulsator characteristics

Ineffective pulsation consistently results in an increase in new infection rate (21-24). Short teatcup liners can create a form of pulsation failure and result in an increase in new infection rate. The mechanism of infection is unknown. It is possible that many of the common defects in milking equipment such as pulsation failure result in undermilking or the leaving of high levels of residual milk in the gland cistern after normal machine milking. A high incidence of udder diseases in cows which were undermilked has been reported (25). Numerous workers have investigated the effect of pulsation rate on milking rate showing an increased milking rate when pulsation rates were increased from 20 to 80 cycles per minute (26); that increasing pulsation rate increased milking rate (27); and that pulsation ratio had

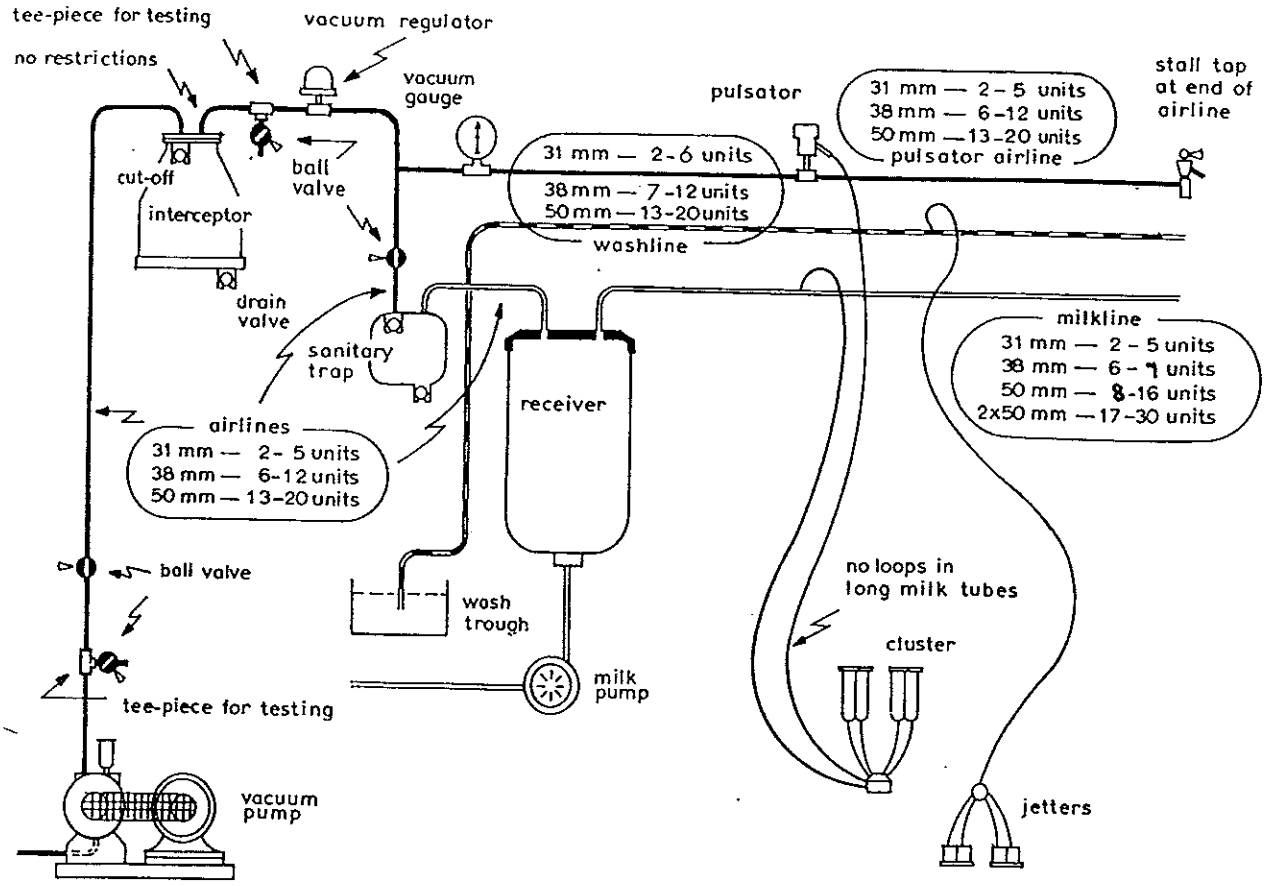

Figure. 1. Pipeline milking machine - main installation specifications.

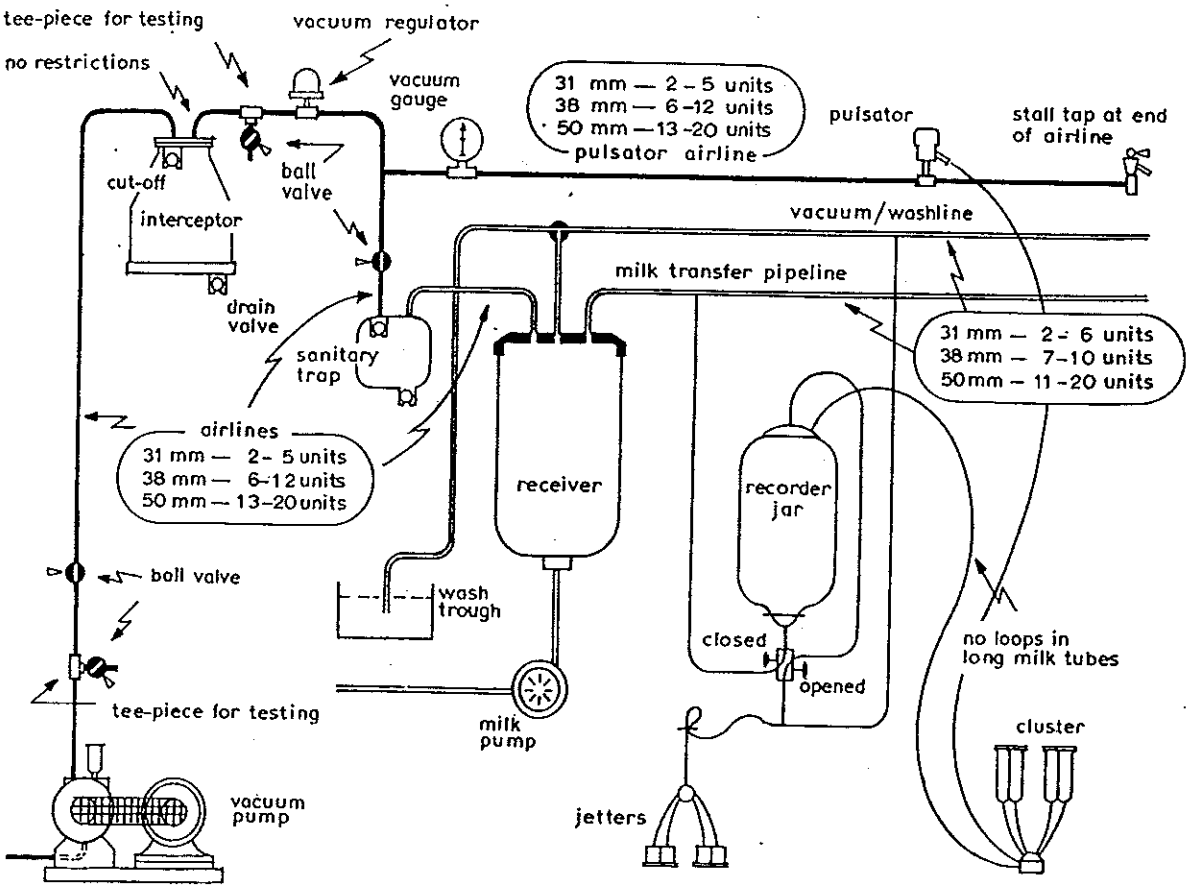

Figure. 2. Recorder milking machine - main installation specifications.

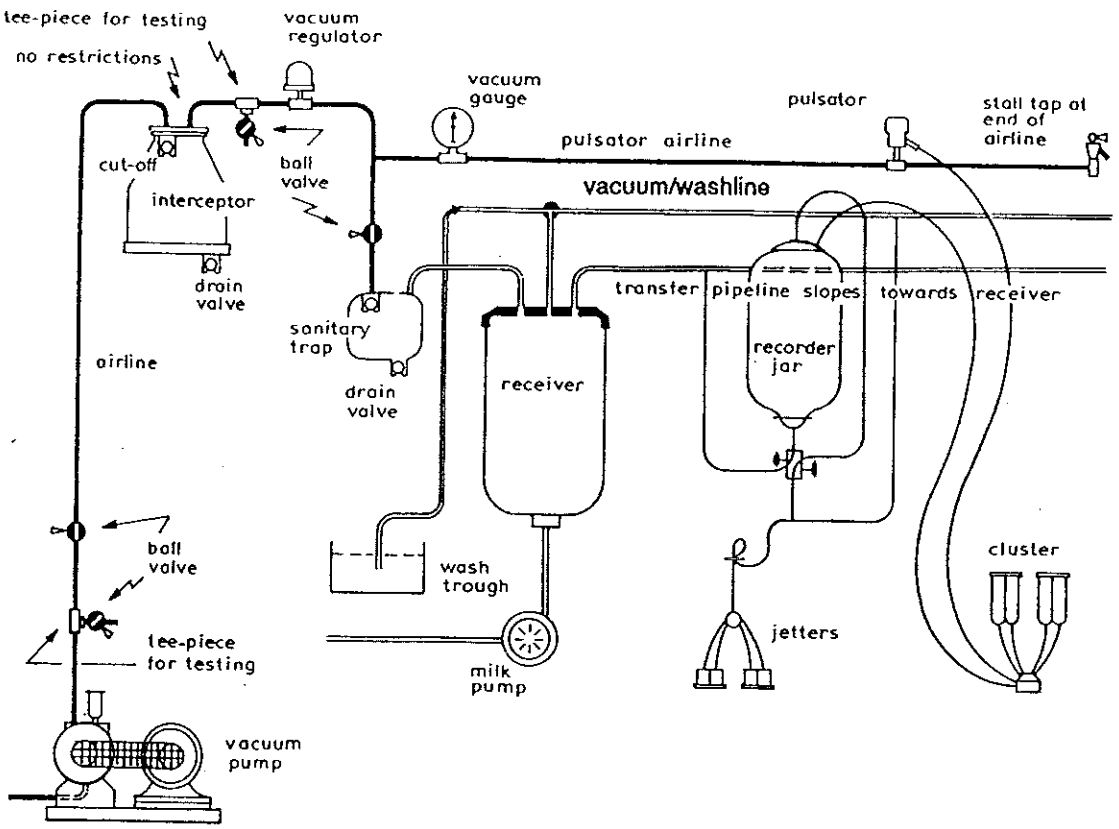

Figure . 3 Recorder milking machine suitable for milking 'through the jars'.

a larger impact on milking rate than pulsation rate (28,29). However, liner design appears to have a much greater effect on milking characteristics than pulsation characteristics or vacuum level (30,31). The ISO standard (1) specifies that the "d" value or the collapsed phase of the pulsation graph should not be less than 15%. Many workers have assumed that liner wall movement directly follows the pulsation chamber waveform. Correlations between pulsation characteristics and liner wall movement are however greatly affected by liner type (32,33) and by milk in "transit" in the system (6-8,11,33). Measurement and analysis of liner wall movement during pulsation provides a much better indication of liner operation than evaluation of pulsation chamber characteristics as commonly used in testing milking systems (34,35).

It has been shown that a 70:30 ratio produced lower machine on-time than 50:50 and 60:40 ratio and pulsation rate did not affect milking rate measurements or milk production (36) and that a pulsation rate of 52 c/min with pulsator ratio of 60.9% gave a 3.73% reduction in machine yield compared to a pulsation rate of 60 c/min with pulsator ratio of 60.9% (35).

Liner design and teat-liner interactions

The main requirements of an ideal liner are high machine yield and hence minimum strip yield, low machine-on time and a low frequency of liner slippage particularly with high yielding cows. In the 1970's liner design evolved from a trial and error method to use of analysis of the effect of specific changes in liners. Mein (37) suggested that most of the differences in the performance of liners could be explained by the depth of teat penetration into the liners. It has been shown (31) that the milking characteristics of commercial clusters were more affected by liner design rather than by pulsation characteristics and that an increase in the number of liner slippages occurred when the operating vacuum was lowered from 50 to 44 or 42 kPa in a milking plant with 1.2 m milk lift (38).

A research programme was carried out from 1985-1989 (35)

(a) to measure the effect of liner design factors and some physical settings of the milking machine on cluster stability, machine yield and milking time,

(b) to explain the variation in cluster stability in terms of teat liner interactions and

(c) to provide criteria for the design of milking machine liners and milking systems.

The following conclusions were drawn:

1. There was an interaction between the cluster stability expressed as the percentage of cows with liner slips and the mean machine yield/cow/milking. The cluster stability improved with reductions in milk yield.

2. The bore at the top of the barrel and the mouthpiece bore of liners were the main factors that affected cluster stability. The barrel bore was negatively correlated whereas the mouthpiece bore was positively correlated with the percentage of cows with liner slips.

3. Generally the percentage of cows experiencing liner slip was higher with narrow-bore liners compared with wide-bore tapered liners. The machine yields and milking times obtained with narrow bore liners were comparable with those obtained with wide bore liners. Thus the main practical limitation of using narrow bore liners is their association with high levels of liner slippage.

4. Altering pulsation characteristics, vacuum reserve or opening valve ("a" value) of the pulsation chamber waveform had only a minimal effect on cluster stability, liner design was the major factor affecting cluster stability. The vacuum levels in the mouthpiece cavity of a liner in a cluster were correlated with the stability of the cluster, high mouthpiece cavity vacuum levels (> 20 kPa) at the start of milking were associated with liners that gave good cluster stability.

5. Teat penetration into liners was similar with wide and narrow bore liners, and teat penetration levels increased during milking.

6. With all liner types the teat expanded on entering the liner to fill the barrel of the liner.

7. The additional teat expansion that occurred with wide bore liners compared with medium bore and narrow bore liners was accompanied by higher levels of mouthpiece cavity vacuum and higher levels of mouthpiece deflection. The friction between the teat and wide bore liners was derived from forces on the teat due to mouthpiece deflection and teat expansion at the top of the barrel. The improvement in cluster stability that occurred with most liner types corresponding to a reduction in machine yield results from an increase in teat penetration levels into the liner and an increase in mouthpiece deflection at the reduced machine yields.

8. With wide bore liners the opening and closing times of the liner wall were generally longer than indicated by the pulsation chamber waveform, the opposite effect occurred with narrow bore liners. The opening and closing time of medium bore heavy wall liners were shorter with alternate than with simultaneous pulsation patterns. The ISO Standard (1) specifies that the "d" value of the pulsation chamber waveform should be at least 15%, the closed times of the liner wall during milking with some wide bore liners were as low as 5% of a liner wall movement cycle.

9. Measurement of liner wall movement characteristics during milking required simultaneous measurement or monitoring of teat position, with some cows the teat penetrates sufficiently into the liner during milking preventing liner wall movement.

10. The optimum liner should have the following characteristics: (a) a barrel bore at the top of the barrel in the range of 30-31.8 mm, (b) a mouthpiece bore in the range 22-23.5 mm, (c) a mouthpiece cavity width in the range of 47-48 mm, (d) a shoulder radius in the range of 22-24 mm, (e) a mouthpiece radius in the range of 1.3-2.0 mm, (f) a volume of the mouthpiece cavity in the range of 28-30 ml, (g) length to bottom of barrel in the range 135-140 mm, (h) wall thickness 2 - 2.3 mm, (i) mouthpiece softness in the range 3.4-4.0 mm, (j) mouthpiece cavity height in the range of 24-29 mm, (k) 15% stretch and (l) the liner material should be durable and should give optimum response to pulsation during milking.

11. The milking characteristics were similar with simultaneous and alternate pulsation patterns.

12. Liners with barrel bores of 30-31.5 mm require cluster weights of 3-3.2 kg in order to have acceptable milking characteristics in midiline plants with system vacuum of 48-50 kPa and milk lifts 1.2-1.6 m above to cow standing level.

Selection of liners and clusters

A commercial testing service is available at Moorepark for liners and clusters. Generally a cluster weight of 3.0-3.2 kgs with claw weights of 750-800 grams are recommended for wide bore liners. The measured milking characteristics include machine yield, machine-on time, percentage of cows with liner slippages allowing air admission of $\geq$ 35 l/min, percentage of cows with liner slippages allowing air admission $\geq$ 90 l/min and percentage of cows with "fall-offs" and "push-ups". These trials are carried out with cows yielding 16-20 kg/cow/milking. Similar milking characteristics were obtained when claw volumes of 140 ml and 323 ml were compared. Thus a claw volume of 140 ml is adequate for cows with mean yields of 16 kg/cow/milking milked in a midiline plant.

Despite considerable liquid flow and cyclic vacuum fluctuation, the Moorepark designed liners gave good cluster stability. This low level of liner slippages allows an operator in a 2 stall/unit direct-to-line pipeline plant to cope with up to 18 milking clusters without the use of automatic cluster removers. The poor stability obtained with narrow bore liners can generally be improved by the use of very light cluster weight (1-1.5 kg) or by increasing vacuum levels. There is however a risk that this approach could reduce the machine yield. While good cluster stability is important a high machine yield and low levels of teat damage and minimal discomfort to the cow are a vital element of a milking machine cluster. In trials at Moorepark liners with wall thickness of 3 mm with low levels of tension (5%) gave poor liner wall movement. A wall thickness of 2-2.3 mm with 15% tension is now recommended.

Selection of components

The selection of high performance components is a prerequisite to ensuring consistent performance of a milking machine. Independent endurance test data for components such as vacuum pumps, vacuum controllers and pulsation systems can help the design engineer to identify high performance components that require minimal maintenance. A list of recommended components based on endurance tests is provided by Teagasc, Moorepark. This list is updated annually and published in the farming papers.

Summary

The use of high performance liners and clusters minimizes rates of slippage and gives good milking characteristics despite the presence of considerable liquid flow and cyclic fluctuations at the teat end. While shorter milking times can be obtained with milking plants in which minimum vacuum instability occurs in the main milking system during milking, these designs have little scientific or economic justification. The forces applied to the teat and the cluster stability are more related to liner and cluster design rather than the degree of vacuum stability at the teat end. The high incidence of liner slippage obtained with narrow bore liners may limit the number of units in a milking shed and can result in unnecessary vacuum

reserves. The use of high system vacuum levels or high teat end vacuum levels during milking can improve the cluster stability with narrow bore liners, there is however a considerable risk that the exposure of the teat to high vacuum levels can result in increased teat damage and hyperkeratosis. For optimal design of a milking machine more attention should be given to the performance characteristics of the critical components and less emphasis placed on minimizing the normal vacuum fluctuations in the milking system. There is however a need to minimize the number of abrupt vacuum fluctuations at the teat-end as these fluctuations contribute to new infections.

References

1. INTERNATIONAL STANDARDS ORGANIZATION (1983) Milking Machine Installations - construction and performance. ISO 5707.

2. O'CALLAGHAN E (1988) Processing Symposium - Milking Systems and Milking Management, Harrisburg, Pennsylvania, U.S.A., pp.11-23.

3. INTERNATIONAL ASSOCIATION OF MILK, FOOD AND ENVIRONMENTAL SANITARIANS, UNITED STATES PUBLIC HEALTH SERVICE, THE DAIRY INDUSTRY COMMITTEE (1990) Dairy, Food and Environmental Sanitation 9 679-689.

4. COWHIG, M J (1969) Proceedings of the Symposium on "Machine Milking" National Institute for Research in Dairying, Reading, p.15.

5. COUSINS C L (1972) The relationship between the milking machine and new intramammary infection. Ph.D. Thesis, University of Reading, p.157.

6. COUSINS C L, THIEL C C, WESTGARTH D R & HIGGS T M (1973) Journal of Dairy Research 40 289-292.

7. THIEL C C, COUSINS C L, WESTGARTH D R & NEAVE F K (1973) Journal of Dairy Research 40 117-129.

8. O'CALLAGHAN E, O'SHEA J, MEANEY W J & CROWLEY C (1976) Irish Journal of Agricultural Research 15 401-417.

9. O'SHEA J & O'CALLAGHAN E (1978) Proceedings of the 3rd International Conference on Machine Milking, Louisville, Kentucky, p.262.

10. O'SHEA J & O'CALLAGHAN E (1982) Proceedings of Seminar on "Milk Production from Pasture", Ruakura Agricultural Research Station, Hamilton, p.77.

11. O'SHEA J, O'CALLAGHAN E, MEANEY W J & CROWLEY C (1976) Irish Journal of Agricultural Research 14 372-399.

12. O'SHEA J, O'CALLAGHAN E & MEANEY W J (1979) Irish Journal of Agricultural Research 18 225-235.

13. O'SHEA J, O'CALLAGHAN E & MEANEY W J (1981) Irish Journal of Agricultural Research 20 163-183.

14. THOMPSON P D & SCHULTZ W D (1975) Journal of Dairy Science 58 752-351.

15. WOOLFORD M W, WILLIMSON J H & PHILLIPS D S M (1980) Proceedings of the International Workshop on "Machine Milking and Mastitis", Moorepark, Teagasc p.45.

16. O'SHEA J, O'CALLAGHAN E & MEANEY W J (1984) Irish Journal of Agricultural Research 25 155-171.

17. GRINDAL R J (1987) Proceedings of the International Mastitis Symposium, MacDonald College, Str. Anne de Bellevue, Quebec, Canada, p89.

18. FELL L R (1964) Dairy Scence Abstacts 26 551-569.

19. NEAVE F K, OLIVER J & DODD F H (1957) Report of the National Institute for Research in Dairying, Reading, England, p42.

20. WALSH J P & NYHAN J F (1967) Animal Production Research Report, Moorepark, Teagasc, p54.

21. BRAMLEY A J, GRIFFIN T K & GRINDAL R (1978) Proceedings of the International Symposium on Machine Milking. National Mastitis Council, Arlington, VA.

22. KINGWILL R G, DODD F H & NEAVE F K (1978) Technical Bulletin 1 Machine Milking, National Institute for Research in Dairying, Reading, England, p262.

23. REITSMA S H, CANT E J, GRINDAL R J, WESTGARTH D R & BRAMLEY J (1981) Journal of Dairy Science 64 2240-2245.

24. WOOLFORD M W, & PHILLIPS D S M (1978) Proceedings of the National Mastitis Council, Arlington, VA.

25. EBENDORFF W, KRAM K, MICHEL G & ZIESACH J (1987) Kieler Milchwissenschaft 42 23-25.

26. CLOUGH P A, DODD, F H & HUGHES, E W (1953) Journal of Dairy Research 20 375-380.

27. STEWART W E & SCHULTZ L H (1958) Journal of Dairy Science 41 849-856.

28. SCHMIDT G H, GUTHRIE, R S, GUEST , HUNDTOFT, E B, KUMAM A & HENDERSON C R (1963) Cornell Agricultural Experimental Station Bulletin 983.

