BRITISH MASTITIS CONFERENCE 1992

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GENUS ANIMAL HEALTH
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I N D E X

INTRODUCTION

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The fifth British Mastitis Conference has again been based on the feed-back of delegates to the previous Conference and it is hoped that the subjects chosen will go a long way towards meeting those requests.

There has always been much interest in the development of natural resistance to mastitis and speakers will give the latest information on this topic.

Cell counts have been discussed on many occasions in the past but a new aspect will be to give a review of the current position in other countries. Practical examples of how cell count information can be used and how to tackle a mastitis problem on the farm will offer valuable advice for problem herds.

As at previous conferences, there will be an update on current research and a reintroduction of the poster display covering a wide range of subjects.

This Conference has become an important forum for giving up to date information on mastitis control and for delegates to discuss both at the plenary sessions and amongst themselves the many areas of interest. The organisers hope that you will continue to feed-back your views on the content of the Conference and subjects of interest so that future Conferences provide delegates with some of the subjects on which further information is required.

NATURAL RESISTANCE TO MASTITIS

PROSPECTS FOR BREEDING FOR IMPROVED RESISTANCE TO MASTITIS

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Introduction

Mastitis represents a major cost to the dairy industry, through lowered milk production, loss of income because of withheld milk, costs associated with prevention and treatment, and as a major reason for culling cows. While a variety of preventative measures are currently in use, breeding has often been presented as a strategy for achieving permanent gains in resistance. This paper will attempt to review the available information on the potential and feasibility of breeding for increased resistance to mastitis. It will also attempt to describe steps in other countries to present information on the genetic merit of bulls for mastitis resistance or somatic cell count (SCC), and consider the importance that should be given to these traits in commercial breeding programmes.

Genetic variation for resistance to mastitis

Despite the economic importance of mastitis, there are comparatively few good heritability estimates for either incidence or related measures of susceptibility. The most comprehensive set of estimates are from Scandinavian studies, where mastitis has been recorded as part of their routine health and fertility monitoring programmes for up to 20 years. Heritability estimates from these programmes, and recent studies elsewhere, are summarised in Table 1. This list is not comprehensive (see 1), but focuses on studies providing heritability estimates for both mastitis and SCC.

While higher estimates have been reported (2), mastitis incidence has generally been found to have a low heritability, usually less than five per cent. This low estimate is not surprising. Firstly, the condition can be brought about in various ways, and different pathogens may be implicated. Animals are usually not challenged in a standard manner. Finally, where the outcome is recorded in an all-or-none manner, heritability will be reduced, especially at low incidences (3).

These studies show that there is genetic variation in susceptibility, so that genetic gains in resistance are possible. But because heritability estimates are so low, ranking of cows is relatively inaccurate, and progeny testing of sires is the only feasible route to genetic improvement. Even here, progeny groups of several hundred daughters are needed to obtain accuracies comparable with those achieved for production traits with many fewer daughters. Nevertheless, in Scandinavia, with progeny groups of 150-200 daughters (4), there are routine genetic assessments of bulls for mastitis.

Table 1: Heritability estimates for measures of clinical mastitis and Somatic Cell Counts or Scores

Record	Heritability of		Reference
	Mastitis	SCC	
All parities	0.12	0.20	15
1st parity	0.01	na	16
1st parity	0.03	na	17
2nd parity	0.02	na	17
1st parity	0.01	0.08	7
1st parity	0.03	0.12	18
2nd parity	0.05	na	18
1st parity	na	0.11	10

Selection on somatic cell counts

Somatic cell counts or scores (5) have commonly been proposed as an alternative to direct measures of mastitis. This is a more feasible option, now that we have routine testing of cell counts for individual cows in many countries. While information on both clinical mastitis and SCC can be used to predict genetic merit for the former, the benefits of using SCC are especially persuasive where there is no routine recording of mastitis.

For indirect selection to be effective, the indirect measure of susceptibility must be heritable, hopefully more heritable than the character (mastitis) that we want to improve, and the two should be highly correlated. Average heritability estimates for SCC are higher (see Table 1), and they are also higher in the three studies which presented heritability estimates for both clinical mastitis and SCC. An overall estimate of 10 per cent seems a reasonable assumption. There has been conflicting evidence on the relative heritability estimates for individual test samplings or foe lactational averages (6 & 7). The genetic correlation between SCC results for different lactations appears to be high (8).

Critical to the success of a breeding programme based on SCC or Somatic Cell Scores (SCS) is the genetic correlation between SCC or SCS, and clinical mastitis. The general consensus is that this correlation is high, of the order of 0.6 or more (see 1 for review), although much lower estimates have also been reported (9). This is an area where further estimates would obviously be helpful.

Ranking animals on susceptibility

Where there is widespread use of AI, the most obvious path to genetic progress is through the ranking of the available bulls on the character we wish to improve. In this section I will consider sire evaluation procedures used in Sweden, as an example of the Scandinavian countries, where there is routine disease recording, and recent proposals for ranking sires on somatic cell counts in the USA. The latter situation provides a model for what might be achieved in the UK and other countries where there is no routine recording of mastitis.

In Sweden breeding values (BVs) are routinely predicted for both mastitis and somatic cell count for bulls of both the Swedish Red and White (SRB) and Swedish Friesian (SLB) breeds, based on records of their daughters. Swedish proofs are assessed on the performance of an expected 150-200 daughters, and bulls must have information on 70 daughters for their proofs to be published.

The information on mastitis incidence in Sweden comes primarily from the treatment of clinical cases by veterinarians, although information from the AI technician or farmer (reasons for culling cows) can also be used. Cows are recorded as having or not having mastitis between 10 days before calving to 150 days after first calving, a period when 75 per cent of all cases during first lactation occur. Focusing on first lactation information avoids bias due to culling of cows, and enables breeding values of sires to be calculated as quickly as possible. SCC data is now also collected over this same period.

In their assessments BV are predicted for both mastitis and SCC, while at the same time correcting for year/herd effects, and month and age of calving. Heritability estimates assumed for mastitis and SCC are 2 and 8 per cent respectively. These two BVs are then combined, assuming a genetic correlation between the traits of 0.7. Combining the information is expected to improve the reliability of the proofs, and increase the variation in BV predictions for a group of bulls.

Table 2: Breeding Values for Unproven Swedish Bulls for mastitis traits - (from Ref. 4)

Breed	Number of bulls	Breeding Value for	Mean BV	Standard dev. of BVs
SRB	874	Improved mastitis	99.8	5.2
		Mastitis	100.0	4.4
SLB	520	Improved mastitis	100.9	6.4
		Mastitis	100.9	6.0

These points are well-illustrated in Table 2, which summarises information presented by Eriksson (4) for unproven bulls evaluated in January 1990. BV are presented for mastitis and for mastitis resistance using information on SCC as a correlated trait ("improved mastitis"). Proofs for all traits are expressed relative to a moving base of 100. The variation in genetic merit is measured as the observed standard deviation in BVs, with the great majority of bulls falling within two standard deviations around this mean. Thus we would expect to find bulls with BVs ranging from say 88 to 112. A change in BV for improved mastitis by one (eg from 100 to 101), is expected to decrease mastitis incidence in heifers by approximately 0.6 cows per hundred cows. Thus, taking our extreme bulls on BV, and assume a mean incidence of mastitis in heifers of 15 per cent, the expected difference in mastitis between the daughters of the best and worst bulls might be from as low as eight, up to 22 per cent. The benefit of including SCC in the predictions for "improved mastitis" is shown by the higher standard deviations in BV for this trait, relative to "mastitis".

In the United States SCC testing has been widely available for over 10 years. Currently over 3 million cows are tested, or approximately 75 per cent of cows milk recorded. Recently the Animal Improvement Programmes Laboratory (AIPL) has commenced a national study, to develop procedures to predict the genetic merit of both bulls and cows for SCC, with a view to providing routine genetic predictions within two years.

Boettcher et al (10) have recently published the results of analyses on lactation average values for SCC, for samples tested at five Dairy Records Processing Centres (DRPC). Among these were almost 250,000 samples from first lactation cows, sired by 778 bulls, and these heifer records were used for genetic analysis. While various conditions were imposed when screening the data, animals were only required to have one test result to be included, to ensure that animals culled early in the lactation were not excluded.

Of the environmental effects examined, month of calving, and age at calving and days in milk on last sample day were found to have significant effects on lactation average SCS. After preadjusting for these effects, the heritability estimate averaged 0.11, and varied between .08 and .16 for the different DRPCs. The authors also predicted the Transmitting Ability (ie predicted daughter performance) of the 778 bulls, which had an average of 245 daughters in the study. Future work at the AIPL will attempt to incorporate information on later lactations into these genetic assessments. Decisions also need to be taken as to how to present the information on genetic merit for SCS, whether as part of a continuous distribution, as categories (eg high, medium and low), or whether only extreme individuals will be identified.

In an associated article, Hansen (11) pointed out that progeny testing schemes in the US were normally geared to producing a proof for a young bull based on the performance of 60 daughters in different herds. For the production traits, with heritability values of the order of 40 per cent, the expected reliability of a proof based on 60 daughters would be over 80 per cent. However, the heritability of SCC is much lower, and so the reliability of a proof for SCC, based on these same daughters, will also be lower. As a consequence, for the same amount of information, we can be less confident about proofs for SCC, and more mistakes will be made in ranking bulls for SCC. Given the way progeny testing programmes are currently designed, it is going to take longer, perhaps only when second crop daughters are producing, before we get proofs for SCC which are of the normally accepted level of reliability.

The relative importance of breeding for mastitis resistance

The increased availability of information on the genetic merit of sires for mastitis or SCC will highlight the need to establish the importance of mastitis in breeding programmes, when there are many factors influencing profitability. This paper can only touch briefly on some of the major issues involved.

It is generally recognised that there are unfavourable genetic correlations between the production traits and resistance to mastitis. For example, the genetic correlation between milk yield and either SCC or mastitis appears to be of the order of 15 to 30 percent. Thus if we continue to select solely for increased yields, somatic cell counts and the incidence of clinical mastitis are expected to increase (5). If we attempt by breeding to limit the increase in susceptibility to mastitis, then the expected gains in milk production will be reduced.

We also have to throw into this equation information on linear type traits. A number of US studies (12) have shown genetic associations between udder and teat traits and SCC. This has prompted Rogers (13) to argue that type traits such as udder depth and teat placement should be included, along with SCC and production traits, in indexes designed to maximise profitability. While it would be very useful to also have information on genetic associations between the type traits and mastitis, as opposed to SCC, the need to include in a set of breeding objectives all traits influencing profitability is well taken, as is the need to use all available sources of information to predict genetic merit for those traits. These estimates of genetic merit can then be multiplied by the economic importance of the trait, and the results summed to predict an animal's genetic merit for overall economic value.

Discussion

In this paper I have attempted to describe breeding programmes elsewhere which incorporate information on mastitis and SCC. In this section I want to focus specifically on likely developments in these areas in the UK. All of the activities described are either already underway or planned for the immediate future.

Major changes are currently being made to genetic evaluation procedures used in the UK. From July 1992, assessments made by the National Animal Data Centre (NADC) on both bulls and cows for the production traits will be from an Animal Model form of BLUP analysis, which will make use of information on all relatives, as well as records on up to the fifth lactation. Similar procedures are used by the Holstein Friesian Society for linear type traits.

Plans for the NADC analyses include the use of test day records for the production traits, as well as the genetic evaluation of other traits, including somatic cell counts. While SCC information on individual cows only became available in the UK in 1990, already we find that these records are being requested on over 70 per cent of all milk recorded cows. Thus a substantial body of data is accumulating, which can be used to predict genetic merit under UK conditions. Accurate predictions will require the identification of environmental factors of importance, and hopefully will make use of test day records for SCC.

Breeding objectives for the UK dairy industry have to date received little attention. However, in 1990 Genus launched a Profit Index (PIN), for ranking bulls and cows for production traits. Research is currently underway, principally using data from the Langhill herd, to expand that index, to include either longevity or health and reproduction costs in the objective, and to make use of linear type information to predict genetic merit for such traits (14). Clearly, mastitis and SCC should fall within this overall framework.

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DEFENSE MECHANISMS OF THE UDDER

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Introduction

Mastitis is inflammation of the mammary gland. The sequence of events which must precede a case of clinical mastitis is bacterial invasion and despite frequent emptying of the gland at milking the bacteria must multiply to numbers which are capable of inducing an acute inflammatory response. Although the environment and husbandry methods associated with the dairy cow result in continual challenge of bacteria to the teat end clinical mastitis is a relatively rare event. Numerous defence mechanisms have been described which have the potential to interfere either individually or by collaboration with invasion and disease development (pathogenesis).

Although it can be demonstrated that prior infection or vaccination can increase resistance of the mammary gland to mastitis (1), this paper will only consider mechanisms of normal animals which have not been actively immunised. These systems can be divided into inflammation-independent and -dependent; the former comprising the intrinsic mechanisms of a normal healthy gland and the latter being inducible and centred around the mobilisation of polymorphonuclear leucocytes (PMN) from the peripheral circulation into the milk duct system. Inflammation is a complex process which depends on the interaction of many factors, some of which dependent on the immune system.

The efficient functioning of one or more of the intrinsic systems, obviates the need for the induction of the latter and hence the inflammation. The early signs of mastitis can be thought of as evidence of the mobilisation of a defence mechanism. If one of these systems does not eliminate the bacteria or at least restrict their growth, a much more severe form of mastitis ensues which may cause long term or permanent damage to mammary tissue and systemic reactions which can be fatal.

Intrinsic defence mechanisms

Teat duct

This 1.0 cm long link between the sinus of the gland and the environment is not a simple tube, but a tight orifice with deep longitudinal invaginations lined with keratin. It acts as the primary barrier to infection which involves chemical and physical mechanisms.

Although electron microscopic studies reveal a mesh like organisation of the keratin which may impede the progress of micro-organisms (2) the major physical barrier of the teat may be dependent on the effective closure of the teat canal after milking when the gland is most vulnerable to infection. The nature of duct closure remains unresolved. It has been suggested that the teat duct has a poorly defined muscular sphincter giving an active closure in a spiral fashion (3), although Nickerson and Pankey (4) suggest that the musculature follows the longitudinal ridges of the canal. There is a plethora of often conflicting information regarding the relationship between teat patency in the inter-milking period, ease of milking and the rate

of new intramammary infections. McDonald (5) reported that teat ducts from quarters susceptible to infection had a larger diameter than resistant quarters, whereas no relationship was found between duct length and susceptibility (6). Temporary increase in the patency of the duct by reaming increases susceptibility to infection, and loss of integrity of the duct by physical damage such as a cut almost invariably results in intramammary infection. For a more comprehensive review see Craven and Williams (7).

It has been suggested that constituents of keratin such as fatty acids and basic proteins with known *in vitro* antimicrobial properties may contribute to the effectiveness of the teat canal as a barrier to infection. Their importance *in vivo* is not proven; the data relating to fatty acids is conflicting (8 & 9), and although basic proteins are antimicrobial *in vitro* (10) and bind to bacteria within the teat duct (11), whole keratin lacks antimicrobial activity *in vitro* (12). It is clear that the effect of the dynamic situation within the teat canal is difficult to assess, but it is worth noting that teat canal colonisation by Gram +ve cocci is relatively common (13). It appears that the defensive mechanism of the teat can be attributed largely to its physical properties.

Non-specific humoral systems

Once bacteria have entered the teat and lactiferous sinuses, mastitis is not a certainty; there are several antimicrobial systems which have been shown to exist in milk drawn from the udder. Unfortunately, their efficacy within the udder is less clear.

Lactoferrin

Lactoferrin (LF) is an iron binding protein which *in vitro* can produce bacteriostasis by removing the iron necessary for bacterial growth. Under certain conditions LF can combine with antibody to be bactericidal (14). Any inhibitory activity of LF is lost in the lactating gland, not only because it is present in low concentrations, but also because the high citrate concentrations in milk interfere with the iron binding. It is unlikely therefore to be effective as an antimicrobial system in the lactating cow, but in the non-lactating udder, where levels of LF are much higher and citrate lower, there is evidence that the system may have a direct defensive role. The resistant of the dry udder to *Escherichia coli* infections has been associated with LF (15).

Lactoperoxidase/thiocyanate

All milk contains the enzyme lactoperoxidase (LP) which in the presence of thiocyanate (SCN) and hydrogen peroxide (H_2O_2) can produce a short-lived highly oxidative system which is bacteriostatic for Gram +ve and bactericidal for Gram -ve bacteria (16). The levels of SCN in milk depends on the feeding regime of the animal, being particularly high when the diet contains legumes and brassicas. The H_2O_2 is either generated by the bacteria growing within the gland or cellular or enzymic constituents of milk. Gram -ve organisms produce little or no H_2O_2 and there is no evidence to suggest that the system is of any value in the udder against these organisms. There is evidence that it may partially protect the lactating mammary gland from *Streptococcus uberis* infections (17). However, the levels of oxygen in the milk of a healthy mammary gland is low, and reduced even further by bacterial growth (18); this may restrict the formation of peroxide within the udder and so limit the value of this potential defence mechanism *in vivo*.

The enzyme zanthine oxidase, found in the squamous epithelium and keratin of the teat duct (19), can, in the presence of purines, generate H_2O_2. It has been suggested that this system may locally generate H_2O_2 for the LP system, so providing another antimicrobial system within the teat canal. Oxygen availability may also limit the activity of the system.

Complement

The haemolytic tests designed to measure complement (C) activity in serum are too insensitive to measure activity in normal milk (20). However a more sensitive microassay (21) detected very low levels in milk throughout lactation, even though its activity is usually masked by anti-complementary factors. There is, however, circumstantial evidence which suggests that C has a significant protective role in the udder.

Low numbers of serum-resistant strains of *E. coli* introduced into the udder almost invariably grow and induce mastitis, whereas comparable numbers of serum-sensitive organisms fail to produce disease (22). This implies that on entering the gland, serum-sensitive organisms are killed by the alternate C pathway. However, *in vitro* tests fail to support this, since serum-sensitive strains of *E.coli* grow in milk drawn from the udder. Nevertheless, in a study by Sanchez-Carlo (23), all 184 strains of *E.coli* isolated from cases of bovine mastitis were found to be serum-resistant. It is possible that *in vivo* a very mild inflammatory response is induced following infection, and this results in a slight increase in serum derived C in the milk. Although C cannot be directly proven to be a defence mechanism in the udder, even as an inducible system, it is clear that organisms susceptible to C do not grow.

Immunoglobulins

Varying concentrations of all classes of immunoglobulin can be found in milk throughout lactation. They are dramatically elevated in colostrum. There is no evidence to suggest that in the normal udder immunoglobulin is an independent defence system, but in the role as an opsonin for the uptake of bacteria by PMN, they are a vital element in what is perhaps the most important single defence mechanism, (see below).

Milk cells

The proportion of cell types making up the low numbers ($<150,000$/ml) in normal milk remains a topic of debate. Paape (24) suggested that up to 26% were polymorphonuclear leucocytes (PMN), with the remainder equally divided between macrophages and lymphocytes. However, many groups believe M and L to be the predominant cell types. Discrepancies in results may be as a consequence of animal variation. Macrophages and lymphocytes do not have a primary defensive role, but both are mediators or initiators of other vital defence mechanisms, and will be considered below. The phagocytic PMN (see below), is an important element of a defence system, but the numbers in normal milk would be insufficient for the system to function effectively.

Inducible defence mechanisms

Following bacterial infection of the udder, and failure of the existing (intrinsic) defence systems to inhibit growth, an inflammatory response is initiated. This results in recruitment of PMN from the blood, with phagocytosis and killing of opsonised bacteria. Although this system often limits a bacterial infection of the udder to a mild or sub-clinical disease, there are occasions when it fails and severe, peracute mastitis ensues.

The inflammatory response and PMN recruitment

The existence of bacteria or their toxins within the udder causes the generation of inflammatory mediators which lead to increased perfusion of blood to the mammary tissue, local attachment of PMN to capillary walls or endothelia (margination) and an opening of the junctions of the capillary walls in an area near to the original stimulus. Factors produced within the udder act as an attractant and this results in the migration of PMN and leakage of plasma into the tissue of the udder. It is likely that the first PMN to arrive also release factors which promote or amplify the process. The PMN accumulate in high numbers in the sub-epithelial connective tissue and finally pass into the lumen of the gland, where the bacteria are growing, by penetrating between intact cells or passing through areas of damaged epithelium. Apart from the complex mediators of inflammation generated by the interaction of bacteria with components of the normal mammary gland, chemotactic or attractive factors for PMN are also produced. Certain cleavage products of C, particularly C5a are potent chemotaxins in many species, including cattle (25); but it is likely that C levels are too low in normal milk to initiates a response. Once inflammation has started serum-derived C may potentiate the mobilisation of PMN. However cells present in the normal udder may be the major source of chemotactic factors. Macrophages, which represents the predominate cell type in normal milk and dry secretion, interact with bacteria or their products, and rapidly release chemotactic factors. Many substances have been identified, and are referred to as interleukins or cytokines (For a review, see 26). Whatever initiates the mechanism, once migration has started, release of lysosomal granules will undoubtedly sustain the process (27).

Opsonisation and Phagocytosis

After arrival at the site of infection the PMN must phagocytose (ingest) bacteria and internalise them within a phagosome. To do this they must be activated by the Fc region of an immunoglobulin molecule or a fragment of a C molecule. This requires bacteria to be coated with opsonin; either an immunoglobulin molecule attached to the bacteria by its Fab region, so exposing its Fc portion, or a suitable C fragment. On contact between an opsonised bacteria and a PMN, attachment takes place and phagocytosis is stimulated.

In decomplemented non-immune serum, milk and colostrum, the major opsonins for phagocytosis of mammary gland pathogens by PMN are IgM and to a lesser extent IgG2 (28 & 29). All samples of milk are opsonic for staphylococci, non-encapsulated *E. coli* and some strains of *S. uberis*. Thus for a large number of mastitis causing organisms, opsonin levels in milk are not limiting. It should be emphasised that the importance of opsonin levels in normal milk is questionable, since inflammation and the recruitment of PMN from the peripheral circulation also allows serum leakage into the milk. This soon elevates IgM in

milk to levels which can opsonise the capsulate *E. coli*, however it is likely that some strains of *S. uberis* remain resistant to phagocytosis and present a totally different problem (30).

