MASTITIS IS AN ECONOMIC PROBLEM

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SUMMARY

Mastitis is an economic problem. As with many other cattle diseases, the economic damage of mastitis, either clinical or sub-clinical, can be brought down to a few categories: milk production losses, drugs, discarded milk, veterinarian, labour, milk quality, culling, clinical mastitis, sub-clinical mastitis and other diseases. Management decisions can be taken at various levels: the quarter level (e.g. drying off a single quarter), the quarter/cow level (e.g. treating clinical or sub-clinical mastitis), the cow level (e.g. culling a cow with clinical or sub-clinical mastitis), the herd level (e.g. changes in management such as barn and milking hygiene) and the national or regional level (e.g. improving extension services). Using the basic cost elements around mastitis and mastitis management, costs and benefits can be calculated for different circumstances. The average costs under Dutch circumstances of a case of clinical mastitis are estimated to be €277 and €168 for cows in early and late lactation respectively. For UK circumstances the average costs are estimated to be £203. On average, treatment of chronic sub-clinical mastitis caused by *Streptococcus uberis* is €15 more expensive than not treating. The outcome of this calculation is very dependent on assumptions regarding transmission of infection. Blanket dry cow therapy, as a strategy is economically preferred over selective dry cow therapy. Factors affecting this decision are the level of intramammary infections during the dry period, the cost of antibiotics and the (spontaneous) cure rate. However, it is difficult to translate this type of generic advice to individual farmers. Many times generic economic calculations are not specific enough. Each cow, farm and region differs for production circumstances and price levels. Therefore, economic calculations should be as specific as possible.

INTRODUCTION

Mastitis is an animal welfare problem. Also, mastitis might be a food safety problem, but mastitis is clearly an economic problem. Being an endemic disease on dairy farms all over the world, mastitis is an important cause of a less efficient milk production. Moreover, mastitis affects milk quality directly through changes in technical and hygienic milk quality and indirectly through the intrinsic milk quality. This makes mastitis a concern for the dairy industry. Mastitis management, therefore, should have the goal of improving milk quality and the efficiency of milk production and thus make the production of milk more sustainable. Given the multi-factorial nature of

mastitis, management consists of a wide range of activities, amongst others the treatment of diseased cows (clinical or sub-clinical), dry cow therapy, prevention of transmission of infections (either from cow to cow, or through the environment) and improvement of the immune system. There is much scientific literature on mastitis management. However, there is less scientific literature on the economics of mastitis and often this literature is a calculation of economic damage of mastitis, or the benefits of one or two management factors. Most studies are normative (using simulation modelling to estimate economic effects). Only a few studies are positive (using collected data to estimate economic effects).

Economic calculations for costs and benefits of mastitis and mastitis management depend very much on the specific situation of a country or region. Therefore, clear economic statements are very hard to give. Recently, IDF published an extensive review on economic consequences of mastitis (16). The aim here is to present a comprehensive overview of economic considerations around mastitis management. First the economic damage caused by mastitis is described in general. In the second part of the paper economic concepts are illustrated at the cow level, herd level and regional level.

ECONOMIC DAMAGE OF MASTITIS

As with many other cattle diseases, the economic damage of mastitis, either clinical or sub-clinical, can be brought down to a few categories: milk production losses, drugs, discarded milk, veterinarian, labour, milk quality, culling, new cases of clinical mastitis, new cases of sub-clinical mastitis, other diseases. Although costs for these factors might differ between countries and regions, the economic principles behind these factors are the same and will be explained below.

Milk production losses

In both clinical and sub-clinical mastitis, there is a loss in milk production. There is a large amount of published research on these changes in milk production (8, 9, 19). Moreover, the loss in milk production does not only occur during the case itself, even after the mastitis case is cured, the milk production level of the cow stays lower. Milk production loss is not obvious to the producer, because this is milk never produced, and therefore never seen. It is a hidden cost or lost income opportunity. The economic damage of a lower milk production per cow depends on the structure of the farming business. First of all, milk payment systems may differ (payment based on amount of fluid milk or based on milk constituents such as fat and protein). Secondly, the calculation of the economic damage of decreased milk production differs between a quota system (e.g. such as in place in the EU, Norway or Canada) or a non-quota system.

In a quota situation, the production potential of a farm is in most situations the quota and not the herd size, therefore, the returns of milk sales are for a large part defined by the quota and the goal of the farmer is to produce the milk within the quota as efficiently as possible. With a decreased milk production a farmer has several options (depending on the legislation associated with the quota system):

- Milk more cows to fill the quota. In this case, economic damage is calculated as the additional costs to milk more cows. These costs are not easy to estimate and consist amongst others of additional feed costs, additional veterinary costs, additional labour and additional costs for use of the barn. Many times additional costs for the barn are nil. However, with a crowded barn, costs might be associated with a lower level of animal welfare. When the farmer uses the full capacity of a barn for additional earnings (for instance to raise heifers for sale), the costs associated with higher barn use are the decrease in earnings for these additional activities.
- Increase the production of the cows (e.g. by feeding more concentrates) to fill the quota. In some situations, milk production of the cows can be increased by application of a better (more expensive) feeding regime. Additional costs are associated with the higher amount of (more expensive) feedstuffs to do this. In some cases, depending on the management capabilities of the farmer, a higher milk production per cow can lead to more health disorders.
- Lease out quota to other farmers when the quota will not be filled by own production. In some quota systems, farmers can lease or lease out milk relatively easy. This makes the quota system much more flexible. When this is done due to mastitis because milk production has decreased, the returns from milk sales will be decreased and some savings might occur because of less feeding.

Drugs

This is a straightforward economic damage. Drugs, necessary to treat a cow with mastitis, cost money. Depending on the legislation and the infrastructure in a country, costs of drugs may vary between countries.