29. SCHMIDT G H & VAN VLECH L D (1969) Journal of Dairy Science 52 639-645.

30. BRANDSMA S (1968) Proceedings of Symposium on "Machine Milking", Reading, p.51.

31. O'SHEA J & O'CALLAGHAN E (1980) In "Experiments on milking machine components at Moorepark, 1976-1979", Teagasc, p.115.

32. LE DU J (1977) Association of Veterinary Medicine 121 1.

33. REITSMA S Y & BRACKMAN D K (1985) Canadian Agricultural Engineering 27 no.2.

34. STROHMAIER HERMANN A & GRIMM H (1987) Experimental studies to improve effective pulsation in milking machines. ISBN 3-8001-8196-7 Engen Ubner GmBH & Co.

35. O'CALLAGHAN E (1989) Measurement of cluster stability and its relationship to liner design and physical setting of the milking machine. Ph.D. Thesis, National University of Ireland.

36. MEIN G A (1978) Proceedings of the 3rd International Conference on Machine Milking, Kentucky, p107.

37. THOMAS C V, FONCE D K, BREMEL D H & STRESSEN S (1991) Journal of Dairy Science 74 1243-1249.

38. SPENCER S B & ROGERS C W (1991) Journal of Dairy Science 74 429-432.

HOW THE MILKING MACHINE AFFECTS MASTITIS

John Baines, ADAS Dairy Development Centre, Government Buildings, Lawnswood, Leeds

Introduction

The milking machine is one of many factors affecting mastitis incidence. It is often the link between deficiencies in one or more areas and an increasing likelihood of intramammary infection. This paper describes the ways in which the milking machine affects transfer of mastitis pathogens and the invasion of the teat by these bacteria.

Machine milking effects on mastitis incidence

The milking machine can affect mastitis in two main ways:

i. as a vector by actively transporting bacteria to and sometimes into an udder quarter;

ii. by affecting the condition of the teat and therefore the functioning of the first line of udder defence (1).

Milking machine as a vector of mastitis pathogens

The milking machine acts as a vector of bacterial transfer by a number of mechanisms. Much has been published about the significance of impacts (2,3,4). Vacuum fluctuations within the milking machine have been implicated as a major cause of impacts. The results of trials published indicate that mastitis incidence was only significantly increased when large irregular fluctuations in plant vacuum level coincided with wide cyclic fluctuations in vacuum level beneath the teat. Some manufacturers and many advisers in the United Kingdom concluded that efforts should be directed at ensuring adequate vacuum stability within a milking machine whilst not becoming too concerned with large cyclic fluctuations during milk flow.

Much of the trial work supporting these conclusions was done in the late 1960s and 1970s. Reference to Table 1 shows how milk yield has increased since then. It is fair to speculate that milk flow rates from modern higher yielding cows are much higher than from the cows used as a basis of the trial work (5).

Table 1. Milk yields for various EC countries (6).

	average yield/lactation (kg)			
COUNTRY	1960	1970	1980	1991
Germany	3395	3800	4548	4810
France	2145	3110	3606	5013
Italy	3105	2642	3384	3852
Netherlands	4275	4340	5035	6224
Belgium	3811	3597	3847	4422
Luxembourg	3375	3496	3975	4786
United Kingdom	3577	3929	4766	5135
Denmark	3708	3940	4855	6212
Irish Republic	2233	2513	3228	3794

Investigations into mastitis problems show that wide cyclic fluctuations in vacuum level in the short milk tube are very often associated with liners being flooded with milk. This is related to milk flow rate from the teat. It is highly likely that when this situation arises, the milk picks up bacteria from the liner surface, from the teat surface and from other quarters of the udder thereby increasing bacterial challenge at the teat end.

Large increases in milk yield and milk flow rate in recent years have prompted further developments in the design and construction of milking machine clawpieces and pulsation systems.

Initially, various shields and ball valves were developed in an attempt to interrupt the reverse flow of milk towards the teat. The use of shields in liners produced promising results. However, as milk yields have increased further, the liners themselves have become likely to be flooded. It seems likely that this negates the effects of the shields. Further developments using ball valves appeared to give a benefit, but changes in milking vacuum characteristics have outweighed the benefits.

One of the significant features of traditional cluster design was the size of the short milk tube. It has been normal to use short milk tubes of 7-8mm internal diameter. On certain farms, where in spite of the use of shielded liners, the amount of clinical mastitis has increased, it is the narrowness of the short milk tube resulting in flooded liners which subsequently negates the effect of the shield. Increasing the bore of the short milk tube has a dramatic effect in reducing the amplitude of vacuum fluctuation within the liner (7) thereby reducing the likelihood of milk moving towards the teat.

Liner Slip

Liner slip is important for a number of reasons.

- as a cause of impacts, particularly in the context of small capacity clusters with narrow bore short milk tubes;
- it is associated with incomplete milking out;
- as a cause of disruption to milking routine.

The design of liners, and in particular liner mouth-pieces, is the most significant factor relating to liner slip (8). In many cases, cluster position, udder shape, teat condition and the absence of support for weighty long milk tubes are also significant features.

Reverse flow of milk (impacts) produced as a consequence of liner slip has been shown to be associated with an increased infection rate. This situation should be avoided if at all possible. There are a number of conflicting strategies. Moorepark have developed a design of liner which minimises liner slip. There does appear to be a high incidence of tissue congestion associated with this liner. Other manufacturers of milking equipment have tackled this problem by provision of support for the cluster and milk hose, and use of lighter clusters.

In addition to liner slippage, incomplete milking can also be a result of poor cluster position. It is vital that the cluster hangs squarely on all 4 teats.

Teat Condition

The milking machine teat cup liner comes into direct contact with the teat. Lesions produced as a result of machine milking have been associated with increased levels of clinical and sub-clinical mastitis (9,10,11). The milking machine must be installed and operate so as to minimise the likelihood of any teat lesions or adverse effects on teat conditions. In particular, preventing circulatory impairment which is associated with increased susceptibility to mastitis infection. It is essential that an adequate massage to the teat is provided by liner wall movement.

Oedema/congestion

One significant change in teat condition often overlooked in mastitis investigations is the possibility of teat congestion and oedema. Circulatory impairment appears to be associated with an increase in the likelihood of a teat to admit bacteria (9,10,11,12,13).

Hyperkeratosis

Hyperkeratosis is one of the most common conditions giving rise to concern during mastitis investigations. This is characterised as a smooth or roughened ring of tissue around the teat orifice (11). The degree of hyperkeratosis is related to the compression of the teat orifice during milking. A mild degree of hyperkeratosis alone is not usually associated with an

Table 2. Relationship between teat end lesions, hyperkeratosis and infection levels - field observations (14).

Teat End Classification	Number of teats	% Infected
Normal	554	30.8
Smooth chronic rings		
- very mild	1177	24.8
- mild	1003	22.5
- moderate	312	26.6
- severe	72	34.7
Rough chronic rings		
- very mild	125	20.8
- mild	199	26.1
- moderate	151	32.5
- severe	80	26.3
Acute	16	43.8
Traumatized	72	59.7
Other	221	33.0
Normal	1231	21.0
With lesions	3473	19.8

increased rate of intramammary infection. Table 2 shows the results of field observations of a number of teats. The general trend is for teats with rough/smooth chronic rings of a very mild/mild severity to have lower levels of infection than teats considered to be normal. It is only when hyperkeratosis becomes severe or is associated with other forms of teat trauma that a higher rate of mastitis infection is noted.

The flow of milk exerts a sheer force on the keratin lining of the teat canal. Where the teat displays severe hyperkeratosis it is likely that the flow of milk exerts an even greater sheer force on the keratin lining of the teat canal. This seems to produce a vicious circle of events whereby milk flow rate becomes even slower.

Black Spot

Black spot is a term often misused. It describes a bacterial infection of the teat orifice. The organisms involved are often associated with dirty bedding and in particular, foot rot. Lesions of this type are often heavily colonised with these bacteria. This could have significance as far as new infection rate is concerned (M.F.H. Shearn, pers. comm.).

Petechial haemorrhage

Petechial haemorrhage is often associated with excessive vacuum level. Inadequate closure and excessive liner open times are predisposing factors. This type of lesion can be regarded as an indication of circulatory impairment. It is likely that it is also associated with damage to the teats' internal defence mechanism. This condition is often seen, with no evidence of hyperkeratosis, in association with hydraulic milking systems. In conventional milking systems this type of lesion may be associated with hyperkeratosis.

Wedging of Teats

Wedging of teats is characterised by a white line across the end of the teat. This may develop further to roughened callused skin along the line of the wedging. This line may itself crack and become subject to further colonisation. All of these signs are associated with milking machine operation either in terms of degree of compressive load on the teat or duration of closure. This may be an indication of incorrect setting of vacuum level pulsation rate and ratio.

Chaps

Chapping of teats is usually associated with atmospheric conditions and hygiene. The milking machine may aggravate chapping where incorrect cluster position pulls and twists the teats. This can be indicated where the chaps occur predominantly on one or two teats. Where chaps become infected this increases bacterial challenge to the teat during milking, particularly if inadequate sized short milk tubes and claw pieces result in flooded liners.

Pseudocowpox

Pseudocowpox is a viral infection. Where lesions of this type become secondarily infected, it is likely that there will be an increased bacterial challenge to the teat. Pseudocowpox can normally be controlled effectively by using Iodophor teat disinfection ensuring adequate teat coverage.

It is vital that the milking machine is installed and operates correctly to minimise the likelihood of any form of teat lesion or adverse effect on condition. In particular circulatory impairment of the teat must be avoided.

Teat Cup Liners

The operation of the teat cup liner in massaging the teat to relieve congestion is essential in maintaining teat condition. Whatever type of milking machine is installed, it is vital that the correct pulsation rate and ratio is employed for that particular combination of liner and shell. Whilst it is possible to produce guidelines relating to liner-open time based on flow rate measurements (15) the minimum liner-closed time necessary is much more specific to the liner design in use. ADAS investigations into problems on farms confirm that where incorrect rate and ratio of pulsation are applied, teat lesion problems are almost inevitable.

The way in which the liner actually compresses the teat is important in maintaining teat condition. The mechanical properties of the liner rubber change with use. A sensible guideline for liner usage is 2,500 cow milkings. In general, liner performance rapidly deteriorates after this point.

Hygiene

Contamination of the milking plant usually contributes little to total bacterial counts. Intramammary infections are far more important as a source of bacteria in bulk milk. However, when plant cleaning is not adequate, it is often the teat cup liners and clusters which are not being disinfected properly. They become a reservoir of bacteria which can spread to teats when liners flood.

Over-milking

No published work, so far, has shown any association between over-milking on its own and an increased rate of intramammary infection. It is vital to stress that over-milking combined with inadequate or unsatisfactory liner movement is associated with an increase in infection rate (16). ADAS experience in the early 1980s provides anecdotal evidence to suggest that where automatic cluster removers were fitted, thereby reducing over-milking, Somatic Cell Counts tended to reduce. The most important benefit of automatic cluster removal is to take much of the perceived pressure off the operator in large parlours.

Operator Technique

The effect of the operator on the milking machine must not be underestimated. Minimising air admission during cluster application and attention to cluster position are vital.

Teat hygiene, prior to cluster application, is a major factor relating to infection. Ideally, the cows should be clean on entering the parlour. If teats are soiled then they should either be washed and thoroughly dried or wiped using a disinfectant impregnated towel. Alternatively, pre-milking teat disinfection can be used subject to thorough wiping of teats with paper towels.

Investigating problems

The first stage in investigating any problem is to identify the scale and the nature of the problem. Machine milking problems may be indicated by a number of signs:

increasing Somatic Cell Count;
erratic Total Bacterial Counts;
increasing incidence of clinical mastitis;
development of teat lesions and worsening teat skin condition;
cows in discomfort during milking;
uneven milking.

A clear indication of the extent and the time of occurrence of problems can often point towards the specific factors involved. The next stage is a full appraisal of all aspects of machine milking including operator routine and milking hygiene. A full assessment should also be made of the effectiveness of housing management.

ADAS Mastitis Monitor records clinical mastitis, Somatic Cell Count and Total Bacterial Counts. This can be useful in identifying trends and particularly vulnerable times of the year.

Conclusions

On-farm investigations into mastitis problems combined with published literature lead to a number of conclusions.

1. Inadequate hygiene regimes in milking plant, operator routine and cow housing have serious implications for udder health.

2. The transfer of mastitis-causing organisms within the milking machine should be limited by:

   using clawpieces and liners designed to minimise flooding of liners;
   minimising liner slip;
   optimising liner movement.
   minimising air pressure changes inside the machine during milking;

3. The correct settings of pulsation rate and ratio should be employed for the liners and shells in use in order to optimise teat condition. Teat hygiene and post-milking teat disinfection are essential components.

References

1. SPENCER S B (1989) Journal of Dairy Science 72 1907-1917.

2. THIEL C C, THOMAS C L, WESTGARTH D R & REITER B (1969) Journal of Dairy Research 36 279-297.

3. THIEL C C, COUSINS C L, WESTGARTH DR & NEAVE F K (1973) Journal of Dairy Research 40 117-129.

4. COUSINS C L, THIEL C C, WESTGARTH D R & HIGGS T M (1973) Journal of Dairy Research 40 289-292.

5. GRINDAL R J & HILLERTON J E (1991) Journal of Dairy Research 58 263-268.

6. EC DAIRY FACTS AND FIGURES (1992), Milk Marketing Board, Thames Ditton.

7. THIEL C C, CLOUGH P A, WESTGARTH D R & AKAM D N (1968) Journal of Dairy Research 35 303-316.

8. O'SHEA J, O'CALLAGHAN E & MEANEY W J (1983) Proceedings, Annual Meeting National Mastitis Council 22 4-14, National mastitis Council, Kentucky. 1976-9 p115.

9. HAMANN J & DICK J (1984) Personal communication.

10. HAMANN J, MEIN G A & WETZEL S (1993) Journal of Dairy Science 76 1040-1046.

11. HAMANN J (1987) International Dairy Federation Bulletin 215, 33-55. IDF, Brussels.

12. TOLLE A & HAMANN J (1975) International Dairy Federation Document 85 193-199.

13. JASPER D E & WHITTLESTONE W G (1972) Journal of Dairy Science 60 1979-1981.

14. O'SHEA J (1987) International Dairy Federation Bulletin 215 5-32, IDF Brussels.

15. WILLIAMS D M, MEIN G A & BROWN M R (1981) Journal of Dairy Research 48 7-21.

16. MEIN G A, BROWN M R & WILLIAMS D M (1986) J Dairy Res 53 17-22.

REVIEW OF YOUR KNOWLEDGE OF MASTITIS

SO YOU THINK YOU KNOW ABOUT MASTITIS? REVIEW OF OUR KNOWLEDGE OF MASTITIS

A.H. Andrews & J. Fishwick, Department of Large Animal Medicine and Surgery, Royal Veterinary College, Hawkshead Lane, North Mymms, Hatfield, Herts AL9 7TA.

Introduction

Mastitis is a continuing problem of the dairy industry. While the prevalence and incidence of the disease varies from year to year, it still remains one of the major causes of production loss. As such, it tends to be accepted and not treated with the same interest as a newer, more exotic disease. The problem involves many different sectors of the industry, including the farmer, stockman, veterinary surgeon, the milk boards, manufacturers of milking machine equipment, the pharmaceutical industry, disinfectant producers, nutritionists, management specialists and consultants in varying disciplines. Thus a herd with a mastitis problem can be the focus of much attention from different people, each of whom is likely to concentrate mainly on one aspect rather than the whole of this complex problem, which ultimately is simply seen as inflammation of the udder. It is also possible that the assumption will be made that the experience of others dealing with the problem will be similar to their own.

It was in the above context that we decided to organise a lighthearted quiz on mastitis at the 1992 British Mastitis Conference. It was called Mastimind and, to add interest, we were privileged to be loaned the Mastermind chair by the British Broadcasting Corporation. The co-operation of many of last year's audience is gratefully acknowledged and it is hoped that delegates at future events will be equally willing to participate.

The quiz

The aim of the quiz was to ascertain the background knowledge on mastitis of delegates at the 5th British Mastitis Conference. This was undertaken by a series of twelve multiple-choice questions. Several of the problems were illustrated by pictures and some of these would have required a certain amount of guesswork. The ingenuity of the participants was employed in the tie breaker question, which showed an apparently double headed Hereford cross Friesian cow and asked "which end of this cow do you milk?"

The quiz participants remained complete anonymous and this was achieved by having each paper numbered and a duplicate number being kept by the delegates. This allowed a winner to be identified who could be presented with a bottle of champagne donated by Ciba Agriculture. Delegates were asked to provide their background as farmer, veterinary surgeon, adviser or other. They then answered the question sheet by ticks or single or few word answers to the questions which matched those on the display boards.

Table 1. 1992 Mastimind Quiz Questions.

1) Is foremilking a contractual condition of milk sale?

Yes [ ] No [ ]

Is there anything wrong with this technique?
Yes [ ] No [ ]

If yes, what is wrong? .....

2) Which bacterium is the most common cause of chronic mastitis?

Is it:
Strep. agalactiae [ ]
Strep. uberis [ ]
Staph. aureus [ ]
E.coli [ ]

3) This cow has developed mastitis in this straw yard. What is the likely cause of infection?

Is it:
Staph. aureus [ ]
Pseudomonas spp. [ ]
E.coli [ ]
Strep. agalactiae [ ]

4) For England and Wales to be in Band A, milk must have a total bacterial count of less than?

[ ],000 bacteria/ml.

5) This finger is infected from a cow. What is the infection?

Is it:
Cowpox [ ]
Ringworm [ ]
Pseudocowpox [ ]
Dermatophilus [ ]

6) Approximately how many cows in England and Wales have *Streptococcus uberis* mastitis infection annually?

Is it:

50,000+	[ ]
100,000+	[ ]
200,000+	[ ]
250,000+	[ ]

7) In England and Wales, what is the maximum rolling mean cell count to be in Band 1?

[ ],000 cells/ml

8) Has this cow mastitis?

Yes [ ] No [ ]

If no, what is the problem? .....

9) Approximately what percentage of UK dairy herds teat dip?

Is it:

Over 50%	[ ]
Over 60%	[ ]
Over 70%	[ ]
Over 80%	[ ]

10) Under the terms of milk sale in England and Wales, how many clear days must elapse after calving before milk can be included for sale?

[ ] clear days

11) This goat has mastitis. Which gland is affected?

Is it:

Right	[ ]
Left	[ ]
Both	[ ]

12) The Tiebreaker:

Which end of this cow do you milk? .....

The questions used are shown in Table 1.

The completed questionnaires were marked by the College team at the meeting so that Robert James, Ciba Agriculture, could present the prize. Subsequently, the questionnaires were analysed using our interpretation as to the correct answers.

Findings

It was a credit to the audience that about a third of them took up the challenge. It is fair to say that many entered into the spirit of the quiz and it is re-assuring to know that those interested in mastitis have an excellent sense of humour. The questions (see Table 1), which were considered to be a relatively fair test of knowledge, were 1 a,b,c, 2 3 4 5 6 7 9 and 10. The results, distributed between the various categories of delegates, are given in Table 2 and these are given in percentages in Table 3.

The total mark for the questions was 16 and the overall winner, Dr. Douglas McEwan, received 14. However, several others were in strong contention.

Question 1 (a) Is foremilking a contractual condition of milk sale?". This was difficult to answer and it was therefore not surprising that most were wrong in their answer. Many advisers were, however, correct, but the veterinary surgeons appeared to be lacking the knowledge.

Question 1 (b) A visual question concerning foremilking produced a high (80%) correct reply. However, in the third question as to what was wrong with the technique 1(c), most people did not give the main reason for this. A degree of interpretation was allowed in this answer, but overall success was low with only veterinary surgeons having more than half the answers correct.

Question 2 "Which bacterium is the most common cause of chronic mastitis?" produced two thirds with the correct answer, although 95% of veterinary surgeons were right.

Question 3 "This cow has developed mastitis in this straw yard. What is the likely cause of infection?" It was pleasing to see most people providing the correct answer, with advisers being 100% right.

Question 4 "For England and Wales to be in Band A, milk must have a total bacterial count of less than [ ],000 bacteria/ml?"
The advisers were again best at this question, although the overall percentage correct was surprisingly low.

Question 5 "This finger is infected from a cow. What is the infection?" This produced about the anticipated figure for being correct, with the veterinary surgeons doing best.

Table 2. The number of correct replies to the Mastimind 1992 Quiz.

PARTICIPANTS (number) QUESTION NUMBER	FARMERS (12)		VET. SURGEONS (21)		ADVISORS (6)		OTHERS (16)	
	Right	Wrong	Right	Wrong	Right	Wrong	Right	Wrong
1a	4	8	5	16	2	4	9	7
1b	8	4	19	2	6	0	11	5
1c	2	10	12	9	1	5	5	11
2	5	7	20	1	3	3	9	7
3	7	5	20	1	6	0	14	2
4	7	5	11	10	4	2	7	9
5	5	7	16	5	3	3	7	9
6	4	8	6	15	3	3	4	12
7	7	5	10	11	3	3	8	8
9	3	9	3	18	0	6	3	13
10	12	0	12	9	2	4	1	15

Table 3. The percentage correct replies to the Mastimind 1992 Quiz.

Question	Subject	Farmers	Veterinary Surgeons	Advisers	Others	Overall
1 (a)	Foremilking	33%	24%	33%	**56%**	36%
1 (b)	Foremilking	66%	90%	**100%**	69%	80%
1 (c)	Foremilking	17%	**57%**	17%	31%	36%
2	Bacteria	42%	**95%**	50%	56%	67%
3	Bacteria	58%	95%	**100%**	88%	85%
4	TBC	58%	52%	**66%**	44%	53%
5	Zoonosis	42%	**76%**	50%	44%	56%
6	Strep.uberis	33%	29%	**50%**	25%	31%
7	Total cell count	**58%**	48%	50%	50%	51%
9	Teat dip	**25%**	14%	0	19%	16%
10	Milk sale after calving	**100%**	57%	33%	6%	49%
	OVERALL:	**48%**	**58%**	**50%**	**44%**	**51%**

Question 6 "Approximately how many cows in England and Wales have *Streptococcus uberis* mastitis infection annually?" This was a very difficult question in that it involved a knowledge of the approximate percentage and incidence of cows affected with mastitis per year, the percentage of those which were due to *Streptococcus uberis* and the number of cattle in England and Wales. It was not surprising that those managing the correct answer was low. However, half the advisers were correct.