The immunoglobulins which function as opsonins are all produced by the animals immune system in response to antigen. The reason why "normal" milk contains opsonins is the continual exposure of the cow to bacteria. In the case of encapsulated *E. coli*, the levels of antibody in milk appear to be simply not high enough.

Microbicidal activity of PMN

Following phagocytosis, the environment within a phagosome soon becomes very hostile for microorganisms. Many different antimicrobial system have been identified, with different bacterial species showing different susceptibilities to them. They can essentially be divided into (a) oxygen dependent systems which rely on a respiratory burst and the generation of short lived products of reduced oxygen, which in the presence of halides and myeloperoxidase produces a potent killing system and (b) oxygen independent systems relying on the release of factors such as lactoferrin, cationic proteins and proteases into the phagosome. As mentioned earlier, oxygen levels in milk which are naturally low, are reduced even further by bacterial growth, and this may impair the activity of oxygen dependent systems. It has been suggested that the ingestion of casein by mammary PMN compromises their subsequent ability to kill bacteria (31). It is likely that these factors restrict their microbicidal activity, but for many infections of the mammary gland PMN are an important defence mechanism.

This short review gives an indication of the variety of potential defence mechanism of the udder. It is by no means exhaustive, and has no more than touched on the wealth of detailed information available regarding the intricacies of how each system functions. Many of the systems have, however, been dissected and studied *in vitro*, and it is not always wise to assume the same biological activity in the environment of the udder.

The best documented, and perhaps most important defence mechanism of the udder is the inflammatory response which culminates in the killing of bacteria by PMN. Rapid recruitment can reduce *E. coli* mastitis to a self-curing disease, which experimentally can be shown to be visually undetectable (32). Failure or delay in PMN recruitment, as can occur in newly calved cows, may have dire consequences (33). Although inflammation (mastitis) is not apparent, the animal may succumb to the direct and indirect action of toxins which are produced by the unrestricted growth of bacteria. The reason for this failure of PMN mobilisation in early lactation continues to elude researchers. The value of PMN in controlling *Staphylococcus aureus* mastitis was well demonstrated by Schalm et al (34), who showed that chronic staphylococcal mastitis could be converted to a fatal gangrenous form by inducing systemic leucopenia.

As was pointed out by Anderson (35), it is unfortunate that the indicators of a vigorous host response are also regarded as the symptoms of the disease.

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USING CELL COUNTS

CELL COUNTS AROUND THE WORLD

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Early in 1992 the International Dairy Federation (IDF) issued one of its regular questionnaires to 32 member countries requesting mastitis cell count data for the two previous years. Twenty-five countries replied to questionnaire, albeit four of them to state that they had no national statistics available. The replies of the other 21 countries provide the basis for this paper.

Cell count data

Due to the many different ways in which mastitis cell counts have been analysed in the past, it is often difficult to find comparable data. However, probably because cell counts are now used widely as a measure of milk quality, the presentation is becoming more uniform. Nevertheless, differences remain, so that caution must be exercised in attempting comparisons. There are also large differences in the structure of dairy farming in different countries. Within the 21 countries the following differences were observed:

Number of dairy herds: a range from 1,380 state herds in Hungary to 249,200 herds in West Germany.

Herd size: a range from 10 cows in Austria to 296 cows in the kibbutz herds in Israel. The median herd size was 39 cows. Proportion of herds being regularly cell counted: a range from 10% in Russia to 100% in 12 countries.

Number of cell counts per herd per year: a range from 5 in Russia to 52 in England and Wales. Most had 12 or 24 counts, and the median was 13.

Mean cell counts: calculated as arithmetic in 10 countries, geometric in 7, both in 2, not stated in 1 and not available in 1. Some were selective about the herds included, such as only the herds contributing to co-operatives in Finland or only the herd-book registered herds in Israel. Others were selective on the cell counts, such as Northern Ireland which included only the lowest 3 of the 4 counts in the month. Two were estimates, Russia at 300-600 thousand and USA at 300-400 thousand cells/ml, and these figures have not been included in the comparisons.

Cell count instruments: now very little variation, as all countries used the Fossomatic, exclusively in all but five countries.

National cell counts

In responding to the IDF questionnaire, different parts of the UK replied separately so the data relate to England and Wales, Scotland, and Northern Ireland. They have not been combined, partly because of the selective counts for Northern Ireland noted above, and partly because England and Wales used the geometric mean in order to provide a comparison with previous years. For the purposes of this paper, the arithmetic mean from the central testing laboratories has been used, as this is a milk quality parameter and more comparable with the rest of the UK.

With all the many reservations noted, Table 1 presents the comparison of national cell counts grouped into EC and non-EC countries.

Table 1: National mean cell counts (1991)

		Count (000/ml)	Mean*
EC:	West Germany	230	G
	Northern Ireland	235	A
	England & Wales	288	A
	Denmark	295	G
	Belgium	301	G
	Netherlands	301	G
	Scotland	314	A
Non-EC:	Switzerland	114	G
	Norway	204	A
	Sweden	233	G
	Finland	247	A
	Austria	260	A
	Japan (1990)	260	A
	Canada	280	A
	New Zealand	298	A
	Australia	335	A
	Israel	398	A
	Hungary	433	A
	Czechoslovakia	450	G
	South Africa	465	A

* A = Arithmetic, G = Geometric

Most countries were able to provide the distribution of their herds according to their annual mean cell count. In view of the use of 400 thousand cells/ml as the threshold in Step 2 of the EC Health and Hygiene Regulations, admittedly as the geometric mean over three months, the available data are presented at this level in Table 2.

Table 2: Percentage of herds with annual mean cell count below 400 thousand cells/ml (1991)

EC:	West Germany	84
	Denmark	81
	Netherlands	80
	Northern Ireland	80
	Scotland	79
	France (1990)	72
	Belgium	70
	England & Wales	67
Non-EC:	Norway	97
	Switzerland	95
	Sweden	94
	Finland	89
	Canada	80
	Russia	57
	South Africa	56
	Israel	53
	Hungary	52

Tables 1 and 2 provide broadly similar ranking of countries. The Scandinavian countries, Switzerland and West Germany have the lowest cell counts, and the UK as a whole is about average within the EC. Eastern European countries, Israel and South Africa have the highest cell counts.

It is possible of course that the countries replying to the questionnaire have lower cell counts than those unable to supply data, simply because of the improvement resulting from attention to mastitis control and/or payment on cell count. Three EC countries could not provide national cell count data, Greece, Italy and Spain, and three did not reply to the questionnaire, Ireland, Luxemburg and Portugal (not a member of the IDF). Of the remaining IDF member countries, Poland could not provide data, and Bulgaria, India, Iceland, Kenya and Kuwait did not reply to the questionnaire.

The mean cell counts of Denmark and England and Wales are similar in Table 1 but the percentage of herds below 400 thousand cells/ml is quite different in Table 2. Table 3 shows how this arises.

Table 3: Distribution of herds by annual average cell count (%)

Cell count(000/ml)	Denmark	England & Wales	Difference
< 100	1	2	+ 1
100- 199	13	21	+ 8
200- 299	37	26	-11
300- 399	30	18	-12
400- 499	12	11	- 1
500- 699	6	12	+ 6
700- 999	0	6	+ 6
> 1000	0	3	+ 3

Interestingly, 23% of herds in England and Wales have a cell count below 200 thousand cells/ml, which represents very good control of mastitis, whereas only 14% of Danish herds have. However, 21% of herds are over 500 thousand cells compared to only 6% in Denmark, and none of the latter are over 700 thousand 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.

A few countries have national cell count data for more than ten years. Table 4 presents this and the progress made over that period.

Table 4: National cell counts in 1980 and 1991

	Cell counts (000/ml)		
	1980	1991	Change (%)
England & Wales	469	288	-39
Northern Ireland	387	235	-39
Switzerland	171	114	-33
Denmark	390	295	-24
Austria	320	260	-19
Scotland	384	314	-18
Sweden	282	233	-17
Norway	236	204	-14

All these countries can show good progress in reducing cell counts over the last decade with, understandably, a tendency for the countries with the highest cell counts to have made the best progress. Sweden and England and Wales have data extending back to 1970; they reduced their national cell count by a further 17% and 18% respectively during that period.

It might be expected that a reduction in national average cell count indicates a reduction in the prevalence of subclinical mastitis infection. However, when a cell count payment scheme is in operation, this is not always the case as the high cell count milk from infected cows may be withheld from the bulk supply to try to ensure the premium is received. As cell count payment schemes and single cow cell counting spread, this situation is likely to increase, so

that cell counts may cease to be more than a very approximate reflection of herd infection levels.

Due to the cost of carrying out national mastitis surveys, few recent data are available. Austria reduced the prevalence of subclinical mastitis from 27% in 1980 to 25% in 1987, a 7% reduction, and Norway reduced from 36.4% to 36.0%, a 1% reduction. This covered more than half of the period when, as noted in Table 4, their national cell counts were being reduced by 19% and 14% respectively, and seems to indicate that the trend in the national cell count had ceased to be a true reflection of the national subclinical mastitis situation.

Payment on cell count

Of the 21 countries replying to the cell count payment question in the IDF survey, ten paid all their farmers on cell count in 1991, seven paid some, and four paid none, although two of these had plans to start payment schemes. The lowest and highest cell count categories for payment are shown in Table 5, ranked within EC and non-EC countries according to the lowest cell count category.

Table 5: Cell count payment categories (cell counts in thousands/ml)

		Lowest category	Highest category	Farmers paid
EC:	France	200	750	All
	Northern Ireland	200	1,000	All
	Scotland	250	600	All
	Denmark	400	750	All
	England & Wales	400	1,000	All
	Netherlands	400	500	All
	Belgium	500	750	All
	West Germany	500	500	All
Non-EC:	USA	100	300	Some
	Canada	200	750	Some
	Norway	250	750	All
	Finland	250	500	All
	Japan	300	1,000	Some
	Austria	350	750	All
	Switzerland	350	350	All
	Hungary	400	1,000	Some
	Sweden	-	500	Some
	Australia	-	750	Some
	South Africa	-	750	Some

Table 5 demonstrates the very wide range of cell counts used for payment purposes. Even within the UK there is considerable variation. Aesthetically and from a mastitis control point of view there would seem to be some virtue in paying for milk below 200 thousand cells/ml, although this can be difficult to justify from a product value standpoint unless it is used for

a premium milk product. Many American factories pay a bonus down to 100 thousand cells/ml.

The upper cell count limit ranges from 300 thousand up to one million cells/ml. A good case can be made for the selection of 500 thousand cells/ml as the upper limit on both a mastitis control and a milk quality basis. Certainly one million cells/ml seems far too high, and has probably been selected only as a first step in improving milk quality.

There seems to be little relationship between the cell count payment categories and the national mean cell counts in Table 1. However, no information was obtained on the fundamental question of how long the cell count payment schemes had been operating. Suffice to say that the countries with no payment schemes, Czechoslovakia, Israel, New Zealand, and Russia, had some of the highest national cell counts.

Single cow cell counts

The final question in the IDF survey related to the use of single cow cell counts. All 21 countries replying carried out single cow cell counts on a proportion of their herds, as shown in Table 6, which includes the three UK replies separately.

Table 6: Herds having single cow cell counts (%)

EC:	Denmark	69
	West Germany	42
	France	35
	Netherlands	35
	England & Wales	25
	Northern Ireland	21
	Belgium	20
	Scotland	20
Non-EC:	Norway	78
	Sweden	64
	New Zealand	55
	Finland	54
	Hungary	54
	Israel	51
	Canada	46
	Austria	30
	USA	23
	Japan	22
	Australia	20
	Russia	20
	Switzerland	8
	South Africa	4
	Czechoslovakia	2

On average approximately one-third of the cows in the countries replying to this questionnaire were cell counted individually. Although there was a greater range in the non-EC countries, the means were similar (33% in EC, 35% in non-EC) and the medians identical (30%). Again there seemed to be virtually no relationship between usage and the national mean cell count data in Table 1, except that the high uptake in the Scandinavian countries was noteworthy.

Most countries carried out these tests either 10 or 12 times a year, although the range was from one to 15 times. Three countries included the cost in the fee for milk recording. Otherwise the count per test ranged from 8 pence to about 50 pence, although most were in the range 10-16 pence and the median was 14 pence.

Information on the uses of single cow cell counts was requested and 11 possibilities were given. The results from the 20 countries plus the three separate UK replies are given in Table 7.

Table 7: Purpose of single cow cell counts

	No. countries
To improve FARMERS' awareness of mastitis	23
To identify COWS with mastitis problems	23
To investigate HERDS with mastitis problems	20
To identify HERDS with mastitis problems	19
To identify COWS for bacteriological examination	18
To select COWS for culling	18
To identify COWS for treatment during dry period	15
To identify COWS for treatment during lactation	13
To assess the effectiveness of therapy	12
To assess a HERD's economic losses	9
For genetic studies or sire selection	7
Others:	5
To exclude high cell count cows from bulk milk	
To assess milking and management practices	
To estimate prevalence and incidence of subclinical mastitis	
To enable producers to meet EC standards	
For quality assessment	

Progress in England and Wales

In the context of this paper, a note on recent progress in reducing cell counts in England and Wales is relevant. Table 8 provides one measure of progress.

Table 8: Herds with annual average cell count below 400 thousand cells/ml at June

	% herds	Change over previous year
1987	63.7	-
1988	60.6	-3.1
1989	63.0	+2.4
1990	67.3	+4.3
1991	67.0	-0.3
1992	68.4	+1.4

The table indicates good progress in 1989 and 1990 but, surprisingly, virtually no change in 1991 which was the advisory period for the cell count payment scheme. Since payments started, in October 1991, there has been an improvement and the data over the first six months of 1992 show that the average rate of improvement has picked up to 2.2%.

Two related factors which are considered to have had some influence on the slow rate of improvement of cell counts are the need to fulfil milk quota, which resulted in some reduction in culling, and the 29,000 cattle slaughtered under BSE regulations in 1991, which on some farms will have affected the replacement situation and thus the freedom to cull cows for mastitis.

Despite the overall improvement in cell counts there is still a considerable seasonal variation. Table 9 shows the distribution of herds by cell count payment band, based on their geometric mean counts over three months, for the best (May) and worst (September) months during the last year.

Table 9: Distribution of herds according to cell count payment band

Band	% herds		
	May	September	Difference
1	73.5	64.2	-9.3
2	19.1	25.7	+6.6
3	4.9	6.9	+2.0
4	2.5	3.2	+0.7

The table shows that the proportion of herds receiving the cell count bonus was 9% lower in September, representing a direct financial loss to more than 2,000 farmers. Almost 1,000 more farmers suffered the cell count penalty in September, and incidentally the situation was similar in August. This emphasises the need to aim for a cell count below 300 thousand cells/ml, otherwise many herds will fall out of the bonus ban at certain times of the year.

There is considerable variation between regions of the country. Even in the best month only 65% of herds in South Wales achieved the bonus and this fell to 54% in the worst month. At the other extreme, the respective figures for the Yorkshire/Lancashire region were 78%

and 74%. In all months the proportion of milk achieving the cell count bonus throughout the country was approximately 10% higher than the proportion of herds, indicating that the larger herds were more likely to be receiving the bonus.

Conclusion

The evidence suggests that there has been a continuing improvement in mastitis cell counts around the world over the past two years, although there are no data available to indicate any corresponding improvement in mastitis infection over the same period.

The UK compares well to the 20 other countries replying to the IDF questionnaire, and there is some evidence that this reflects lower infection levels. The UK is also very competitive amongst the EC countries replying to the questionnaire, and these countries are possibly better than the EC average.

Most countries now include the cell count as a criterion for milk payment, and most have stricter standards for cell content than England and Wales, although not than Scotland and Northern Ireland.

Single cow cell counts are used in about one-third of all herds and their use is continuing to increase in most countries.

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HOW WE ARE USING COW CELL COUNTS

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The following paper describes the approach to a herd with a Somatic Cell Count (SCC) and Total Bacterial Count (TBC) problem with particular reference to the use of monthly individual cow cell counts. **(For more detail of the herd, see the previous paper of these Proceedings.)**

The TBC for the milk produced by the herd was invariably in Band B and occasionally in Band C and the 12 month rolling average for the bulk tank SCC had been 800,000/ml for the last 18 months (see Fig. 1).

The monthly cell counts were in fact approaching 1,000,000/ml in the months leading up to the introduction of the payment scheme in October 1991. It was this last fact that lead to the decision to approach the author in July 1991 to discuss ways of bringing down both bulk tank SCC and TBC with specific reference to bonus payments.

After an initial appraisal of the situation in July 1991, a number of interim management changes were discussed, but the two major points were to (a) identify cows in the herd with recurrent mastitis or very high SCC and cull them as soon as possible and (b) to start routine dry cow antibiotic therapy. Dry cow therapy was introduced in August 1991. More detailed use of bacteriology and individual cow cell counts was started in February 1992.

Interpreting the effect of culling on bulk milk SCC

All figures quoted in this paper referring to calving, drying-off and culling, are taken to be the month in which the bulk tank SCC would be effected. Any event after 25th of the month is considered to effect the next months bulk tank reading, (21st of the month for calvings to allow for the first 4 days of milk being discarded).

It was felt that before any detailed program could be devised, the obvious chronic, sub-clinically infected cows must be culled. These were identified on clinical grounds and/or persistently high monthly SCC from the National Milk Records (NMR) individual cow cell count service. A total of 6 cows were culled during July and August 1991, including 4 cows with high cell counts (of which 2 cows were three quartered). Despite these cullings the monthly bulk tank SCC **increased** from 800,000/ml to over 1,000,000/ml (see Fig. 1). From July to November 1991 a total of 16 cows were culled for various reasons. Although 10 of the 16 cows (62%) had SCC in excess of 1,000,000/ml the monthly bulk SCC for November 1991 reached 1,100,00/ml; the highest level for 7 months.

To the casual observer the policy of culling seemed to be having no beneficial effect. However, it must be remembered that although the bulk tank SCC is a single value, it is the net effect of many factors. Furthermore the situation is dynamic and when comparing monthly bulk tank SCC, one must consider not only the number of freshly calved cows entering the herd and the culled and dry cows leaving the herd, together with their individual SCC and yields, but also the total volume of the bulk tank and the change in milk yield of the animals which remained in the herd throughout the period. A complete and accurate

interpretation of what is happening in a herd requires complex calculations, but a consideration of numbers, yield and mean cell count of animals leaving and joining the herd can often indicate the general trends seen in bulk tank SCC on a month to month basis.

The two sets of consecutive bulk tank cell counts are considered in more detail below:-

Between **September** and **October** 1991, 5 animals calved, 14 were dried off and 4 culled, but the bulk tank cell count was unchanged with 1,000 in September and 980 in October (Fig. 1).

Between **November** and **December** 1991, 12 animals calved, 12 were dried off and 4 culled, but the bulk tank cell count fell dramatically from 1,100 in November to 600 in December (Fig. 1).

The calculations below, which assume constant yield for animals remaining in the herd, accurately predict the bulk tank cell counts changes observed between September and October and November and December.

Changes in the SCC reading of a bulk tank between consecutive months is described by the equation below with a factor to take into account the variation in total bulk tank volume.

Total number of cells in tank in Month A	+	Total cells added by fresh calved cows	-	Total cells removed by culls and DO cows	=	Total number of cells in tank in Month B
1	**+**	**2**	**-**	**2**	**=**	**1**

To calculate **1,** Volume in Bulk Tank X Bulk Tank SCC

2, Av. cell count of group X Av. yield of Group X number of cows in Group

These formulae can be used to calculate the changes between September and October '91:-

	September '91	October '91
Recorded Bulk Tank SCC	1,000	980
.. .. .. Yield (Kg)	740	650

Total cells in Bulk Tank for September '91				740,000
	Av. Group cell count	Yield	Number in group	
Calving Group (added	500	23.5	5	+55,700
Drying off Group (Removed)	860	7.0	14	-84,159
Culled Group (Removed)	1,000	13.6	14	-79,659
New total cell number in Bulk Tank in Oct. '91				660,341

Allowing for Bulk Tank Volume for October '91 of 650 Kg

Predicted SCC = 1,015
Actual SCC = 980

Similarly for November '91 to December '91.

	October '91	December '91
Recorded Bulk Tank SCC	1,100	600
.. .. .. Yield (Kg)	570	660

Total cells in Bulk Tank for Nov.'91				627,000
	Av. Group cell count	Yield	Number in Group	
Calving Group (Added)	316	23.5	12	+88,846
Drying Off Group (Removed)	900	8.0	12	-85,320
Cull Group (Removed)	3,500	14.2	4	-198,000
New total cell number in Bulk Tank for Dec. '91				431,726

Allowing for a new bulk tank Volume of 660 Kg.

Predicted SCC = 655
Actual SCC = 660

(NB. all values in these calculations are a factor of 10^3 too low.)

The results show that although there was a reduction in the total number of cells going into the bulk tank in October, the drop in total herd yield from the previous month eliminates any benefit in terms of cell numbers/ml, ie the Bulk Tank SCC. However in December, the overall effect is a much greater reduction in the total number of cells being put into the tank and this together with an increased yield from the new calvers combines to greatly reduce the Bulk Tank SCC.

Recording of bacteriology and treatments

A database of all samples, antibiotic sensitivities, treatments and culls was set up and this was linked to data from NMR (calving dates, individual cell counts etc.) using a relational database. The NMR data was updated by disc each month from February '92 onwards (HERDFILE).

Samples cultured in practice laboratory.