Discarded milk

Economic damage due to discarded milk is comparable with the damage of a decreased milk production. However, there is one difference; the discarded milk is actually produced by the cows, which means that feeding costs for that amount of milk has to be taken into account with the calculations. The economic damage of 100 kg discarded milk is therefore larger than for 100 kg decreased production. Although not advisable from a veterinary point of view (e.g. there is an increased risk of developing resistant bacteria in the calves), discarded milk is often fed to calves instead of milk replacer. This will save costs of milk replacer.

Veterinarian

Besides delivering drugs (in many countries), the veterinarian might have to spend time on diagnosis of a (clinical) mastitis case or supportive therapy.

Labour

Costs for labour are, from an economical point of view, difficult to interpret. Opportunity costs for labour, e.g. to treat an animal, may differ from farm to farm. When the number of hours of external labour can be decreased by preventing mastitis, the opportunity costs are easy to calculate: hours x hourly wage. When it is the labour of the farmer himself, opportunity costs are much more difficult to estimate. If the labour comes from his own free time, it is the value that the farmer himself gives this free time. If the farmer, because of mastitis, spends less time on other tasks, opportunity costs are the decreased income because less effective management. Finally, perception of the value of labour might be important. Treating mastitic cows, while other cows are waiting in the milking parlour, is work that a farmer does not like to do. So (s)he is willing to spend money to prevent that. Labour costs are not only made at the farm level. When there are national programmes or programmes by a dairy company to decrease the level of mastitis, these costs can be associated with mastitis.

Milk quality

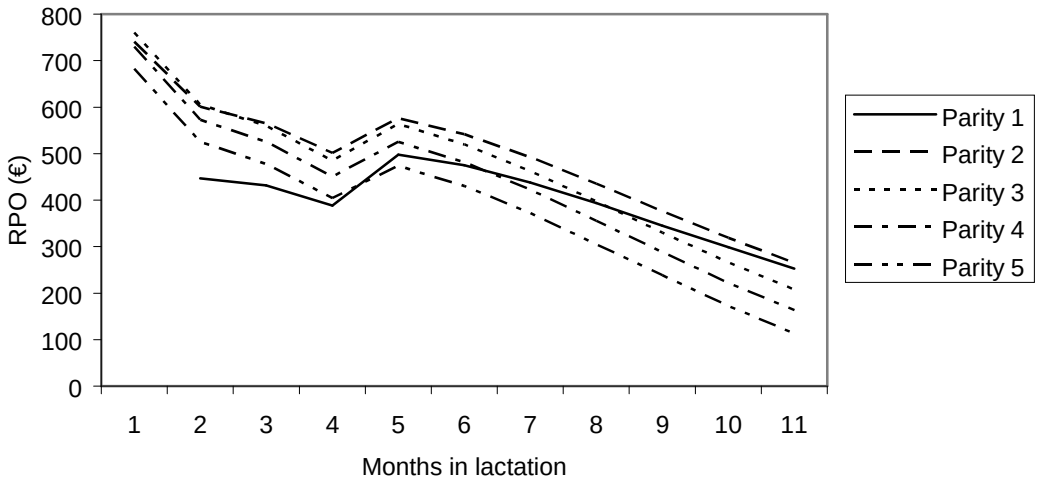
Mastitis influences the quality of milk. Some of these changes cause a less efficient processing of milk and might result in products with less favourable properties. Examples are an unstable and rancid taste of milk, a lower cheese yield and a decreased shelf life (14, 20). The associated economic damage is difficult to calculate and moreover, the direct effect of this economic damage for the individual dairy farmer is even more difficult to estimate. The only changes in milk quality that have a direct effect are the ones influencing factors that are part of the milk payment system, for instance bacterial count and somatic cell count. In most countries there is a regulatory limit for bulk milk bacterial count and bulk milk somatic cell count (BMSCC). In relation to mastitis, BMSCC is an especially important milk quality factor.

Besides BMSCC and bacterial count, most milk payment schemes test and apply penalties for antibiotic residues. Although the mastitis in itself does not affect growth inhibition, the use of antibiotics in treatment of mastitis does increase the risk of penalties. Different countries and milk processors use different rules for antibiotic residues, but the financial consequences of antibiotic residues in the milk can be considerable.

Culling

Cows with mastitis have a higher risk of being culled. The cost due to premature replacement of animals due to mastitis is probably one of the largest areas of economic loss. However, it is also a hidden cost. It is very difficult to calculate in a correct way (cf. 5, 10, 13). When a cow is culled, direct costs are the costs of rearing or buying a replacement animal (mostly heifers). Indirect costs are a decreased efficiency of milk production by the replacement animal, since the milk yield of multiparous cows is higher than that of primiparous cows. Moreover, the milk production of a heifer might be disappointing (heifers have a relatively high culling rate). On the other hand, there are also possible returns from culling a cow, mostly the price of meat. The costs of involuntary culling of a cow differ over time, depending on milk production, parity, lactation stage and reproductive status. This is illustrated in Figure 1, where costs of involuntary culling are given for different parities and lactation stages.

Figure 1 Costs (€) of involuntary culling as represented by the retention pay-off (RPO) for a cow under Dutch circumstances with a calving interval of 13 months and an average production level (source: Van der Walle (26), based upon the model of Houben *et al.* (10)



Clinical mastitis

For some management decisions, prevention of clinical mastitis can be an important benefit. Clinical mastitis in itself is not an economic factor. The factors as described above (milk production, drugs, discarded milk, labour, veterinarian, culling and milk quality) are the economic consequences of clinical mastitis. Much mastitis management aims at prevention of clinical mastitis. Specific management at the cow level can also prevent clinical mastitis in the same cow or can prevent spread of mastitis pathogens. Because of the contagious nature of mastitis, a cow with mastitis increases

the risk that other cows get mastitis. The costs of these new mastitis cases may be attributed to the original mastitis case.