Question 7 "In England and Wales, what is the maximum rolling mean cell count to be in Band 1?" [ ],000 cells/ml. It was disappointing that only about half the questionnaires were right, although the farmers came top with 58%.

Question 8 This question was not analysed, because it was a problem which we did not expect many people, except veterinary surgeons, to be aware of.

Question 9 "Approximately what percentage of UK dairy herds teat dip?" This question was included not because it was thought people would know the answer but to see their perceptions. The answer was the least right of any in the quiz after Question 8 and in most instances the level was underestimated.

Question 10 "Under the terms of milk sale in England and Wales, how many clear days must elapse after calving before milk can be included for sale? [ ] clear days." This question was surprisingly badly answered with under half being correct. What was equally interesting was the range, with the farmers all being correct, but only 6% of the "other" group, 33% of the advisers and 57% of the veterinary surgeons.

These results suggest that, even at a conference specialising in mastitis, the degree of knowledge will be variable and it is wrong to suppose familiarity with the facts. It is likely that those attending the Fifth British Mastitis Conference were motivated to do so because they were already familiar with some aspect of the problem. It is thus likely that, if this exercise were repeated more widely outside the Conference, results would be if anything worse. It does indicate that one section dealing with mastitis problems should never assume another section is as familiar with the subject as themselves. This was particularly seen in the different sectors attending the conference.

Overall, the percentage correct answers was only about half. As mastitis is a disease entity, it was perhaps not surprising that overall the veterinary surgeons obtained most correct answers, although the percentage was only 58%. The answers were likely to be indirectly slightly biased in favour of veterinary surgeons, because of those compiling the competition. However, when looking at individual questions, advisers came top with the highest marks in four questions, followed by farmers and veterinary surgeons (three each) and others (one). The knowledge of delegates interpreted by their correct answers could be rationalised on the basis of working experience. Thus, veterinary surgeons tended to be best in the areas of disease, spread of disease, bacterial cause and zoonoses. Farmers were best in areas affecting their direct profitability, ie total bacterial count (second best), cell counts, teat dipping and days after calving before milk can be sold. Surprisingly, veterinary surgeons were not as well acquainted with these figures as might be expected for those assisting in improving these areas. The same comments could be made for the other groups.

It is difficult to know the interests of those under the category of advisers, although from the attendance list, most were probably from ADAS, or the pharmaceutical companies. The category involved only six people, but they produced correct results mainly in line with the average. They provided the best answers in some areas of disease, eg environmental mastitis, mastitis incidence as well as profitability, eg bacterial counts. It is likely that the "others" group included some advisers, as well as manufacturers etc. Overall, they produced the poorest result, but they were highest in the area of milk technique concerning foremilking.

Conclusion

The fact that mastitis is multifactorial is reflected in the different emphasis of knowledge and response from various parts of the agricultural industry. Knowledge of all aspects of the disease is not necessarily required to give good advice. Thus, veterinary surgeons appear to be most knowledgeable about disease, whereas farmers are more aware of their contractual obligations. Advisers were the second most knowledgeable about disease. It does seem that there is a need to share and use expertise from all sectors to ensure the continual control of this common production problem. To sum up then, "The delegates during the year have tried hard, but there is room for improvement".

TWO APPROACHES TO MASTITIS CONTROL

RESULTS OF OUR APPROACH TO MASTITIS CONTROL IN SCOTLAND

David Logue*, John Gunn* & David Fenlon#, * Dairy Health Unit, SAC Veterinary Services, Auchincruive; # Department of Bacteriology, SAC, Aberdeen

Introduction

Before addressing the given title it must be said that it implies that there is some authority or overall control over how mastitis is tackled in Scotland and that somehow this is vested in the particular group that is being represented here. Since this is certainly not the case it is best to set the record straight right from the start. However we are delighted to have the chance to share with you our results, conceptions and, perhaps misconceptions about this fascinating and frustrating disease. In this paper we will be presenting data, particularly that pertaining to subclinical mastitis, which were collated with the help of the three Milk Marketing Boards in Scotland and other colleagues. This project was coordinated by John Gunn and his part has been indispensible. The other collaborators are listed in Table 1. However there are a considerable number of others who have contributed in some way to the information presented here.

Table 1. Collaborators in the study of subclinical mastitis in Scotland.

D. Taylor, Glasgow University, Vet. School	D. Todd SMMB
J. McIssac A&DMMB	D. Platt, Glasgow University, Dept. Bacteriology
M. MacLeod NoSMMB	

Having been to all of the British Mastitis Conferences since they started in 1988 it would also be unwise to give the impression that we have been hiding some magic cure distilled from something or other in Scotland and have not let on to the rest of the UK. Our approach to mastitis is therefore quite predictable and follows some well trodden paths. Because of our interest we will tend to discuss the general rather than the particular. We appreciate that some would prefer the individual farm problem-solving presentation. In our experience they generally concentrate on one aspect and the overall message which we hope those farmers here will take away fails to be fully stated.

Our approach and thus this paper can be summarised under three headings:

1. Define the problem

2. Identify the main methods of control and prevention

3. Convince the farmer

1. Define the problem

In the past Scotland has always had larger herd sizes than most other parts of the UK but in recent years this difference has become less with quite a number of regions in England matching the Scottish average herd size of 91 cows. Despite the impact of quotas slowing the trend there continues to be a steady increase in the number of cows in the herd. One of the reasons for this last change has been the decline in the cowshed and in the number of herds. In the last decade in Scotland there has been a reduction in the proportion of producers using a byre milking system from 46% to 31%, a fall of nearly one third. The small rise in the number of parlours (see Table 2) belies the overall fall of around a fifth in the number of herds. One consequence of this is that the number of cows being milked, and by inference cared for, by one person has risen from 60 in the early 1980s to 70 now (Table 3). Within Scotland and indeed the UK the area around Aberdeen (A&DMMB) perhaps shows the future with an average herd size of 121 cows.

Table 2. Proportion of different milking systems in Scotland.

SYSTEM	1981	1990	Av herd size in 1990
Byre	46%	31%	55 cows
Parlour	54%	69%	106 cows

[adapted from The Structure of Scottish Milk Production 1990]

Table 3. Milking system and its effect upon number of cows per milker.

SYSTEM	No. cows/milker		Av herd size in 1990
	1981	1990	
Byre	37	39	55
Parlour	79	85	106
Overall	59	69	91

[adapted from The Structure of Scottish Milk Production 1990]

The first problem we must come to terms with when discussing inputs for mastitis diagnosis, control and prevention is the need to understand the management systems and in particular the economic and labour pressures in these larger herds. It is the demands of time which are paramount and especially those related to managing cows to reduce mastitis. Often the latter measures are seen as of less immediate priority than other farm needs such as silage making.

The second part of defining the problem is to attempt to monitor the extent of mastitis. All are well aware that there are essentially two types of mastitis:

i) *clinical*

ii) *sub clinical*

The distinction between the two is somewhat arbitrary and can be misleading

i) Clinical mastitis

The most accurate appreciation of clinical mastitis can be achieved by examining farm records, the details of mastitis treatments and most importantly the bacteriological identification of the cause. Unfortunately, in our experience, sufficient information on the last parameter to give an authoritative estimate of the predominant organism acting on the farm is rare. However even without bacteriology some insight into the problem can be achieved providing that the farm records properly and preferably puts this information in an easily accessible database such as DAISY. For example, on one farm there were a substantial number of cases of mastitis occurring around 100 days and not, as is often the case, in early lactation (Figure 1). Indeed this breakdown substantially changed the perception of both farmer and vet. In another instance there appeared to be a relationship with lameness. Unfortunately in neither example were there sufficient laboratory results available to relate these findings with a particular pathogen though both were thought to be of 'environmental origin'.

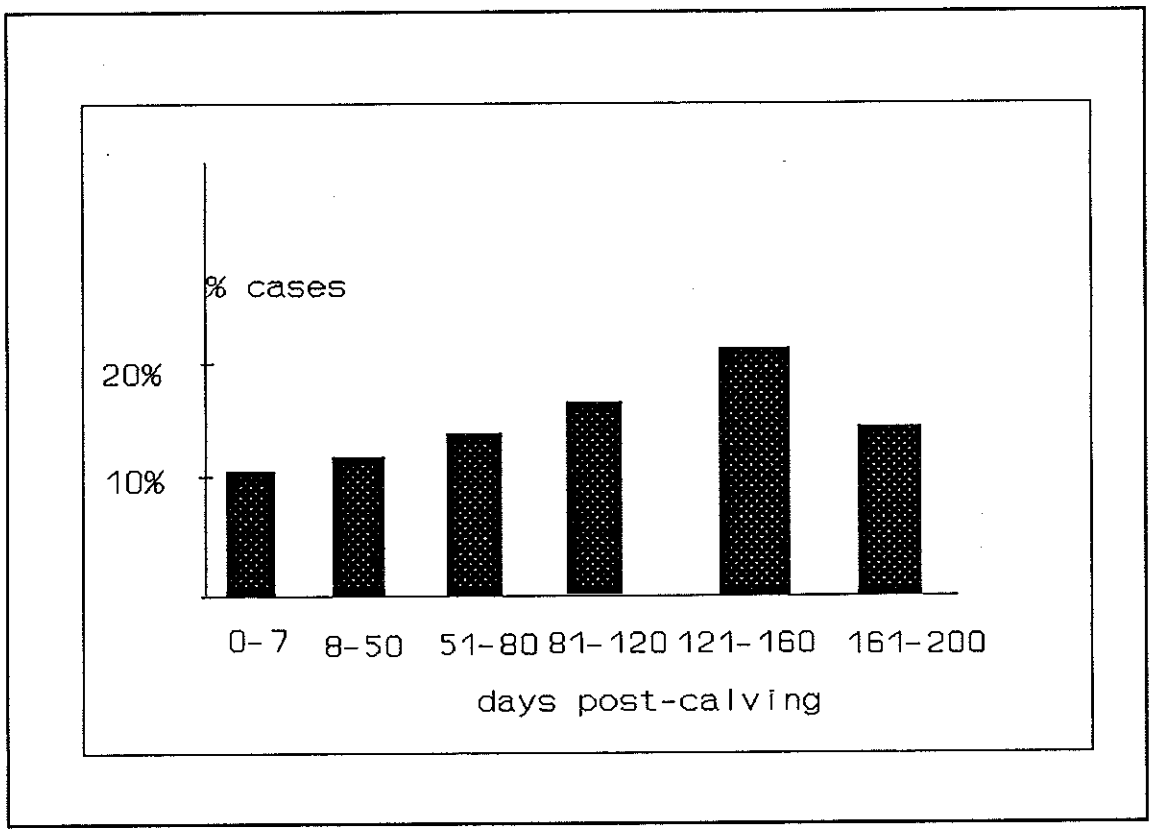

Figure 1. Incidence of clinical mastitis by days post-calving.

This reluctance to examine sufficient milks is a combination of three factors:

a) cost

b) inconvenience

c) hope.

Since in our project investigating herds with high Somatic Cell Counts (see later) we offered farmers free bacteriological examination of clinical mastitis samples and received hardly any, we are forced to the conclusion that, despite what might be thought, inconvenience and hope have as large an influence as cost! The hope is that the results from one sample will be representative of the whole. In many cases it is further compounded by the hope that they will be valid from one year to the next. Unfortunately neither need be the case. Table 4 shows the apparent change in proportion of isolates from the main herd at the SAC Auchincruive Crichton Royal Farm in two successive years (1987-89). Thus we would recommend that any farm interested in mastitis control should aim to sample about 20% to 25% of their clinically affected quarters for bacteriological examination and take the sample PROPERLY. The fresher the sample the more accurate the bacteriology so it is worth trying to ensure that it is kept cool and processed as soon as possible.

Table 4. Clinical mastitis and sampling variation between years.

Mastitis	Year 1	Year 2
Number of incidents	63	86
Number of samples	23	30
E. coli	17	11
Staph. aureus	3	6
Strep. dysgalactiae	1	11
Strep. uberis	0	1
Others	2	4

The problem of obtaining good information about the organisms acting on any one farm has forced us to another method of defining the problem. This involves taking a more general view and using the pooled results of similar laboratory tests on mastitic milk samples submitted to the 8 veterinary investigation centres in Scotland by farmers and their veterinarians. We rely totally on diagnostic field samples to give us this background and under these circumstances the data can present problems of interpretation; but some comparisons are more acceptable than others. For example comparison of the proportions of diagnoses of the different mastitis causing organisms throughout the Great Britain has remained remarkably steady over the last decade. However there does appear to be a consistent difference between these overall figures and those for Scotland alone which show firstly that there would appear to be more 'cowside' or 'contagious' organisms diagnosed in Scotland (*Streptococcus agalactiae, Staphylococcus aureus* and to a lesser extent *Streptococcus dysgalactiae*) and secondly that one particular environmental organism (*Streptococcus uberis* or SPUB in Figure 2) appears to be less common. Obviously one

could blame sampling bias for this difference but cynically it seems unlikely that Scottish farmers are any more interested in mastitis than their counterparts elsewhere.

So this raises a number of questions:

i) why is there this difference?

ii) why, if there is more cowside mastitis in Scotland, is this not readily detectable in differences in the Somatic Cell Counts in Scotland compared to the rest of the UK. (See Figure 3).

iii) why are the proportions of these major organisms in the UK so consistent when we are told that the incidence of mastitis, whether clinical or sub clinical, is declining (2) and bulk tank somatic cell counts appear to confirm this.

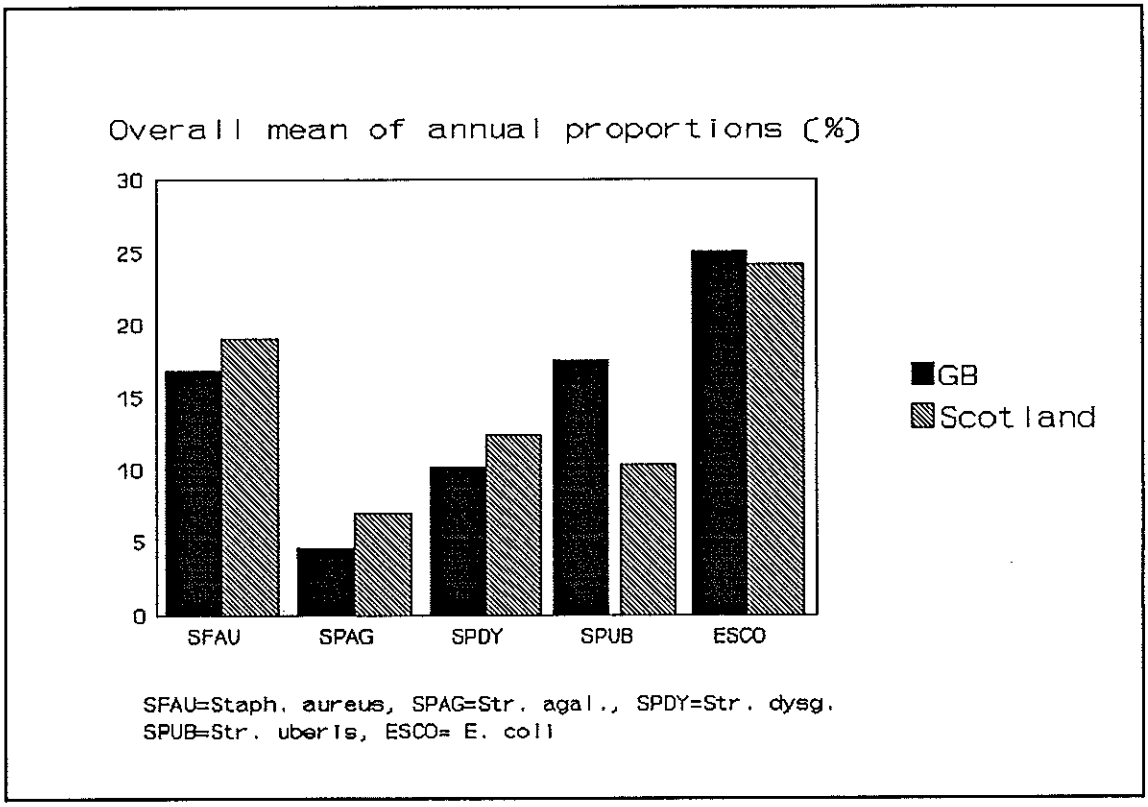

Figure 2. Mean proportion of bacterial isolations for major pathogens in milk samples in Britain and Scotland, 1980-1992.

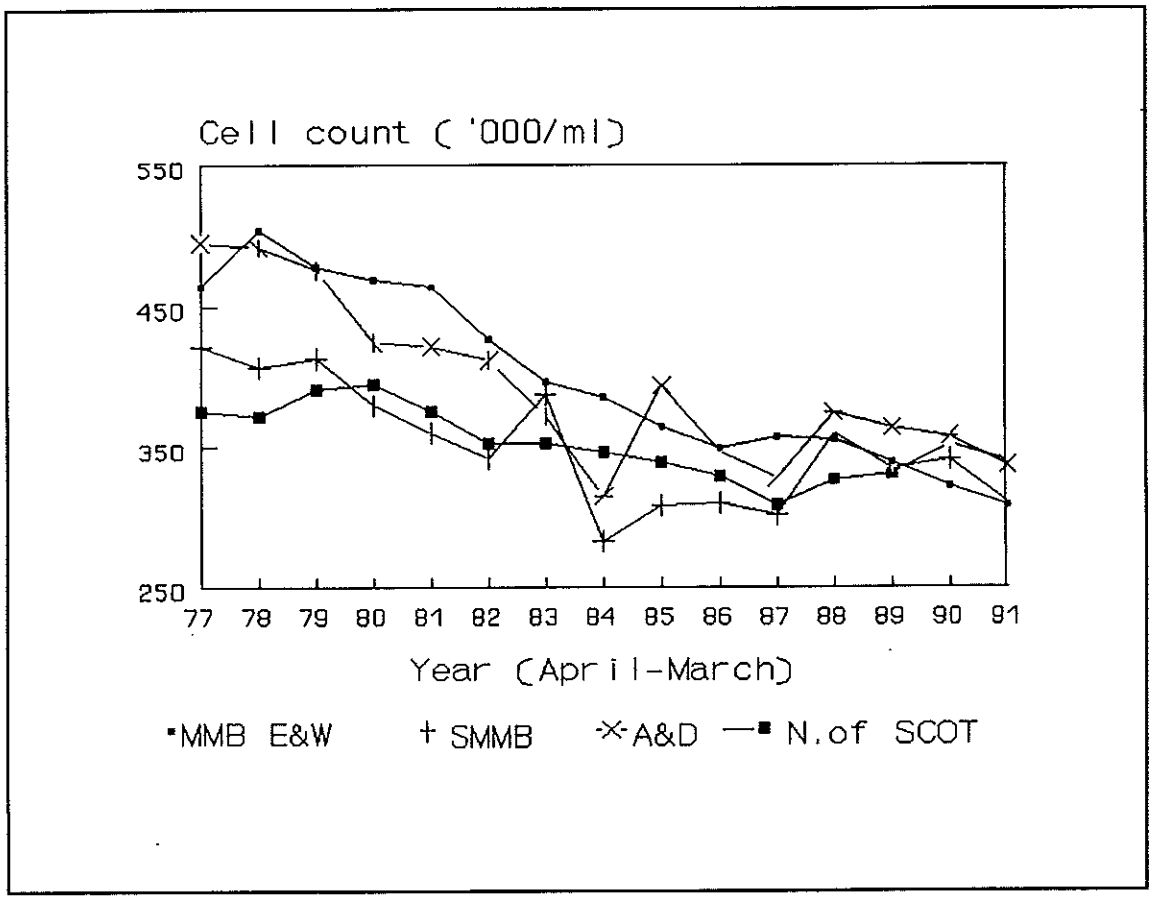

Figure 3. Changes in mean somatic cell count.

ii) Sub clinical mastitis

These are some of the questions which have been plaguing us for some time and indeed still do! The more so when at the very first of these conferences in 1988 Beck & Dodd (1) reminded the audience that the main cost of mastitis to the farmer was the level of hidden (subclinical) mastitis. However it was not until the industry realised somewhat belatedly the full implications of the first EC Directive on milk quality in 1986 that there was sufficient interest to examine this problem from a broader perspective than just trouble-shooting on an individual farm.

In attempting to define subclinical mastitis there are some advantages over the clinical disease in that a visit and bacteriological examination of milk samples can identify cases and give fairly accurate prevalence figures. Examination of data from old herd investigations by SAC Aberdeen has shown that although there were difficulties at the beginning and end of lactation there was a good correlation between the presence of infection (virtually irrespective of the organism) in the quarter and a high somatic cell count and this relationship could still be drawn for the udder as a whole (Figure 4). In other words high individual cow somatic cell counts mean an infected cow and the higher the mean cell count over a period the more likely the cow was to be infected (Figure 5). Thus it is possible to sample cows regularly

to determine their individual somatic cell count (by comparison to bacteriology this is quick and cheap) and rank them in a 'pecking order' of likelihood to be subclinically infected. Samples taken from among this group are more likely to yield a significant pathogen and so indicate which are the major subclinical pathogens acting in the herd. This information is vital in determining the best immediate control strategy.