Code	Title		Description
S	Screen samples	-	Cow samples taken from all cows in the herd mainly during February '91 and March '91 (not clinically affected).
S LF	Quarter samples	-	Individual quarter samples from any cows with significant isolates on a screen sample, cows with a persistently high individual cell count or cows clinically identified as a problem (recurrent cases).
C RF	Clinical sample	-	Individual quarter sample from a clinical case of mastitis.
D LF	Pre-Drying off	-	Individual quarter sample from a problem cow just prior to sample drying-off. Used to select quarter treatment prior to drying-off and dry cow tubes.
P LF	Parturition sample	-	Cows quarter treated at drying-off and sampled at calving.

Treatments. Drug used and number of tubes recorded. Treatments selected on sensitivity testing

T RF	Treatment of clinical case.
D LF	Quarter treatment of problem quarter just prior to drying off.
DOFF	Routine dry cow therapy (DCT).
K	Cull cow. Date and reason for cull recorded. The following is an example of the data extracted from the various databases.

The following is an example of the data extracted from the various databases.

Cow 0178 calved at 20.7.91, lactation No. 2

Date	Sample	Bacteria	No.	Sig	Con	CCFeb	CCMar	CCApr
02.03.92	C RH	Staph C+	9			861	714	1312
02.03.92	T RH	SYNULOX	4					
09.03.92	S	Staph C+	30		Y	861	714	1312
12.03.92	T RH	ERYTHROCIN	4			861	714	1312
16.03.92	S	C. bovis	13			861	714	1312
26.03.92	S	Staph C+	40			861	714	1312
30.03.92	S LF	Staph C-	3	?		861	714	1312
30.03.92	S LH	Staph C-	4		Y			
30.03.92	S RF	Staph C-	2	?				
30.03.92	S RH	Staph C+	44	*				
28.04.92	D LF	C. bovis	20			861	714	1312
28.04.92	D LH	No growth						
28.04.92	D RF	No growth						
28.04.92	D RH	Staph C+	21	*				
30.04.92	D LF	STREPTOPEN MC	5			861	714	1312
30.04.92	D RH	STREPTOPEN MC	5					
01.05.92	DOFF	STREPTOPEN DC	4					
Calved 16.7.92								
10.08.92	P	Staph C+	10	Cell count July 198, August 66				

All clinical cases were recorded from January '92 onwards and sampling of all clinical cases was started at the end of February '92 just before the first whole herd screen. The type of information above will continue to be recorded for every cow in milk up to October '92. For simplicity the 2nd and 3rd most numerous bacteria, although recorded, have not been printed. The column headed "Sig" is set to ? for less than 3 bacteria isolated. This column contains a * for any quarter with a significant isolate. The column headed "Con" is set to Y if *Streptococcus faecalis* or *E. coli* is isolated (except for an *E. coli* clinical case).

The failure to isolate a coagulase positive Staphylococcus on 16.03.92 may be due to the sporadic excretion of quarters infected with this pathogen or the infection may have been temporarily suppressed by the antibiotic given a few days before. Ten days later on 26.03.92 a coagulase positive Staphylococcus was isolated again.

Approach to high cell count cows

The treatment of known infected quarters during the last week of lactation is an attempt to save high cell count cows from the cull list. In this practice BSE has put considerable strain on maintaining cow numbers in some herds. History has shown quite clearly that many cows once infected are unchanged by conventional DCT with long-acting preparations. By treating known infected quarters with quicker release milking cow products at the end of lactation, selected on the basis of sensitivity testing of samples taken in the last week of lactation, it is hoped to improve bacteriological cure rates. Now that bulk milk cell counts influence the value of the milk directly, perhaps a slightly different approach to "End of Lactation" therapy is needed. It is unlikely that during the current economic climate that drug companies will feel able to launch a new product for this purpose, thus at present at least we must make use of milking cow products already on the market.

Analysis of bacteriological samples results and associated individual cell counts

The correlation between the Monthly Bulk Milk cell counts and the Average Individual Cow cell counts was good (see Fig. 2) and it seemed that interpreting the effects of management changes on bulk milk cell count could be backed up by detailed analysis of individual cow cell counts. Any discrepancy in these two figures will be related to yield variation.

When all the cow screen samples were broken down by bacterial type, either coagulase +ve and -ve Staphylococci were isolated from over 60% of the samples (see Fig. 3). This was not surprising in view of the high cell count and that *Streptococcus agalactiae* had never been isolated from this herd. What was perhaps more surprising was that *Corynebacterium bovis* featured in 15% of cow screen samples. Even if drying-off and clinical samples were added to the data, *C. bovis* still featured in 13% of samples (Fig. 4).

Most of the cow **screen** samples were taken on 9.3.92 and so a breakdown of March '92 individual cow cell counts by bacteria was graphed (see Fig. 5). It is noticeable that although *C. bovis* featured in 15% of clinically normal cow screen samples, the 12 cows had an average cell count of 447. This is higher than any other bacteria isolated. Only the coagulase positive Staphylococci group of 28 cows had an average cow cell count in a similar range at 424.

C. bovis is considered to be a minor pathogen, even a commensal organism. It can be isolated from clinical cases and is known to produce a significant elevation of cell counts in a high proportion of cows in an affected herd. Although the organism is highly infectious and spreads readily in the absence of teat disinfection it is easily eliminated by antibiotic dry cow therapy. This pattern is consistent enough to be useful in indicating which herds are not effectively applying teat disinfection and/or dry cow therapy. It is the authors opinion that this herd had a significant proportion of cows infected with *C. bovis* and it was this that was a major contributory factor to the high bulk milk cell count. As a consequence the economic impact from reduced yields may not have been as great as if the problem had been associated with infections such as Staphylococci. However the resulting losses from cell count and TBC penalties were amounting to approximately £150 per month, not to mention the higher incidence of clinical mastitis.

Interpreting the effect of dry cow therapy on individual cell counts.

Since the cows where *C. bovis* was isolated had the highest average cell count and very few cows received DCT before August '91 a comparison was made between the average cell counts broken down by bacteria for cows which did and did not receive DCT. Although routine DCT was started in August '91 no accurate records were available so the animals were split according to calving date. Only those cows calving after 1.11.91 were deemed to have had dry cow therapy at the end of their previous lactation. The difference was marked. The average cell counts for cows which had DCT at the end of the previous lactation were all significantly lower than cows which did not receive DCT (Figs. 6 & 7). All the groups which received therapy had a mean somatic cell count **below** 300,000/ml, whereas all the groups which did not receive therapy had a mean somatic cell count **above** 300,000/ml.

The average for *C. bovis* had reduced from 650 without DCT to 250 with DCT. The corresponding figures for coagulase positive Staphylococci were 690 without DCT and 290 with DCT. It should be remembered that these did not represent bacterial cures, but the numbers of bacteria isolated after DCT was much reduced (data not included).

The graphs (Figs. 5, 6 & 7) were repeated for February '92 with similar results. (The February individual cow cell counts were taken on 20.2.92 and most of the screen samples were taken on 9.3.92).

To confirm that this was an effect of dry cow therapy, other possibilities were investigated. Cell counts vary with the stage of lactation and so the calving pattern was examined (see Fig. 8) but no obvious effect on cell count could be postulated. Herd size and number of cows in milk was also checked (see Fig. 9) and again there did not seem to be any obvious effect to cause such a marked difference between cell counts for cows calving before or after 1.11.91. Two further graphs were examined to confirm the improvements in cell count in cows receiving dry cow therapy. The average cell counts were compared for cows which had, or had not received DCT for each months weighing, from November '91 to May '92 (Fig. 10). As more cows calved that **had** received dry cow therapy, the average cell count of these cows never exceeded 400,000/ml despite the total number of cows in this group reaching 44. The average cell count for the cows which **had not** received DCT remained consistently higher and only fell below 400,000/ml when the number of cows in milk in this group fell. This is despite the peak number of cows in milk which **had not** received DCT only reaching just over half the number of cows in milk which did have DCT. The monthly bulk milk cell count fell as the proportion of cows in the milking herd which had received DCT increased (cf Fig. 1). If the cows that were dried off each month are traced as a group through to calving and the average of their last cell counts before drying off compared with their first cell count in the next lactation (Fig. 11), one can see that cell counts are generally lower after the dry period once DCT was started. Average calving cell counts for cows dried off in May and June are incomplete. (At the time of going to press the average calving cell count for 3 cows which have calved out of the 5 which dried off in May is 98.)

Total bacterial counts

At the end of March '92 TBC figures were still in Band B (March '92 average was 53) despite bulk milk cell counts being below 400,000/ml for the first time. (The 3 month geometric mean was 402 for March '92 just missing the bonus payment!). Often when bulk

milk cell counts are elevated (500,000/ml or greater) it is difficult to keep the TBC in Band A. This is probably as a result of a high level of sub-clinical mastitis. However the TBC had not followed the downward trend of the bulk milk cell count and a visit to examine the plant revealed that the washing procedure was inadequate. The ball valve on the supply to the water heater was not functioning correctly and the volume of water circulating was insufficient to effectively reach the last jar in the plant. Once this had been corrected, together with long milk tube replacement, the next TBC was in Band A. The TBC for the end of March '92 was 103 and the first for April '92 was 7! The rest of the parlour and plant rubberwork was replaced the following week.

What of the future?

Having set out to achieve cell count Band 1 and TBC Band A and having reached our target in 8 months, the question is obviously will the improvements be maintained?

Providing the NIRD 5 point plan is maintained the herd cell count and TBC should remain in Band 1 and Band A respectively.

By the time this paper is presented at the British Mastitis Conference in October '92, the cows with infected quarters treated with milking preparations at the end of lactation just prior to DCT will have calved long enough to assess the true success. These cows will be monitored very carefully, including quarter sampling at calving and monitoring their monthly cell counts. The author awaits with considerable interest to see how many cows with persistently elevated cell counts during the last lactation can be permanently bacteriologically cured and thus removed from the cull list.

There are also other cows from other herds within the practice that have been treated in the same way. These cows will also have calved long enough to asses the true success by October '92

The results so far look encouraging. Cell counts in cows treated in this way appear to fall more dramatically and more consistently than they did in these cows in the previous dry period where only DCT was used. In some cows where only conventional DCT was used the cell counts were actually higher during the following lactation.

Cost benefit of the exercise

Extra Revenue

1) From bonus a) Cell count 0.2p/litre
b) TBC bonus 0.23p/litre

Total extra bonus = 0.43p/litre assuming that 3 month geometric mean never exceeded 700,000 or the TBC never entered Band C ie no penalties incurred previously.

Quota is for 400,000 litres.

Thus extra revenue from bonus = £1,720/annum

2) Reduced expenditure from reduced clinical mastitis - no records kept.

From practice invoices 200 tubes purchased from December '90 to June '91

Estimate 3.5 tubes per case ie 57 cases treated.

From farm records 41 cases treated from December '91 to June '92

This is a reduction of 16 cases during a six month period.

At £50/case this represents a saving of £800/annum

This is a very conservative estimate. Further reductions throughout the year are likely although the incidence is generally higher during the winter months and so reductions during the summer months will be less significant.

Not including any improvements in yield or quality because of reduced cell counts.

Total annual saving £2,520.

Extra expenditure

1) Routine Dry Cow therapy @ £5.50/cow/year or **£400/annum.**

2) Detailed bacteriology - sponsorship from NMR was up to £600. This included monitoring all cows as they calved in. If a more commercial approach were taken with less repeated sampling to monitor problem cows the cost could easily be halved for the 12 month period. **ie £300**

3) Continue teat dipping - no extra cost. Although chemical costs may increase as teat dipping was not carried out routinely throughout the year.

4) Cost of culling cows. 24 cows were culled during 12 months, often for mixed reasons (eg Lame and high cell count). Thirteen of these cows had cell counts of over 1,000,000/ml, some of which had lost quarters or suffered from recurrent mastitis.

Estimate replacement cost of £300 per cow.

Annual cost of culls £3,900

Total annual cost £4,610.

COST BENEFIT in first year = (£1,720 + £800) - (£410 + £300 + £3,900)

Net <u>cost</u> in the first year £2,090

Providing the improvement in bulk milk cell count and TBC is maintained the cost benefits will be evident in the following years as the number of cows culled for mastitis or persistently high cell count should be minimal. Laboratory fees will also be reduced to clinical cases and some monitoring of problem cows. There should be no need to screen the whole herd in the future.

ESTIMATED SUSTAINED COST BENEFIT IN FUTURE YEARS

£1,500 to £1,750/annum

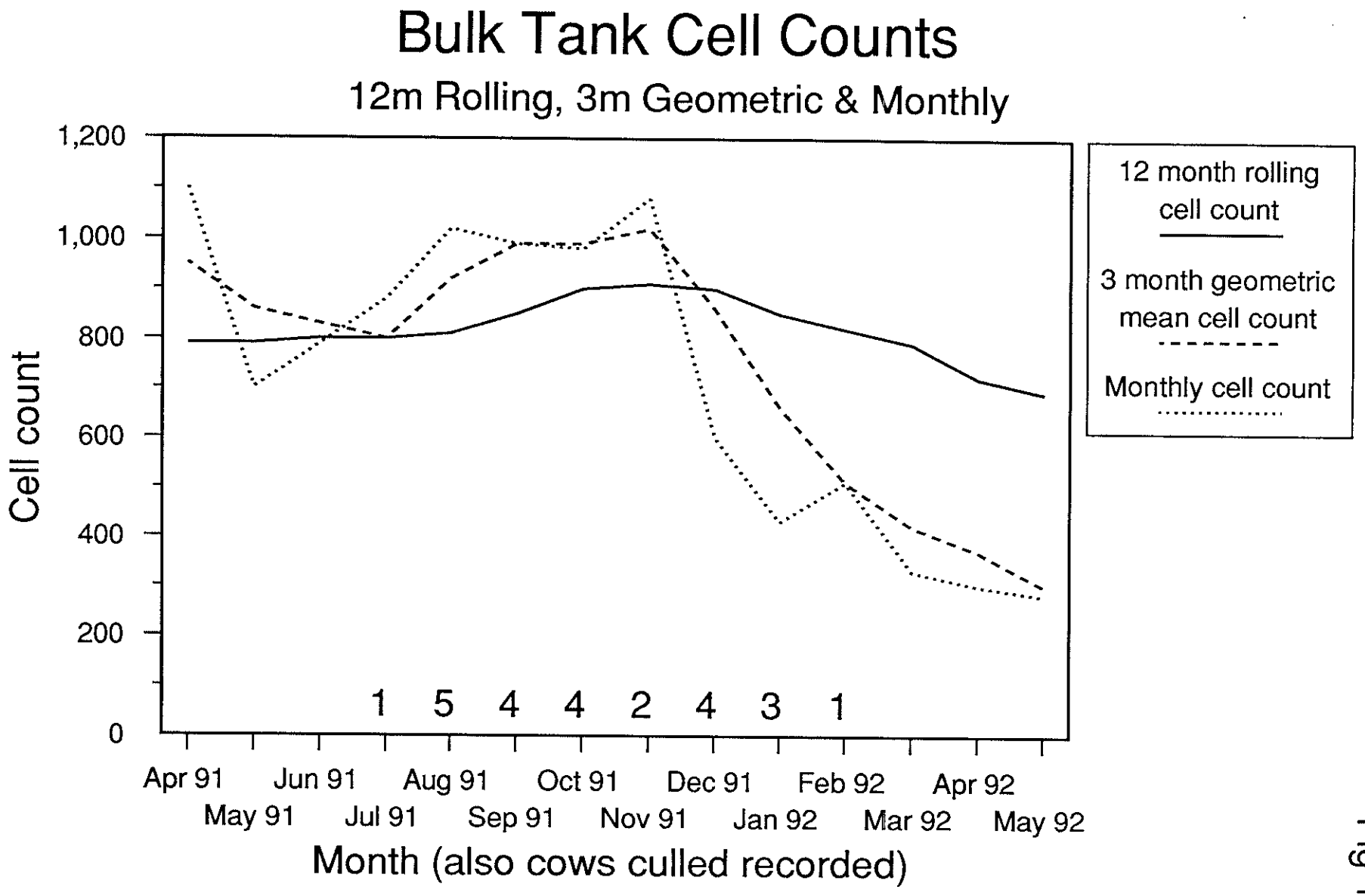

Bulk Tank Cell Counts
12m Rolling, 3m Geometric & Monthly
1,200
1,000
800
600
400
200
0
Cell count
12 month rolling cell count
3 month geometric mean cell count
Monthly cell count
1 5 4 4 2 4 3 1
Apr 91
May 91
Jun 91
Jul 91
Aug 91
Sep 91
Oct 91
Nov 91
Dec 91
Jan 92
Feb 92
Mar 92
Apr 92
May 92
Month (also cows culled recorded)

Fig 1

Comparison of Average Individual Cow and Bulk Tank Cell Counts

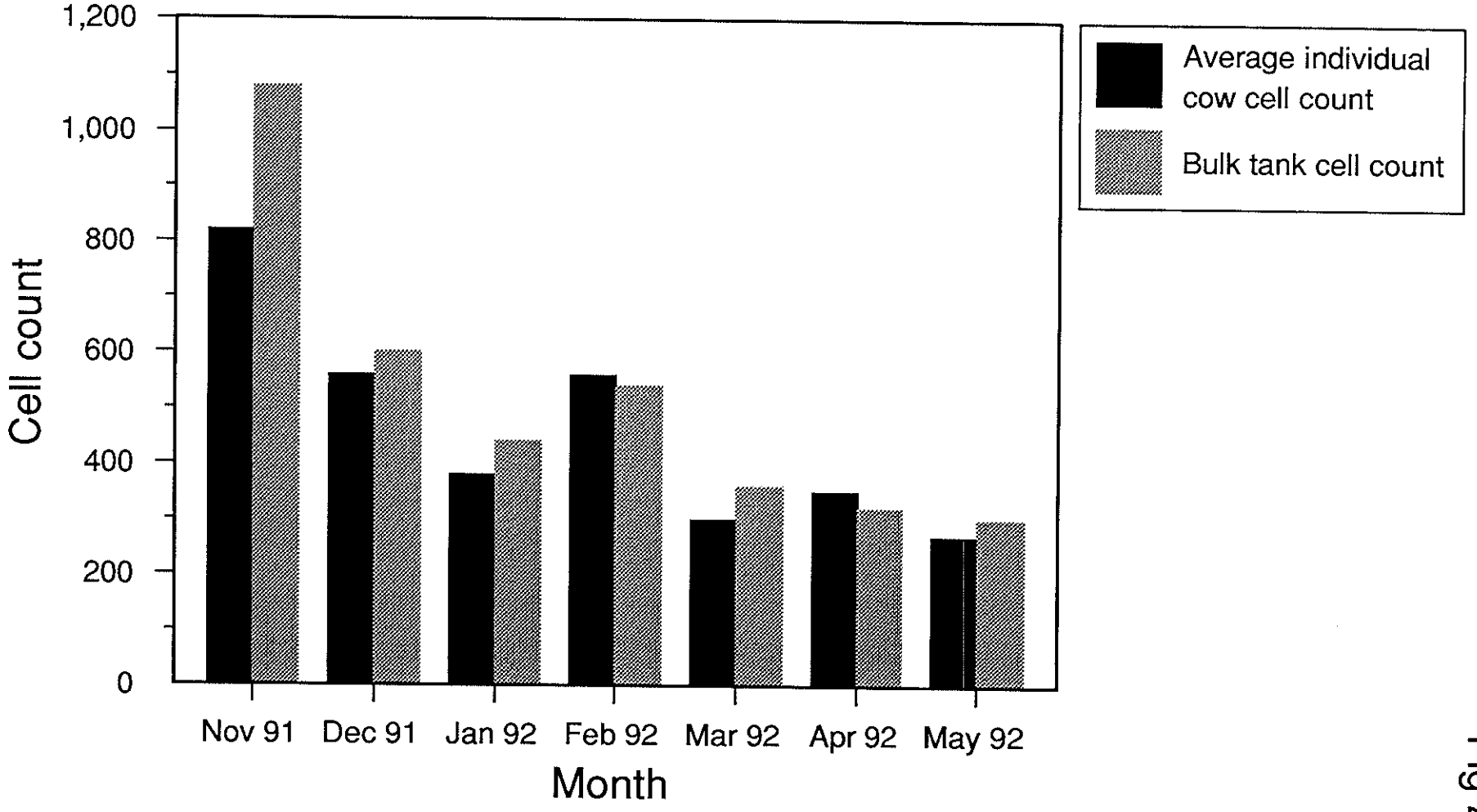


Fig 2

Breakdown of 89 Cow Screen Samples Taken During February and March 92

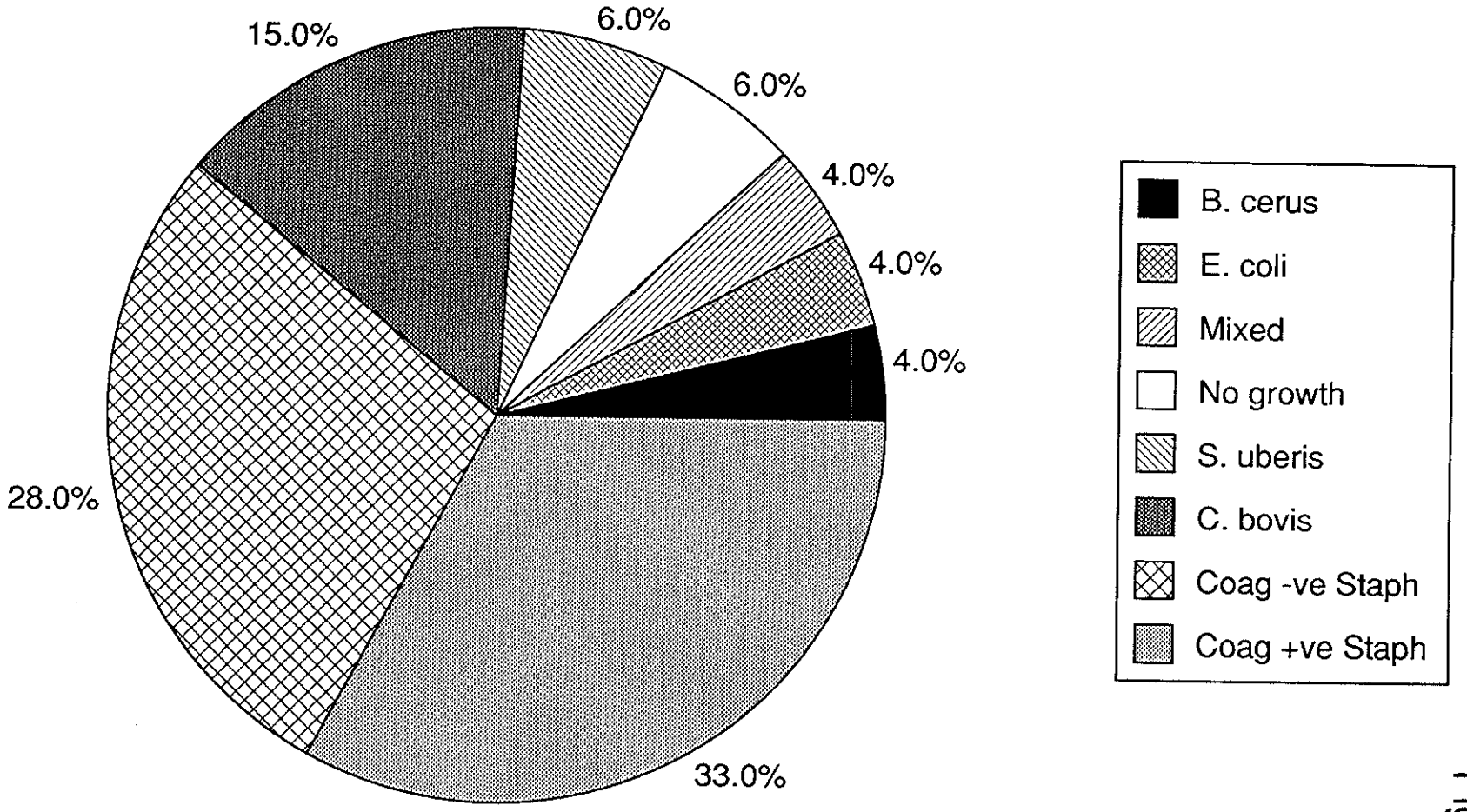


Fig 3

Breakdown of 130 Samples Feb to May 92

(Samples include screen, clinical & drying off)