Sub-clinical mastitis

By the same reasoning as for clinical mastitis, prevention of sub-clinical mastitis can be an important benefit of mastitis management at various levels.

Other diseases

An association exists between mastitis and other cattle diseases. The causal relation however, is difficult to determine. When the risk of other diseases is increased by mastitis, economic damage of other disease cases attributable to mastitis can be seen as economic damage of mastitis. However, this damage is very hard to establish because the interactions between various diseases are hard to establish and will not be further discussed in this paper.

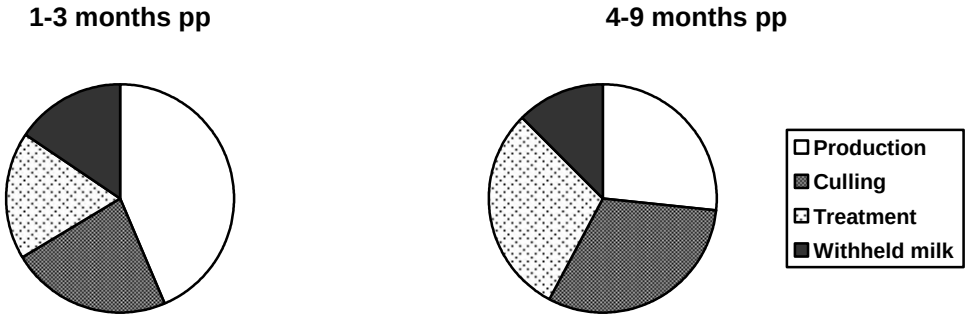
EXAMPLES OF ECONOMIC DECISION SUPPORT AT VARIOUS LEVELS

Economic calculations serve only one goal: to support decisions. As such, calculations should take two situations into account, for instance: doing nothing (*laissez faire*) and treatment. However, as a starting point (before comparing management alternatives) it is a good habit to get insight in the economic magnitude of a situation: how much does mastitis cost at this farm? A starting point for this is a calculation of the cost of a single case of mastitis. When there is room for improvement, decisions at various levels can be supported by economic calculations. This can be done at the cow level (e.g. instance treatment of sub-clinical mastitis), or at the herd level (e.g. selective dry cow therapy versus blanket dry cow therapy). Economic calculations can also be carried out at the regional level (e.g. investments in a mastitis control programme). Examples of various economic calculations are given later.

Economic damage of clinical mastitis

For the Dutch situation, the most recent cost estimations of clinical mastitis were made in 1997 (3). Factors taken into account for this estimation were: decreased production, culling, treatment (including labour) and withdrawal of milk. The economic damage of clinical mastitis depends, amongst others, on parity, lactation stage and causing pathogen. The economic damage of an average mastitis case was calculated as €277 and €168 respectively for a cow 1-3 months after calving and 4-9 months after calving. The distribution of the damage is given in Figure 2. The most important difference in the two lactation stages for the economic damage of clinical mastitis, is the difference in milk yield. Both production losses and kilograms of withheld milk are lower for a cow later in lactation.

Figure 2 Distribution of average economic damage of clinical mastitis for cows in the beginning (1-3 months *post-partum*) and the end of lactation (4-9 months *post-partum*)



Losses from clinical mastitis differ according to causative pathogen. Based upon the literature, de Vos and Dijkhuizen (3) estimated the economic damage of clinical mastitis for a cow early in lactation as begin €263 for *Escherichia coli,* € 270 for streptococcal, € 293 for staphylococcal and €272 for bacteriologically negative mastitis cases. Using this type of calculation (costs per clinical mastitis case) combined with incidence data from a specific farm, an estimate can be made of the total economic damage of clinical mastitis on that farm. However, one should not forget that assumptions underlying the factors used to calculate the economic damage of an average mastitis case may differ between farms as described earlier.

For the UK situation, estimates of direct costs of endemic animal diseases have been made by Bennett (1). The results of these calculations were at country level and existed both of direct costs and control costs. Based upon a spreadsheet available on-line (www.rdg.ac.uk/livestockdisea/), the economic damage of an average clinical mastitis case for UK circumstances was estimated to be £203. This is higher than the Dutch estimate. Partly this can be explained by different price levels. It is the author's opinion however, that the UK calculations are overestimated given the handling of costs of lower milk production.

Treatment of sub-clinical *S. uberis* mastitis

Traditionally, sub-clinical mastitis cases were not treated with antibiotics except during the dry period. However, recently this practice has been changing with some veterinarians regarding treatment of some types of sub-clinical mastitis to be effective. Various factors play a role in the cost-effectiveness of treatment, amongst others probability of spontaneous cure, probability of the cow becoming clinically diseased, spread of infection to other cows, cure rate under treatment and physiological effects of the infection. Since the decision on antibiotic treatment of sub-clinical mastitis involves much uncertainty and variability, the economic calculations were carried out with a stochastic Monte Carlo model (22). This model is based

upon an earlier described deterministic simulation model (7, 23) and simulates the dynamics of an infection for a cow known to have sub-clinical mastitis caused by *S. uberis*. Besides the effect of treatment on the infection status and economic damage of the cow, possible infections in other cows are also taken into account. The average economic damage (with basic input parameters) when a cow with chronic sub-clinical *S. uberis* mastitis (diagnosed after 2 subsequent cow somatic cell count measures above 250,000 cells/ml) is not treated is €88.47 (Table 1). With a short (3 day) treatment, the average economic damage was higher. With a long (8 days) treatment, the average economic damage was even greater. For the average cow, treatment is not economically efficient. Sensitivity analysis showed that this might depend on some specific cow and farm factors. Moreover, the spread of economic damage (Table 1) indicates that the risk of higher damage is much greater when a cow with chronic sub-clinical mastitis caused by *S. uberis* is not treated. This indicates that, for the (Dutch) average situation treating a sub-clinically infected cow with *S. uberis* is not cost-efficient. However, when for instance the costs of clinical mastitis are estimated to be higher, or when the risk of mastitis spreading is higher than in the average situation, or when the costs of culling a cow are higher, the optimal decision might change. Therefore a general answer cannot be given.