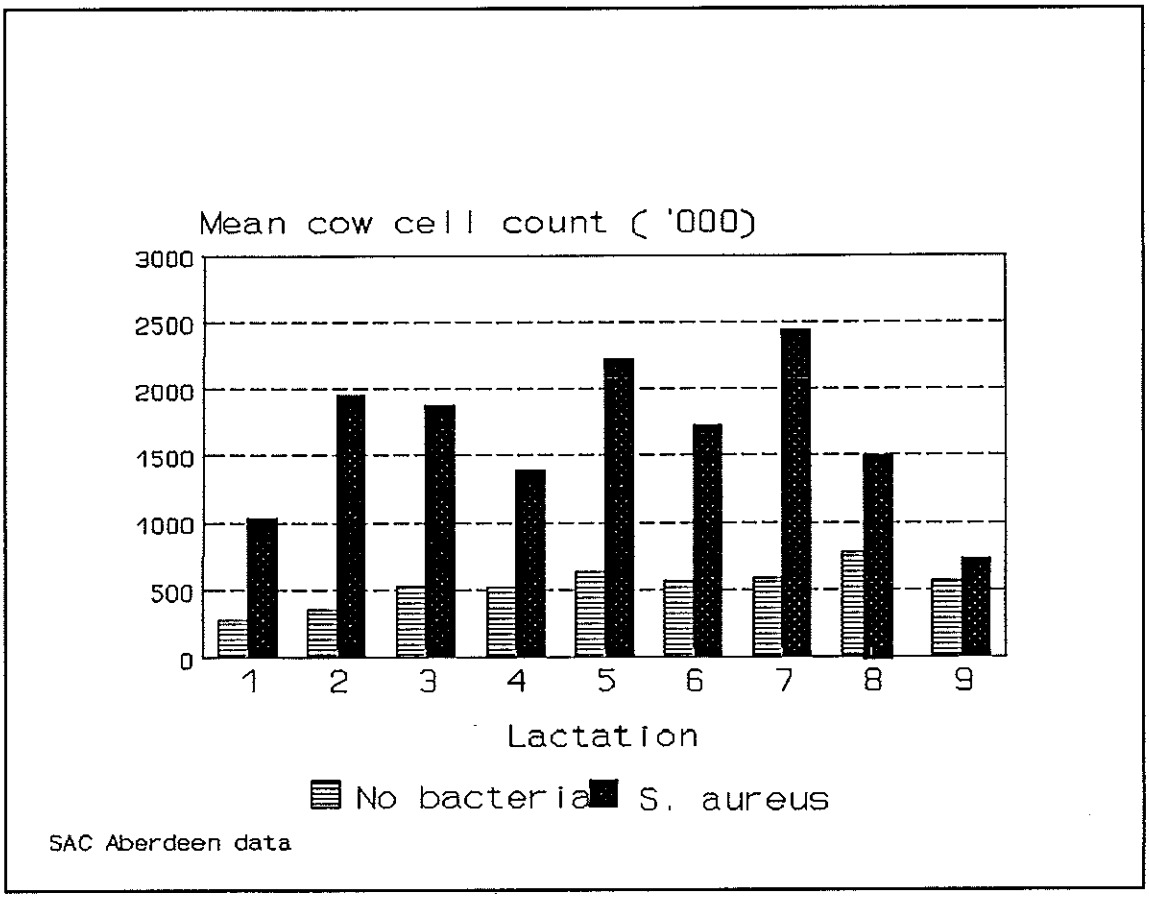

Figure 4. Effect of presence of a major pathogen (*S.aureus*) upon individual cow somatic cell count by parity.

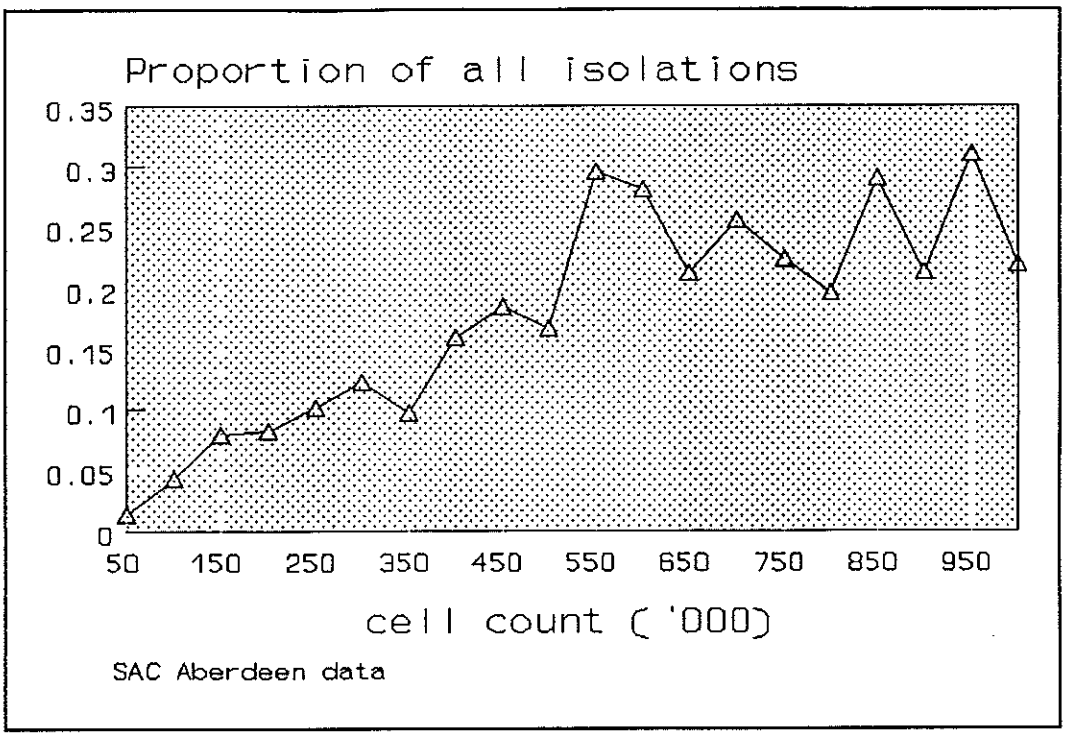

Figure 5. Likelihood of obtaining a bacterial pathogen with increasing individual cow cell count.

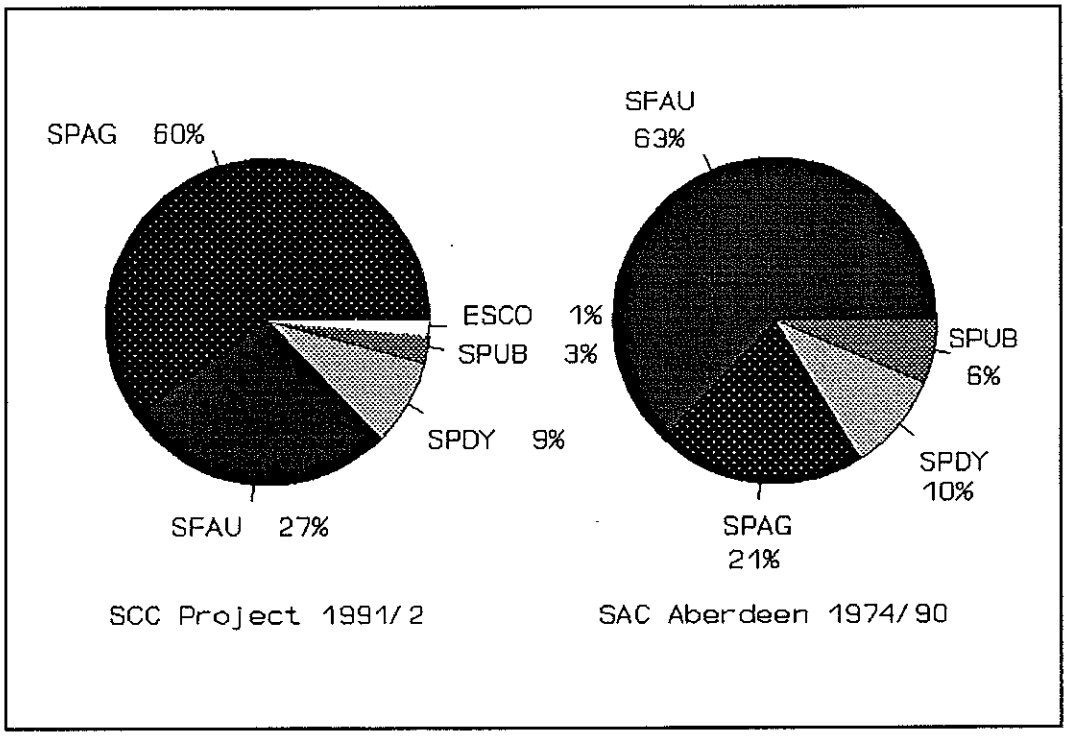

Figure 6. Frequency of isolation of different bacterial pathogens from samples taken in the Cell Count Project and by SAC, Aberdeen. SPAG - *Streptococcus agalactiae*, SPDY - *S.dysgalactiae*, SPUG - *S.uberis*, SFAU - *Staphylococcus aureus*, ESCO - *Escherichia coli*.

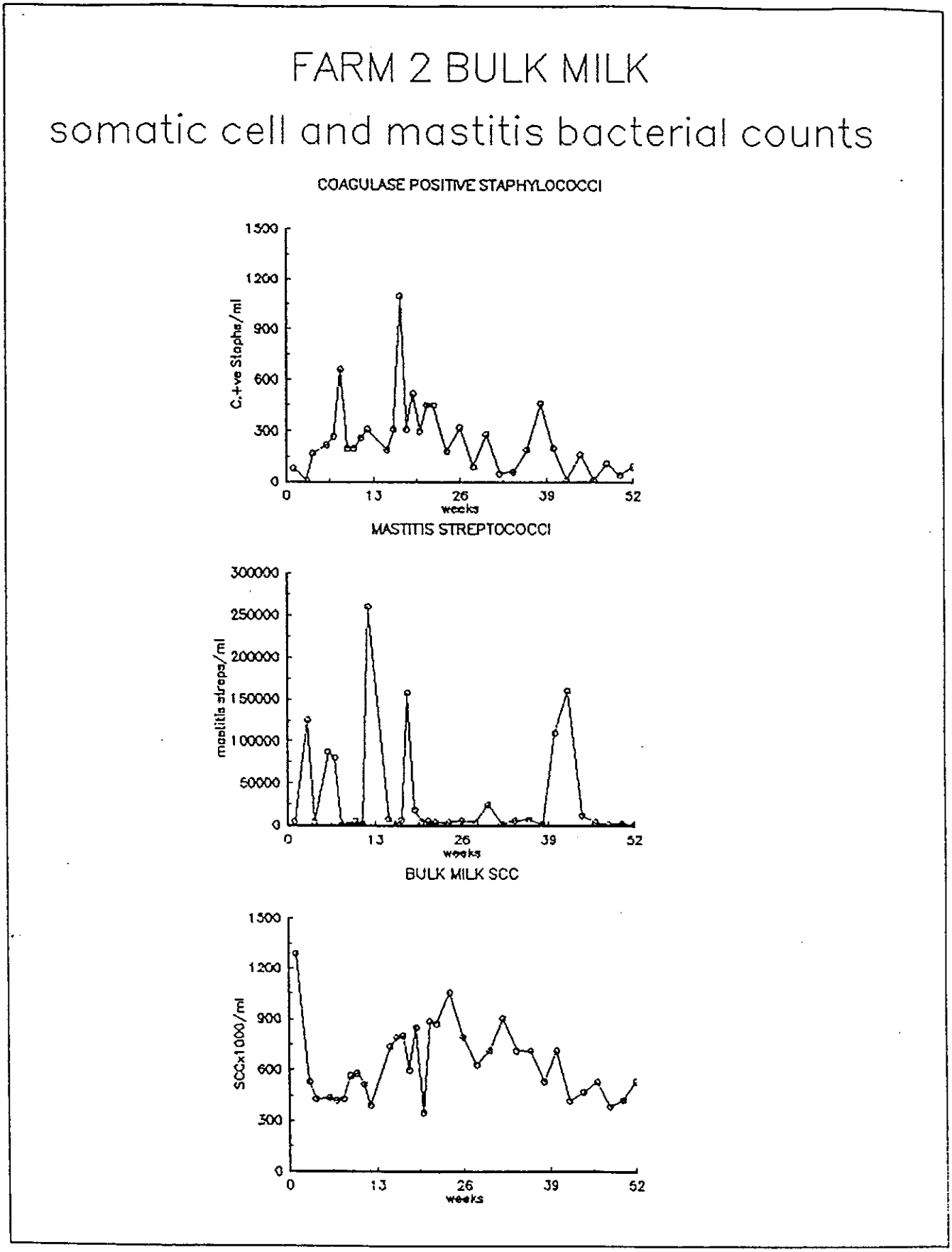

Figure 7. Variation in weekly bulk milk cell count, recovery of staphylcocci and streptococci on one farm.

Study of the Aberdeen data showed that *Staphylococcus aureus* was the most common isolate (Figure 6) however these herds were investigated because they were experiencing a 'mastitis problem' they were not selected specifically because they had a high bulk tank somatic cell count (BTSCC). The equivalent results taken from a representative group of high cell count herds identified and examined over the last two years shows that the pecking order of these major organisms has changed with *Streptococcus agalactiae* now the predominant isolate (Figure 6). It was present in 84% of these high cell count herds and accounted for 60% of all the significant isolates.

Surprisingly we could find no clear correlation between monthly herd BTSCC and Total Bacteria Count using the full database of the SMMMB. Nevertheless it was possible to use a milk sample taken from the bulk tank for culture as an 'advance' warning that mastitis-causing streptococci and particularly *Streptococcus agalactiae* were endemic. However the erratic nature of its recovery from bulk tank milk means that a negative result cannot be conclusive (Figure 7). The relatively lower numbers of *Staphylococcus aureus* shed by the infected udder mean that this organism is not reliably identified by this technique.

2. Identify the main methods of control and prevention

Since the Veterinary Investigation Centre (VIC) data indicate that almost half of all clinical cases and virtually all the sub-clinical cases in Scotland are caused by the three major so-called 'cowside' 'contagious' or 'parlour' organisms and there are now financial incentives and penalties for low and high bulk tank somatic cell counts it would seem prudent to make controlling these the highest priority. Furthermore in this endeavour we are fortunate because comprehensive measures for controlling these forms of mastitis have been developed from research which began over half a century ago - The FIVE POINT PLAN. The major problem is in persuading the farmer to apply them without using some short-cuts or ignoring them when it does not suit. We have to try to educate the farmer that success depends upon using ALL of these strategies in combination, not just what suits, and instil a COMMITMENT to the cause. Finally we must communicate a plan of action to those farms having problems; a plan which is simple, is tailored to the individual farm, states the priorities clearly and gives sensible targets. All are much easier to talk about than to do!

Unfortunately these five strategies seem to be less successful for the one third of cases of clinical mastitis in Scotland caused by the 'environmental organisms'. These have very much caught the farmers imagination, in some cases to such a degree that all thoughts of the others are ignored or certainly pushed to the bottom of the priorities list. One reason for this is the intractable nature of this latter group. Since a whole conference was devoted to environmental mastitis in 1989 and we have nothing new to add we propose to concentrate on the former save to make what might appear to some to be the facetious comment that one important method of control is to clean up the environment and look hard at your calving facilities! If you like a sixth point to add to the 5 point plan is REDUCE ENVIRONMENTAL CONTAMINATION!

Education

The very fact that so many farms have bulk tank somatic cell counts (BTSCC) in excess of 400,000 is evidence that we have not been as successful as we would have liked. But is it all the fault of the advisors? It is interesting to note that those farms in the SMMB who milk record have a lower BTSCC than the rest (Figure 8). In fact milk recorded herds are 3 times

less likely to incur a cell count penalty. We believe that much of this greater advantage stems from the greater attention to detail which these herds have learned either from hard experience or from listening to good advice (or both). The discipline of recording is something else which is very hard to impart yet it is what separates the best from the rest. Finally there is the need for tighter reproduction management which brings the veterinarian onto the farm more regularly with his attendant advice and discussion. All these are 'education factors' which should not be ignored. However the best factor is getting the farmer off his farm and on to 'good' farms to see how other farmers manage to do it better. For some reason that always makes a bigger impact than all the pontificating that this paper represents.

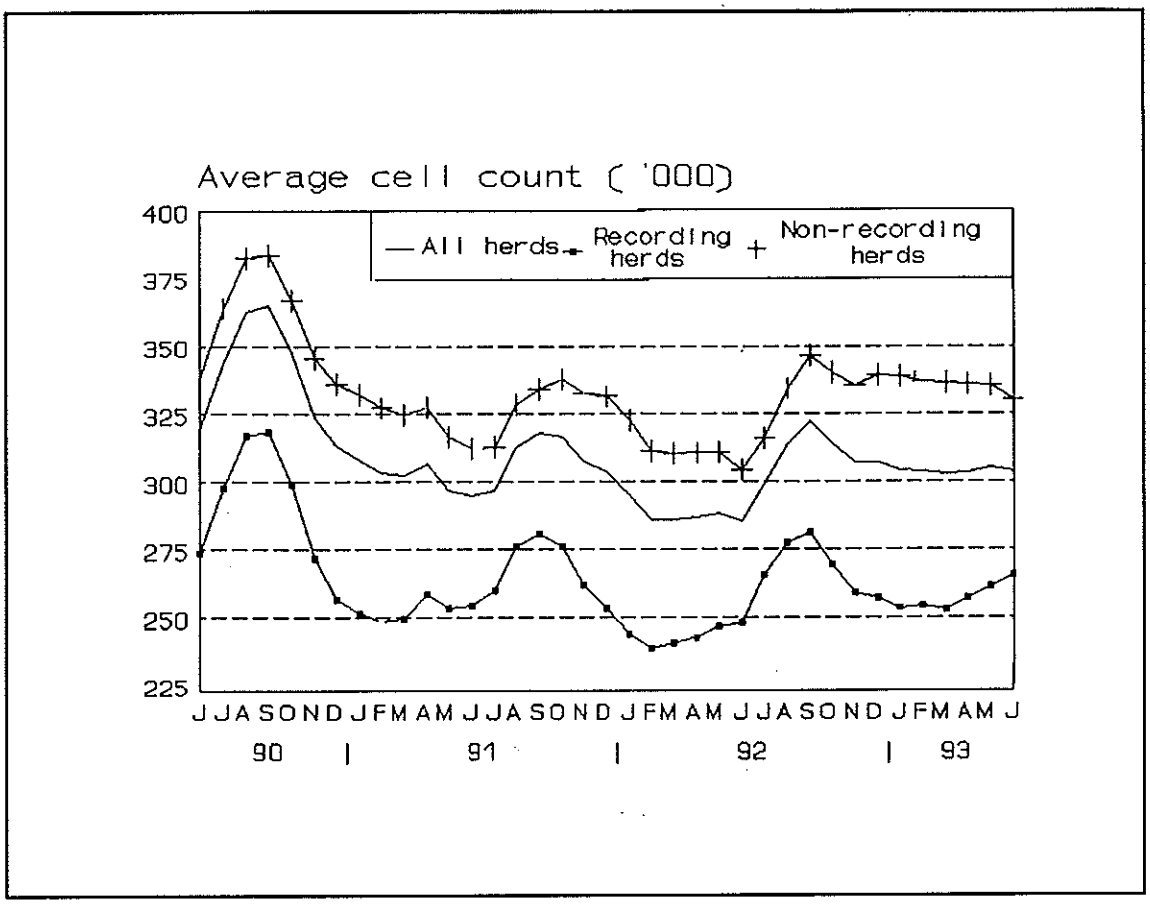

Figure 8. Bulk tank cell counts from the SMMB: Effect of milk recording.

When asked in a postal survey, the number of farmers who admitted to not following the two vitally important aspects of the 5 point plan in the control of *Streptococcus agalactiae,* post milking teat dipping and dry cow therapy, was in our opinion staggeringly high (Table 5)

Table 5. Proportion of farms failing to fully apply good mastitis control measures.

Area	Do not teat dip/spray	Do not use DCT
SMMB	44%	19%
A&DMMB	23%	8%
NoSMMB	22%	26%
Scotland total	42%	18%

[Adapted from 'The structure of Scottish milk production, 1990]

Worse, this survey did not ask whether the farms teat dipped ALL year round. Our experience is that many do not teat dip or spray in the summer. A further illustration of this failure and its importance was seen in a small survey by SAC Aberdeen and the A&DMMB comparing farms with a BTSCC greater than 400,000 (i.e. in penalty) and similar sized farms which have either had some 'borderline results' in the last year or have maintained a low value throughout (Table 6).

Table 6. Relationship between BTSCC and some management parameters.

Type of herd	High	Borderline	Low
Mean BTSCC (x10)	633	374	191
Mean annual yield	5322	6393	6278
Buy in replacements	80%	20%	10%
Post milk teat dip/spray	30%	100%	100%
Possess ACR	50%	50%	100%
Yearly machine test	20%	70%	90%
% herd >5th lactn	10%	22%	21%

The high BTSCC farms obviously do not control their management inputs as well as they should and particularly they do not disinfect teats after milking.

Commitment

The most important part of any control programme, particularly for those farms attempting to reduce their BTSCC, is COMMITMENT. We can identify the major organisms, advise treatment; milking of high risk cows last, early drying off and dry-cow therapy, checking the milking machine, and in some cases culling and so on. This is the easy part, it is the man at the sharp end who must really want to do it not just talk about it! Application of these measures to a greater or lesser extent in a small number of statistically representative farms which were given a specific advisory input (essentially one visit and regular visual updates of BTSCC and individual cow cell count [ICSCC]) has shown that progress can be made and that it was greater than in an 'unhelped' control group (Figure 9). It can be seen that at least initially the latter were also making progress so all the articles in the farming press and other advisory inputs have had a positive effect. However these mean values hide our failures.

Figure 10 shows that within the SCC Project some farms have made excellent progress and unfortunately some have not. We are still investigating the reasons for this difference but it is clear that some are more COMMITTED than others!

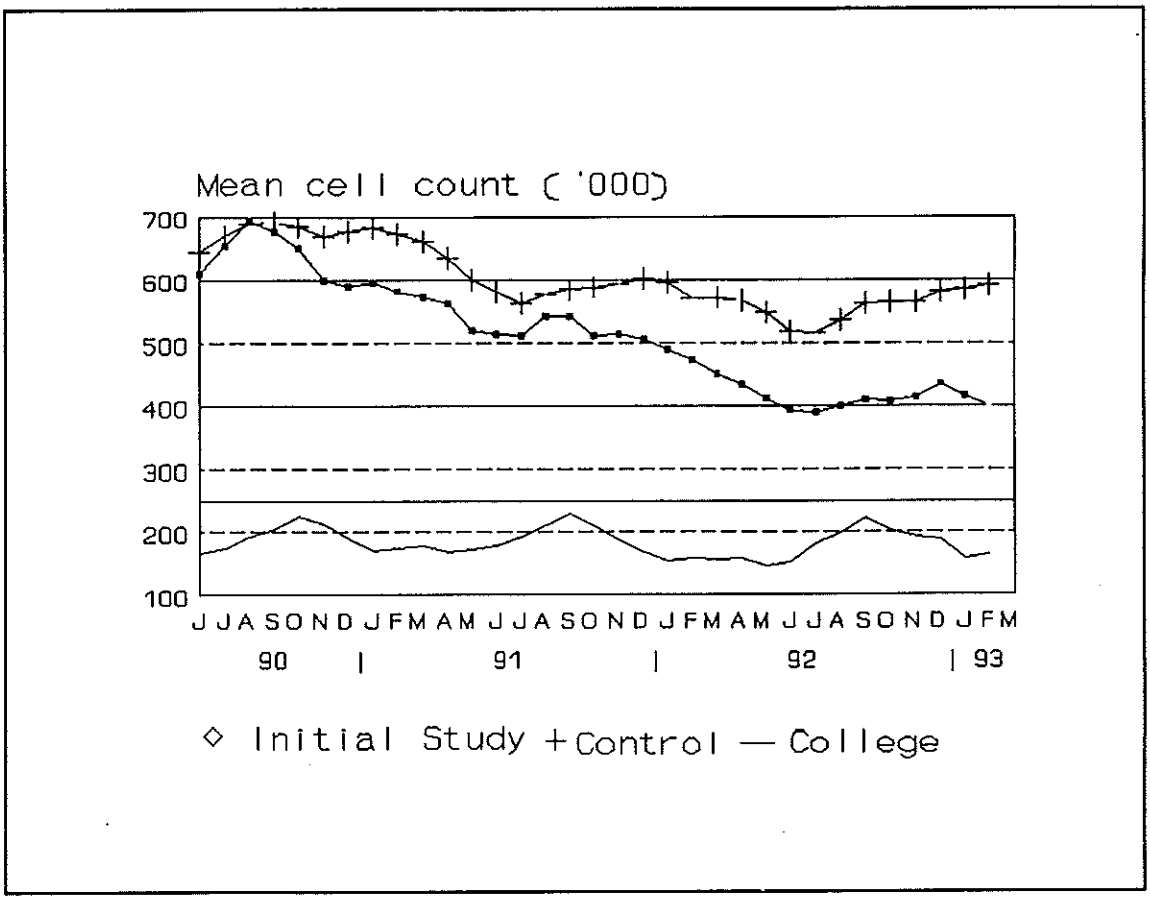

Figure 9. Progress in the Cell Count project showing changes in the bulk tank cell count for herds in the initial study and the control group (and also the SAC herds).