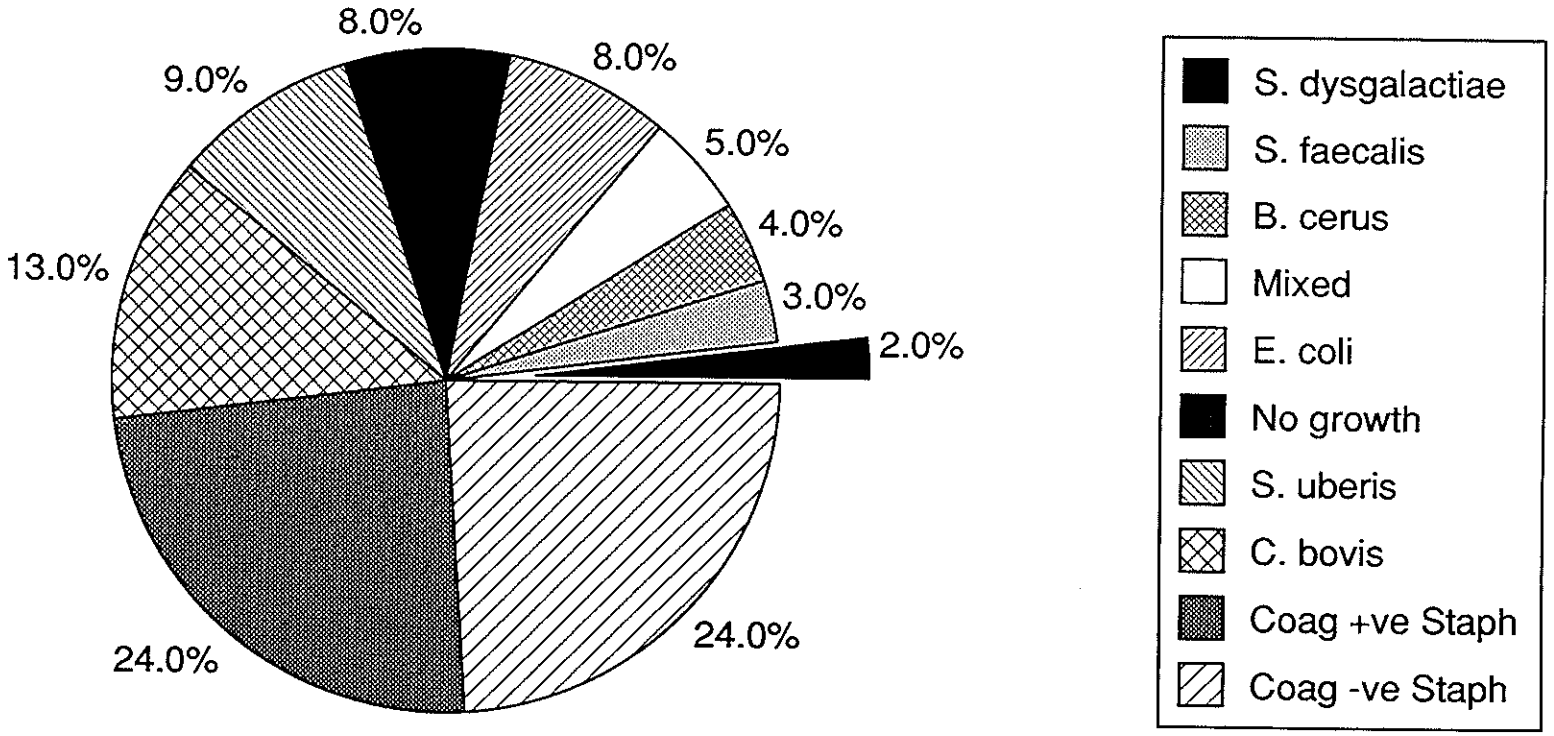


Fig 4

Average Individual Cell Count for Each Type of Bacteria Isolated

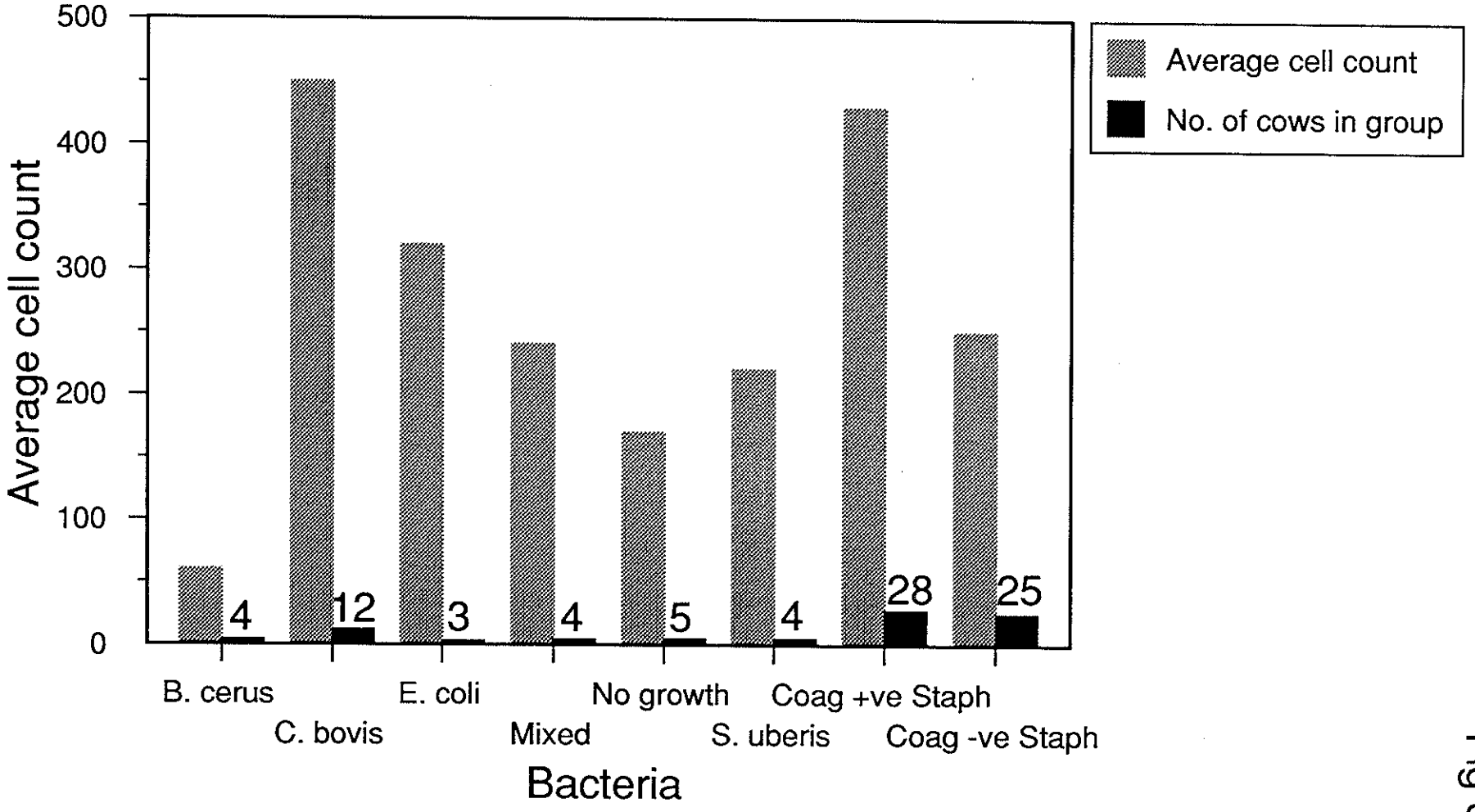


Fig 5

Average Individual Cell Count for Cows which had Received Dry Cow Therapy

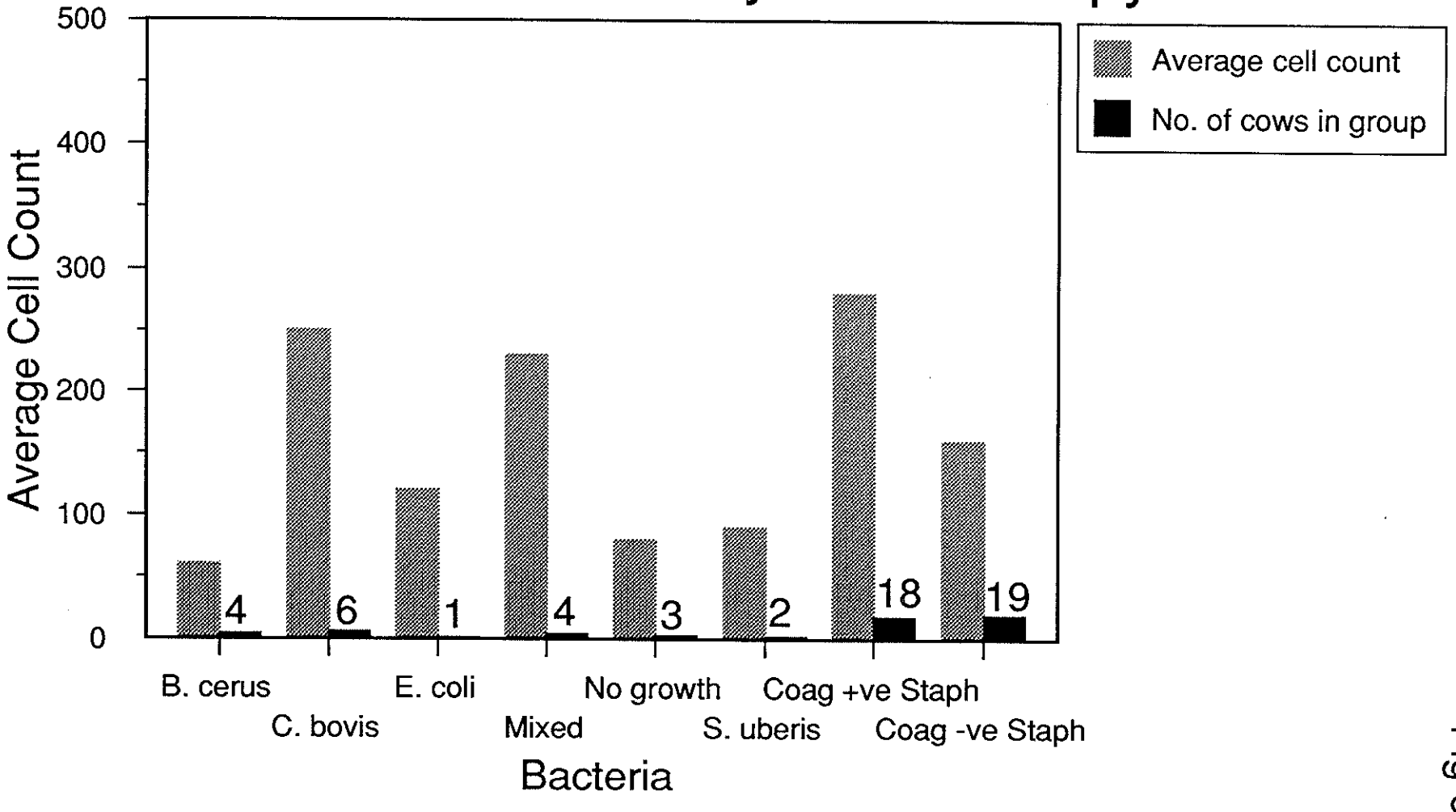
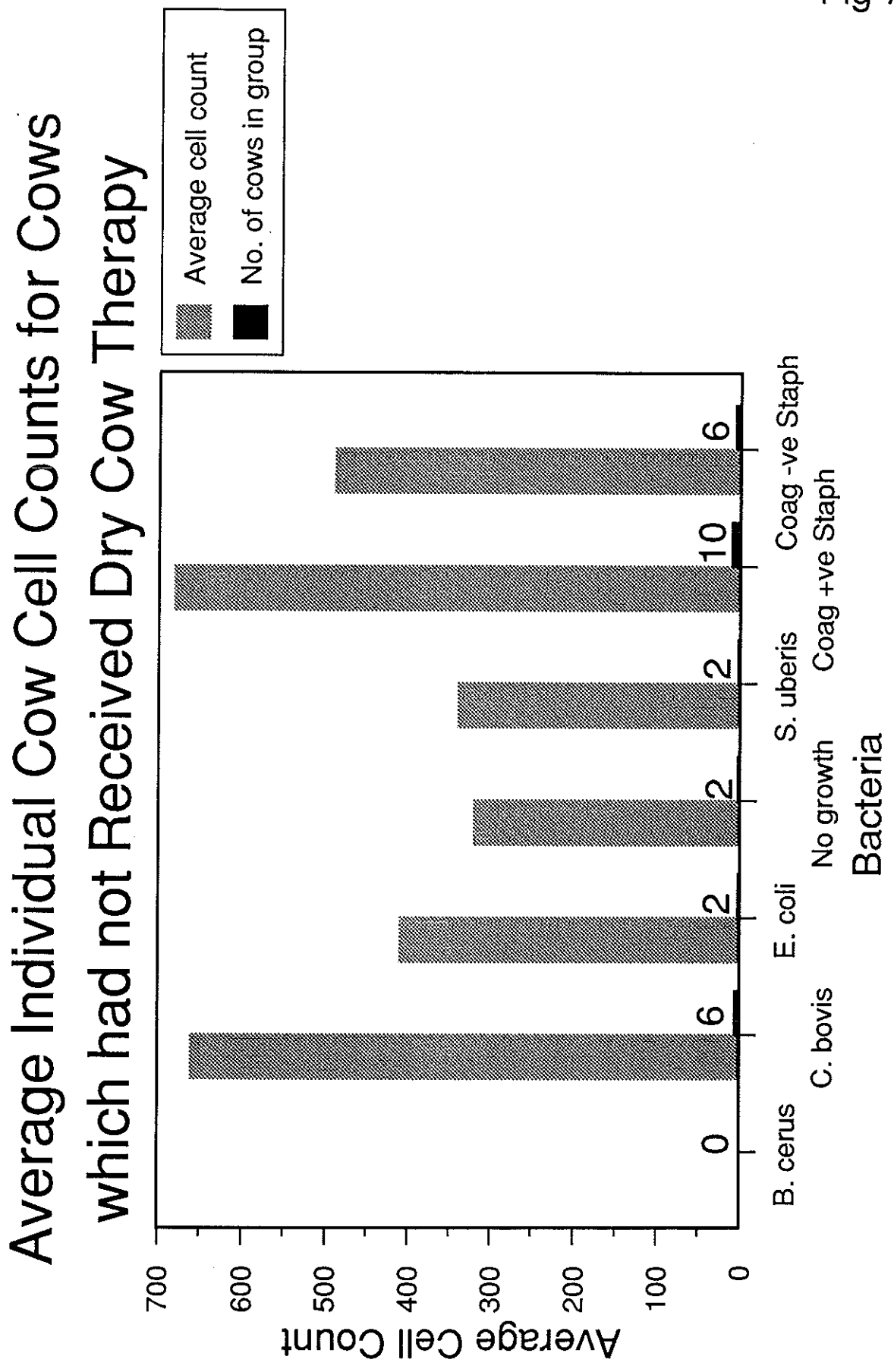


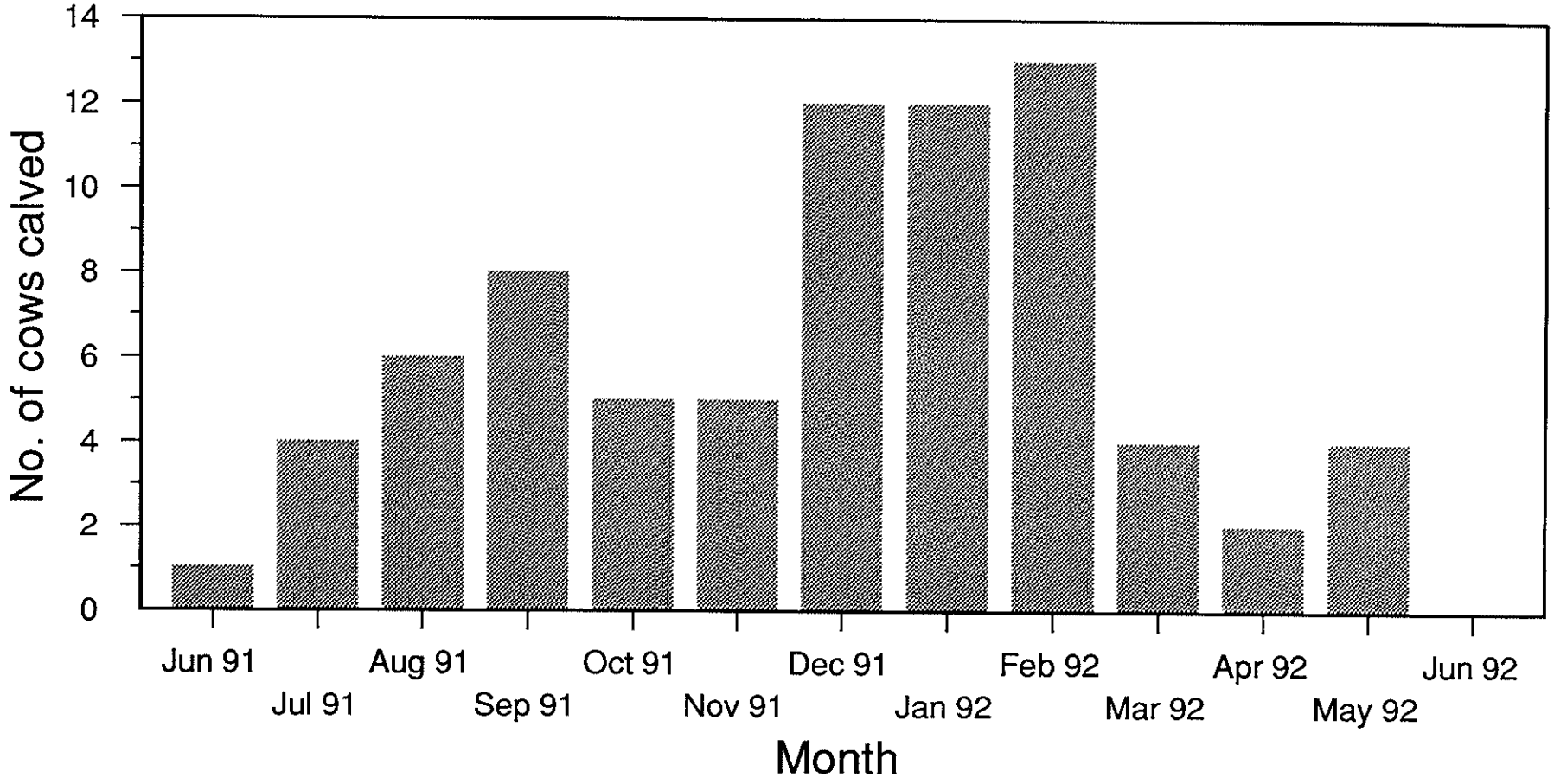
Fig 6

Fig 7


Average Individual Cow Cell Counts for Cows which had not Received Dry Cow Therapy
Average cell count
No. of cows in group
Average Cell Count
0
100
200
300
400
500
600
700
0
6
2
2
2
10
6
B. cerus
C. bovis
E. coli
No growth
S. uberis
Coag +ve Staph
Coag -ve Staph
Bacteria