Similar calculations have been carried out for *Staphylococcus aureus* (24). In this study it was concluded that in general treatment of chronic sub-clinical mastitis caused by *S. aureus,* is also not cost effective. Cow factors and transmission of infection are important variables in this case.

Table 1 Total economic damage (€/cow with chronic *S. uberis*) for different treatment methods. Given are the average and the spread (extremes and percentiles)

Treatment	Average	Minimum	Maximum	5%	95%
None	88.47	0	1,149.26	7.52	416.12
Short	103.47	0	1,019.38	9.13	294.78
Long	142.85	0	1,240.14	8.90	234.33

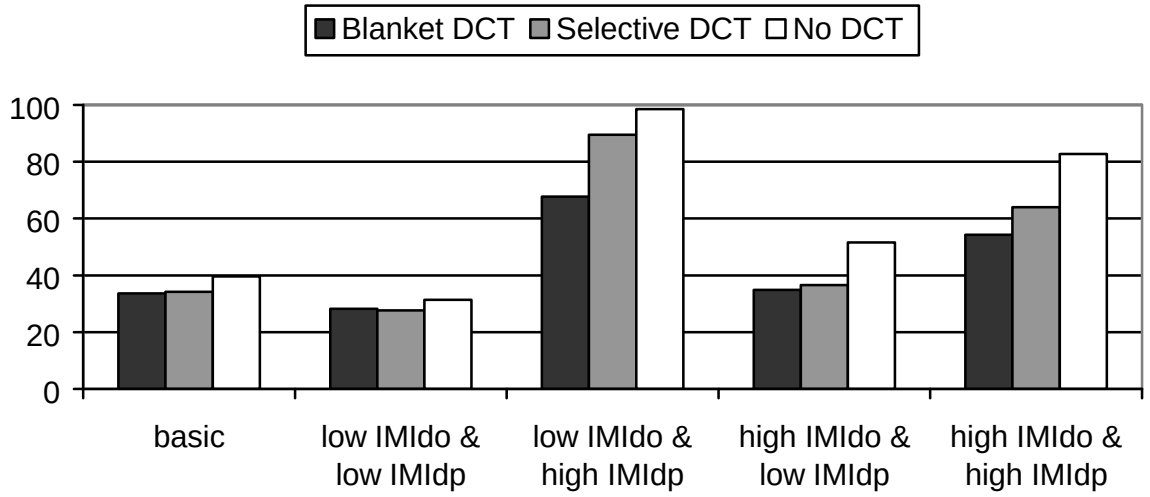
Optimal dry cow therapy

In many countries (including the Netherlands and the UK) blanket dry cow therapy (DCT) is the standard way to dry-off cows. However, due to concerns on antibiotic resistance, selective DCT is proposed as an alternative. An economic benefit may occur from using selective dry cow therapy, since not all cows have to be treated with antibiotics, which saves on the costs of antibiotics. An initial economic model on DCT indicated that for most circumstances blanket DCT was more cost-efficient that selective DCT (6). In order to account for variation and different types of pathogens, a new model was developed (11). This stochastic Monte Carlo model simulates the

dynamics of intramammary infections (IMI) around the dry period in order to predict the economic consequences of DCT for various types of pathogens (*Streptococcus agalactiae*, *Streptococcus dysgalactae*, *S. uberis*, *S. aureus* and *E. coli*). The parameters milk production, distribution of pathogens, risk of IMI during the dry period, prevalence of IMI at the moment of drying off, effectiveness of cow selection for selective DCT and probabilities of cure, prevention of new infections and economic values of these factors can be varied within the model. The probabilities for the basic situation were gathered by interviewing experts. The expert opinions are translated into a minimum, most expected and maximum value (Pert distribution) of the different probabilities.

For Dutch circumstances, the average costs associated with mastitis and mastitis control around the dry period were €39.62, €33.59 and €34.25 per cow for respectively no DCT, blanket DCT and selective DCT (basic situation in Figure 3). The largest proportion of these costs was caused by the costs of clinical mastitis after calving (92%, 65% and 85% respectively). However, the mastitis situation at a farm is an important factor when deciding on a DCT strategy (Figure 3). Moreover, costs of antibiotics and cure rates (spontaneous as well as after treatment) showed a large influence on the optimal strategy.

Figure 3 Sensitivity of costs (€ per average cow on a farm) for different strategies of dry cow therapy (DCT). Given are the basic situation and situations with a high level of intramammary infections (IMI) at drying off (IMIdo) and during the dry period (IMIdp)



In a calculation for the UK situation, using decision tree analysis, the use of blanket DCT showed to be highly favourable over selective DCT (2).

Mastitis control at a regional or country level

The type of mastitis control to be applied is usually regarded as something that the individual farmer should decide upon. However, it might be beneficial to establish regional or national programs in order to reduce the level of mastitis. Currently a number of initiatives to improve the udder health of farms in a country are undertaken (4, 17, 21, 27). The Dutch program (27) was initiated by the Dutch dairy industry in order to put extra focus on the production of health milk from healthy cows. A good udder health is regarded as one of the most critical factors of this. Besides that, it was estimated, using the calculations of de Vos and Dijkhuizen (3) that the yearly economic damage of clinical mastitis for Dutch farmers exceeds 100 million Euros, assuming a yearly incidence rate of 25% and 1.6 million dairy cows at risk each year. Investing a few million Euros in a project aiming at a reduction of clinical mastitis with 10% seems to be a good investment. However, figures like these (the 100 million Euros) should be handled with care, especially in a situation without a quota, where the free market regulates milk prices. A decrease of disease costs will result in lower production costs, which changes the supply curve. This change in the supply curve will result in lower prices. So a large part of the benefits of lower diseases costs in a free market system will be for the consumers and not for the producers (consumer surplus). In a quota situation, the shift of the supply curve is also influenced by the quota, so this consumer surplus effect is hardly a problem under quota circumstances.