The main difference in the strategy used in this project over that propounded in the '5 point plan' has been the availability and application of ICSCC as means of identifying those cows which contribute most to the general level of infection in the herd and then attempting to reduce this by treating and/or culling as many as possible within the restrictions of quota and the organism(s) identified. Whereever possible these cows should also be milked last. It must also be pointed out that the higher the herd average is over 250,000 the more likely it is to incur frequent penalty (>400,000) (see Figure 11) so our target is not to get herds under 400,000 but under 200,000! It must be emphasised that treatment during lactation, in our experience, will not necessarily result in a dramatic reduction of ICSCC what we are trying to do is REDUCE THE LEVEL OF INFECTION in the herd. This will only be TEMPORARY unless the herd applies all the other strategies we have already mentioned.

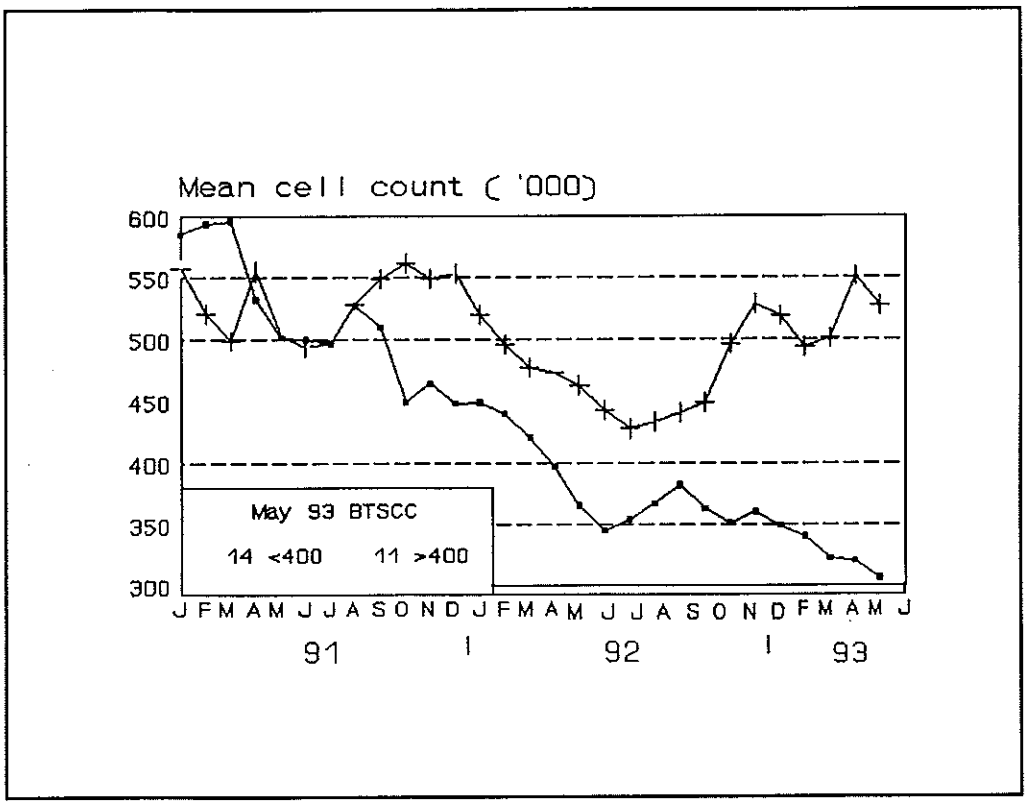

Figure 10. Cell Count Project herds, comparison of the bulk tank cell count between 14 'responding' herds and 11 'non-responding' herds.

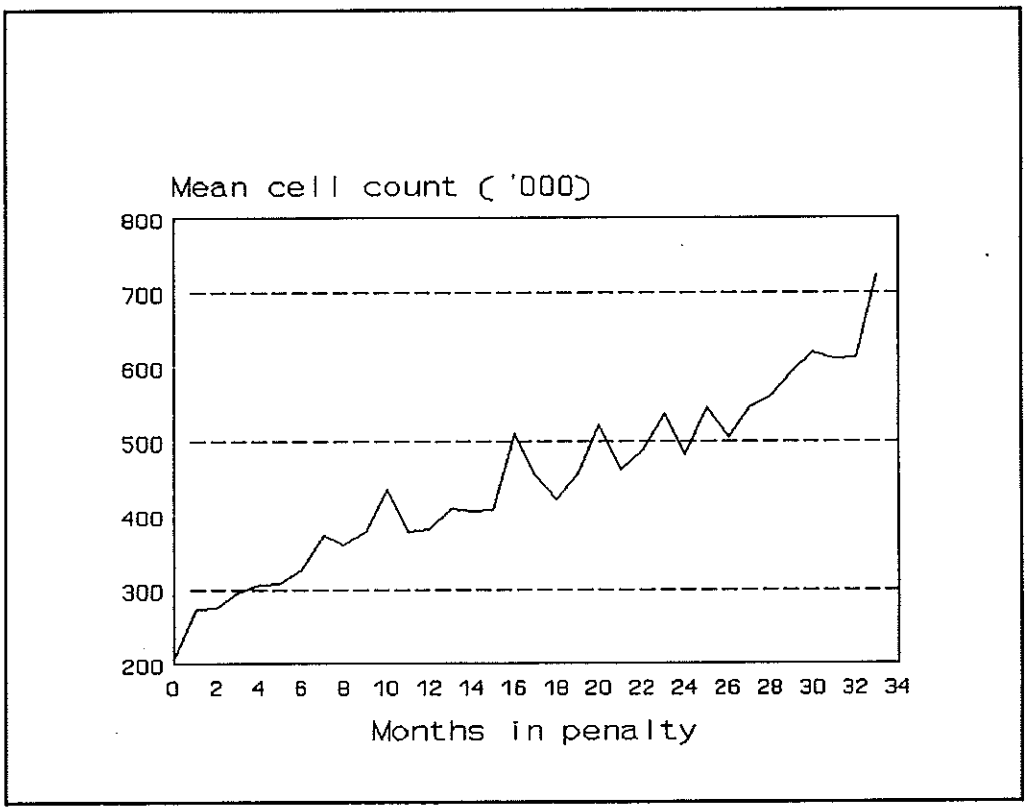

Figure 11. Mean bulk tank cell count versus months in penalty bands for SMMB herds.

Further strategies

During this study a number of the participants expressed an interest in pre-milking teat dipping and thanks to the collaboration of a commercial company we were able to allow them the use of an experimental product over the period November to January last winter. We stress that this was not a controlled trial but we took the rather pragmatic view that anything that concentrated the farm on the need for good hygiene was worthwhile. Examination of the BTSCC and TBC profiles of these few herds with the winter before and comparing them with similar 'non users' illustrated that this was probably the case though these few results are hardly a compelling argument for their widespread recommendation (Tables 7 & 8). Nevertheless it merits further more controlled study.

Table 7. Effect of premilking teat dipping on bulk tank TBC.

Comparison with same period in previous year	Better	Same	Worse
User farm	4(44%)	4(44%)	1(11%)
Non-user	4(27%)	4(27%)	7(47%)

Table 8. Effect of premilking teat dipping on BTSCC.

Comparison with same period in previous year	Better	Same	Worse
User farm	6(67%)	0	3(33%)
Non-user	8(53%)	3(20%)	4(27%)

In a similar vein we have examined whether there was any merit in giving a second treatment of dry-cow therapy, 3 weeks after drying off, to cows with a higher than average ICSCC. In this case the cows used were our own and we were able to pair the 40 cows and impose some experimental discipline but we were not able to prevent animals from being culled! The extra treatment had no significant effect upon infections caused by *Staphylococcus aureus*, nor did it significantly lower the ICSCC in the subsequent lactation, though there was a trend towards a lower figure. However there was a significant reduction in the number of 'missing' i.e. culled quarters at the end of the post treatment lactation ($p < 0.05$). Thus the general trend was that this extra treatment gave a slight advantage and we would like to repeat this with larger numbers. Smith and colleagues (3) also reported a small advantage when they gave two long-acting cloxacillin treatments with 2 weeks between finding 10.6% of staphylococcal infections persisting to calving compared to 20.3% in those treated singly. The problems in eliminating this organism are considerable. Examination of isolations at drying off and calving indicated a cure rate of just over 40% substantially lower than some reports (3,4) but still well within other estimates which range from as low as 20% (5).

These very low cure rates make it clear that we need to understand more about the relationship between this organism and our cows for it is a rare farm indeed which does not have some cows infected by it. We have just started examining isolations of *Staphylococcus aureus* from our own herds and from other herds and subjecting them to bacterial DNA fingerprinting. The results are very preliminary but we have found both within and between herd differences and similarities, for example apparently the same organism has been identified in herds as far apart as Ayr and Perth. We believe that this type of information will be needed to help us understand why this organism is so persistent and also to augment the present research into the production of a vaccine, which perhaps is not so far away now as it was ten years ago (6).

In short there is no new breakthrough, no magic injection, just confirmation that the only way to control mastitis is by hard work, application of simple principles and attention to ensure that these are correctly applied. To recap these are:

1. REDUCE THE LEVEL OF INFECTION (OR CONTAMINATION)

2. LIMIT THE POSSIBILITIES OF TRANSFER OF INFECTION

3. LIMIT THE EFFECT OF PREDISPOSING FACTORS

3. Convince the farmer

As can be seen some farmers have been sufficiently convinced to enter into a control programme with real commitment and have consequently been very successful. The problem is convincing the rest who are in cell count payment penalty bands that it is worth their while to do the same. We have made a calculation of the cost of BTSCC penalty last winter using information from the two groups of herds i.e. those given project advisory input and those without. The advantage in terms of cell count penalty alone between these two groups was of the order of £20/herd/month over last winter. Note that this ignores the higher numbers of antibiotic failures, higher TBC figures and lower milk fat and protein which we estimate could cost a further £10/month. These comparisons are not theoretical they were based on actual milk sales data and mean a loss of around £3/cow/year at present quality prices. No big deal some might say- and that is one reason why some farms are not actively reducing their sub clinical mastitis and why penalties for high BTSCC figures will continue to rise! However these same farms should note that Beck & Dodd (1) estimated halving the incidence of mastitis i.e. both clinical and sub clinical would result in a benefit of £33/cow/year. Taking all these figures together then improved control of mastitis should mean some £35 to £40 improved gross margin per cow. Of course to achieve this involves extra labour, teat-dip, dry cow tubes, and ICSCC counts but, on the plus side, there would be less treatment long-term.

These sort of figures are being continually being thrown at farmers to convince them of the need to think more deeply about a number of aspects of their management of their dairy cows and if they are all added up the figure would make one wonder if it is possible to make a profit at all! However even if those associated with the loss of efficiency are somewhat optimistic and the losses are only half of this estimate or even a third a loss of margin of £10/cow is not something to be ignored in these recessionary days.

Remember these calculations were done with last years milk quality payments. All the signs are that whoever is buying the milk this winter is going to make the financial returns for high quality milk even more attractive and of course equivalently less attractive for the lower quality and that this trend is going to continue as long as the buyers can choose their source of milk. Under commercial conditions, it takes something like two years to get the level of infection down sufficiently to be sure that there is good control of the BTSCC. Can those in penalty bands, or indeed near the borderline, afford to wait? We hope that those farmers in the audience have been convinced, and also that veterinarians and other advisors have been stimulated to try and instil a greater awareness, of the need for, and reasons behind, the present strategy of mastitis control.

Acknowledgements

We gratefully acknowledge the help and support of a considerable number of SAC staff. SAC receives financial support from SOAFD.

References

1. BECK H S & DODD F H (1988) Proceedings of the British Mastitis Conference pp27-33.

2. BOOTH J M (1988) Proceedings of the British Mastitis Conference pp 2-7.

3. SMITH A, RAUTENBACH H F P, DODD F H & BRANDER G C (1975) International Dairy Federation Document 85 pp345-348

4. EBERHART R J (1986) Journal of Dairy Science 69 1721-1732.

5. PANKEY J W, BARKER R H, TWOMEY A & DUIRS G (1982) New Zealand Veterinary Journal 30 13-15.

6. WATSON D L (1992) Research in Veterinary Science 53 346-353.

INTRODUCING A NATIONAL MASTITIS CONTROL PROGRAMME

E.F. Logan, BVMS PhD FRCVS, Milk Marketing Board, Northern Ireland

In October 1990, the Milk Marketing Board for Northern Ireland decided to introduce a Somatic Cell Count Payment Scheme (Table 1) in order to encourage milk producers to meet the proposed EEC Milk Hygiene and Quality Directive due to be introduced in January 1994. The Somatic Cell Count Payment Scheme provides premia or penalties dependant on the bulk milk somatic cell count.

Table 1. Somatic cell count payment scheme.

Depending upon the monthly average somatic cell count of the milk supplied the following financial adjustments will apply:

Class	Average Payment ('000)/ml	Adjustment ppl
C+1	200 or less	+0.1
C	201- 400	Nil
C-1	401- 600	-0.3
C-2	601-1000	-0.8
C-3	More than 1000	-1.6*

*An additional deduction of 9.0 ppl will be applied to the month if supplies in the preceding two months are in class C-3.

The payment adjustments shown will be effective from 1 October 1993.

It was also realised by the Milk Marketing Board for NI that farmers would need expert advice and a veterinary surgeon was appointed to set up a mastitis control programme. Meetings were held with representatives of the Association of the Veterinary Surgeons Practising in Northern Ireland and the Northern Ireland Veterinary Association to explain the role of the veterinary surgeon and to seek their approval and support. The dairy farmer's veterinary practitioner receives a copy of all results and it is hoped that he will co-operate fully with the farmer to discuss the problem and to supply advice.

Somatic cell counts and sub clinical mastitis

What is a normal cell count? I consider that a herd of healthy dairy cows, milked correctly, should have a monthly bulk somatic cell count of 100,000/ml or less. The top herds in Northern Ireland have little difficulty achieving this figure (Table 2).

Table 2. Annual somatic cell count (SCC) & total bacterial counts (TBC) of regional winners & runners up for high quality milk awards in 1991-92, and 1992-93.

	12 Month average SCC ('000/ml)	12 Month average TBC ('000/ml)
1991-92		
22 herds	62	6
Overall winner	47	3
1992-93		
26 herds	63	8
Overall winner	36	4

High bulk milk cell counts are caused by individual cows with high somatic cell counts. The more cows with high counts, the higher the bulk milk cell count. The cause of a high somatic cell count in a cow can be either physiological or pathological; the former usually being an individual cow problem, whereas the latter is a herd problem. Cows which are newly calved, or drying off, have very high somatic cell counts and cows which, for whatever reason, do not reach peak yield, usually have higher than normal cell counts.

The pathological cause is sub-clinical mastitis which is an inflammation of the udder. The cells in the milk are white blood cells which migrate from the blood to the udder. It should be appreciated that other substances also pass into the inflamed udder from the blood and that milk from a cow with sub-clinical mastitis is not normal.

Sub-clinical mastitis can be caused by repeated mechanical damage, by infectious bacteria, or, not uncommonly, a combination of both, the former predisposing the udder to invasion by the bacteria. The milking machine and its components are the commonest cause of physical damage, although rough handling of cows cannot be ruled out. However, sub-clinical mastitis, caused by infectious bacteria, is the most important cause of high bulk milk somatic cell count.

The mastitis control programme

The aim of the Mastitis Control Programme is to examine each herd, identify the problems in the herd and to provide a solution for that individual herd. The scheme operates as follows:

All milk producers are encouraged to make use of the Mastitis Control Programme but farmers with cell counts above 400,000ml regularly receive letters informing them that their somatic cell count is too high. They are advised to sample the herd to identify the problem. The distribution and collection of the milk sample bottles are organised and coordinated by the Regional Field Officers who operate from 4 regional centres in Northern Ireland.

Initially, composite milk samples are taken from the collection jar from each milking cow in the herd or, if jars are not available, individual quarter samples are taken. These samples, which are preserved, are examined for somatic cell count.

The samples are accompanied by a form which provides the identification and relevant history for each cow (Table 3). It also gives the name of the farmer's veterinary surgeon. The results are analysed and appropriate comments added, both for individual cows and a general comment on the herd. The farmer then re-samples either individual quarters which have high cell count or samples each quarter of the individual cows with high counts and a somatic cell count and bacteriological examination are then carried out on these samples. If *Staphylococcus aureus* is isolated then a drug sensitivity pattern is sent to the veterinary practitioner.

All milk recorded herds receive a somatic cell count for individual cows at each monthly recording and herd owners can send individual quarter samples to the laboratory for bacteriological examination and somatic cell count.

Ideally, one would like to carry out a full bacteriological examination of each quarter of every cow in the herd, but this would probably not be economically viable and in the case of high somatic cell count herds, would probably be too detailed an examination.

What information does the Mastitis Control Programme provide to the herd owner?

Somatic Cell Count

The initial screening of the herd, when composite milk samples are taken from each cow and tested for somatic cell count, identifies those cows with normal counts, and those with sub-clinical mastitis. Frequently, the somatic cell count pattern helps to identify major defects either in the milking plant or the milking technique.

If the milking plant is working properly and the milking technique is good, almost every cow in the herd will have a somatic cell count under 200,000/ml and cows, in full milk, will have counts under 100,000/ml and can be as low as 9,000/ml. Only cows with infectious, sub-clinical mastitis will have high cell counts.

If overmilking is a problem, then cows in full milk will have very good counts i.e. under 100,000/ml but the counts of cows in mid/late lactation will rise to between 300,000 - 600,000/ml or higher. Overmilking occurs more frequently than dairy farmers realise and it is significant that when automatic cluster removers are added to a milking parlour, the somatic cell count in succeeding months usually falls by at least 100,000/ml.

If the milking plant is not working properly, then most of the herd will show evidence of milking machine damage to the udder. Very few cows will have counts under 200,000/ml and the majority will have counts between 500,000 and 1,000,000/ml. In these cows usually all four quarters will be affected but it is not uncommon to find only one quarter affected.

Heifers are particularly susceptible to milking machine damage and, as the lactation progresses, their cell count will gradually increase. Instead of having counts of around

Milk Marketing Board for Northern Ireland

MASTITIS CONTROL SCHEME

TABLF 3

Producer's name _____

Address _____

_____

Ident No _____

Date sample taken _____

No of cows in herd _____

No of cows in milk _____

No of cows dry _____

Veterinary Surgeon's name

_____

Address _____

_____

Ident No of cow	Lact No	Date of last calving	If not available: is cow					Has cow had Mastitis? How many times ?	Any other information	FOR OFFICE USE ONLY	
			Freshly calved	Full milk	Mid lact	Late lact	Drying off			cell count thous/ml	Remarks

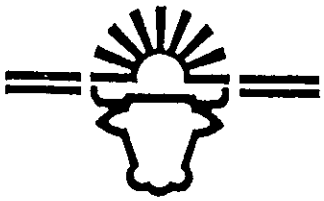

TABLE 4

MASTITIS CONTROL SCHEME

Farmer's name _____ Veterinary Surgeon's name _____

Address _____ Address _____

_____ _____

Ident No _____ Tel No _____

INDIVIDUAL QUARTER MILK SAMPLES FOR BACTERIOLOGICAL EXAMINATION

Total number of samples taken _____ Date sampled _____

Ident No of cows in this box	Lact No	Date of calving	If not available: is cow				
			Freshly calved	Full milk	Mid lact	Late lact	Drying off

SAMPLING OF MILK FOR BACTERIOLOGY

Samples must be taken carefully.

I realise that it is impossible to obtain aseptic samples under field conditions but suggest the following routine to keep teat infections and extraneous infection to a minimum:-

1. Discard first strones of milk

2. Clean the teats only - DO NOT WASH - ensure that they are dry when you sample.

3. Clean end of teat with 50% spirit* and cotton wool.

4. Take a few strones of milk (cleanly and quickly) from each quarter into a sterile bottle; ¾ fill the bottle.

5. Dispose of used cotton wool

E F Logan
Veterinarian

*50% spirit = ½ methylated spirits + ½ water

30,000 - 50,000 ml they will have a 10-fold increase with counts consistently between 400,000 and 500,000/ml.

In some herds, the older cows will have a higher somatic cell count and the younger cows will have very good counts. In these herds it is usual that the milking machine is functioning properly, but the milking routine and hygiene are poor, allowing pathogenic bacteria to pass from cow to cow at milking time, and thus new infection rate is high.

Bacteriology of milk samples

To take full advantage of the Mastitis Control Programme it is essential that the farmer sends in follow-up samples from cows with a high somatic cell count to the laboratory. These samples must be taken aseptically and a leaflet (Table 4) giving instructions is sent with the sample bottles. From these samples, a variety of bacteria can be isolated; some are related to infections and others to the hygiene and milking routines. Nevertheless, in most herds, one pathogenic species will predominate. The distribution of bacteria recovered from milks in Northern Ireland is shown in Table 5 where it can be seen that the commonest organism is *Staphylococcus aureus* which was found in 98.4% of herds. *Streptococcus agalactiae* was found in 19.6% of herds. *Streptococcus dysgalactiae* and *Streptococcus uberis* were also isolated consistently. These latter organisms tend to be found in only a few cows in each herd and were the predominant organism in only two and one herds respectively.