Calvings per Month

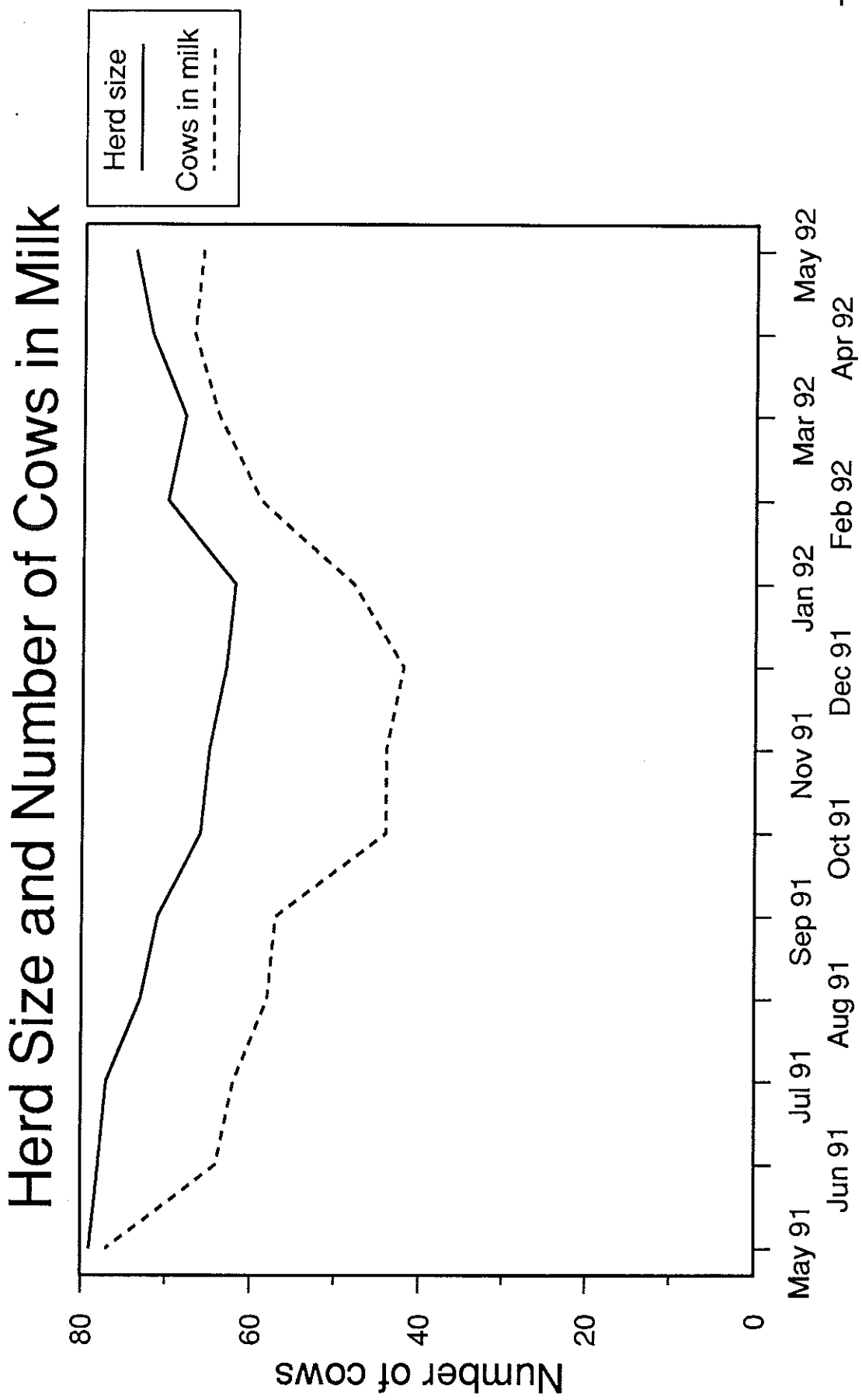
taken as first Month milk suitable for bulk tank



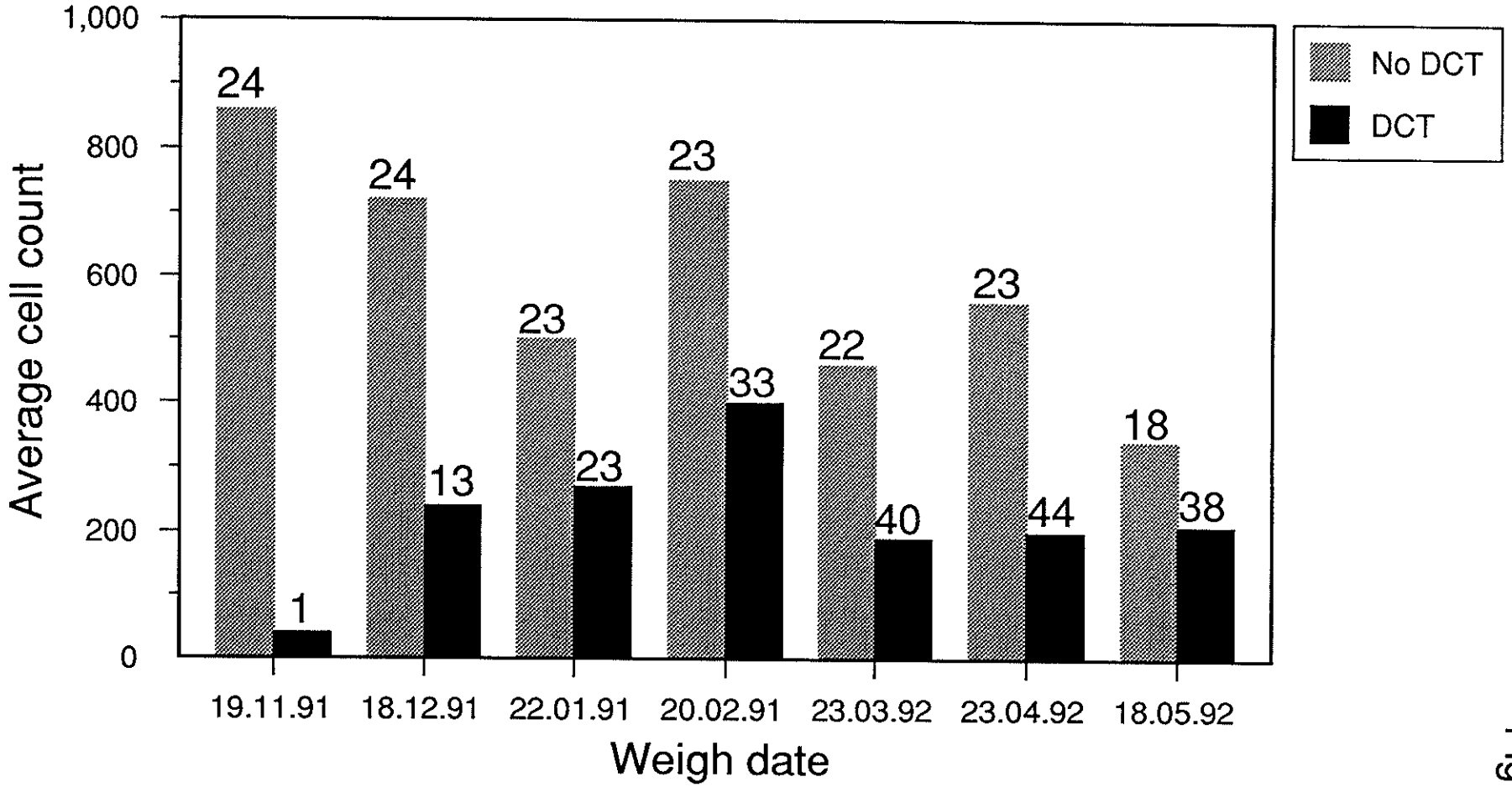
Note: animals allocated to the month where milk first influenced bulk tank reading

Fig 8

Fig 9


Herd Size and Number of Cows in Milk
Herd size
Cows in milk
Number of cows
0
20
40
60
80
May 91
Jun 91
Jul 91
Aug 91
Sep 91
Oct 91
Nov 91
Dec 91
Jan 92
Feb 92
Mar 92
Apr 92
May 92
Month

The Effect of Dry Cow Therapy on Average Individual Cow Cell Count in Next Lactation

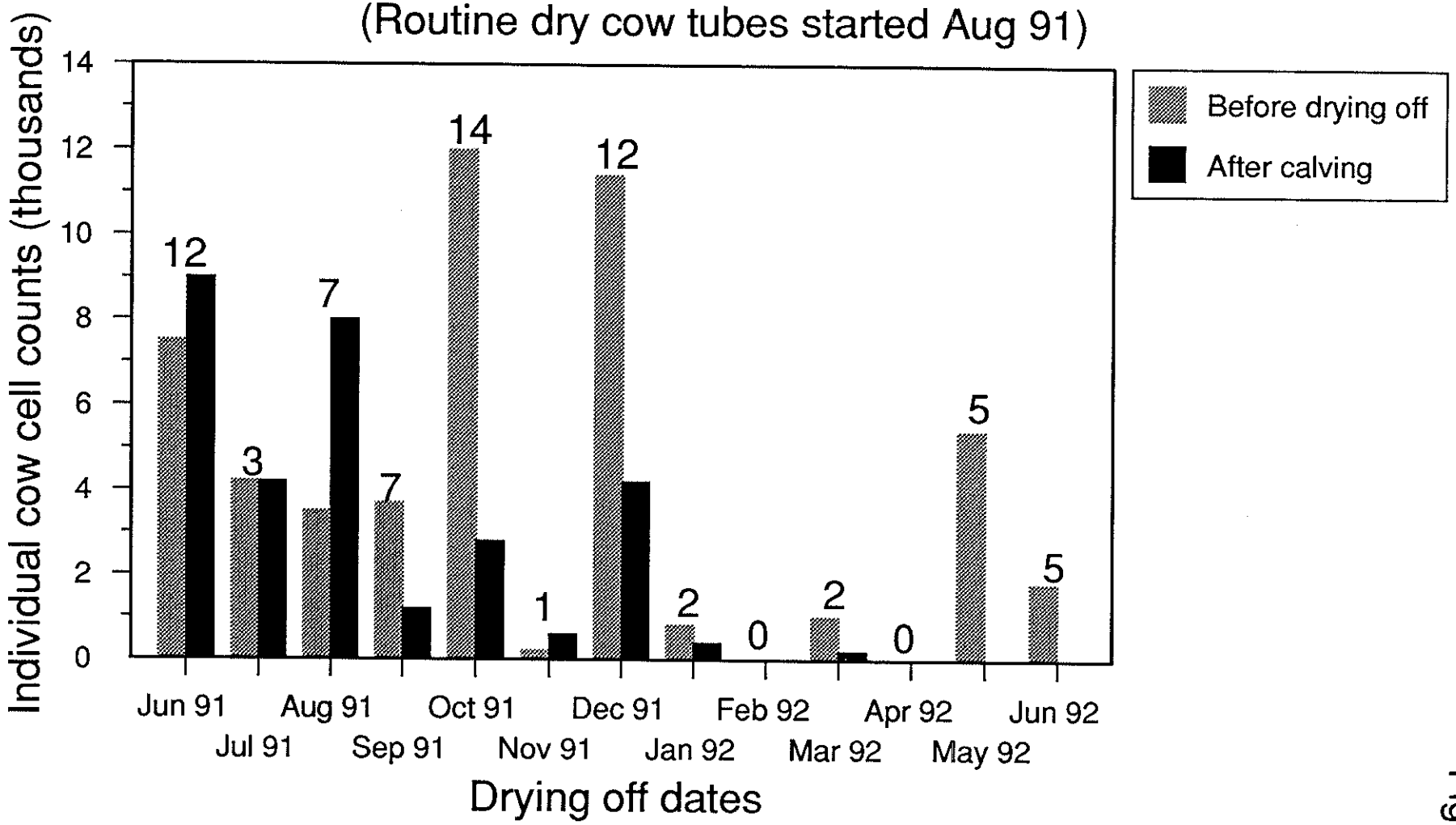


n = number of cows in group

Fig 10

Cell Counts Before Drying Off & After Calving

(Routine dry cow tubes started Aug 91)



n = number of cows in group

Fig 11

HOW WE ARE USING COW CELL COUNTS The Farmers viewpoint:-

Robert G.N. Harris, Southwood Cottage Farm, Bickleigh, Tiverton, EX16 8HE.

The Farm

The farm can be described as a family concern; run by myself and my wife when she is not looking after our young family. In other words there are one and a half full time staff. Our land is made up of 180 acres in a ring fence at 500 ft. above sea level. Part is woodland and steep, rough grazing land, but 70 acres is river valley meadows, in 3 separate blocks. The land supports 80 Friesian cows plus all followers. We aim to rear 20-25 heifers to calve into the herd at 24-27 months of age. A considerable number of beef cattle are also reared and sold on as forward stores.

Some 10 years ago we installed a new milking plant with the aid from the Farming and Horticulture Development Service. We opted for a Fullwood 6/12 eye level herring-bone parlour, complete with automatic cluster removers, automatic milk transfer and electric feeders. At the same time the cows winter housing system was also changed from a cubicle system to a loose straw yard. Dry cows are kept separate. The first 12 months following this major change everything went very well, with only isolated cases of clinical mastitis, which we considered to be acceptable. Although in the years that followed cell counts were rising, we were happy that we were keeping mastitis at bay.

The Problem

By April 1991 we had a bulk tank cell count of 950,000, and we had fallen into Band B for the Total Bacterial Count (TBC) in the milk. During the next few months with the cows going out to grass, I was hoping that the situation would improve. However, by August 1991 our TBC was still in Band B and our milk cell counts had not improved. I thought that with such a high cell count it was impossible to achieve a TBC in Band A. With bonus payments coming in for cell count in October of that year, it was decided that positive action was needed. We had discussions with our Veterinary Surgeon, and by January 1992 decided to work together to try and beat the problem. **(The veterinarians account of the problem and the solution appears in the next paper of these proceedings.)**

The Solving of the Problem

The Milk Marketing Board tested the parlour, but as with previous annual tests they found it to be working satisfactorily. We changed to a policy of treating all cows with antibiotics at drying off; previously we had selected cows for treatment based on cell counts from National Milk Records. Despite the culling of 20 cows between July and December 1991, 50% of which were directly because of high cell count we still had a bulk cell count of 1,002,000 (Band 3) and a TBC of 80,000. We could not afford to cull more cows, furthermore, we realised that this did not appear to solve the problem! In an attempt to improve the cell count, 6 cows with counts of over 1,000,000 were treated as for clinical mastitis, 3 were dried off early and one sold. This panic measure did not solve much as 5 out of the 6 treated cows still had a high cell count in the following month and 4 of them returned as clinical cases in January 1992.

My new years resolution for 1992 was "to get the cell count down". I was going to:-

- Write things down.
- Dry wipe with paper towels.
- Carry out post milking teat spraying.
- Work more closely with the vet.

The behaviour of the cows improved and our cell count was heading in the right direction; the monthly count was 427,000 with a geometric average of 637,000, but we were still in Band B for TBC.

So that we were not working totally in the dark, all clinical cases were sampled prior to treatment. February was another bad month with 11 new cases recorded. The whole herd was sampled in early March to give an overall bacteriological picture. Following this the problem cows were identified and quarter sampled separately.

At this stage the water heater was checked and it was found that the volume of wash water was inadequate. The correction of this, together with rerubbering of the milking plant lowered the TBC overnight from 103 to 7! The new rubber was translucent Silclear Sibcone Dairy Tubing allowing milk flow to be observed and any problems with the wash routine noticed. Clearly, the major contribution to the high TBC was not due to mastitis and unrelated .to the high cell count

With the arrival of Spring, the cows were turned out, and there was a reduction of clinical mastitis. Our cell count was more respectable, but the cows with a cell count of over 200,000 were quarter sampled at the end of lactation and infected quarters treated prior to drying off.

During the 7 months of recording clinical mastitis we had the grand total of 41 cases (see Fig.), 5 cows being responsible for 50% of these. One of these cows was culled; the remaining 4 are in the herd under a very watchful eye.

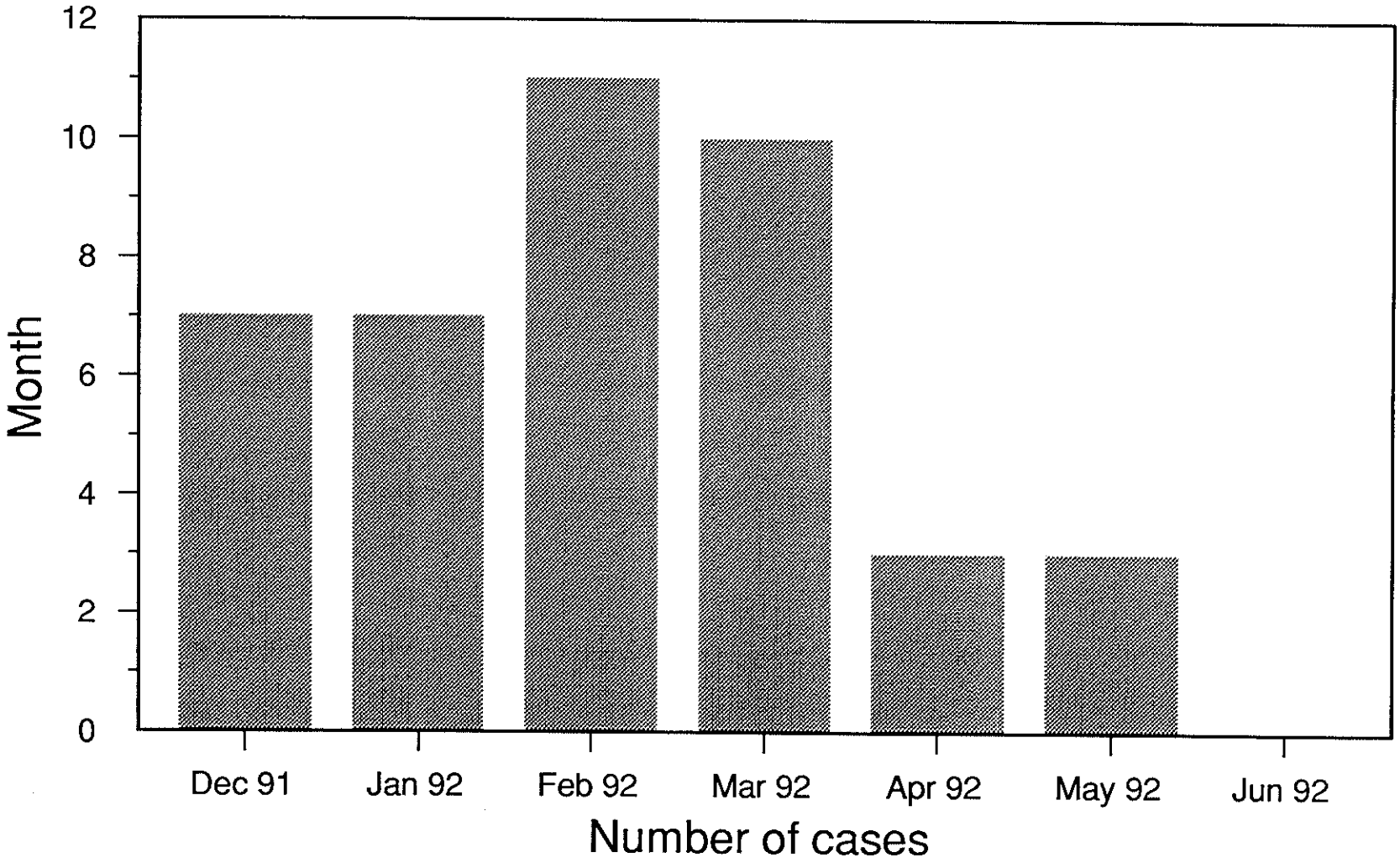
At the time of writing this paper, 25% of the herd are dry. All of them received dry cow therapy and we are keeping our fingers crossed that when they re-enter the herd they will produce clean low cell count milk.

We had arrived at our goal and are receiving bonus payments for both TBC and cell count thank to veterinary advice and his laboratory staff, MMB records and new machine tubing.

I believe that the main points which helped us to get our bonus payments were:-

- Dry cow antibiotic therapy for all cows.
- Better use of MMB records
- Laboratory investigation of clinical samples.
- Post milking teat spraying.
- Dry towel wiping.
- Correct temperature and volume of wash water.
- New rubber on milking machine.

Number of clinical cases treated per month from November '91 to June '92



PROBLEM SOLVING

TACKLING MASTITIS ON THE FARM

Peter Edmondson, Eddy Williamson & Partners, Allyn Saxon Drive, Shepton Mallet, Somerset BA4 5QA

Mastitis is not a man-made disease, however, man can greatly influence the incidence of mastitis through management practices. Good management practices tend to result in low levels of mastitis and good milk quality.

The veterinarians perception of a problem frequently differs from that of the dairyman. The veterinarians aim is to prevent a problem from occurring or to intervene at the onset of trouble, whereas some dairymen will delay seeking advice to see if the situation resolves itself.

Below is a set of target levels that should be attainable in every dairy herd;

TBC levels under 10,000/ml
Coliform counts under 150/ml
12 month rolling herd cell count under 300,000/ml
Mastitis rate under 35 clinical cases/100 cows/year

If these targets are exceeded in a herd, then in my opinion, there is a problem. Mastitis is a management problem, and it is up to the veterinarian to work with the dairyman to modify his management practices to resolve these problems.

The majority of dairy farmers in England and Wales are content with their mastitis management when they are receiving their TBC and somatic cell count "bonus" payments, irrespective of clinical incidence. Others see these bonuses as just that, if TBCs are under 20,000 for a month, then that is a bonus. The "Bonus scheme" is misleading as it is a penalty scheme and should be called such. The payment system should be changed so that producers whose cell counts are over 400,000 and/or TBCs over 20,000, see that they have been penalised and a deduction has been made from their milk cheque. Hopefully, this may encourage more of these producers to seek advice on ways to overcome their problems.

There are three reasons for getting involved with the problem herd; high cell counts, high TBCs and/or a high incidence of clinical mastitis. In this herd study, the problem was one of a rising herd cell count.

The first temptation for any eager veterinarian is to jump into the car immediately and try and solve the problem then and there. This approach to problem solving is likely to be unsuccessful. In order to succeed, an organised approach to herd investigation is required. I use the same procedure no matter what investigation is being carried out. The first place to start is in the practice. Some practitioners fail to realise the amount of information that they already have available on individual herds. This includes:-

VCCIS - the Veterinary Cell Count Information Service from Genus Animal Health

Records of lactating and dry cow intramammary sales

Bacteriology reports

Many practices now have computerised herd health recording systems with detailed mastitis information

So before you leave the practice, you can get a fair idea as to what is happening on the farm. Are the lactating cow tube sales high for the herd size? Is the cell count increasing? Is dry cow therapy used on the entire herd? All this gives invaluable background information.