DISCUSSION

In this paper the basic elements to calculate the economics of mastitis and decisions around mastitis has been described with some examples of the use of economic calculations for decisions at various levels. This paper does not provide a conclusive answer of the costs of mastitis and the benefits of certain mastitis management options. These costs and benefits depend on the specific situations (price levels, production circumstances) of a country region or the farm. Decisions might even differ from cow to cow, given milk production levels, age and reproductive status of that specific cow. Economic calculations should therefore be very specific. Current developments in the use of computers in dairy farming provide opportunities for farm or cow specific economic calculations. The elements described in this paper can be used to calculate costs and benefits of mastitis and mastitis management for different situations.

When economic calculations are used for decision support (which is the primary goal of animal health economics), there are a number of assumptions, such as transparency, perfect information and a clear definition of a utility function. Under these assumptions, the (rational) decision maker follows the most optimal advice. However, in reality, people take other decisions than the most optimal one from an economic point of view. Anecdotal evidence from veterinary practice does support these

observations. Neo-classical economists might argue that the problem and choices were not transparent, that there was no complete information, or that the definition of the used utility function was not correct. However, from the field of behavioural economics, where psychological insights are combined with economic theory, there is an argument that behaviour of people might be irrational from an economic point of view, but is rational from a psychological point of view. In this field many experiments are carried out describing the economic behaviour of, mostly, consumers (12, 15, 18). Since farms are small "family companies", the private household and the business are closely interrelated and the decisions are often taken by one person. Therefore, the economic behaviour of consumers and of farmers might be comparable. Finally, deserving more attention are the gain/loss disparity (consumers regard the value of a loss higher than the value of a gain, which shows some resemblance with cure or prevention) reasoning under uncertainty and the time preference of money (discount rates unconsciously used by consumers are much higher than the "economic" discount rates (25). Insight into this economic behaviour of dairy farmers can explain deviations of economic optimal behaviour. However, to enhance the profit of dairy farms, correct economic calculations for mastitis management remain very important.

CONCLUSIONS

Mastitis is an economic problem. The economic damage of mastitis, either clinical or sub-clinical, can be reduced to a few categories: milk production losses, drugs, discarded milk, veterinarian, labour, milk quality, culling, clinical mastitis, sub-clinical mastitis and other diseases. The costs for these factors might differ from farm to farm. Therefore, it is hard to give conclusive answers on the costs of mastitis and the benefits of mastitis management for individual farms. Management decisions can be taken at various levels: the quarter level (e.g. drying off a single quarter), the quarter/cow level (e.g. treating clinical or sub-clinical mastitis), the cow level (e.g. culling a cow with clinical or sub-clinical mastitis), the herd level (e.g. changes in management such as barn and milking hygiene) and the national or regional level (e.g. improving extension services). Using the basic cost elements around mastitis and mastitis management, costs and benefits can be calculated for specific circumstances.

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MASTITIS IS A WELFARE PROBLEM

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SUMMARY

Mastitis is a welfare problem. It is reported as being the most common disease of dairy cattle contributing to culling, and may be the cause of one in eight cattle deaths. Studies have demonstrated that even mild cases of mastitis result in alteration of pain processing. It is therefore important that cases of mastitis are identified and treated rapidly, with analgesics when appropriate.

INTRODUCTION

Mastitis is a common disease of dairy cows worldwide. In the UK the national incidence of clinical mastitis is approximately 40 cases per 100 cows per year (3, 10, 18, 19). The majority of dairy farmers are required to have in place herd health plans, for which incidences of disease are recorded. In recent years pressure has increased from Government and consumers to improve animal welfare, and reducing endemic disease is one aspect of this. The problem faced by farmers is quantifying and controlling pain caused by mastitis in the face of increasing manpower shortages.

Welfare

Over the last decade, partly as a consequence of the BSE crisis and the Foot and Mouth Disease outbreak, the Government has shown increasing interest in the health and welfare of livestock. The Farm Animal Welfare Council's (FAWC) third freedom is 'Freedom from pain, injury or disease' emphasising the importance of being able to quantify and control pain in mastitis. This has lead to the development of an 'Animal Health and Welfare Strategy', which aims to manage the impact of animal disease and improve the welfare of animals kept by man, whilst protecting the economic and social well-being of people and the environment. The aim of this initiative is to promote disease prevention and allow animals to produce higher yields, but at the same time remain productive for a longer period of time.

Traditional methods for disease detection rely on visual observation, however increasing herd sizes and a shortage of available skilled labour (1) has resulted in reduced manpower and increasing reliance on mechanised systems. Clinical mastitis is characterised by grossly abnormal milk and was traditionally detected by visual observation of the milk. Due to less time being available per cow, many farmers do not examine the foremilk, despite this being a legal requirement. The need for automated systems to monitor animal health and fertility has been recognised both for economic and

welfare reasons and there has been increasing interest in the use of biosensors (13).