Table 5. Pathological bacteria recovered from 250 herds 1/12/91-30/11/92.

	NUMBER OF HERDS		
Bacterium	Isolations	Only isolate	Predominant isolate
S.aureus	246 (98.4%)	72	210 (84%)
Str.agalactiae	49 (19.6%)	3	33 (13.2%)
Str.dysgalactiae	93 (37.2%)	1	2 (0.8%)
Str. uberis	72 (28.8%)	-	1 (0.4%)
L. monocytogenes	2	-	-
Group L Streps.	2	-	-
Group G Streps	1	-	-

Other bacteria, isolated from quarter samples, give an indication of the quality of the milking routine. In herds where teat dipping is not practised or is ineffective, two types of bacteria are often found in quarters; certain coagulase negative staphylococci, predominantly *Staphylococcus epidermidis* and the other streptococci. *Staphylococcus epidermidis* lives on the skin of the teats and udders of cows and some researchers believe it is a minor pathogen which causes some udder damage raises somatic cell counts to around 500,000/ml. I believe it is an opportunist which inhabits already damaged quarters - it is often isolated in quarters which have been damaged by a previous, usually coliform mastitis.

Several types of streptococci contaminate skin sores and scratches and from these are transferred into the udder during milking. They too can cause sub-clinical mastitis.

Drug sensitivity tests are carried out on isolates of *Staphylococcus aureus*. Some researchers consider these to be of little value, but drug sensitivity patterns do vary between herds (Table 6) and in some herds there may be 2 or more strains with different drug sensitivity patterns.

Table 6. *Staphylococcus aureus* drug sensitivity - 846 subclinical isolates from 250 herds.

Antibiotic	AMP	CEP	CLX	ERY	NEO	NOV	PEN	TET
Concentration μg/IU	2	5	5	5	1	5	1.5	10
No of isolates								
215	S	S	S	S	S	S	S	S
503	MS	MS	S	S	S	S	MS	S
94	MS	MS	S	S	S	S	MS	R
21	R	MS	S	S	S	S	R	S
6	S	S	S	S	S	S	R	S
3	R	S	S	S	S	S	R	R
2	R	S	S	S	S	S	S	S
2	S	S	S	S	S	S	S	R

S = Sensitive
MS = Moderately Sensitive
R = Resistant

Action by the milk producer

Having carried out the mastitis investigation and established the problem it is then necessary for the farmer to take action. In the short term, where the milk quality leads to a financial penalty, it is important to reduce the cell count quickly. This can be done by drying off cows in late lactation which have high cell counts and in other cases selectively feeding the milk from high cell count cows to calves.

I cannot over emphasise how important the milking machine is in relation to sub-clinical mastitis. Studies carried out both in Scotland and in the Veterinary Research Laboratories in Northern Ireland have shown that 68%-75% of milking machines were faulty in high cell count herds, the commonest faults being:

1) insufficient vacuum reserve
2) vacuum fluctuations
3) too high a vacuum due to a faulty regulator

In Table 7 are examples of milking machine damage. The monthly somatic cell counts are quoted for two herds, one where a new milking parlour had been built, the other where an existing plant was faulty. In the former herd the plant was installed in 1990. Following an initial drop the somatic cell count rose to over 1 million/ml by February 1991. The herd was sampled, the problem identified, an MMB technician adjusted the milking machine and the

bulk milk counts fell as the damage to the udders was resolved. In the second herd, the plant was updated and serviced in October 1992. Currently both herds have somatic cell counts of less than 200,000/ml and receive premium payments. If the milking machine has been causing serious problems it would be necessary to resample the cows in a few months time to establish that the cell counts of the individual cows were falling before taking any further action.

If he is not already doing so, the farmer should be encouraged to treat all cows with antibiotics at drying off. Dry cow therapy is the most effective way of removing infectious bacteria from the udder. Cows with a history of infection or visible damage to the udder should be culled.

It is important to improve the milking routine in the herd. There is little point in treating cows with sub-clinical mastitis if re-infection of these cows is not prevented. Owners are encouraged to follow the 5-point plan which is designed to minimise the spread of pathogenic bacteria during milking. Where *Staphylococcus aureus* is the causative organism, producers are advised not (except in particular cases) to treat the milking cows having a high cell count with antibiotics. The cure rate is very low (approximately 20-25%) and if more than one quarter in a cow is involved it is unlikely that a bacteriological cure is possible. In the longer term, old cows which have a history of mastitis and/or have been treated previously during the dry period and show improvement in cell count should be culled - there is no alternative.

In the case of herds where *Streptococcus agalactiae* is the predominant organism the possibility of "blitzing" the herd should be considered. That is, treat all cows with an appropriate antibiotic preparation and withhold the milk for the required time. This is a very effective means of reducing and sometimes eliminating the bacterium from a herd. Herds in which *Streptococcus agalactiae* is a problem rarely follow the 5-point plan. It is essential once the level of infection has been lowered that good milking practice is established to prevent re-infection of the cow. *Streptococcus dysgalactiae* and *Streptococcus uberis* are usually associated with poor environmental conditions. Cubicles will be dirty and passages and collecting areas badly soiled with slurry.

Since the inception of the Mastitis Control Programme over 2,500 herds and over 50,000 cows have been tested up to March 1993. In addition, a further 850 herds on milk recording receive monthly individual cow cell counts.

Conclusions

What effect has the Mastitis Control Programme had on somatic cell count in Northern Ireland? The annual figures are shown in Table 8 where it can be seen that prior to the scheme the cell count was relatively constant at around 350,000 cells/ml but since 1991 there has been an overall fall of approximately 100,000 cells/ml. In the 5 months January - May 1993 the monthly average cell count has been very steady in the range 218,000 - 224,000 ml.

Table 7. Milking machine damage in 2 dairy herds - somatic cell count (x1000) before and after servicing milking machine.

Herd 1		Herd 2	
Month	SCC ('000)/ml	Month	SCC ('000)/ml
1990		1992	
May	357	April	433
New Milking Parlour Installed		May	442
June	265	June	599
July	215	July	678
August	357	August	618
September	376	September	430
October	312	October	414
November	615	Milking Machined Serviced	
December	452	November	258
		December	422
1991		1993	
January	688	January	167
February	1148	February	165
Milking Machine Adjusted & Serviced		March	112
April	537	April	119
May	423	May	106
June	339		
July	251		
August	400		
September	337		
October	309		
1993			
May	197		

Table 8. Annual average somatic cell counts in Northern Ireland.

1981-89	Count ('000/ml) range 369-327
April - March	
1988-89	347
1989-90	359
1990-91	300
1991-92	236
1992-93	260

The increasing number of herds in the lower somatic cell count bands (Table 9) also shows the improving position.

Table 9. Numbers of herds by somatic cell count class.

	C+1 ≤200	C 201-400	C-1 401-600	C-2 601-1000	C-3 >1000
	No %	No %	No %	No %	No %
Oct. 1990	1566(25)	2687(43)	1195(19)	650(11)	141(2)
Oct. 1991	2011(33)	2857(47)	818(14)	303(5)	58(1)
Oct. 1992	1699(29)	2540(43)	1010(17)	515(9)	100(2)
Apr. 1993	2642(46)	2003(35)	652(12)	355(6)	50(1)
May 1993	2791(49)	2055(36)	637(11)	225(4)	24(<1)

At the individual herd level Table 10 shows the typical results of seven herds which have participated in the Mastitis Control Scheme. In each herd there is a marked sustained improvement in the monthly somatic cell counts.

Table 10. Monthly somatic cell count of 7 herds which have participated in the mastitis control programme.

	Herds - SCC ('000/ml)						
Month	1	2	3	4	5	6	7
1990							
Oct	716	810	794	442	1066	972	904
Nov	566*	1268	675*	439*	804	998*	1115
Dec	320	1331	380	462	883	697	1067
1991							
Jan	246	1057	205	354	400*	537	993
Feb	216	1219	226	290	619	461	1255
Mar	154	1214	281	321	252	296*	1052
Apr	74	1274*	222	271	172	247	1071*
May	106	428	126	200	116	218	668
Jun	71	335	140	315	499	206	418
Jul	93	339	276	268	305	342	280
Aug	140	539	243	245	228	284	303
1993							
May	176	332	197	95	312	108	241

*Month of sampling under mastitis control programme

Of particular interest is the effect of treating milking cows infected with *Streptococcus agalactiae* (Table 11). This herd has moved from the penalty bands in October 1992 to premium band in April 1993 - a ten-fold decrease in somatic cell count.

Table 11. Somatic cell count (scc) in a dairy herd before & after treatment of cows for *Streptococcus agalactiae* infection.

Month		Monthly SCC ('000/ml)
1992	June	1000
	July	1227
	August	1195
	September	1468
	October	1000
	Herd Sampled & Cows Treated	
	November	570
	December	270
1993	January	331
	February	265
	March	208
	April	139
	May	127

Herd owners should be encouraged to test their herd annually. In this way a somatic cell count profile can be established for the individual animals and the efficiency and effectiveness of the milking routine monitored.

In conclusion, it should be realised that mastitis is a multi-factorial disease and if it is necessary to continually use antibiotics to treat cows in a dairy herd, then it is an indication of failure of management by the herd owner. Antibiotics should be used to treat cows as a last resort and not a first line of defence.

A good somatic cell count is the art of milking cows.

Acknowledgements

I thank Mrs Rose Teague and her staff in the MMBNI Central Laboratory for carrying out the somatic cell counts and the microbiologist Mr Paul Gillespie and his staff of the DQF Central Quality Control Laboratory for their co- operation and expertise in carrying out the bacteriological examination of the milk samples.

THE TREATMENT OF DIFFICULT MASTITIS CASES

THE TREATMENT OF DIFFICULT CASES OF MASTITIS

R.W. Blowey BSc BVSc MRCVS, 124 Stroud Road, Gloucester.

Introduction

The treatment of mastitis has occupied hours of discussion and has certainly been the subject of numerous research papers. In this conference alone, there have been four papers dealing with treatment in the past three years. As a practitioner, I am frequently asked for "something stronger" for those cases which are difficult to treat and I am sure it would be to the combined delight of the farming, veterinary and agricultural industries if a 100% successful, one application, non-milk withholding treatment could be found!

At what is treatment directed?

A simplified series of events relating to the pathogenesis of a new case of mastitis could be described as:

1. Arrival of pathogen at the teat end.

2. Bacterial penetration of the teat canal and arrival in the teat cistern

3. Bacteria elicit a host response (which is primarily a cellular invasion of the teat and gland cisterns by white blood cells).

4. Some bacteria (e.g. coliforms) produce toxins which may result in changes in the host such as

    - heat and swelling in the quarter
    - generalised illness i.e. toxaemia, pyrexia and shock

5. Other mastitic bacteria (e.g. *Staphylococcus aureus* and *Streptococcus agalactiae*) adhere to the mammary duct epithelium. At this site they are less likely to be "washed away" by the flow of milk from the udder and hence may become established as chronic infections.

6. Finally, certain bacteria, notably *Staphylococcus aureus* and Pseudomonad sp., are able to persist within a cell (typically within macrophages) where they are protected from the action of antibiotics.

In the treatment of mastitis therefore, therapy could be directed against

- killing the bacteria (i.e. use of antibiotics)
- flushing out the bacteria (i.e. regular stripping)
- enhancing the cow's own ability to overcome the bacteria
- control of the secondary effects of toxaemia (i.e. a hard quarter and/or sick cow)
- preventing the establishment of a carrier cow which would otherwise remain a risk to other animals.

There is a body of opinion which considers that the treatment of all mastitis except in clinically sick cows is unnecessary. The logic of this is that approximately 60% of clinical cases of mastitis are caused by environmental organisms and that many of these cases undergo "self cure", with elimination of the causative organism, in the absence of treatment (1). Supportive evidence comes from the fact that up to 40% of all clinical cases sampled for bacteriology are found to have no significant organisms present. This subject was extensively reviewed by Craven (2) at a previous Mastitis Conference. He calculated that only 37% of infections would bacteriologically undergo self-cure in the absence of antibiotic therapy and he concluded that, on balance, treatment was worthwhile. Benefits obtained from those cases which required treatment outweighed the losses associated with those cases where treatment was unnecessary.

The definition of a "difficult case"

As the subject of my paper is the treatment of "difficult" cases, it is obviously necessary to clarify my definitions. My initial reaction was to take the scientific approach, in other words, define the organism and discuss possible treatments of mastitis caused by that particular organism. However, this would not be the herdsman's approach. He has to deal with a case on the clinical signs of mastitis as presented to him. As the audience at this Mastitis Conference is essentially a practical one, I have chosen the latter route.

Much of the following material is a subjective clinical assessment of cases and of a client's perception of response to treatment. My comments should therefore be interpreted accordingly. The "difficult cases" which I intend to consider include:

1. Acute coliform infections
2. Gangrenous mastitis
3. The hard, swollen, non-responsive quarter
4. A chronic recurrent case.

1. Acute coliform mastitis

"*E. coli* mastitis" has become almost a household term on dairy farms and is often a cause of alarm. Infection can produce a range of clinical signs, varying from sudden toxaemic death at one extreme to chronic carrier cows at the other. Cows which are able to mount a significant cellular response develop a hard, hot quarter, often with a characteristic straw-coloured, watery secretion. These cows are often not particularly ill, although their temperature may be raised. However, in cows where the quarter stays soft and changes in the milk are almost undetectable, the host cellular response is likely to be poor, the toxaemia more severe and the cow generally sicker.

There is a body of opinion which states that antibiotics are not indicated in the treatment of coliform mastitis because many, or the majority, "self cure", as shown experimentally (1). However, I would not support this view because -

A When the case is first presented it is not always possible to be sure which organism is the causative agent.

B Even though some 40% of clinical cases of mastitis are sterile by the time that clinical changes are first seen, this still means that the organism is present in the majority (60%) of cases.

In the initial stages at least therefore, I would recommend treatment with both parenteral and intramammary antibiotics. Theoretically, bacteriostatic antibiotics which inhibit bacterial growth and therefore rely on natural bacterial death or bacterial removal by host cells are better than bacteriocidal antibiotics, which specifically kill bacteria, since the latter can lead to the further release of toxin. However, it is very difficult to assess the practical importance of this, since much of the generalised illness associated with *E. coli* infection is due to the release of endotoxin. Two extremely important factors in treatment are -

(i) Drugs to control shock, e.g. flunixin (Finadyne, Schering Plough Animal Health Ltd.)

(ii) Fluid therapy.

Fluids are essential and if the cow will not drink voluntarily, "calf scour" type electrolyte solutions can be easily administered orally, using a watering can. In the USA a commonly used piece of equipment is a protected stomach tube attached to a stirrup pump. This will deliver 50 litres of fluid quite quickly and easily and is considered to improve the response in cases of toxaemia. Intravenous fluids may be given by drip or, more commonly, by pumping 5-10 litres at speed using a converted garden sprayer. Others have used a 200ml intravenous infusion of 7.0% w/v sodium chloride, to stimulate the cow to drink.

Frequent stripping is also an essential part of treatment. This removes toxins from the udder and also removes bacteria, thus reducing further toxin production. Ideally, affected cows should be hand-stripped every hour and the quarter massaged to remove as much debris as possible. Injections of oxytocin immediately prior to stripping causes milk ejection which helps to flush infected milk from deep within the gland.

2. Gangrenous mastitis

This is usually sudden in onset, and the cow is found by the herdsman to have a cold quarter and teat, and a bloody mastitic secretion, often mixed with gas. If casualty slaughter is possible, then this is the best option. Schedule 2 of the Meat Inspection Regulations states that animals with toxaemia are not suitable for human consumption. In the early stages disease may be localised to the quarter and provided that the cow is not pyrexic or showing other signs of generalised illness, then, on discussion with the OVS at the abattoir, slaughter might be considered an option. However, many cases show systemic changes and the decision is one of whether to cull or attempt treatment. If the latter route is selected, veterinary attention would include the use of both parenteral and intramammary antibiotics, with systemic antishock therapy, fluids and frequent stripping.

Clinical assessment of the cow needs to be very careful. A bruised udder can look very similar to the early stages of gangrenous mastitis, but recovers well without treatment and certainly does not need to be sent off! At the other extreme, when gangrene and sloughing becomes extensive, aggressive antibiotic therapy is needed against secondary infections, plus repeated flushing of the affected area with water or warm saline. Procedures for amputation of the quarter are described in the literature, but this is not commonly practised.

Infection by *Baccillus cereus* may initially appear similar. The teat and udder can appear 'bruised'. This type of infection can be readily distinguished by a 'port wine' appearance of the milk. The prognosis is usually very poor.

3. The hard, swollen, non-responsive quarter

This may be a consequence of an earlier obviously coliform infection, or may have been more insidious in onset. It is a frequent problem presented to the clinician. My own approach to such cases is is to start by taking a sample for bacteriology. While a major proportion are sterile, it is still surprising how many samples yield useful results, even if the cow has only just finished a course of intramammary therapy. Common organisms found include:

(i) **Yeasts**, often Prototheca species. These respond moderately well to iodine therapy. One regime is to infuse 100mls of a mixture of 1.8g iodine crystals in 2.0 litres of liquid paraffin plus 23mls ether, into the quarter, leave for 30-40 minutes and then strip out. If it is left longer than this, severe damage to the quarter may result. Concurrent oral or intravenous iodine therapy may also be administered. Yoghurt infusions into the udder have also been suggested as a treatment for yeast mastitis.

(ii) ***Streptococcus uberis*** This appears to be becoming an increasingly common cause of both chronic indurating mastitis and recurrent cases. Therapy with high levels of parenteral and intramammary penicillin for 5-6 days produces reasonable results, but even then, some cases remain refractory to treatment.

(iii) **No growth** This is the most common finding and may occur for a variety of reasons including:

- the original cause of the mastitis is no longer present because it has been eliminated naturally or has not survived the time lag from sampling to the laboratory or
- residual antibiotics have inhibited bacterial growth, or
- antibacterial substances from the mastitic udder have inhibited growth or
- the organism is present in such low numbers that it is not detectable by standard procedures.

A proportion of these hard quarters respond well to cortisone or other anti-inflammatory therapy. This can be given parenterally or infused into the quarter, for example with 50ml of sterile water. Concurrent antibiotic therapy should be administered.

Cows which fail to respond should not necessarily be culled. It is surprising how many will eventually come back into milk. Even if the quarter has been unproductive for several months, a proportion can be stimulated back into milk production, simply by regular stripping. Many come back into full production at the next lactation.

4. A chronic recurrent case

This presents as slight induration of the quarter, repeated bouts of clots in mastitic milk and a moderate to high cell count. It is probably the most difficult case to deal with and with current high animal values there are frequent requests for suitable treatment regimes.

A Bacteriology. Samples should be taken using strict aseptic precautions and a larger volume of milk used for the initial culture, since the causative organism may only be present in low numbers. Although many samples produce "no growth", useful results can be obtained and include:

(i) *Staphylococcus aureus* (= coagulase positive staphylococci). This remains an individual cow problem in many herds and is the most difficult organism to treat. Response to treatment during lactation is particularly poor (3) and even dry cow therapy may be disappointing. This is especially the case in older cows and in cows which have had repeated attacks of mastitis previously (4). Causes of poor responsiveness have been listed as

- antibiotic resistance. Many strains of staphylococci are beta-lactamase producers and therefore resistant to penicillin and some of the semi-synthetic penicillins such as ampicillin and amoxycillin. To date, there have been no reports of resistance to cloxacillin nor to cephalosporins and hence products such as Orbenin Extra (SmithKline Beecham Ltd.) and Cepravin (Pitman Moore Ltd.) should be effective, provided that they are able to reach the organism. An alternative is to use a beta-lactamase inhibitor such as clavulanic acid (e.g. Synulox, SmithKline Beecham Ltd.).

- fibrosis within the udder, leading to failure of the antibiotic to reach the organism.

- intra-cellular survival of staphylococci within macrophages. Most antibiotics penetrate cells very poorly. The exception is enrofloxacin (Baytril, Bayer Ltd.) and there are a few anecdotal reports of intramammary infusion producing a good response. Enrofloxacin is primarily effective against gram negative organisms. The lincosaminide antibiotic pirlimycin also penetrates cells and has been shown to be effective against experimentally induced staphylococcal mastitis (5).

(ii) *Streptococcus uberis* As with the "hard quarter", it is interesting that an increasing proportion of chronic recurrent mastitis cases are found to be infected with *S. uberis*. Treatment with high doses of penicillin is not consistently successful.

(iii) *E. coli*. Although typically an environmental pathogen, cases of chronic recurrent coliform mastitis have been reported (6) and are becoming increasingly common. As with *S. uberis*, response to therapy during lactation may be disappointing. Insufficient data are available to be able to know whether these "chronic recurrent" strains of E. coli can persist through the dry period to cause an acute toxaemic mastitis at the start of the next lactation. However, if this is the case, then perhaps there is a need to review current thoughts on dry cow therapy, where the choice of antibiotic is currently made on the sensitivity patterns of gram positive organisms only. In addition, if *S. uberis* and *E. coli* are both becoming increasingly common causes of "chronic" mastitis, then perhaps predipping will become a more important mastitis control procedure.

B Dry off early. Quarters showing a consistently high cell count are highly likely to be infected and hence are a risk both to other quarters in the same cow and to other cows. Standard texts suggest that contamination of a liner by an infected cow may spread bacteria to the next 6-8 cows to be milked. If treatment has been unsuccessful therefore, it would be beneficial to the other quarters, and the remaining cows in the herd, if the infected quarter was dried off. Dry cow therapy should be administered when the remaining three quarters are dried off.

C Aggressive antibiotic therapy. Because dry cow therapy is generally more effective than lactation treatment (3), aggressive therapy is often administered just prior to drying off. Suggested regimes include tubing with a lactation intramammary daily for five days and then a dry cow tube on the 6th day (A. Biggs, personal communication), or repeating dry cow therapy two weeks after drying off. The antibiotic chosen should be effective against staphylococci and this will then cover streptococci. Costs can be minimised by carrying out bacteriology, or even a CMT test on all four quarters and then targeting the aggressive therapy against affected quarters only. If three or four quarters are affected, then parenteral therapy may also be indicated. The use of products such as tilmicosin (Micotil, Elanco Ltd.), which are specifically concentrated in the udder and which persist at significant concentrations for 2-3 weeks following a single dose, could be indicated. Trial work on their efficacy is needed.

Conclusions

Although the search continues, there is no wonder-cure for mastitis. Treatment remains based on antibiotic therapy, with supportive treatments such as anti-inflammatories, fluids and management practices such as frequent stripping. However, perhaps the overall disappointing response to treatment does have some benefits. The fact that mastitis is difficult to treat must mean that extra efforts are directed at udder hygiene and general preventive measures, with resulting benefits on milk quality and animal welfare.