Unless there is an urgent need to get to the farm, a bulk tank milk sample is brought into the practice laboratory for analysis. The following tests are carried out:

Coliform count to measure milking hygiene

TBC - Total bacteria count

Culture of the sample to identify all pathogens

In this case study which relates to the problem described in the previous paper, *Streptococcus dysgalactiae* was identified from the bulk tank, and so one pathogen was known before the farm visit. However, there is a need for caution, if a particular pathogen has not been identified from the bulk sample, it does not exclude its presence from the herd.

Once all the above information is available, it is entered into a mastitis investigation checklist, see Figure 1. This checklist is invaluable and ensures that all aspects relating to mastitis management are examined. Furthermore, if a client rings up with further queries following a visit, then you can refer to the checklist to see what was happening and what recommendations were made. This is very useful when dealing with many herds at the same time where confusion can easily occur.

The Farm visit

The visit takes place in the afternoon, arriving about an hour before milking. If the machine has not been tested in the last six months a static machine test will be carried out to check plant performance. All results are recorded on the test sheet, see Figure 2.

Farm records of clinical cases, recent milk quality results, and individual cow cell counts, where available are examined. It is useful to see how the farm mastitis records correspond to the practice lactating cow sales information. Blowey assumes an average of 5 tubes per clinical case. In this study the figures corresponded closely. In this herd, 41 cases of mastitis had been recorded in the past 12 months, giving a mastitis rate of 27 cases of mastitis per 100 cows per year. This is below the target figure of 35.

The source of replacements is important. This herd rears nearly all its replacements, but some fresh calvers are purchased and there is the risk of introducing new pathogens such as *Streptococcus agalactiae*.

During milking, the milker's routine, treatment regimes for dry and lactating cow therapy, wash-up routines are all discussed and observations noted to check how well the milker's description and actual procedure correspond. The method of mastitis identification is discussed. In some parts of the country, few farmers foremilk cows. Heavy reliance is placed on the use of in-line mastitis detectors. These tend to give a false sense of security, as they are rarely checked, and many cases of mastitis go undetected resulting in mastitis milk in the bulk tank.

In this herd, the milker informed me that he always checked each mastitis detector after each cow was milked, however, at the rear of the parlour one detector was totally clogged and the herdsman was unable to identify the offender. He has since started foremilking!

Samples are collected from any new clinical cases identified during the visit, and from any suspected problem cows for bacteriological analysis. The only reliable way to identify mastitis is by foremilking. Early detection and treatment of clinical cases will increase the speed of recovery, maintain milk quality, and reduce the risk of spreading infection to the rest of the herd.

Clinical cases and other cows undergoing therapy that necessitate milk withdrawal from the bulk tank should be milked last to avoid accidental contamination of milk with drug residues. This also eliminates the risk of spreading infection to the rest of the herd by contaminated equipment and hands or gloves, and speeds up milking.

Particular attention is paid to the administration of intramammary preparations. The teat-end is scrubbed clean with a Mediwipe to reduce the risk of accidental infusion of mastitis pathogens into the udder via the tube. Some farmers occasionally report that a cow under treatment makes good progress for a couple of days, but then develops a more severe form of mastitis. This can occur with poor preparation where organisms present on the teat-end are forced into the udder resulting in a more severe infection than the one for which the treatment was originally intended.

Teat-end preparation is especially important with dry cow therapy which remain in the udder for long periods of time. Any introduced pathogens could cause problems during the dry period or after calving. Intramammary preparations will only be effective if administered in accordance with the manufacturers recommendations. There is the belief that as these preparations contain antibiotics, they will overcome any inadequacies in their administration - not so!

The culling policy of the herd is discussed. In some seasonable calving herds, where up to 20% of the herd will be culled due to poor fertility, there is little room for culling persistent mastitis offenders. This hinders the progress of any mastitis programme. Chronic mastitis cows are unprofitable irrespective of milk yield and only act as a reservoir of infection to the rest of the herd. They must be removed. In this herd, mastitic cows are removed if they have three or more episodes of mastitis per lactation.

Irrespective of any equipment tests that may have been carried out, a dynamic machine test is always conducted on at least three high yielding cows to check teat-end vacuum levels and fluctuations. A few pulsation traces are checked, as is the vacuum stability in the plant. The static test is useful in highlighting problems such as poor vacuum reserve or pulsation, however, it must be remembered that it is carried out on a machine that is not milking cows. The dynamic test identifies problems during milking that may not be shown up on the static test.

The milking machine is the dairy farmers combine harvester. It should extract all milk from the udder rapidly with minimal risk to udder health. Despite the fact that it generates the majority of the dairy farmers income, it is probably the most neglected and misunderstood piece of equipment on the farm. The average milking machine runs for four hours every day. If you compare the milking machine to the average motor car, assuming that the milking parlour travels at 40 mph, it will "travel" 58,400 miles with only one service a year! How frequently are tractors serviced?

On some farms, faults identified at the machine test are left unattended and come low down on the list of farm priorities. It must be remembered that most faults that occur with milking equipment are gradual and so the operator is unaware of their presence, or the slow drop off in plant performance. Parlours must be regularly serviced, at least twice a year. Rubberware should be regularly checked and replaced whenever perished. Liners need to be renewed after every 2,500 milkings or six months, whichever is soonest. In all herds, the frequency of liner life is checked. In this herd liners were changed after every six months by which time they had carried out 2,750 milkings, and were collapsed.

All recommendations are entered on the checklist, and are discussed with the herdsman and the owner, if possible, before leaving the farm.

Reporting and follow-up procedure

A detailed report is sent to the owner and the herdsman to arrive about a week after the visit. This report comes off a word processor where an introduction is inserted, and a standard list of recommendations amended as necessary to produce a report that covers all aspects of mastitis management. It is important that any suggested management changes are justified.

One month later, providing that no problems have occurred in the meantime, a follow-up phone call or visit is made to check on progress. This helps clear up any difficulties that may have been encountered since the visit.

Over the past fives years in this practice, substantial improvement has been made in improving the mastitis management and milk quality of our dairy clients. Over 78% of herd mastitis and milk quality problems have been resolved totally, while 12% continue to make good progress. The others have made no improvement whatsoever, and their situation has deteriorated. These are the dairymen who want an instant solution to a chronic problem, with no input or expense on their part. These tend to be farmers with little or no borrowings, content to make an average living today, with no concern for tomorrow.

In the future, the demands on the dairy farmer will be for even higher standards of milk quality as the large supermarkets continue their upward retail sales of liquid milk. It is essential that dairy farmers continue to work with their veterinarian to prevent and overcome mastitis and milk quality problems, which will ensure a healthy and prosperous dairy industry in the future.

Figure 1. Mastitis Investigation Checklist

Name: Date / /

Address:

Reason for visit: TBC/ SCC / CLINICALS / ROUTINE

Initial visit / Date of last visit: / / .

Background information:

No. milking in herd: _____ No. dry: _____

12 month RHA SCC: TBC range:

_____ mastitis cases in last _____ months from _____ cows

Tube sales: Past 12 months _____ Milking cow, _____ Dry cow.

Mastitis rate: cases per 100 cows per annum (est/actual)

Replacements: all homebred / in-calf/calved down heifers / cows

NMR individual cow cell counts available: Yes / No

Culling policy: Clinical cases / SCC / Conformation / None

Bulk Tank Analysis: Date of sample: / /

TBC Coliform Count SCC

Ag / dys / ub / faec / *Staph aur* / other Staph / Coli / Bacillus

Comments:

Housing and Yards:

No. of cubicles: Loose housing

Cubicle design and length (7'3" x 4')

Bedding: Straw / Shavings / Sawdust / Paper / Other

Condition of housing:

Calving facilities: good / fair / poor Wind exposure: Yes / No

Well drained yard free from stagnant water: Yes / No

Premilking procedures:

Condition of cows entering the parlour: good / average / poor

Willingness of cows entering parlour: good / average / poor

Long tails / hairy udders

Washing: none / dry wipe / pre-dipping / hose / udder cloth

Pre-dipping: Yes / No Iodine / HypoCl

Drying: none / individual paper towels / cloth

Stripping of cows: all / some / none

Milking procedure:

Do all milkers wear gloves: Yes / No

Mastitis identification: stripping / detectors / visual / filters

Are mastitis detectors checked: Yes / some / none

Units attached within one minute of preparation: Yes / No

Cows milked out rapidly: Yes / No (3 to 5 minutes)

Magic water / undermilking / overmilking

Liner slip / unit fall-off / stray voltage / ACR function

Throughput: _____ cows / hour with _____ milkers

Hygiene during milking: good / average / poor

Vacuum shut-off prior to unit removal: Yes / No / ACRs

Teat dipping / spraying. RTU dip / diluted correctly / storage

Dip type: Iodine / Chlorhexadine / Hypochloride / Other

Teats dipped all year round: Yes / No

Teat lesions: No / Black spot / Prolapse / BHM / PseudoCP

Milking Order: One group / Fresh / Highs / Low

Clinical mastitis:

Proper records kept: Yes / No

Treatment regime: Full course (3 tubes) / Part course / 2nd choice

Teats disinfected before infusion: Yes / No

Treated cows properly identified: Yes / No

Mastitis milk into dump bucket / recorder vessel / separate cluster

Recorder vessel rinsed between milkings: Yes / No

Clusters sterilised between cows: Yes / No

Dry Cow Therapy:

Type of preparation used: None/ SCDC / SPMC / LeoRed / Other

Blanket treatment of herd: Yes / No

Abrupt / Gradual drying off. Cows starved: Yes / No

Cows dried by calving date and/or yield (5 ltrs/day): Yes / No

Wash-up Routine:

	MORNING	AFTERNOON
Rinse:	none/cold/warm	none/cold/warm
Main wash	hot/cold	hot/cold
Temperature:		
Circ Time:		
Chemical:		
Concn:		
Volume:		
Final Rinse:	none/cold	none/cold

Boiler capacity: _____ litres for _____ units (10 ltrs/unit)

Temperature of hot water: F / C

Milking Parlour:

Type: Hi line / Lo line / DTL / Abreast Age:

General Condition of plant: satisfactory / needs attention

Vacuum level: _____ fluctuating _____ kPa. Gauge reads _____ kPa.

Controller function: adequate / needs attention

Pulsation rate: per minute, Pulsation ratio:

Last test date: by MMB / dealer. Checked every _____ months

Liners changed every _____ months or after _____ milkings

Recommendations

1.

2.

3.

4.

5.

6.

7.

8.

9.

10.

11.

12.

Figure 2.

MILKING MACHINE TEST

Name: Date / /

Address:

1. Vacuum level and stability:

_____ kPa near pump _____ kPa on parlour gauge

_____ kPa at teat-end _____ kPa fluctuation at teat-end

2. Controller function: Adequate / Inadequate

Clean controller / Change filter / Controller is defective

3. Vacuum reserve: Adequate / Inadequate

_____ litres/minute needed for system

_____ litres/minute at receiver jar (Actual reserve)

_____ litres/minute at receiver jar (Effective reserve) >90% of actual

4. Pulsation system: Master / Individual pulsators

_____ functioning normally _____ malfunctioning

_____ Pulsation rate (55 ideal) _____ Pulsation ratio (60/40 ideal)

_____ B, milkout phase (40 ideal) _____ D, massage phase (20 ideal)

5. Condition of equipment:

_____ short air, _____ short milk, _____ long pulse, _____ long milk tubes perished

Condition of clusters and lines: good / average / need attention

Liners changed after every _____ months or _____ milkings,

Liner condition: good / perished / split

PULSATION ANALYSIS

Name: Date: / /

Pulsator Number	A %	B %	C %	D %	Ratio A+B/C+D	Rate
01	___	___	___	___	___/___	___
02	___	___	___	___	___/___	___
03	___	___	___	___	___/___	___
04	___	___	___	___	___/___	___
05	___	___	___	___	___/___	___
06	___	___	___	___	___/___	___
07	___	___	___	___	___/___	___
08	___	___	___	___	___/___	___
09	___	___	___	___	___/___	___
10	___	___	___	___	___/___	___
11	___	___	___	___	___/___	___
12	___	___	___	___	___/___	___
13	___	___	___	___	___/___	___
14	___	___	___	___	___/___	___
15	___	___	___	___	___/___	___
16	___	___	___	___	___/___	___

Pulsation Rate: _____ cycles per minute

TARGETS:

Pulsation Ratio:	60 / 40
Milkout "B" phase	Over 40% of cycle
Massage "D" phase	20% of cycle
Pulsation rate	55 cycles / minute

TACKLING MASTITIS ON THE FARM

Neil Christensen, Steanbow Farm, Pilton, Shepton Mallet, Somerset.

At Steanbow Farm we have 450 Friesian Holstein dairy cows divided into three herds, two of 150 cows and one of 100 cows. The average yield for all herds is 6,000 litres. The calving season runs from June to February. Most replacements are homebred, and additional freshly calved heifers are purchased to maintain herd size.

When cows are housed, they are fed a complete diet based on grass and maize silage, straights and a mineral mix. Silage is fed from July onwards while the cows are at grass. Maize gluten is fed in the parlour.

Each herd is divided into a high and low yielding group and fed accordingly. All cows are housed from October to April in cubicles which are bedded with a generous amount of chopped straw. Cubicles are dusted weekly with hydrated lime powder to keep beds dry. There are automatic scrapers in all sheds.

The aim for milk quality is to have a cell count under 200,000, and TBC levels in single figures. We follow the five point mastitis programme of teat dipping, treatment of clinical cases, dry cow therapy, milking machine maintenance and have a culling policy. The overall incidence of mastitis in all herds is low.

We have routine fertility visits every fortnight when any problems relating to health and production are discussed, and herd progress monitored.

In July 1991, the cell count in the New Park herd, which has 150 cows, started to rise to over 400,000 over a period of one month. The rising cell count was discussed with our veterinarian, Peter Edmondson, during one of his routine visits. He collected a bulk tank milk sample from this herd to identify any possible problems, and arranged to visit the herd early the following week.

Prior to the mastitis visit our milking routine was as follows:

Cows were sprayed with a diluted post dip iodine solution which was left on the teats for about 30 seconds, and was then wiped off with a single service disposable paper towel. The herdsman relied on mastitis detectors and visual swelling of the udders to pick out cows with mastitis.

The practice of applying this diluted post-dip solution before milking had been practised for a period of about six months. After milking, the teats were again sprayed with the diluted iodine solution.

The remainder of our mastitis management is as follows:

Treatment of clinical mastitis

Pre-treatment milk samples are collected from infected cows from time to time, to identify the organisms responsible for mastitis, and to ensure that the most efficient treatment is being used.

Cows with mastitis are milked into a dump bucket using a separate cluster which is disinfected between cows. Mastitic cows always receive a full course of treatment, and treated cows are marked with red tail tape. All treatments are recorded in the medicines book.

Dry cow therapy

Dry cow therapy is used on all animals. Cows are dried off according to calving date giving a six to eight week dry period, or if the milk yield drops below 5 litres a day.

Milking Machine

The herd is milked through a 16 x 16 high level direct to line herringbone with ACRs and milk meters. The herdsman milks on his own with a throughput of 60 cows per hour.

The milking machine is tested twice a year by the MMB, one test is a static test while the other is a combined static and dynamic test. Any problems identified are corrected immediately. Liners are changed every six months.

Culling policy

Problem mastitis cases are culled from the herd. These tend to be the older cows who have additional reasons for being culled such as bad feet or poor fertility. Most cows who have three or more cases of mastitis per lactation are culled.

During the mastitis visit it was explained to us that *Streptococcus dysgalactiae* had been isolated from the bulk tank, and that this bacteria was probably responsible for the rising cell count. The significance of this organism was fully explained.

All aspects of our management was examined to see what improvements could be made to control the spread of infection within the herd.

The recommendations made were as follows:

1. All milkers were to start wearing gloves to cut down the spread of infection from cow to cow on milkers hands.

2. The pre-milking dipping was to be stopped as it was ineffective and costly. It was not working as post-dip solutions have a slow speed of kill and are not intended to be used as pre-dips.

3. The herdsman was to dry wipe the clean cows, and to wash and dry the dirty cows. All cows were to be foremilked to improve the mastitis detection, as early detection will help reduce the spread of infection to the rest of the herd on liners etc.

4. After milking, cows were to be sprayed with full strength teat dip after milking. The importance of coating the entire surface of every teat after every milking was emphasised to control this particular bacteria.

5. We had been cleaning the ends of dirty teats with cotton wool soaked with surgical spirits before administering dry cow therapy. The importance of thorough cleaning of each teat-end before administering any tubes, either milking or dry cow, was stressed, and we now use Mediwipes for this purpose.

6. We had been changing liners every six months, but these needed to be changed every five months as the liners were collapsed at the time of the visit and were six months old.

Since we have modified our milking routine and management, our cell count has continued to drop and is now below 200,000. TBC levels remain in single figures and the incidence of mastitis in the herd has dropped.

We have also tried some novel ideas:

One of our herdsmen was keen to try a homeopathic approach to prevent mastitis in the herds. We tried this in all three herds for a period of a year. There was no real difference, and only one herd continues as the herdsman likes the idea.

Two years ago we put in a suspended head rail 8" below the withers, and 5' 6" from the back of the cubicle. We have found that since this was installed a lot more cows use the cubicles for longer periods of time, and they are cleaner, drier cows.

The cubicle bed base is solid concrete, and recently we have put some old quarry conveyor belting on the cubicle floor while continuing to place the same amount of chopped straw on top. This has added to cubicle comfort and has resulted in less cubicle sores. We have also installed some DORSDUNN cubicles which are very popular with our cows.

CURRENT RESEARCH

IMMUNE RESPONSES IN THE BOVINE MAMMARY GLAND

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Understanding the basic defences and immune mechanisms of the mammary gland is instrumental in developing measures to prevent mastitis. Future development of mastitis vaccines and selection of animals with natural resistance to mastitis requires a detailed knowledge of how the mammary gland responds to invasion by bacteria and how protective responses may be maximised.

Vaccination of the mammary gland must overcome some major problems (1). These include:-

a) the relatively low concentrations of cells, antibodies and non-specific substances present in the mammary gland.

b) cells which are involved in taking up and disposing of bacteria also often contain fat and casein derived from milk, preventing effective killing of the bacteria.

c) the large surface area of the mammary gland requiring surveillance especially during lactation.

d) most bacteria grow well in milk.

e) the wide range of different organisms and their products which may cause mastitis.

Most attempts to vaccinate cows against mastitis have come from studies where the stimulating antigen (usually killed or modified bacteria) is injected into the cow at a site distant from the mammary gland. In this way the antigen is taken up into the bloodstream and circulates round the body where it will hopefully stimulate an immune response. However cells or antibodies which have been stimulated by this method then have to cross into the mammary gland itself if they are to be present in the gland ready to attack incoming bacteria in the future.

Another method of producing a protective immune response may be to introduce the antigen directly into the mammary gland itself. In this way the antigen is mimicking real infection and this has been previously shown to produce some significant immunity to mastitis pathogens (2).

The bovine udder consists of four quarters, which are clearly physically divided into left and right halves by a suspensory apparatus. There is no visible division between the two quarters of the same side but infusions of different coloured fluids into the two teats of one side of the gland demonstrate that the cavities drained by them do not communicate. Tissue fluid from the udder drains via lymphatic vessels into two lymph nodes (or lymph glands), called supramammary lymph nodes, one on each side of the gland. The gland is therefore physically and immunologically divided into left and right portions.

Bacteria usually enter the mammary gland via the teat duct. Their multiplication may be inhibited in the teat duct or mammary tissue by non-specific substances (See paper of A.W. Hill delivered at this conference) or by specifically produced antibodies or cells directed against them. Antibodies produced by B lymphocytes coat bacteria and allow them to be ingested and killed by other mammary gland cells such as neutrophils and macrophages. Stimulation of specific T lymphocytes may also occur. These T cells are responsible for killing infected cells and helping to attract other immune cells to the area. T cells are also required to give help to B cells in order for them to produce antibody.

The three components involved in producing an immune response are the antigen-specific T cell, an immune recognition molecule (called an MHC class II molecule) and the antigen itself (3). The immune recognition molecule is expressed on cells called 'antigen presenting cells'. Antigen presenting cells take up antigen, process the antigen and express the altered antigen on their cell surfaces in association with the immune recognition molecule. The T cell which responds to the antigen in combination with the immune recognition molecule is involved in both producing T cell responses and B cell responses. An investigation of antigen presentation, can therefore be considered as an examination of the immune response at an early stage.

The outcome of successful antigen uptake, processing and presentation is an immune response which can be either primary (on the first exposure to the antigen) or secondary (on subsequent exposures to the antigen) in nature. In secondary responses, increased antibody levels or cellular responses usually appear more quickly and reach higher levels than primary responses.

In order for an immune response to occur, antigen must be transported to a site in the body where it can interact with immune cells. Antigens taken up by the mammary gland have the opportunity to react with cells both within the mammary gland tissue and/or in the local supramammary lymph nodes.

Our studies have shown that when antigens are infused into dry mammary glands, antigen is taken up extremely rapidly from the cavity of the gland. We infused two different types of antigen into dry cows 1) ovalbumin (or egg white) which is a soluble protein antigen and 2) killed *Streptococcus uberis* bacteria. Both types of antigen could be identified in the supramammary lymph node within one hour of infusion. The soluble protein antigen ovalbumin, identified by a specific staining method, was found accumulated in the outer part of the lymph nodes in areas where B cells are concentrated, whereas *S. uberis* could be seen in the centre of the lymph node, where the phagocytic macrophage tends to be the predominant cell type. This shows that antigens do not distribute evenly throughout immune tissue and this may reflect previous exposure to the antigen or different handling mechanisms depending on the form of the antigen.