Clinical mastitis

Clinical mastitis is characterised by observable inflammatory changes in the mammary gland, such as heat or swelling, alterations in the appearance of the milk (4, 14, 16) and systemic effects in some cases. Cases can be further classified as peracute, acute, sub-acute or chronic and are often also defined as mild, moderate or severe. Approximately 10% of clinical coliform infections result in peracute or toxic mastitis and 50% of the affected cows will die (9). Cows affected by the peracute form of mastitis are generally considered by veterinary surgeons and farmers to be in severe pain and distress, and veterinary surgeons often use analgesics in the management of the disease, especially the non-steroidal anti-inflammatory drugs (NSAID), in conjunction with antibiotics and supportive therapy. In stoical species such as ruminants, it has been difficult to assess whether less severe mastitis, such as a mild or moderate case, also causes pain, or if NSAID therapy would improve recovery. In field cases of clinical mastitis, it was reported that 2 g of ketoprofen administered intramuscularly once daily for a maximum of five days, significantly improved recovery from clinical mastitis in dairy cows; recovery was categorised as a return to at least 75% of the pre-mastitis daily milk production (17).

Culling

Mastitis is reported as being the most common disease of dairy cattle resulting in culling and may be responsible for 10% of premature disposals (7). A cow suffering clinical mastitis has an increased risk of being culled (5). In the UK mastitis was found to be the most common infectious disease contributing to culling rates, and was responsible for 10% of disposals (6). This increased in a linear relationship with increasing age of animal, with nearly 14% of cows culled for mastitis in their sixth lactation.

Assessment of pain in cattle

Pain assessment in animals and man is inherently difficult. It has been suggested that in animals, neonates and non-communicative humans, it might be the reaction to pain we are assessing, whereas in communicative man it is the perception of pain (2). Pain has been defined by the International Association for the Study of Pain (IASP) as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage' (11). Building on this, animal pain was described as 'an aversive sensory and emotional experience representing an awareness by the animal of damage or threat to the integrity of its tissues; it changes the animal's physiology and behaviour to reduce or avoid the damage, to reduce the likelihood of recurrence and to promote recovery' (12). Species-specific guidelines for the assessment of pain were suggested by a working party for the Association of Veterinary Teachers and

Research Workers to be used in the regulation of animal experiments (15). The assessment of the severity of pain was based on the evaluation of a wide range of parameters by trained and experienced observers (15). They suggested that cattle in pain often appear dull and depressed with little interest in their surroundings, can be inappetant with subsequent weight loss and, in milking cows, a sudden drop in milk yield. In severe pain, they noted that rapid shallow respirations were observed and that on handling cattle may react violently or adopt a rigid posture possibly designed to immobilise the painful region. Grunting and grinding of the teeth may be heard.

EXPERIMENTAL WORK

Over 100 dairy cows with naturally-occurring mild or moderate clinical mastitis were followed to assess pain associated with the disease. Clinical and laboratory parameters were assessed to evaluate their usefulness as objective markers of pain.

The heart rate, respiratory rate and rectal temperature of cases with moderate clinical mastitis were significantly higher than those of mild cases of clinical mastitis; demonstrating that moderate cases of mastitis resulted in altered physiological responses. Cases with only one quarter affected were recruited, allowing the response to mechanical stimulation of the hindlegs on the affected and unaffected sides to be compared. Results from mechanical threshold testing showed that there were differences in the response to mechanical stimulation between the ipsilateral leg and the contralateral leg. In other words, alterations in the pain processing pathways are a feature of acute clinical mastitis in dairy cows. The use of the NSAID, meloxicam, had a significant beneficial effect on the pain threshold levels in cows with mild and moderate clinical mastitis. Cases treated with meloxicam reached normal threshold levels earlier than cases treated with antibiotics only ($p=0.04$). The concentration of the acute phase protein, haptoglobin, in milk increased with increasing severity of clinical mastitis - a phenomenon that has also been reported in respiratory disease in calves (8). Prostaglandins are recognised mediators of inflammation that are released via the arachidonic acid pathway as a consequence of tissue damage and/or inflammation (20). Prostaglandin concentrations were significantly increased with increasing severity of mastitis.

The finding that meloxicam-treated animals reached normal threshold levels earlier than animals treated with antibiotics, only suggests that NSAIDs may help improve recovery in cases of clinical mastitis and that by alleviating the pain associated with mastitis, will improve welfare.

The fact that physiological and laboratory parameters were significantly different between mild and moderate cases of mastitis could be investigated further. This could lead to the development of non-invasive technologies to recognise early signs of disease. They could also be used to assess the most

appropriate therapy and to monitor the response to therapy. Such techniques would serve to reduce the impact of mastitis and improve the welfare of dairy cows.

CONCLUSIONS

Mastitis is an important and common disease of dairy cattle. Work must continue to reduce the prevalence of the disease and at the same time, to develop new techniques for the rapid identification of cases and for the quantification of their severity. The ability to assess clinical severity of mastitis may allow prompt use of more appropriate treatment for mastitis, such as parenteral analgesics and parenteral antimicrobial treatments. The use of some physiological and laboratory parameters as indicators of mastitis could have value in automated milking systems and be of particular value at a time of reduced manpower in the dairy industry. Increasing use of analgesics in all cases of clinical mastitis should be encouraged to minimise pain and improve recovery.

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HUMAN HEALTH RISKS ASSOCIATED WITH HIGH SCC MILK

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SUMMARY

Drinking high cell count milk and consuming dairy products made with high cell count milk does not pose a known, direct human health risk. However, the secondary relationships between human health risks and indirect factors such as ingestion of human pathogens, bacterial toxins, and residual antibiotics associated with high cell count milk cause potential alarm for consumer safety. Reducing mastitis and cell count in milk are both public safety and food quality initiatives.

INTRODUCTION

To question openly whether high cell count (SCC) milk poses a direct human health concern is important to reassure the protection of consumers. Limits on the maximum SCC in bulk milk collected from farms are applied in many countries with a common maximum limit of 400,000 cells/ml. In the United States the national standard for milk sanitation is contained in the Grade "A" Pasteurized Milk Ordinance, 2001 Revision. The NMC (formerly the National Mastitis Council) has sponsored proposals to the National Conference on Interstate Milk Shipments during four of the last five conferences to lower the legal limit of SCC in milk from the present value of 750,000 cells/ml. Discussions at the National Conference on Interstate Milk Shipments on the value of decreasing the legal limit of SCC in bulk milk led the Board of Directors of NMC to organise a symposium at the 43rd NMC Annual Meeting in Orlando, Florida on January 15, 2005. The purpose of the symposium was to report the current understanding of the possible connections between SCC in bovine milk and human health. Major points from the symposium are reviewed here.