References

1. HILL A W, SHEARS A L & HIBBITT K G (1978) Research in Veterinary Science 25 89-93.

2. CRAVEN N (1991) Proceedings of the British Mastitis Conference p.42

3. WILSON C D (1980) In **NIRD Techical Bulletin 4** Mastitis Control & Herd Management, p113.

4. MEANEY W (1991) Proceedings of the British Cattle Veterinary Association, Kinsale. p211 .

5. YANCEY R J, KINNEY M L & FORD C W (1985) Journal of Antimicrobial Chemotherapy 15 219-232.

6. HILL A W & SHEARS A L (1979) The Veterinary Record 105 299-301.

CELL COUNTS - THE LOWER THE BETTER!

CELL COUNTS - THE LOWER, THE BETTER

Andrew Sanders, Elton Farm, Elton, Ludlow, Shropshire.

Background

Elton Farm is situated, despite the address, in deepest rural Herefordshire right in the centre of the Welsh Marches. Our land runs to 350 acres, part owned and part rented; it lies between 450 ft and 650 ft, edged by forestry (wonderful fly country!).

Although we are Herefordshire born and bred, we only moved to Elton seven years ago, after ten years in West Wales. There we established the herd and learnt some salutary lessons on how to cope with adverse weather conditions and slurry.

Some have described our start at Elton as difficult, I would have said 'traumatic' e.g. a Class 4 water supply, a milk licence that was teetering on the edge of being withdrawn, a 14th Century house that no one had lived in for 10 years, no forage in store, a slurry pit that had not been emptied for three years, acres of permanent set aside and buildings full of the detritus from years of neglect.

Our first job was to get past the MAFF officials regarding the milk licence in order to buy the farm. I can still remember our first meeting as prospective buyers, with them, the agents and the owners in the dairy, and the man from the ministry twitching his nose at the smell of Jeyes fluid that had been used to clean it up for the occasion!

Our first undertaking on purchasing the farm was to disconnect the existing water supply, there being no mains supply for miles. This led us to three months of hard labour, carrying water for cows from the nearest village, four miles away, in black orange juice containers. We have since put in a bore hole and have a Class 1 water supply.

The highlights of the early months, having arrived in the middle of August, included finding a tractor under the existing heap of rotting silage; burning 7,000 bales of rotten hay, left in the field after the baler; making our new crop of silage in November; explaining to the dairy hygiene lady on one of her frequent visits that we had no loo she could use, pointing her in the direction of the nearest bush etc. etc. Fortunately things have changed for the better.

Back to the present day. The mixed farm has 140 acres of arable, including set aside and maize. We are milking about 125 pedigree Holstein/Friesian cows with a similar number of heifers, reared to calve at 2 years, with all year calving. Present yields are around 7,000 litres at 4.34% butterfat and 3.3% protein, calving interval 364 days, MOC £1220 at present and margin per litre 17.8p.

The cows are cubicle housed and fed a complete diet in winter; set stocked with supplementary fresh cut grass and silage in the summer months. They are milked through a Fullwood 6/12 Herringbone Parlour erected in 1968, but updated in 1986 following a dynamic test by ADAS. The parlour has the original stalls, vacuum pump and pulsation, but we increased the size of the vacuum line and put in a new vacuum regulator, installed new 6 gallon Alfa jars (how we wish we had put in 7 gallon jars) at eye level and added-vision

type Alfa clusters with checkball liners but no ACR's. It takes around 2 hours to milk 100 plus cows.

Most of the field work is done by contractors, so the labour force consists of my wife, an HND Sandwich student from Harper Adams and me. My father helps out in his spare time. Milking is shared between us, my wife 50%, me 25% and the student 25%. Mastitis levels do not seem to fluctuate between different personnel.

Cell counts

Having set the scene, we get to cell counts. My first recollection of cell counts in the 1970's was a figure of 280,000 which reduced progressively to 140,000 for the 60 cows we had when we left Wales. Since then cell counts have fallen continuously to a low of 41 in February this year for 120 cows, the three month geometric mean at the moment of writing is 62 and the Total Bacterial Count is 3.

People used to ask (including our then new vet), and still do, ' Aren't you worried about very low cell counts?'

'No, the lower the better' is my stock reply.

So why am I happy with low cell counts? I have five main reasons.

1. Low numbers of clinical cases of mastitis.

Only 14 cows have had a case in the past 12 months including 2 cases of coliform mastitis. Apart from financial losses incurred by a clinical case, there are the practical problems at milking time, discarding milk, washing out units, taking samples, treating infected quarters, worrying about cross infection, damaged quarters and the risk of putting antibiotics into the bulk tank. Mastitis causes such disruption to the regular milking routine so I am fortunate to avoid much of this.

2. No cows culled for mastitis.

It would be terrible to have to sell your favourite high yielder because she had a high cell count. Replacement costs are the third highest variable cost after concentrates and forage.

3. Longevity.

Cows last longer if they do not get mastitis. Cows in our herd achieve peak lactation yield in lactations six or seven. Maximum lifetime profitability depends on cows reaching this age.

4. Attraction for buyers.

We find that the buyers of pedigree stock are very interested in the animal's cell count. Most young cows have figures below 50.

5. Having high quality milk to sell.

This is going to become increasingly important with the demise of the MMB. I would imagine that it is likely that after an initial honeymoon period, prices will rise to a level where demand for milk starts to tail off. It is at this point that those dairy companies left will become ever more discriminating about the hygienic quality of the milk they purchase.

Our management regime

The milking routine

This comprises

- an annual milking machine testing
- changing liners regularly
- hot and copious amounts of circulation cleaning water
- clear identification of all cows
- units rinsed with hypochlorite after milking cows with cell counts over 200,000
- mastitis detectors in the milk line, we do not strip the fore milk, so this is a good early warning of mastitis, especially for inexperienced students
- dry wiping of cows before milking. After 15 years of dry wiping, I can still remember chapped teats and tubs of udder cream when we were washing and drying.
- care taken not to over milk. We do not pull on units, they are taken off immediately the milk stops flowing
- instant teat dipping of whole teat, using fresh dip every milking
- careful recording of cows with clinical mastitis cases
- ensuring that cows stand for about half an hour after milking doing something useful such as eating on their way to the cubicles or field (the process of drinking, eating and cogitating on their life's experiences takes about half an hour)

Cow comfort and cleanliness

In winter, management is based around the cubicles. The shed we inherited when moving to Elton had been up for 20 years, with cubicles measuring 3' 9" x 7' , earth beds and a concrete heelstone. Clearly the chopped straw that we had used in Wales was not an option in this type of cubicle. Initially we made these more acceptable by removing the bottom rail and replacing with rope. In order to make the bed more comfortable we used large volumes of straw which led to very clean cows (far cleaner than in the summertime) and a much reduced incidence of foot problems due to passageways strewn with straw. Cows spent a lot more time lying down. The cows have got progressively bigger. The shed was designed for 550 kg British Friesians, but we now keep 750 kg Holstein/Friesians. Modifications have been made to the existing shed and recently we have built another cubicle building with Dutch comfort cubicles 8' x 4'2", (the concrete heelstone is 12" high and 18" wide), earth bed and a neck rail. These appear to be even more comfortable and the cows are even cleaner. Suggestions have been made that the heelstone is too high from the point of view of the weight transfer and damage to feet when cows are standing half in and half out, but

from the cubicle hygiene perspective they are much drier and cleaner. Most cows stand in with all four feet. The Dutch comfort design seems to ensure all sizes of cows position themselves very accurately when lying down, whilst giving them plenty of room to lie comfortably and get up easily.

The secret of our whole cubicle system is the quantity and regularity of re-bedding. We use 20 to 30 conventional bales of straw each day, depending on the type, 7 days a week. If this is not done every day, then one can tell in the milking parlour next morning. Wads of straw are thrown into the front of the cubicles, the cows eat some of it and some is dragged to the back of the cubicle when cows reverse out. Thus fresh straw is regularly spread where the cows position their udders. Furthermore, straw is also dragged out into the passage, next to the heelstone, keeping cows' feet clean. Usually we do not have to wash a single cow in the winter. If we do, it will only be due to falling down or bulling. Our system depends on a relatively cheap supply of straw and we are fortunate to live in an area of mixed farming where straw is readily available. We have tried 'Cubisan' and hydrated lime in the past, but gave up the practice because we were worried about burning the cows' teats, preferring instead to rely on a dry bed and fresh clean straw.

In my view it is the regularity of bedding that is most important, not the quantity.

From my experiences in West Wales, where we had no fines-concrete cubicle beds and two precious bales of chopped straw daily for 60 cows, this keeps cows clean and cell counts down. For cow comfort, rubber mats could be an advantage, but they cannot compare with our 6" mattresses of straw.

Animal health and welfare

1. Closed herd

After years of breeding for a high genetic herd, who wants somebody else's problem cases? I think that it would be very risky to introduce new strains of bacteria with cows from different herds.

2. NMR individual cell count testing

This is equally important to low cell count as to high cell count producers, as it highlights genetic influences on cases. Ours are limited to three families. Beware - one case of undetected clinical mastitis on milk recording day can distort the cell count. Back in June, one cow was responsible for 39% of our herd cell count.

3. Dry cow therapy

We use tubes that last the longest, re-tubing cows that are dry for more than eight weeks with shorter acting antibiotics, 4 weeks before calving. We even tube in-calf heifers 8 weeks before calving in the summertime to help protect against summer mastitis, thus avoiding a case in the last seven years, despite all the woodland and tremendous fly populations.

4. Foot trimming

We make regular use of preventative foot trimming, trying to eradicate lame cows which tread on teats in the cubicles, are generally unfit, lie down too much, and are more prone to mastitis.

Stress-free environment

In the winter the majority of cows tend to filter from the cubicles, to the collecting yard and to the parlour on their own. After milking there is a race to the feed passage stocked with fresh silage, then they wander off to the cubicles through the furthest gate (the nearest one is closed for the duration of milking) where they lie down in freshly bedded and scraped cubicles.

The complete diet in the winter makes the cows quiet, contented and relaxed with no fighting, bullying or hungry irate cows (have you ever seen one?) hanging about for food.

The cows in summer behave as a herd and follow a strict timetable. They always appear, (who didn't shut that gate?) half an hour to an hour before milking, depending on the weather and take themselves out again crocodile fashion, picking their way carefully down the track, sated after a feed of silage or fresh cut grass. In very wet conditions (cast your mind back to this spring) we are quite willing to house them, avoiding the need to have them standing in muddy gateways bawling, standing under hedges, writing off tracks and having to be driven out through the mud, udders flapping against their legs.

Looking at my own experiences over the last 17 years, it was concentrating on all the routines to do with the milking parlour, dry cow therapy etc. that enabled us to get the herd below the 200,000 cell count. It was not until we paid more attention to cubicle hygiene and comfort that we saw cell counts approaching the 100,000 mark. Having reached that milestone, we tried to reduce stress factors, which we maintain has allowed the cell count to drop to it present level. Even now, any periods of stress, particularly bad weather during the summer and walking long distances to grazing will push the count back to around 100,000.

As a practical farmer, I think that by necessity, people have had to concentrate on the wrong end of the job, 'shutting the stable door, after the horse has bolted'. If more time and money was spent on cow hygiene, cow comfort and welfare, the bulk of the costs and problems associated with mastitis could be avoided.

Finally, if one wants a really low cell count of 50,000 or less, I think one has to become a cow psychologist.

MASTITIS AND YOUR FUTURE MILK PRICE

MASTITIS, MILK QUALITY AND MILK PRICE

Mike Hurst, Central Testing, Milk Marketing Board, Thames Ditton, Surrey KT7 0EL

This paper provides an overview of the reasons for and purpose of somatic cell count payment schemes and examines the current position. Details of schemes and data are taken from those being operated by the Milk Marketing Board (MMB) in England and Wales although very similar schemes are in use in Scotland and Northern Ireland.

The question that you might ask is, "Why bother?" Although we have had payment or penalty schemes for compositional quality, hygienic quality and antibiotics for many years, why did we not have a cell count payment scheme until October 1991? Clearly the requirements of the market place and recent EC legislation are factors but it is worth considering the views of the major players in a little more detail.

The farmer

It is probably fair to say that cell count payment schemes have not been introduced because of a strong demand from individual farmers. Although the cost of mastitis to British dairy farmers has been estimated at £70 million is it not an individual farmer's right to run his business as he wants to, even if it is inefficient? Are not dairy farmers already penalised quite adequately through loss of yield, lower compositional results and hygiene scheme penalties if they do not adopt good mastitis control? The impact for instance of poor hygienic quality on payment as a consequence of mastitis can be seen from Table 1 which details the current deductions for milk with poor Total Bacterial Count (TBC) results.

Band	2-Month Geometric Mean TBC/ml	Milk Price Deduction (ppl)
A	20,000 and less	NIL
B	20,001 - 100,000	0.25
C	Above 100,000	2.00 (If first occasion in previous 6 months.) 6.00 (If 2.00 in previous 6 months.) 10.00 (If 6.00 in previous 6 months.)

Table 1. Hygienic Quality Payment Scheme penalties in England and Wales.

As much as 10p per litre can be deducted if results are consistently above 100,000 colonies/ml. - a very severe dent in the milk cheque but a real situation that some dairy farmers find themselves in.

However, most if not quite all dairy farmers appreciate the value of a scheme which provides routine cell count information to aid herd management and mastitis control. Many dairy

farmers take a great deal of pride in their achievement of top quality milk and it is also apparent that many of them are becoming a little irritated that a significant proportion of their number is letting the side down and tarnishing a good image. Figure 1 clearly illustrates this point and shows that there are a significant number of dairy farmers with ground to make up.

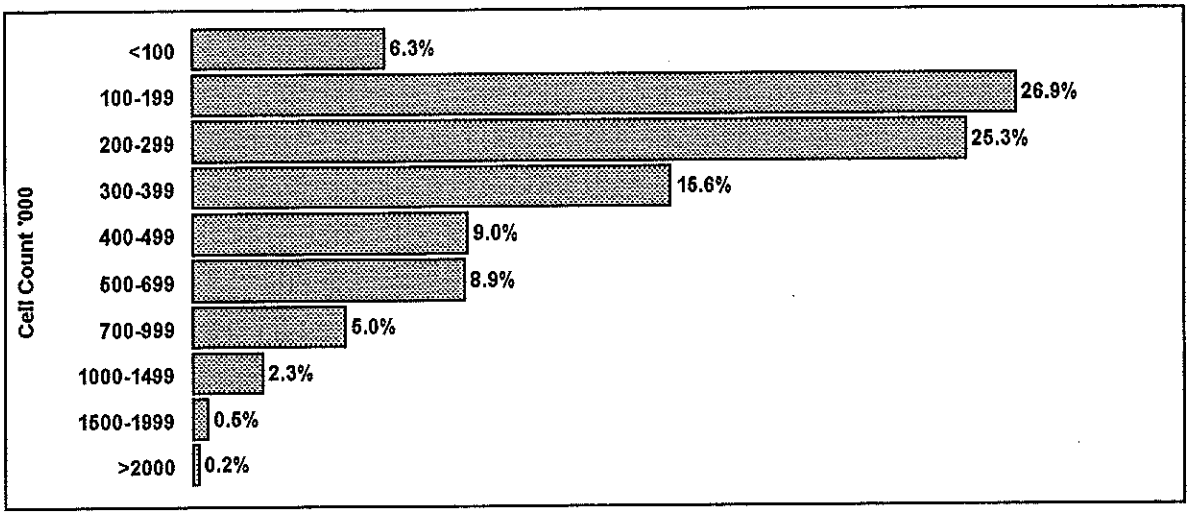

Figure 1. The distribution of ex-farm supplies by cell count in England and Wales in April 1993.

The purchaser

For simplicity the purchaser in this definition includes the processor and the retailer of the finished product to the consumer.

It is generally accepted that mastitic milk can affect the processing properties of milk and influence product quality, although there continues to be a debate about the point at which this occurs. Various problems have been reported:

- Development of "off flavours" in liquid milk and milk products.
- Poor heat stability affecting condensed and powdered milks.
- Renneting and clotting affected in cheese and yoghurt manufacture.
- Casein reduction affecting cheese yield.
- Sediment in homogenised milk.
- Higher levels of bacteria, some of which can be pathogenic and obviously undesirable although they should be eliminated by pasteurisation.

Clearly for simple commercial reasons the processor would prefer to have none of these problems and he might feel it was worth his while to purchase his raw milk only from a source with a low cell count to avoid them.

The retailers, and in particular the larger multiples yield great purchasing power. They can and do demand that ingredients in products made for them are of high specification. This is not necessarily driven by the effect that the quality of an ingredient, (in this case milk), may have

on the product but also by the need to maintain a wholesome image and possibly gain an edge over a competitor.

The consumer

Many of the retailer's requirements are of course what they perceive their customer, the consumer, wants to buy. The consumer, usually wants a wholesome product made from pure and natural ingredients and in this case this surely means milk from a healthy cow. What would the average consumer think if they were told that a teaspoonful of milk included about 2 million dead body cells from the inside of a cow's udder? It is too easy to think that the consumer does not know or concern themselves about these things but to ignore what the consumer might perceive about the product that provides one's livelihood is a very dangerous line to adopt in this day and age.

The regulator

After a considerable period of discussion the EC Commission has now produced its Health and Hygiene Directive (92/46/EEC) "laying down the health rules for the production and placing on the market of raw milk, heat treated milk and milk products". In consultation with other interested parties within the dairy industry, the Ministry of Agriculture (MAFF) is currently establishing an interpretation of these "rules" that will then be enshrined within our own domestic legislation. In simpler terms there will be standards set for raw milk that will have to be met if it is to continue to be sold. Table 2 details the standards that have been laid down for somatic cell count.

	Three Month Geometric Mean Somatic Cell Count	
Raw cows' milk for the production of heat-treated drinking milk, fermented milk, junket, jellied or flavoured milk and cream.	from 1.1.1994 ≤ 400,000 cells per ml	
Raw cows' milk for the manufacture of other milk based products.	from 1.1.1994 ≤ 500,000 cells per ml	from 1.1.1998 ≤ 400,000 cells per ml

Table 2. Somatic cell count standards to be met for raw cow's milk.

The Directive and the standards come into force on 1 January 1994 when effectively milk for the liquid market must have a cell count of no more than 400,000 cells/ml and for manufacture of no more than 500,000 cells/ml based on a geometric mean over a period of 3 months. It should be noted that by 1 January 1998 all raw milk must meet the lower standard.

The Directive states that the standards are to be "met for the collection of raw milk from the production holding or for acceptance at treatment or processing establishments" and for a time it was thought that a strict interpretation of the standard would be used in the UK. From the data shown above in Figure 1 it can be seen that in April 1993, a good month for average cell count, a considerable number of individual ex-farm supplies (25.9%) would have failed to

meet the liquid milk level and a significant number (16.9%) would have been above the level set for manufacture, effectively making them unmarketable if the standards in Table 2 were applied at individual ex-farm supply level. Fortunately MAFF is proposing that for the time being they will assess cell count on the basis of the geometric average of the bulk intake into "treatment or processing establishments". MAFF has however, made it clear that they can only use this interpretation if efforts continue to be made by all sides to improve cell counts and it would seem likely that at some time in the future the standards will have to be applied at individual ex-farm supply level.

What can be achieved?

70 - 75% of dairy farmers would seem to have the matter in hand but a significant amount has to be done by the other 25% or so to ensure that they continue to have a market for their milk. It is not for this paper to suggest practically how this can be achieved but it is worth considering the role of a cell count payment scheme in the light of the foregoing observations.

The purpose of a quality payment scheme is basically twofold:

- To reward dairy farmers differentially according to the market value of their milk.
- To encourage improvements in the quality of raw milk to meet the demands of the marketplace either through bonus or penalty.

Payment schemes do seem to provide a crude but usually effective method of communication. It is often difficult to get the right message over to some farmers and penalties can be persuasive.

We know that dairy farmers are capable of responding to payment schemes. The improvement in TBC (Figure 2) was dramatic after the introduction of a hygienic quality payment scheme with bonuses and penalties in 1982 and the average has continued to decline. Similar improvements in the level of antibiotic contamination have been achieved over the same period despite successive increases in the sensitivity of the test and more frequent testing.

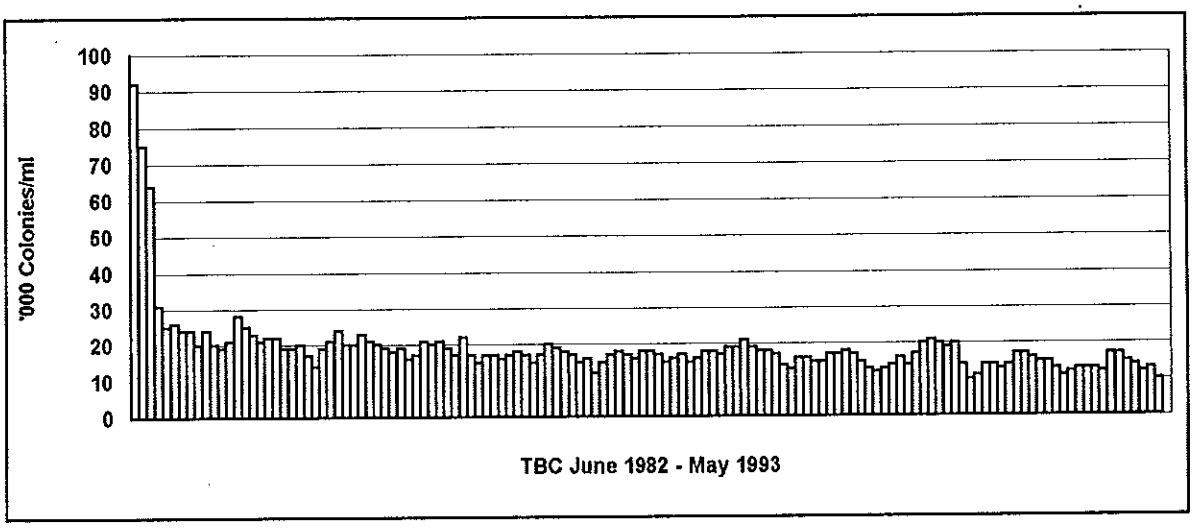

Figure 2. Weighted average TBC in England and Wales.

It has to be said however that the dramatic improvement in TBC seen at the start of the scheme has to be put in context compared with what possibly has to be achieved with cell counts. Before 1982 most dairy farmers had little knowledge of and were under no pressure to respond to TBC results. Virtually overnight they were exposed to weekly information about the bacterial quality of their milk and an intense campaign to clean up their act - milk from clean and healthy cows, milked through clean and well maintained plant. This was backed by a promise of bonuses and a threat of penalties. The majority of farmers were able to respond quite quickly and were rewarded for their efforts.