Since expression of immune recognition molecule is essential in the early immune response it was important to investigate if cells within the mammary gland are capable of this function. Using an immunological staining technique, cells expressing the molecule can be identified. Cells within the supporting connective tissue of the gland, in the epithelium lining the gland and in the cavity of the gland itself, were found to express the immune recognition molecule. During this study, it was noticed that quarters which had been infused with *S. uberis* had the greatest amount of stained cells in both the connective tissue and the epithelium. Quarters

which had been infused with soluble protein antigen or control quarters which had not been infused, generally showed less staining (4). These findings may reflect that the cows were likely to have been exposed to *S. uberis* previously and that immune T cells released substances which induced the immune recognition molecules in the gland.

As the ultimate aim of vaccination is to provide protective immunity before natural challenge with bacteria has occurred, we must know how the unprimed individual (an animal which has never been exposed to the specific antigen) responds to vaccinal antigen. To begin to answer the question 'Can cells of the mammary gland participate in immune responses?' we decided to examine the role of cells derived from mammary gland secretions to act as antigen presenting cells.

It has been suggested that the mammary gland is generally immunologically compromised when compared to the rest of the body (7) and that cells in the gland may not be able to participate in immune responses effectively. Our aim was to take cells from mammary secretions and to investigate if they were capable of acting as antigen presenting cells in this primary system. We took cells from secretions as they are relatively easy to collect and as they are derived from cells in the mammary gland tissue, they should reflect their activity too.

Milk was collected by hand from cows in mid- or late-lactation which had been machine-milked one hour previously. Several cell types could be identified. These included epithelial cells which slough from the tissue lining the gland, neutrophils which digest bacteria and are usually present in large numbers during infection, lymphocytes which may be either T or B cells, and macrophages which also digest bacteria but which are considered to be the cells responsible for antigen presentation. The cell types were then further separated to leave just the lymphocyte and macrophage populations and these were used as the 'milk antigen presenting cells'.

In our study we measured the ability of T cells taken from the bloodstream of cows to respond to antigen in the presence of antigen presenting cells. The response was measured by proliferation of the T cells. Until recently, in order to detect T cell responses to antigen in the laboratory, it has been necessary to use T cells which had been exposed to antigen in the live animal, therefore, only secondary responses could be assessed. However, recent advances in other species have shown that it is possible to measure primary T cell responses in the laboratory (5).

Initially the response of T cells respond to antigen (in this case ovalbumin) was measured in the presence of "blood antigen presenting cells". When no antigen is present the T cells do not respond. When the antigen is added, the T cells show increased proliferation at day 4 of culture, peaking at days 5 and 6, after which the response declines. If the cells are then restimulated with the same antigen, a secondary response occurs which is characterised by a much faster T cell response, in this case peaking at day 2 of culture. When other antigens are added to the stimulated cultures, the T cells either do not respond or respond to a lesser degree, showing that the cells are responding specifically to the particular antigen to which they have been exposed (6).

When the 'milk antigen presenting cells' were added to the blood derived T cells it was found that the milk cells were capable of presenting antigen. The timing of the response was different, with the peak response occuring at day 9 of culture compared with the 6 days of 'blood antigen presenting cells'. The response in the presence of antigen was still higher than control cultures at day 12 of culture. However, five times more 'milk antigen presenting cells', than 'blood antigen presenting cells', were required to produce a response (5).

Conclusions

We have shown that both soluble and bacterial antigens are transported very rapidly to the regional mammary gland lymph nodes following infusion directly into the udder and that the antigens show different distribution patterns within the lymph node.

The bovine udder has been shown to be capable of expressing the immune recognition molecule which is required in order to produce a local immune response and we know that the udder of milking cows contains cells which are capable of taking up antigens and presenting them to T cells.

In our studies so far, we have found no reason to suspect that the bovine udder is not capable of responding to local introduction of antigen, although further work is required.

As the dry period presents the optimum opportunity for local infusion of a potential future vaccine, it is important to extend this work to examine the antigen presenting ability of cells from dry cows. At this time the volume of udder secretions is lowest, removal of secretion has stopped and cellular components of the secretion are most concentrated. Infusion of potential vaccines during the dry period may hopefully stimulate protection prior to calving, a time when the incidence of mastitis is known to increase (8).

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MILK SECRETION

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The British dairy industry stands at the threshold of a new era in milk marketing. We suggest that the same could also be said of milk production; major developments are waiting in the wings, led by the biotechnological advances that have been made in the last few years. This short review will examine one area in which significant progress has already been made, but where further changes are likely to happen; milking frequency. The effects on milk yield and udder development will be described, and the importance of a specific milk protein which acts to regulate milk secretion by feedback inhibition will be explained. The potential for devising new milk production strategies through manipulation of this inhibitor will be explored, and the possibility of using the inhibitor as adjunct therapy in the prevention and/or treatment of clinical mastitis will be briefly discussed.

Milking frequency, and feedback inhibition of milk secretion

Fact; milk yield is increased by more frequent milking, and decreased by less frequent milking.

Fallacy; these responses are controlled by the secretion of milking-related galactopoietic hormones, or else by pressure within the udder.

The first of these statements can be accepted at face value for the time being, although we shall add some detail later. The second statement is more controversial, and requires immediate explanation and validation. Experiments in goats performed some ten years ago established the local nature of the frequent milking response; thrice daily milking of just one gland increased yield only in that gland (1). This was clear evidence against a hormonal mechanism, which would have affected both glands equally. Pressure effects were then discounted by repeating the unilateral three times daily milking whilst reintroducing an inert solution into the gland at the extra milking, in order to maintain intramammary pressure. The stimulatory effect was still evident, proving that it was the actual removal of the milk which was important (2).

The existence of a secretion-inhibiting substance in milk had been proposed by Linzell and Peaker (3), and the search for its identity now began in earnest. Milk whey constituents were fractionated according to molecular mass, and the various fractions were infused into the udders of lactating goats. Inhibition of milk yield was achieved with the fraction comprising components of 10-30 kDa, and this inhibition was both concentration-dependent and reversible, thus fulfilling the major criteria for a biological control mechanism (4). The same fraction was also shown to reduce milk yield in rabbits (5), and to inhibit the secretion of both lactose and casein by rabbit mammary tissue cultured *in vitro* (6). This latter observation was particularly important; it demonstrated that the effect was a direct one of the milk fraction itself, and it formed the basis of a bioassay which has been used in further purification of the inhibitory activity. As a consequence, the goat inhibitor has now been identified as a small molecular weight protein (7) and a similar protein which also has inhibitory properties has been found in cow's milk (8).

Milking frequency, and milk yield

It is well established that milk yield is increased by milking more than twice daily. As a specific example, we cite data from two recent collaborative experiments between ourselves and the Institute for Animal Health, Compton (9, 10) (Figure 1). Both employed a split-udder design, whereby half of the udder (two diagonally opposed quarters) was milked four times daily, whilst the other half remained on twice daily milking as a control.

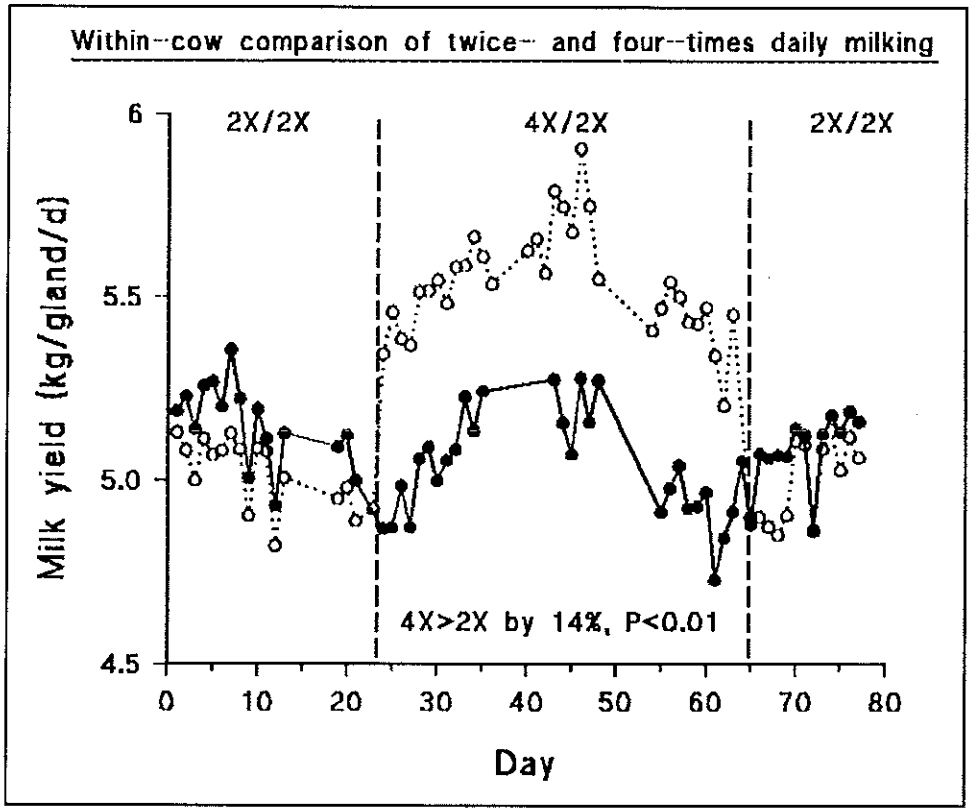

Figure 1. Stimulation of milk yield by frequent milking. Half of the udder (dotted line) was milked four times daily rather than twice daily where shown.

Four times daily milking increased yield by 10.4% in the first experiment and by 13.8% in the second, similar effects to those produced by thrice daily milking (11-13). The response is rapid, occurring within hours (14), can be elicited at any stage of lactation and continues for as long as the treatment is applied. There would be a clear advantage to be gained if the effect were to persist after the frequent milking ceased, and in some cases there is evidence for just such an occurrence. In one experiment, lactation persistency (the rate of decline in milk yield with time after peak lactation) was improved by frequent milking, so that although the incremental and decremental changes in moving to and from four times daily milking were the same, the net effect was that the treated udder half continued to produce more milk than the control half after the treatment finished (9).

Does milk yield increase further with frequencies greater than three times daily? We have not specifically compared three times and four times daily milking, but would anticipate statistically indistinguishable responses from the two. We are aware of one study in which groups of cows were milked either three times or six times daily for the first six weeks of lactation, and in this case the six times daily milked cows produced approximately 20% more milk (mean daily production over the six weeks of 35.3 kg/d for three times milked and 42.6 kg/d for six times milked; U. Bar-Peled, personal communication). Interestingly, six times milking produced a highly significant carry-over response, which continued at least 12 weeks after the treatment finished.

There is now some interest in milking once daily, or three times in 48 h. Studies in New Zealand have reported production losses of 10-25% over short periods of once daily milking (15) and 35% over a full lactation (16). Figure 2 shows data from an experiment in which mid-lactation cows were milked once daily for seven days (17). Yield was reduced from a mean of 21.21 (SE 0.82) l/d to 16.37 (SE 0.97) l/d, a fall of 22.8% There appeared to be no long-term detrimental effects; yield recovered completely within 48 h when twice daily milking resumed and lactation persistency was not affected.

Milking frequency, and variation between cows

Let us regard the udder simplistically as a two compartment structure comprising secretory alveolar tissue and cisternal storage tissue, and further, let us be quite clear that storage of milk actually occurs in both compartments. This is important in relation to feedback inhibition; since the inhibitor must be in contact with the apical membrane of the secretory cell in order to be effective, it follows that only milk which is stored within the secretory tissue will have inhibitory activity. Consequently, milk yield and yield responses to altered frequencies will be influenced by the size of the cistern relative to the mass of secretory tissue. For a constant secretory tissue mass, milk yield is correlated with cistern size (at least in goats; (18)), and because they are normally less affected by the inhibitor, cows with large cisterns respond poorly to frequent milking (19), but tolerate once daily milking well (17).

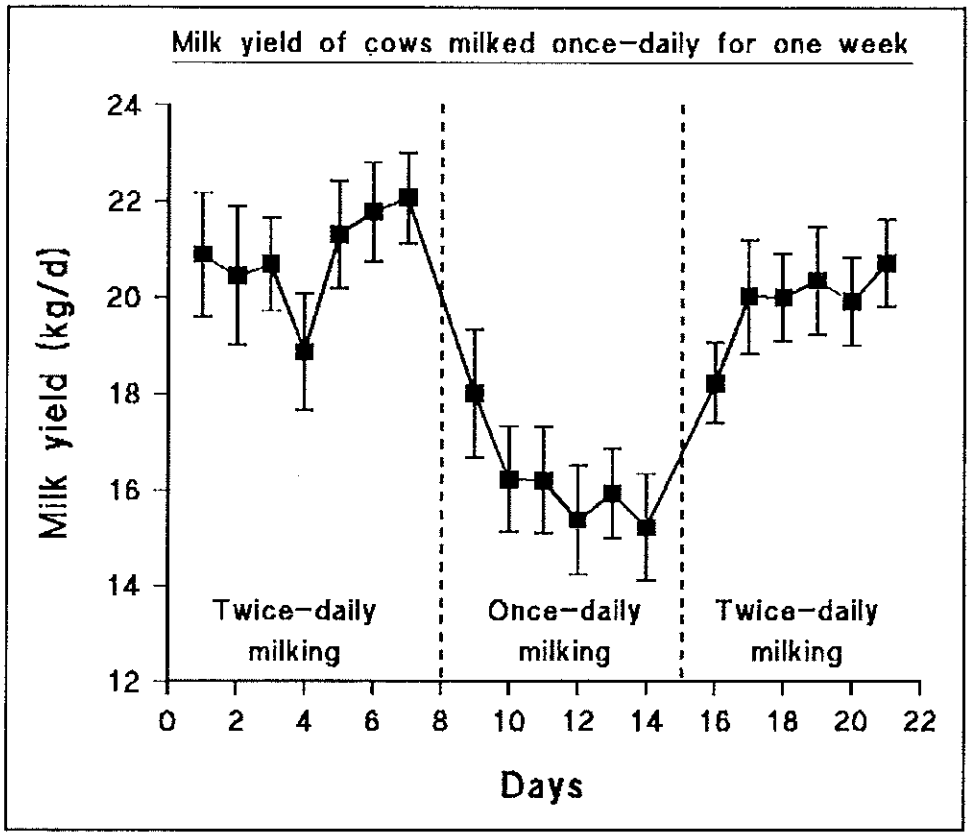

Figure 2. Inhibition of milk yield by infrequent milking. Milking frequency was reduced from twice daily to once daily where shown.

Milking frequency, and mammary gland development

The textbook tells us that mammary development is essentially a pre-partum process, and that the lactating udder cannot grow. The textbook is wrong! There is now ample evidence for lactational mammary growth under a variety of circumstances (20), one of these being as a response to increased milking frequency. Frequent milking has a triphasic action. The effect on milk yield is immediate, in the short term (days to weeks) this increased yield is achieved through an increase in the activity of preexisting secretory cells (ie cellular differentiation), and in the longer term (weeks to months) the frequently milked gland actually grows in size (21). Increases in the cellular content of specific milk synthetic enzymes appears to be a direct consequence of more frequent removal of the inhibitor, since there is *in vitro* evidence for an antidifferentiative effect of the inhibitor (22). There is no evidence for the inhibitor having any anti-proliferative action on mammary cells, and it may be that the longer term

growth response is related to the presence of other bioactive milk constituents; a mammary derived growth inhibitor has been described, for instance (23).

Effects of milking frequency on mammary development have primarily been described in goats. In cows, we have obtained evidence for increased tissue differentiation in four times daily milked glands (9), but the long term experiments required to demonstrate a growth response have not yet been done. Furthermore, whereas body imaging techniques have been used to monitor mammary growth non-invasively in live goats (24), the only reliable means of conducting this kind of work in cows would be as a terminal experiment; they are simply too big for imagers.

Milking frequency, and future strategies

Frequent milking brings economic rewards but also increased costs. Infrequent milking reduces fixed costs, but decreases income and could conceivably compromise animal health. In both cases the disadvantages may outweigh the advantages, so it could be argued that new frequency-related strategies are non-starters. As we shall now see, this could not be further from the truth.

Intensive engineering research being conducted in the UK and throughout Europe will almost certainly lead to the availability of robotic milking systems within the next 5 years or so (25). Two approaches are envisaged. In one, cows will be milked individually in unattended robotic stations at a frequency chosen by the cow (research suggests 4-6 times daily; Rossing (26)). In the other, the robot will milk batches of cows in conventional parlours (most probably of the automated tandem design), removing the drudgery of teat cup attachment and again conceivably opening the way to milking four or more times per day. The perceived benefits are, for the cow, improved health (through automated monitoring), welfare (a more natural milking frequency) and choice, and for the farmer, increased yield, more time for genuine husbandry and more sociable hours.

The physiological and behavioural research which is essential to support the robotic concept is only just beginning, but it should be evident to the reader that the consequences of milking, say, six times a day could be very far-reaching. Milk yield will undoubtedly increase, and as a consequence so will the energetic commitment demanded of the cow. Time available for husbandry will increase, and the good dairyman will make excellent use of that time. He will need to, for health and nutritional management will have to be first rate. Reproductive management is an imponderable. Will cows come into season, if milked so frequently? On the other hand, should we want them to come into season? The evidence is by no means definitive, but there are indications that lactation persistency will be increased markedly by very frequent milking, particularly if used in combination with other galactopoietic stimuli such as growth hormone (27) or oxytocin (28). The whole concept of 305 day calving intervals will certainly need to be re-examined.

Whilst a proportion of future dairy farmers are likely to produce milk in very intensive and very efficient systems utilising robotics, feedlots, biotechnology and computers, it is also just as likely that a proportion will take the opposite road, of extensification. To do so, there will inevitably be a need to reduce fixed costs, including labour costs, hence the attraction of once daily milking. There are welfare arguments in favour of such a system too; the cow will be

disturbed less and spend more time at pasture. However, most cows need to be milked at least twice daily for comfort and efficiency, so if once daily milking is to be successful it will have to be applied only to appropriate cows. The suggestion has been made that particular breeds might be better than others; Jersey cows over Holsteins, for instance. This is not supported by more recent data (S.R. Davis, personal communication). We have refuted any suggestion that low yielding cows will necessarily tolerate once daily milking better than high yielders (17), and instead have demonstrated the absolute importance of cistern size (see above). The next step is to develop a simple technique for measuring cisternal capacity on farm, since this will allow allocation of cows to appropriate milking strategies, and in the longer term will enable selective breeding for a demonstrably efficient biological characteristic.
The third way in which advances will be made is in manipulation of the feedback inhibitor itself. Immunoneutralisation of the inhibitor could potentially allow the benefits of more frequent milking without the additional milkings. On the other side of the coin, the inhibitor itself might be used, judiciously, as a peak-lactation yield limiter in once daily milked herds.

Milking frequency, and mastitis

This topic was reviewed expertly at the 1991 British Mastitis Conference (29), and since we are not mastitis experts it would not be sensible for us to repeat the exercise. Instead, we shall restrict ourselves to (speculative) consideration of two possible roles for the feedback inhibitor of lactation in mastitis therapy.

Invasive challenge to the mammary gland results in activation of a number of host defence mechanisms culminating in the inflammatory response; mastitis. One consequence will normally be a reduction in milk yield, but it is not clear whether this decrease in secretory activity forms part of the defence mechanism. Indeed, we simply do not know whether recovery would be more rapid if the gland were in full secretion, or if it were partially 'shut down'. On purely energetic principles, the latter would seem to be preferable, since the cow would then be able to direct more of its efforts to fighting the disease. A reduction in secretion rate leads to an effective increase in pathogen concentration within the udder, but whether this is detrimental or might actually be beneficial in accelerating the inflammatory response is open to question. ('Flushing' of pathogen away from the udder by regular, preferably frequent, milking is a separate issue, and is undoubtedly essential for speedy recovery (29)). It is, therefore, at least conceivable that the feedback inhibitor itself might have a useful role to play in mastitis therapy, simply by 'turning off' secretory activity. Equally, once recovery is under way then the ability to neutralise the inhibitor could be valuable in speeding up the return to full yield.

The inhibitor may be useful at drying off. Simply by virtue of its yield limiting activity, higher producing cows could be dried off more comfortably at the correct time relative to calving. It is also possible that the inhibitor might have a direct action, for instance in the phagocytosis of dead secretory cells. Until the purified inhibitor is available in reasonably large quantities, such hypotheses are not testable, but the possibilities are very exciting.

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A NEW SLANT ON TREATMENT

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Introduction

The need to discard milk from cows treated with antibiotics for bacterial mastitis is one of the most common frustrations encountered by dairy farmers and their veterinarians. Surveys in the literature from different countries on economic consequences of mastitis agree that this is one of the most important costs of clinical mastitis (1). The period during which milk must be discarded is variable and depends on the drug used and the duration of its application.

These constraints foster an attitude to mastitis therapy that may be considered as self defeating. The pressure to minimise discard time and thus reduce costs and losses, results in shortening of the treatment period. The consequence of this is that bacteria may escape contact with the drug and therefore the number of treatment failures will increase.

Staphylococcal mastitis is known to be refractory to treatment. There are a number of reasons advanced for these treatment failures, the most frequent being resistance of the bacteria, bacterial sequestration in phagocytic cells (2) and also within abscesses (3). These abscesses within udder tissue can be of varying size, from microscopic to rather large, and drugs are limited in their ability to penetrate the fibrous capsule. Bacteria in the glandular network behind plugged ducts are also prevented from coming into contact with the antibacterial drugs. When these abscesses subsequently rupture or ducts unblock, the bacteria are shed and so the disease recrudesces. It is clear that reducing the duration of treatment decreases the likelihood of an encounter between pathogen and drug, and therefore reduces the probability of bacteriological cure.

In economic evaluation of mastitis costs, the greatest single cost is that of lost production due to subclinical disease (1, 3, 4). Surveys agree on this point despite the different criteria used to estimate such losses (1). Measures currently employed in attempts to control subclinical disease include routine hygienic precautions at milking, antibiotic therapy at drying off time, routine bacteriological surveillance with batch milking of uninfected before infected animals and culling of chronic carriers. Antibiotic therapy during lactation is rarely employed due to the requirement for disposal of milk.