HEATH RISKS

Direct effects of neutrophils

The review of literature presented in this paper did not reveal a known, direct health concern with consuming milk with an elevated SCC. The cells in milk are primarily leukocytes (white blood cells) and secretory cells. As the SCC increases, the percentage of cells that is the white blood cell type - neutrophil increases. In this strictest definition of the problem, any potential health risk of consuming milk with an elevated SCC would depend largely on the human health concerns of ingesting bovine neutrophils.

Although the ingestion of large numbers of bovine neutrophils in milk may be objectionable, direct negative effects on the safety of humans have not been described as a result of consuming dairy products made with milk having high SCC.

Transfer of pathogens

The primary cause of a high SCC in milk is intramammary infections. Neutrophils migrate from blood into the mammary gland in response to an infection. The vast majority of bovine intramammary infections are caused by bacteria. Many of these bacteria are also the causative agents of human diseases (e.g. *Escherichia coli, Staphylococcus aureus, Streptococcus agalactiae*). Fortunately, pasteurisation of milk kills the most common mastitis causing bacteria. Proper pasteurisation of milk is very effective in preventing the transfer of viable pathogens from milk of infected mammary glands to humans. However, emerging technology has proposed an additional epizootic path of infectious agents from cow to man despite pasteurisation of milk for public sale. Recent studies have indicated the transfer of bovine strains of *S. agalactiae* to human populations with devastating effects, apparently after the wide spread acceptance of pasteurisation (2).

Evidence has been reported that *Mycobacterium avium* subsp. *paratuberculosis* associated with Johne's disease in cattle and isolated from human patients with Crohn's disease, may survive some accepted milk pasteurisation procedures. Although the possible association between shedding of the *Mycobacterium avium* subsp. *paratuberculosis* in milk and subsequent survival after pasteurisation is compelling, the rate of shedding is low in infected cows and not related to an increase in SCC (5).

Pasteurisation reduces the number of viable microorganisms, but often does not destroy toxins produced by bacterial pathogens. The transfer of heat stable toxins produced by mastitis-causing pathogens in milk is a potential concern. Specifically, enterotoxin produced by *S. aureus* in milk of infected cows has been implicated in cases of food poisoning. As *S. aureus* continues to be a major cause of mastitis in many parts of the world, the frequency of enterotoxin production among strains of this species causing mastitis is a potential concern (1, 3).

Secondary relationships

The established ancillary relationships between SCC and human health concerns are possibly more problematic than the direct health concerns of consuming high SCC milk. Investigators have consistently reported a positive relationship between SCC of bulk milk and antibiotic residue violations (4). Consumption of milk products adulterated with antibiotic residues poses a potential catastrophic risk to people hyper-sensitised to the antibiotic. However, milk is screened for the presence of violating levels of antimicrobial inhibitors. In addition, verified medical cases of humans

having allergenic reactions due to consumption antibiotics in milk products are very rare. Transfer of antibiotic resistant bacteria to humans from milk is unlikely after milk is pasteurised and selection of resistant bacteria in humans after ingesting antibiotics in milk is only speculative.

The use of bulk milk SCC as an indicator of farm hygiene has been related to the potential for human health risk (1). A large and diverse group of human pathogens reside in the cow's environment (3). These microbes are often pathogens or normal flora of dairy cows. Evidence suggests that contamination of milk with most of these pathogens occurs during or after harvest of milk and is not due to intramammary infections. However, herds with high bulk milk SCC are more likely to have these pathogens infecting cows and present in elevated populations in the farm environment (3). The tempting inference is that farms ineffective in implementing hygiene practices to reduce bulk milk SCC tend to be ineffective in other farm hygiene measures aimed to reduce exposure of milk to human pathogens via routes other than intramammary infections. The relative risk to humans is minimised by pasteurisation that effectively destroys the majority of human pathogens that may reach the bulk milk as result of poor farm hygiene practices.

Unpasteurised milk and milk products

The greatest risk of high SCC milk to human health is in the consumption of unpasteurised or improperly pasteurised milk (3). Viable pathogens and their toxins can be transferred from the milk of infected quarters directly to humans. A potentially greater concern for consumer safety arises from transfer of pathogens from the environment during and after harvest into milk that is consumed unpasteurised. Surveys indicate that dairy producers and their families drinking milk produced on their own farms are among the demographic groups at greatest risk to food-borne diseases due to consumption of unpasteurised milk.

CONCLUSIONS

Consuming milk with a high SCC does not pose direct, specific health risks to people. In contrast, the relationship of high SCC milk with poor farm hygiene, antibiotics residues, and presence of pathogenic organisms and toxins offers insight into the potential increase in safety risk factors to consumers when high SCC milk is marketed. Currently, no model exists that will define the magnitude of decreased risk to consumers that would result from lowering the maximum limit of SCC in bulk milk. However, most reports indicate that lowering limits of SCC will positively influence acceptability and suitability of milk as measured by improved safety, milk quality, and value added products. In many countries, especially in European Union, safety, suitability and consumer acceptance each play comparatively important roles as driving forces for lowering SCC. Safety,

acceptability and suitability are not discrete properties of a product, but rather they overlap and intertwine when assessing risk to consumer health.