The situation with cell count is clearly more complex and although many of us hoped that the imposition of a payment scheme would bring about a significant improvement in cell counts we knew it would not be possible to turn things around as quickly as we did with TBC. The lack of a significant improvement since the introduction of the payment scheme is disappointing but to be fair to many it needs to be weighed against the fact that there have been other pressures on herd management in the last couple of years that have overridden some measures that farmers could have taken. Neither should we ignore the improvements that have been made in controlling mastitis over the last 20 years as can be seen in Figure 3.

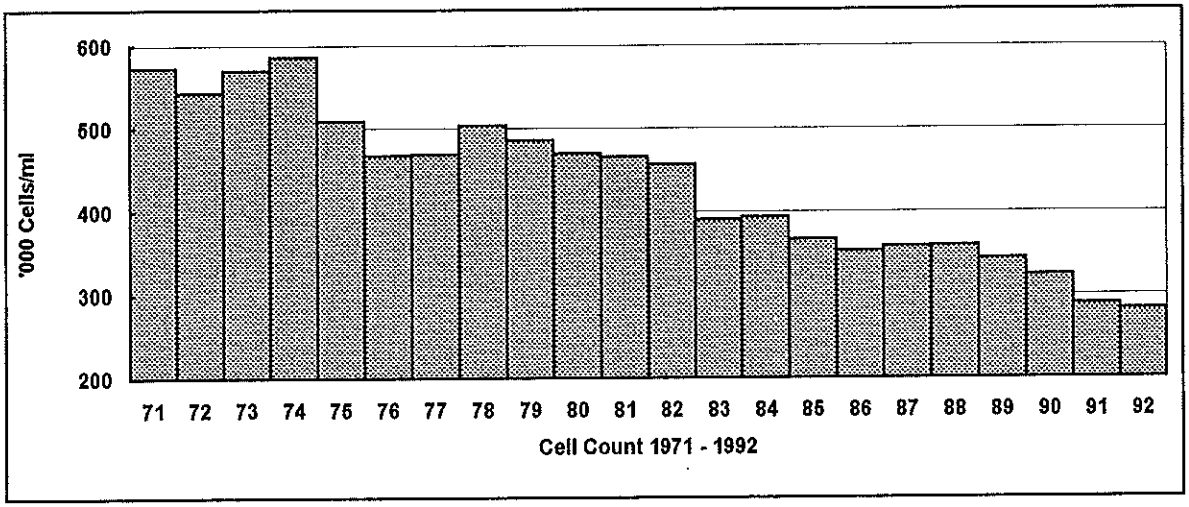

Figure 3. Annual mean cell counts in England and Wales.

Major reductions in cell count occurred in 1975/76 following an increase in the culling of the national herd and in 1983 after the introduction of the TBC payment scheme. The apparent improvement shown in 1991 should be treated with some caution as it was from this point that data began to be generated by Central Testing laboratories and there was a change in the type of instrument used for testing and the procedure adopted for calculating average results.

The cell count payment scheme in England and Wales was introduced in October 1991 after a twelve month trial period when it was becoming apparent that EC legislation would be introduced in one form or another and would impact on our domestic market. In April 1993 the scheme in England and Wales was modified to bring it into line with the EC legislation (Table 2) and at the same time the MMB decided to remove the bonus band, taking the view that as 70 - 75% of dairy farmers were capable of producing top band milk this should be considered the norm and receive the basic milk price and anything less should be penalised. Penalties were increased at the same time to provide some further encouragement to those

outside the top band to improve. Details of the cell count scheme from October 1991 and since the changes were introduced in April 1993 are shown in Table 3.

Band	3-Month Geometric Mean Cell Count/ml (From 1.10.1991)	Milk Price Adjustment (ppl) (From 1.10.1991)	3-Month Geometric Mean Cell Count/ml (From 1.4.1993)	Milk Price Adjustment (ppl) (From 1.4.1993)
1	400,000 and less	+0.2	400,000 and less	NIL
2	400,001 - 700,000	NIL	400,001 - 500,000	-0.5
3	700,001 - 1,000,000	-0.2	500,001 - 1,000,000	-1.0
4	Above 1,000,000	-0.4	Above 1,000,000	-2.0

Table 3. Cell Count Payment Scheme in England and Wales.

The changes introduced in April 1993 not only increased penalties but also had the effect of moving a significant proportion of dairy farmers from a neutral band 2 prior to the changes to a higher penalty band 3 afterwards. 13.9% of supplies were in band 3 in April 1993 compared with 5.1% in April 1992.

The marketplace

Come 1994, dairy farmers will be operating in an open marketplace and the quality of their milk will come under even greater scrutiny when they sell into that market place. A free market also demands that those with a better quality product should receive a better price. Dairy farmers will be competitive if they meet the required standards and present a good and proper image of their product - milk. Most do but some have ground to make up and a few have a lot of work to do or they will not survive.

In conclusion it is worth looking again at an observation made by James Booth at the 1992 Mastitis Conference. Quoting from the results of an International Dairy Federation survey he said. "Interestingly, 23% of herds in England and Wales have a cell count below 200,000 cells/ml, which represents very good control of mastitis, whereas only 14% of Danish herds have. However, 21% of herds are over 500,000 cells/ml compared to only 6% in Denmark, and none of the latter are over 700,000 cells/ml. This seems to be a reflection of the influence of the payment for milk in Denmark having been based partly on cell count for the past 20 years."

Acknowledgements

I am indebted to Frank Harding, Technical Director, Milk Marketing Board, England and Wales and James M Booth, Genus Animal Health both of whom have provided information and helped me unwittingly in other ways with this paper.

ABSTRACTS OF POSTERS

AUTOMATIC MILKING AND TEAT CLEANLINESS

TOBY MOTTRAM, CLIVE ALLEN and NEVILLE PRESCOTT, Silsoe Research Institute, Wrest Park, Silsoe, Bedford MK45 4HS

Automatic milking systems are being developed which will allow cows to be milked more frequently. Research is being conducted into methods of automatically inspecting and cleaning teats. The research is in its infancy and is concentrating on the engineering challenges of routinely deploying automatic machines to inspect and clean living tissue. The biological challenges will also need detailed research. Work on automatic milking offers the possibility of a more predictable teat preparation routine and this may ease the design of experiments to test the effects of hygiene treatments on mastitis.

THE USE OF INDIVIDUAL COW CELL COUNTS

ELIZABETH BERRY, Genus Animal Health, Cleeve House, Lower Wick, Worcester WR2 4NS

There has been an increased interest in bulk milk and individual cow cell counts with the introduction of a cell count payment scheme in England and Wales in October 1991. Individual cow cell counts are available from the Milk Marketing Board's National Milk Records (NMR) and from other laboratories. Approximately 7,000 herds now receive regular monthly individual cow cell counts through NMR.

Several factors other than intramammary infection can have an effect on the individual cow cell count eg stage of lactation, parity and milking interval. Samples taken in the first or last two weeks of lactation may have elevated counts due to normal lactational effects.

A count over 200 thousand cells/ml tends to indicate infection but at least three results for a lactation are needed for interpretation and follow-up action. The lactation average should not be used as this can be misleading. Either quarter cell counts or the California Mastitis Test (CMT) can be used to identify the infected quarters.

Bacteriological examination of milk samples from high cell count cows will reveal the type of bacteria involved and antibiotic sensitivity can be determined. This should always be carried out before making decisions on treatment, if clinical signs are not seen, as treatment of infection during lactation is uneconomical and often ineffective. This will enable control measures to be put into action in addition to those of the Five Point Plan and determine appropriate lactational and dry cow therapy. Such measures range from milking infected cows last or using a separate claw, to the segregation of cows infected with contagious bacteria. If environmental bacteria are isolated this will highlight the need for extra attention to housing and grazing management. Clinical mastitis records should also be used when making decisions on culling.

THE RELATIONSHIP BETWEEN REGULAR MILKING MACHINE TESTS AND BULK MILK CELL COUNTS

ALISTAIR McIVOR, Genus Animal Health, Cleeve House, Lower Wick, Worcester WR2 4NS

A survey was carried out to study the effect of regular milking machine testing on bulk milk cells counts. A random sample of 143 dairy herds was used for the survey and the annual mean cell count for 1992 was calculated for each herd. The geometric mean for all herds was 293 thousand cells/ml whilst the national average was 282 thousand cells/ml.

The data for the herds were divided into two sets: Group A consisted of 68 herds which were members of the Genus milking machine testing service, each receiving a regular milking machine test at least once a year. Group B contained 75 herds which were not members of the service, and most were unlikely to be receiving regular milking machine tests according to market research.

The geometric mean cell count of Group A was 250 thousand cells/ml and 56 (82%) had a mean cell count below the penalty level of 400 thousand cells/ml. Group B had a mean cell count of 339 thousand cells/ml, which was 36% higher than Group A. Forty eight herds were below 400 thousand cells/ml, representing 64% of the group, which was 18% less than Group A.

The difference in cell count between the two groups was statistically significant and indicated that there was a strong association between a regular milking machine test and a lower bulk milk cell count.

AN ANALYSIS OF MASTITIS BACTERIOLOGY ISOLATES FOR 1988-1992

TERRY GREEN, Genus Animal Health, Cleeve House, Lower Wick, Worcester WR2 4NS

Genus Animal Health operates a mastitis bacteriology service to identify the infecting bacteria present in milk samples from cows with clinical and subclinical mastitis. This poster summarises the isolates determined from these samples over the last five years and identifies the trends.

The average number of milk samples received per year was 5495, range 4912-6314. Submissions have increased steadily over the five year period. With the more common mastitis pathogens there has been a marked variation in frequency of isolation from year to year. The only consistent trend has been for the proportion of coliforms to increase steadily over the five year period from 6.4% to 35.1% of samples received. Conversely the frequency of isolation of *Streptococcus uberis* has dropped over the last two years from 8.3% to 1.6%, and of *Streptococcus agalactiae* from 6.3% to 1.1%.

The frequency of isolation of minor pathogens has remained relatively consistent over the five years. The number of samples which failed to yield an isolate has dropped steadily from 10.2% to 6.9% and the level of contaminated samples has fallen from 8.9% to 0.6% due to increased emphasis on the importance of pre-sampling teat preparation.

SUSPECTED ADVERSE REACTION SURVEILLANCE SCHEME

A. GRAY, Veterinary Medicine Directorate, Woodham Lane, New Haw, Addlestone, KT15 3NB

The definition of a Suspected Adverse Reaction:

"A reaction which is noxious and unintended and which occurs at doses normally used in animals. All reports are deemed suspected until proved otherwise".

Scope of the Scheme -

* The Scheme covers all species of animals, fish and bird.

* The Scheme covers all licensed veterinary pharmaceuticals, vaccines and antisera.

* The Scheme covers human operator suspected reactions.

* The Scheme covers the United Kingdom and contributes to the EC Pharmacovigilance (Suspected Adverse Reactions) Committee.

Objectives of Scheme -

* To collect, collate and analyse suspected adverse reaction voluntary reports and act accordingly.

* To prioritise veterinary medicinal products and identify areas of possible concern and identify solutions for action.

* To obtain, analyse and prioritise company product reports for action.

* To develop a regular quarterly report from the voluntary reports for the Veterinary Products Committee and the EC Pharmacovigilance Committee.

* To publicise the Scheme as widely as possible.

TEAT MANAGEMENT

MARTIN F.H. SHEARN, Institute for Animal Health, Compton, Newbury, Berkshire RG16 0NN

THE TEAT IS PROBABLY THE MOST ABUSED PART OF THE COW, ITS CONDITION IS PARAMOUNT TO EFFICIENT MILK PRODUCTION!

Generally little or no thought is given to teat condition until there is a problem either with mastitis or teat lesions. Yet twice or thrice a day a vacuum is attached and milk extracted under physical stress. The tissue expands and contracts this way and that. All year the teat is left to dangle in faeces and urine and in Winter there is also the problem of the cold winds between leaving the parlour and reaching the housing area. In Summer things are fine until the rain turns the tracks and gateways into a quagmire. The teat then gets a free mudbath.

How can MANAGEMENT reduce the effect of this continuous abuse? It should start outside the milking parlour. First the weather, obviously nothing can be done to change the weather, but the effect of inclement weather can be reduced. Do the cows have to plough through the muddy gateways and along deep muddy tracks twice a day? Can the collecting and dispersal yards be enclosed sufficiently to cut down the cold chilling wind and driving rain?

Many of the cubicle housing systems look fine when new, but within a few years become dark, damp, dirty places which any self respecting cow would avoid. Cubicle divisions break and are not repaired. Rarely is the cubicle base and surrounds properly cleaned out during the housed period. And is the area cleaned thoroughly during the Summer ready for the next housing period? What thought is given to the passageways? Even if the housing system is good, are the cows expected to walk through ankle deep in slurry, to reach the feed area and the parlour?

In the parlour - well if the cows' teats are dirty on entering the parlour then the previous paragraph needs re-reading. Hygiene in the parlour starts outside! Are the teats clean when the milking machine is attached to the teats? If not why not! Is the machine well maintained and the liners in good condition, smooth and not like sandpaper? Do the teats get a quick spray As the cow leaves the parlour to be forgotten until the next milking. Why not dip clean teats thoroughly in a good disinfectant with sufficient emollient to keep them in a very good condition. Why DIP when spraying is quicker? Imagine walking quickly through a misty shower, or jumping into a swimming pool, which one gets YOU wet ALL OVER?

THE COST OF MASTITIS: SEPARATE ESTIMATES FOR MILD, SEVERE AND FATAL CASES

R.J. ESSLEMONT, I. SPINCER and E.J. PASMAN, Department of Agriculture, University of Reading, Earley Gate, Reading RG6 2AT

Whilst mastitis is generally recognised as the single most important health problem in dairy cows, it is not known how much a mastitis case costs the farmer. In cost estimates made previously, differences in severity of the cases were not taken into account. Because severity of mastitis cases in a farm can differ, estimates of the cost of a mastitis case were made for three different levels: mild, severe and fatal.

The treatment costs for a mild case of mastitis are based on the cost of 15 minutes of the herdsman's time at £4 per hour and the use of 4 tubes of antibiotics priced at 90 pence per tube. Besides these costs a lowered yield and losses due to discarded milk are taken into account. The direct costs are £60.46. All the costs of a mild case of mastitis can be considered as direct costs; the total is therefore £60.46.

A severe case requires more tubes, a visit by the veterinarian and systemic antibiotics. The cost of treatment and the veterinarian's visit is estimated at £88.60, making the direct costs to be £185.16 and the total cost is £357.26.

The fatal case of mastitis is estimated to require 3 visits from the veterinarian and requires drugs to the value of £30, but the cow is lost and with her a substantial margin. The direct costs are put at £249 and total costs are £2248.

Mastitis is a complex series of diseases, both clinical and subclinical, and it is therefore difficult to estimate economic losses. Often there is a wide variation within a herd between subclinical and clinical levels; for example, a herd might have a high subclinical level, but a low clinical level. The prevalence of the three types of clinical mastitis is assumed to be 70% mild cases, 29% severe and 1% fatal. The average cost is therefore £168. The average direct cost of a case of mastitis is £98.50 (98.50 = 60.46 x 0.7 + 185.16 x 0.29 + 249.00 x 0.01).

MAJOR HISTOCOMPATIBILITY COMPLEX CLASS II EXPRESSION BY BOVINE MAMMARY GLAND EPITHELIAL CELLS

J.L. FITZPATRICK*, C. VILELA**, P.W. BLAND and C.R. STOKES, Department of Veterinary Medicine, University of Bristol School of Veterinary Science, Langford House, Bristol BS18 7DU; *Department of Veterinary Medicine, University of Glasgow Veterinary School, Bearsden, Glasgow G61 1QH; **Faculdade de Medicina Veterinaria, Universidade Technica de Lisboa, 1199 Lisboa Codex, Portugal.

Epithelial cells line the teat duct, teat sinus, gland cistern and the ductular system throughout the mammary gland. The epithelium acts as a barrier to bacterial infection and may induce and regulate local mammary gland immune responses. Epithelial cells were isolated by brushing the surface of the gland cistern of cows following slaughter. The cells were grown *in vitro* as a monolayer and were confirmed as epithelial in origin by immunofluorescence with an anti-cytokeratin monoclonal antibody. Major Histocompatibility Complex (MHC) class II molecules are expressed on the surface of specialised antigen presenting cells and are essential for the induction of immune responses. MHC class II molecules were expressed on epithelial monolayers following incubation with recombinant bovine interferon gamma and mitogen-induced lymphokine supernatants. This shows that mammary gland epithelium can express MHC class II molecules and as interferon gamma is normally produced during the host response to infection, suggests that MHC class II molecules are expressed following bacterial invasion of the mammary gland. These results also suggest a role for the epithelium in the induction of host responses.

HOUSING FOR HEALTH

JOHN BAINES, ADAS Dairy Development Centre, Lawnswood, Leeds, and IAN OHNSTADT, ADAS, Dorchester.

The NIRD/CVL Five Point Plan has been generally effective in controlling bovine mastitis with the exceptions of infections caused by the "environmental" pathogens, *Streptococcus uberis* and *E. coli*. The incidence of mastitis caused by these pathogens is almost unchanged over the last thirty years.

Apart from disastrous effects on herd performance, environmental mastitis can have a serious effect on the marketability of milk. Control will require attention to wider aspects of production, especially both hygiene and animal welfare.

The incidence of environmental mastitis is affected by a number of factors. In addition to plant cleaning, a hygienic milking routine and the effectiveness, and cleanliness of cow housing are major features.

Many cubicle buildings were originally installed to accommodate smaller cows than those of the present as cow size has increased significantly with the introduction of Holstein blood stock. Few buildings have been adapted to compensate for the need for increased cubicle size and improved ventilation. Improving housing can reduce mastitis incidence and consequently lower the herd cell count. In the long term, it is likely that milk buyers will take a much greater interest in the standards of production on the farm.

ADAS have carried out an extensive programme of studies into design and management of cow cubicles. Research shows that dry beds reduce the risk of teat end contamination by environmental bacteria. Emphasis is placed on ensuring that the beds are sloped to enable free drainage, impervious and adequately bedded. The cubicle division itself is of vital importance in not hindering the cow whilst lying down or standing up. The dimensions of the cubicle are also important in ensuring comfort.

The new dairy housing facility at ADAS Bridgets is based on the most up-to-date recommendations arising from those studies. Great attention in the new building is placed on the texture of floor surfaces to optimise grip. Also a high level of planning has been involved in ensuring adequate ventilation.

TEAT DIP TO CONTROL *Corynebacterium bovis*.

J. ERIC HILLERTON, AFRC Institute for Animal Health, Compton, Newbury, Berkshire, RG16 0NN

There is increasing interest in the rate of infection by the minor pathogens, *Corynebacterium bovis* and coagulase negative staphylococci. They, very occasionally, cause mild clinical mastitis, can cause a slightly elevated cell count but probably do not contribute significantly to bulk milk TBC.

On the experimental farm at Compton, where teat disinfectant had been applied by dipping for a number of years, usually some 25-30% of quarters were infected with *C. bovis* at drying off. During a period of using an exit race sprayer to apply disinfectant the proportion of infected quarters increased to 55-60% within 6 months and this persisted. In early 1992 a series of persistent clinical cases, which could only be attributed to *C. bovis,* occurred. The exit race sprayer was removed and teat DIPPING resumed, with no other changes in control methods. The proportion of infected quarters at drying off was reduced to 20-22% but it took nine months to achieve the full effect.

The rate of infection by coagulase negative staphylococci was not influenced by the teat disinfection routine as these infections occur primarily in the dry period.

It appears that control of minor pathogens, which assume much greater significance when the major pathogens are being well controlled, requires continued application of the 5 Point Control Plan and that teats should be DIPPED in disinfectant.

CLINICAL MASTITIS AND STAGE OF LACTATION

J.ERIC HILLERTON, MARTIN F.H. SHEARN, Institute for Animal Health, Compton, Newbury, Berkshire RG16 0NN

FRANK H. DODD, Ashdene, Curridge, Newbury, Berkshire

In spite of the progress in controlling mastitis by the introduction of Dry Cow therapy (DCT), teat disinfection and improved machine milking clinical mastitis still occurs in all herds. By examining the pattern of occurrence of these infections we can discover important areas for research, and new management practices to limit mastitis further.

In the original experiments that developed teat disinfection and DCT the management routine was tested in 30 herds for 3 years. Of the 6200 cases of clinical mastitis that occurred during this trial **over 25% were in the first 5 weeks of lactation.** This higher probability of clinical mastitis in early lactation occurred with all pathogens, but was particularly marked with *Escherichia coli*.

The infections that cause clinical mastitis in early lactation **do not necessarily begin** in early lactation. In fact, if DCT is not used on all cows, most of the infections will either have persisted from the previous lactation, or be new **soon after drying off, the time of the highest rate of new infection.** However, DCT was used in the experimental herds and relatively few infections, mostly caused by *Staphylococcus aureus*, would have persisted through the dry period also new infections during the early dry period would have been infrequent. Therefore, in these herds, most clinical mastitis in early lactation resulted from new infections around the time of calving.

What can be done to reduce clinical mastitis in early lactation?

Unfortunately progress is unlikely through better methods of DCT or teat disinfection. In herds that have been using these for several years, most clinical mastitis is likely to be caused by *E. coli* or *Streptococcus uberis*. These 'environmental pathogens' do not colonise teat skin and are not controlled by teat disinfection. DCT will cure most *S. uberis* infections at drying off, but this pathogen is the most common cause of infection in the late dry period and over calving. DCT will not control *E. coli* infection because it is rare for *E. coli* infection to be present at drying off, or to be contracted in the dry period up to the time that cows begin to 'bag up' before calving.

There is no proven control for environmental mastitis, but the best advice is to take steps to reduce exposure to these pathogens. The **high levels of exposure come from faeces and contaminated bedding.**

Do not bed cows in the late dry period, or early lactation, in deep straw yards or sawdust/shavings. The best method in cubicles is to remove all bedding where udders touch the ground each day and renew bedding. Calving boxes should be cleared out completely each day. The levels of exposure to environmental pathogens will be very small when cows are on clean pasture.

Continue with DCT and teat disinfection to reduce levels of infection by the other major pathogens.

FURTHER COPIES OF THESE PROCEEDINGS CAN BE OBTAINED FROM:

CIBA AGRICULTURE
ANIMAL HEALTH DEPARTMENT
WHITTLESFORD
CAMBRIDGE
CB2 4QT

PRICE: £15 PER COPY
INCLUDING UK POSTAGE