An antimicrobial agent without a withdrawal period therefore has several immediate benefits for the dairy farmer.

The most obvious benefit is that when treating clinical disease, milk can be returned to sale as soon as it becomes normal, avoiding the aforementioned losses due to discard. Perhaps more interesting from the standpoint of general herd health is the ability to treat clinical disease during an extended period and also to treat subclinical disease during lactation. This would increase the percentage of bacteriological cures achieved and result in lower levels of subclinical infection, a reduced risk to other cows, lower cell counts, improved production, and cleaner, healthier milk for the consumer. It should be emphasized that such treatment during lactation could not be expected to replace the husbandry practices of hygiene and

surveillance currently practised. However it would greatly assist clearing up chronic subclinical infections in individual animals and avoid the need to wait until the dry period before treating them.

However products with the advantages associated with no withdrawal period outlined above must also fulfil other criteria. Since there is no withdrawal period there are by definition, residues present in milk. Therefore these residues must be totally innocuous to the consumer. One solution to this problem is to use compounds that are degraded in the digestive tract after consumption. Our investigations have led us into testing the suitability of peptides and proteins for therapeutic use in mastitis. Such proteinaceous agents would be hydrolysed by digestive enzymes to amino acids indistinguishable from those derived from other food sources. Therefore any residues present in milk would be harmless. This enzymic digestion should similarly obviate the effects of such agents on the gastro-intestinal flora of the consumer.

A further, and very important factor to be considered is the effect of such residues on milk transformation in the manufacture of cheese and yoghurt. These processes involve a bacterial fermentation of the milk and inhibition of growth of these bacteria will affect the quality of the end product. It therefore follows that any residues present in milk must have no compromising effect on these fermentation processes.

Sources of antimicrobial proteins and peptides

As already indicated our investigations have led us into considering a number of antimicrobial proteins and peptides as potential therapeutic agents. These derive from natural sources and have phylogenetic origins as diverse as mammalian cells, insects, amphibian skin, and bacteria (see Table 1).

Table 1

Agent	Source
Defensins	mammalian leukocytes
Cecropsins	insects
Sapecins	insects
Magainins	Amphibians
Bacteriocins	Bacteria
Lytic Proteins	Bacteria mammalian leukocytes

A panel of some 16 available agents was tested for antibacterial activity against strains of *Staphylococcus aureus* and streptococci derived from cases of bovine mastitis.

The test was run in brain heart infusion medium and additionally in the presence of milk. Of those agents that had activity against the mastitis pathogens in culture medium few maintained that activity in milk. Two bacteriocins known as Ambicins were least inhibited by the presence of milk. Clearly a minimal requirement for a candidate agent for use as an intramammary therapeutic agent is that it should be active in the presence of milk.

Ambicins N and L

The two compounds that were selected for further investigation and with which we are currently engaged were identified during this phase of our work. These two compounds are known as ambicin L and ambicin N. A protein and a peptide respectively, they are derived from bacterial sources. They were pioneered for therapeutic uses by Applied Microbiology Inc., New York, New York USA. We are investigating their potential use in clinical and subclinical mastitis.

Ambicin L

Lysostaphin, a protein derived from *Staphylococcus simulans* has a molecular weight of 24,000. Its gene has been cloned (5). Ambicin L® (Applied Microbiology, Inc., New York) is a recombinant preparation of Lysostaphin produced by a strain of non pathogenic *Bacillus sphaericus*. Ambicin L has a narrow spectrum of activity being active only against staphylococcal species.

Lysostaphin has been employed in efficacy studies in a number of staphylococcal infections including peritonitis (6) and pyelonephritis (7) in mice to effects on chronic nasal carriage of staphylococci in man (8), and bovine mastitis (9). In these studies no significant adverse effects have been reported.

In a report on the immunogenicity of recombinant lysostaphin (10) it was shown to have no immunogenic effect by the oral route and after intramammary dosing system such effects were only detected after long term, high level dosing.

The use of lysostaphin alone as a mastitis treatment is an attractive proposition. Its narrow spectrum of activity would result in its not having effects on bacteria used in manufacture of cheese and yoghurt.

However mastitis due to *S. aureus* is not readily distinguishable on clinical presentation from mastitis due to other aetiological agents, notably streptococci. Use of lysostaphin with its restricted antibacterial spectrum without precise bacteriological diagnosis would leave these other infections untreated. The clinician cannot afford to wait for bacteriological results before he begins treatment. To maximize the chances of successful treatment it is essential that it is begun promptly upon clinical presentation. Research is currently proceeding into means of rapid, simple, and cheap identification of bacteria causing mastitis in individual cows. To date these tests rely on detection of antibodies to bacteria in milk (11). Antibodies are a consequence of infection and appear some time after an animal has become infected and so cannot indicate precisely what organism is causing disease on a particular day. Such tests will be very helpful in facilitating herd survey work but cannot currently aid in choosing a specific treatment for individual cows.

For these reasons it is important that therapeutic preparations for use in bovine mastitis must have a range of activity that at least covers staphylococci and streptococci.

Ambicin N

Nisin is an antimicrobial peptide of molecular weight 3,400 and is produced by certain strains of *Lactococcus lactis* by fermentation. Ambicin N® (Applied Microbiology, Inc. New York) is a highly purified form of nisin. In the form of nisaplin, an unrefined preparation, nisin is widely used as a food preservative and has been approved for use in some dairy products in USA (12). Extensive toxicological studies required to achieve this approval demonstrated no toxicity. Nisin is a member of a family of Lanthionine containing bacteriocins and its spectrum of activity is broader than that of lysostaphin and includes clostridia, streptococci, corynebacteria in addition staphylococci (13, 14). With suitable formulation this spectrum can be extended to include some gram negative bacteria (15).

Synergistic interactions are reported in vitro between lysostaphin and nisin, and between each of them and non ionic surfactants (15).

The relative broad spectrum of nisin while it brings therapeutic advantages already detailed also brings with it the potential for interference with milk fermentation. We performed experiments where the acidification of milk with commercial strains of *Streptococcus thermophilus* and *Lactobacillus bulgaricus* under the influence of different concentrations of nisin and commercial intra mammary antibiotic preparations was monitored. We found that these strains of yoghurt producing bacteria were less sensitive to nisin than to equivalent doses of an ampicillin cloxacillin combination, by several orders of magnitude. Despite these findings the goal must always be to reduce the quantity of each active ingredient to the minimal level consistent with efficacy. To this end we will exploit any positive interaction between these two agents be it synergistic or additive.

We have investigated the potential efficacy of these two ambicins in a model of subclinical staphylococcal mastitis. Lactating dairy cattle are enrolled into the experiment after 2 successive milk samples from each of their 4 quarters have been demonstrated free from mastitis pathogens on bacteriological culture. These quarters are then inoculated with 50-100 colony forming units of *S. aureus*. We currently use the Newbould 305 strain though other mastitis derived strains have been used.

During the two weeks following inoculation, quarter milk samples are taken 3 times to identify those quarters that have become persistently infected. These quarters are then assigned to specific treatment groups which comprise different ambicin combinations, negative controls and as positive controls we use a combination of ampicillin and cloxacillin (Ampiclox, SmithKline Beecham). The same treatment is assigned to each quarter of an individual cow, and the treatment groups are each milked by a different machine. These precautions are taken to prevent cross infection between groups during the post treatment surveillance period. The sacrifice of "blindness" in this protocol we consider acceptable since our major criterion of assessment, bacteriological cure, is objective. After treatment, quarter milk samples are taken daily for 14 days and subjected to bacteriological culture. Results from a series of experiments using unformulated ambicins are summarised below (Table 2). The % cure is the percentage of quarters becoming culture negative for *S. aureus* and remaining so until 14 days after treatment. The range of % cure in this table represents

the variability of % cure from one experiment to another. The median and overall % cure are estimates of central tendency.

Table 2

Summary of results from a series of experiments comparing efficacy of bacterial cure between a combination of unformulated ambicins, ampiclox and placebo treatments in a model of subclinical staphylococcal mastitis (all treatments were applied 3 times at successive milkings).

Treatment	Number of Experiments	Number of Quarters	Overall % Cure	Median % Cure	Range of % Cure
Placebo	7	58	10	17	0 - 40
Ampiclox	5	34	50	50	29 - 67
Ambicin N 30 mg Ambicin L 10 mg	2	20	70	73	58 - 88

From these results ambicins are seen to perform at least as well as the ampicillin, cloxacillin combination in an unformulated state. Work is currently in progress to optimise and both the dose and formulation of these compounds.

Conclusion

The ambicins, identified in an *in-vitro* screen as potential candidates for a novel intramammary therapeutic agent have upheld their promise in *in-vivo* model experiments. These proteinaceous agents have a rapid bactericidal action and a structural activity and toxicity profile that indicates they may form the basis of a product requiring no period of milk withholding after treatment.

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ABSTRACTS OF POSTERS

MILK FLOW RATE AND MASTITIS

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Recent reports from Compton have demonstrated the relationship between susceptibility to contagious mastitis and the rate of milk flow from the teat. However, flow rate can be dissimilar between quarters of any animal and varies significantly with yield during the lactation of a cow. This relationship does not occur for heifers; they have a similar flow rate throughout the lactation.

Flow rate is not the only property of the teat canal affecting the susceptibility of a quarter to infection. Colonisation of the teat duct by Staphylococci or Streptococci can provide a reservoir of infective bacteria. The risk of colonisation varies with the length of the teat canal, the integrity of its keratin lining and the chemical composition of that keratin. The relationships between such factors and susceptibility to infection are being studied and appear to differ between contagious and environmental bacteria.

AN UPDATE ON THE INCIDENCE OF SUMMER MASTITIS

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An annual survey of the incidence of summer mastitis in dairy herds in England and Wales was carried out over eight years 1984 to 1991. The herds surveyed are involved in a mastitis control scheme.

Summer mastitis is defined as an acute suppurative mastitis, often with a characteristic smell, in dry cows and heifers. The financial losses can be considerable due to the loss of the affected quarter and thus possible increased culling, and sometimes the death of the animal.

Approximately 300 herds were surveyed each year. On average 44% of herds were affected with 2.6 cases per herd. This is similar to the figure of 47% of herds affected found during the previous six years (1978-83). No three year cycle was apparent.

There was a considerably higher incidence of summer mastitis in dry cows (1.7%) and pregnant heifers (1.7%) compared to non-pregnant heifers (0.5%). In the 1991 annual survey 99% of herds used dry cow therapy and 67% of herds used some method of fly control. The use of pour-on insecticidal preparations increased from 2% in 1985 to 44% in 1991, partly due to their ease of use. It was not possible to compare the efficiency of the different fly control measures.

These data show that there has been little change in the incidence of summer mastitis over the past 14 years.

AN INVESTIGATION OF VERY LOW CELL COUNT HERDS

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A survey was carried out to investigate the mastitis control measures in low cell count herds. Eleven herds with an annual mean cell count below 70 thousand cells/ml were visited and a questionnaire completed. The farms were visited in February whilst the cattle were housed.

Herd size varied from 28 to 103 milking cows with an average of 45. Ten herds were milked by the farmer owner, the exception being milked by the owner in the morning and a herdsman in the evening.

Average herd yields varied from 5,300 to 7,000 litres per cow per lactation, with an overall average of 6,150 litres. All reported a low calculated clinical incidence, less than 30 cases per year. This is well below the national average of 40 cases per 100 cows per year.

Nine farmers followed the routine five point control programme: annual milking machine tests, prompt detection and treatment of clinical cases, dry cow therapy, teat disinfection after milking, and culling of chronic cases. The remaining two farms carried out all points except for a regular milking machine test.

Extra attention to detail was noted in all herds. Housing hygiene was good in all the herds visited, ten herds bred their own heifer replacements, in eight herds the cows stood after being milked to allow the teat sphincter to close, and one herd used the California Mastitis Test every month.

BACTERIA IN BULK MILK

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Genus Animal Health operate a bulk milk bacteriology service to identify the cause of high bulk milk Total Bacteria Counts (TBCs). This poster summarises the results of tests carried out on sets of samples from 125 herds submitted last winter, covering the period from October 1991 to March 1992.

Mastitis bacteria were associated with the high TBC in 74 herds (59.2%) and were the sole cause in one herd (0.8%). Milking hygiene was the most common single cause of TBC problems found in 30 herds (24.0%) and plant hygiene was solely responsible for the high TBC in 21 herds (16.8%).

PRE-MILKING TEAT DISINFECTION YES or NO?

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To halve new cases of mastitis infection caused by environmental pathogens apply a teat disinfectant before milking, say USA Research Workers(1). But their results were very variable between farms, with two out of the four showed no significant reduction in mastitis. Trials in the UK(2,3), indicate that pre-milking teat disinfection (PMTD) does not significantly reduce the rate of new infection caused by environmental pathogens.

Which herds might benefit from PMTD? - No one can answer that question, but the following might help.

1. Is there a clinical mastitis problem with environmental pathogens?
2. Does the existing hygiene routine and housing management include **Washing** and **DRYING** dirty teats before milking.
3. Is post-milking teat disinfection being carried out correctly (dipping is best) and with a good quality disinfectant?
4. Are the teats in good condition?
5. Is the milking machine/pulsation system working correctly.
6. Is the housing management good?, with passageways cleaned at least twice daily and back ends of the cublicles kept clean and dry.

If the answer to ALL the questions is YES, then maybe PMTD will help you. You could try your own 'experiment'. Split the herd into two groups, by pairing the cows on lactation age, stage of lactation and known history of mastitis, (Use coloured tail tapes to identify the two groups). Start at commencement of housed period.

The Pre-dip Routine - Fully dip **clean** teats with a pre-dip disinfectant, after a minimum of 30 seconds, dry wipe the teats. Remember you still need to disinfect teats **after** milking with an approved **POST-milking** disinfectant. Keep accurate records of all mastitis and perhaps compare individual cell counts too. PMTD will add 10-15 minutes/100 cows each milking.

Finally, PMTD is not a substitute for sensible udder preparation or good all round cow management. It **will not** work on contaminated teats or just on the teat ends. **It may not work at all in your herd.**

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SUBCLINICAL MASTITIS IN PROBLEM HERDS IN SCOTLAND

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A retrospective examination of data from some 58 herds was undertaken recently. These herds all in N.E. Scotland were subjected to an examination for subclinical mastitis because of either high Somatic Cell Counts (SCC), high Total Bacterial Counts (TBC) or complaints of a high incidence of clinical mastitis. The study covered 15 years up until 1990, but predominantly the middle 5 years. Milk from every cow in the herd was examined either using a composite sample from all 4 quarters (4475 samples), or individual quarter samples (4000). The SCC of each sample was also determined and other epidemiological data noted. A pathogenic organism associated with mastitis was isolated from all herds. In particular *Streptococcus agalactiae* was found in 62% of herds. The proportion of positive samples was highest in the composite group (39%) than quarter (14%), but the decision to take composite samples was not random. In any case the relative order of prevalence of isolates was the same, *Staphylococcus aureus* was the most common accounting for over half of all significant isolates followed by *S. agalactiae* and *Streptococcus dysgalactiae*. The presence of these organisms was associated with a significant rise in SCC and also in the age of the cow. It is evident that standard preventive measures were not fully implemented on these farms.

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A STUDY OF HIGH SOMATIC CELL COUNT HERDS IN SCOTLAND: PRELIMINARY RESULTS

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Monthly Somatic Cell Counts (SCC) and Total Bacterial Counts (TBC) from June 1990 of all 2500 SMMB producers were used to examine trends and to select 24 representative producers who consistently had a Bulk Tank SCC >400,000 cells/ml. Individual cow SCC data was used to selected individual cows for quarter sampling on the basis of a 3-month Geometric Mean SCC >283,000 cells/ml (Linear Score 5 and above). Eight hundred and fifty eight (36%) significant isolations were made from a total of 2392 quarter samples of which 474 (20%) were *Streptococcus agalactiae*, 229 (9%) were *Staphylococcus aureus*, 63 (3%) *Streptococcus dysgalactiae*, 24 (1%) were *Streptococcus uberis* and 10 (0.4%) were *Escherichia coli*. Multiple isolates were recovered from 59 (2%) of the quarter samples. Thus *S. agalactiae* accounted for 55% of all significant single isolations, *S. aureus* for 27%, *S. dysgalactiae* for 7%, *S. uberis* for 3% and *E. coli* for 1%. The prevalence of *S. agalactiae* is very much greater than was found in a group of 4 "control" low SCC herds which were also examined, or in a retrospective study of data from problem herds (see abstract Logue & others). As in the last mentioned study, the quarter SCC associated with a significant isolation was significantly higher than those with no isolate. Since June 1990 the average SCC for the 24 herds has decreased by 28% from 585,000 cells/ml in June 1990 to 420,000 cells/ml in May 1992 (control herds decreased by 11% from 153,000 cells/ml to 136,000 cells/ml for the same period) - adequate control measures consistently reduce the SCC, particularly in those herds with high SCC due to *S. agalactiae*.

A PILOT STUDY FOR A CLINICAL TRIAL OF AN ALTERNATIVE THERAPY FOR THE PREVENTION OF BOVINE MASTITIS

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Whilst alternative therapies are widely used for the treatment and prevention of bovine mastitis, there have been very few statistically valid studies to examine their efficacy. One reason for this is the difficulty of applying the normal rules for a 'scientific' investigation (random allocation to treatment or control groups, double-blind administration of different treatments, etc.) to alternative therapies.

This study was instigated a) in order to demonstrate that it was possible to perform a scientifically valid study of an alternative therapy and b) to identify the possible problems that might be encountered. The treatment chosen was a Nosode (a homeopathic dilution of mastitis-causing bacteria). Two solutions were prepared, one was the Nosode and the other a placebo which consisted just of water: these treatments were labelled A and B and given to the researchers without their knowing which solution was which.

Thirty cows from a small dairy herd were randomly divided into 2 groups. One group was given Treatment A and the other Treatment B: each treatment was given by mouth to individual animals using a disposable syringe. Apart from receiving the experimental treatment the cows were managed according to the farm's usual practice and all treatments (including Dry Cow Therapy and therapy for clinical mastitis) were given as usual. The same dosage regime was used for each group: the treatments were given twice a week for 2 weeks, then once every fortnight. Individual quarter milk samples were taken from each cow before the study commenced: these were subjected to bacteriological examination and their Somatic Cell Counts (SCC) were measured. Individual quarter SCCs were taken on 4 further occasions over the 4 months that the study lasted: bacteriological examination was performed on all quarters at calving, on all cases of mastitis and at the end of the study period.

The information that was collected made it possible to investigate whether there were any differences between treatment and placebo groups in the incidence of clinical mastitis, the quarter SCC, and the fate of quarters infected with bacteria.

This study has satisfactorily demonstrated that it will be possible to perform a scientifically valid full-scale clinical trial of an alternative therapy: it also provided useful indications of the resources required and the potential problems that might be encountered.

CELL COUNT CONTROL - THE ADAS WAY

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In November 1990, ADAS received a request to investigate a milk producer's Somatic Cell Count and Total Bacterial Count problem. Cell counts had been as high as 782,000/ml and Total Bacterial Counts fluctuated between 7,000/ml and 79,000/ml. Milk price bonuses and milk production were being lost. Many factors influence mastitis incidence and bacterial counts in the milk. ADAS believe in dealing with every aspect.

Milking equipment:

- Static and dynamic checks on the installation and performance of the equipment showed a need to improve pulsation and clawpiece size.

- A check on the efficiency of circulation cleaning of the milking plant and also of the bulk milk tank cleaning programme, highlighted inadequate water temperature and flow rate.

Milking routine:

- Observations of the operator routine showed that test spraying was practised but not accurately. Cluster position required improvement.

Cow housing:

- Assessment of cow housing led to improvements in cubicle design and management.

Use of records:

- ADAS Milk Check Mastitis Monitor was introduced to keep a record of clinical incidence.

- Interpretation was provided of individual cow cell count records as a basis for culling.

Staff:

- All staff involved with the dairy cows were instructed in the significance of all of the above points and given an opportunity to join discussions with consultant and with surgeon.

ADAS Mastitis Monitor shows a significant reduction in cell counts with 12 month rolling average reduced from 560,000/ml to 200,000/ml. The Total Bacterial Counts were now consistent at around 2,000/ml.

A METHOD OF MONITORING MASTITIS TO COMPARE THE ACTUAL AND THE EXPECTED RATE OF CLINICAL MASTITIS

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A prototype has been developed of an early warning control system for mastitis. This is part of a larger study into control systems for the management of milk production and common diseases in dairy cows. The mastitis records considered are the number of mastitis cases and the number of first mastitis cases in a lactation per 100 cows. The system will give a warning if either of these parameters becomes "out of control" in that its value lies outside the confidence interval around its expected value.

The basis for the calculation is the average target incidence the farmer wants to keep to. Expected values are calculated taking into account yield, age, stage-of-lactation, and the number of clinical cases of mastitis in the previous lactation and correcting for the calendar month. These adjustment factors were derived from data of 29 selected dairy herds, recorded on the Dairy Information System (DAISY) from the University of Reading.

The expected number of first mastitis cases per 100 cows has been found to rise by 0.085 with an increase of one litre in the average test-day milk yield. The expected overall number of mastitis cases per 100 cows increases by 0.29 for each rise of one litre in the average test day milk yield.

A comparison was made between expected and actual numbers of first and total cases of mastitis for a herd of about 200 cows. The standard deviation of the differences between the expected and actual number of mastitis cases per 100 cows was 2.411. For first cases after calving the standard deviation was 1.384 indicating a lower rate of accuracy of the model when predicting first cases.

The prediction system is currently being assessed through a questionnaire for farmers using DAISY asking them to indicate whether the number of first or total mastitis cases was unusually high or low at any stage in a 12 month period. This data will allow the model to be refined according to its sensitivity and specificity compared to the farmers' own methods of assessing mastitis problems.