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USE OF ACUTE PHASE PROTEINS IN BOVINE MILK

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SUMMARY

Clinical and sub-clinical mastitis result in an inflammatory response and a variety of markers can be used to detect or monitor this response. Somatic cell counts are commonly used to indicate an infection. Milk amyloid A is an acute phase protein which increases in concentration in milk with an inflammatory response in the udder. The specificity of milk amyloid A is comparable to that of somatic cell count. Milk amyloid A may be a more sensitive marker than somatic cell count and influenced less by other physiological factors.

INTRODUCTION

It is important to be able to diagnose, monitor and predict disease and various factors can be used to assist these aims.

Stress and inflammation result in a wide range of biochemical, physiological and behavioural changes and these can be detected by a variety of methods ranging from markers of inflammation, haematological and microbiological parameters to biochemical variables. All of the parameters change at different rates in response to the initial insult and have both a wide range and species variability.

An acute phase response is triggered in response to tissue damage or infection and this can be detected locally or systemically depending on the organ(s) affected. The acute phase response is a non-specific, innate, immune response and has a protective role in limiting disease and injury by minimising tissue damage and enhancing rate of repair. It is stimulated by release of cytokines including Interleukin 1, Tumour Necrosis Factor α and Interleukin 6 from macrophages and monocytes (1).

To be classified as an acute phase protein there must be a change in concentration of at least 25%. In mammals all acute phase proteins increase in magnitude. Acute phase proteins include fibrinogen, serum amyloid A, haptoglobin, α -1 acid glycoprotein and C reactive protein. Species variation occurs in type and concentration of acute phase proteins.

Serum amyloid A is produced by hepatocytes and is a widely monitored acute phase protein in human medicine. It is also being used more widely in veterinary medicine (7). In the cow, extra hepatic synthesis of amyloid A has been reported including from mammary epithelial cells which produce

the isotype, milk amyloid A. Some leakage from the blood to the milk of serum amyloid A may occur in cases of clinical mastitis, but this is negligible in uninfected quarters. Uninfected mammary quarters have very low or undetectable levels of milk amyloid A and the levels rise in response to infection. This response is specific to the quarter and does not necessarily result in detectable levels systemically.

Generally, clinical mastitis can be detected by visual inspection of the milk and palpation of the udder, but sub-clinical mastitis can be harder to identify. The milk somatic cell count is regarded in the dairy industry as an important indicator of intramammary infection, especially sub-clinical mastitis. Other indirect methods include milk electrical conductivity, adenosine tri-phosphate, N-acetyl-β-D-glucosamine. While bacteriology may be considered a gold standard for confirmation of infection, problems may arise even with this, such as number of bacteria below the detection level used, or contamination of the sample. Milk amyloid A has been reported in the literature both for detection of clinical and sub-clinical mastitis.

This paper will compare milk amyloid A with both cell count and other detection methods for uninfected quarters and cows, sub-clinical and clinical cases of mastitis.

MATERIALS AND METHODS

Two data bases were used to analyse the comparative use of milk amyloid A with somatic cell counts, bacteriology, milk electrical conductivity and clinical data.

Trial one

Individual quarter milk samples (10-ml) from 25 cows were taken at the beginning, two minutes after the start of milking and immediately after milking had finished. The foremilk samples were analysed for bacteriology, conductivity, cell count (National Milk Records) and milk amyloid A (Tridelta Phase Series, Tridelta, Dublin). Cows were selected at random from the milking herd at the Institute for Animal Health.

For each milk sample the result of the test was known, but not the actual mastitis status. Therefore, a Bayesian approach was used to estimate the diagnostic parameters of each test and the prevalence of mastitis. For each sample the probability of the milk sample being from a case of mastitis can be calculated given the results of the diagnostic tests. For example, if all 4 tests are positive, the probability $p(m)$ by Bayes law of the source being mastitic is:

$$p(m) = \frac{prSe_{maa}Se_{scc}Se_{bact}Se_{ec}}{prSe_{maa}Se_{scc}Se_{bact}Se_{ec} + (1-pr)(1-Sp_{maa})(1-Sp_{scc})(1-Sp_{bact})(1-Sp_{ec})} \quad (1)$$

Where pr is the population prevalence, Se_{maa} is the sensitivity of milk amyloid A, Sp_{maa} is the specificity of milk amyloid A etc. Similar equations can be used for all other combinations of test results. By imputing starting values for all the parameters a Marcov Chain with Monte Carlo sampling can be constructed which will asymptomatically converge to give the values of these parameters given the data. The first iteration from the starting values, for each sample will give a probability that the sample is mastitic given the test result and the starting values. This probability is then used to generate a new set of samples. Each sample is randomly assigned a "true" mastitis status using a binomial random number generator with the probability given by the series of equations of which equation (1) is an example. From this set of samples the next set of parameters of prevalence, sensitivity and specificity can easily be calculated from the prevalence in this simulated data set and the diagnostic results given in the actual samples. From these new parameter values the next iteration of the Marcov Chain is calculated.

A macro was written in Microsoft Excel to automate this for a Marcov Chain Monte-Carlo. After a period of "burn in" of 1000 iterations, within which the chain had converged, the poster median parameter values and the 95% credibility intervals were calculated from a further 10,000 iterations.

Trial two

A longitudinal study on 21 dairy cows was conducted over 33 days. All cows were pregnant and yielding between 18 and 26 litres of milk daily. At the beginning, after 14 days and at the end of the trial and on detection of clinical mastitis, foremilk samples were collected aseptically from each quarter and examined for bacteria (5).

Representative samples (minimum 100 ml) of whole udder milk from each cow were collected daily from the parlour milk meters during morning milking. These samples were divided, after thorough mixing, into five aliquots with four of the samples sent to three laboratories (one laboratory received a duplicate sample on a three day rotation) and the fifth sample was sent for estimation of the concentration of milk amyloid A (Tridelta Ltd, Maynooth, Ireland).

At least three data points were available for the somatic cell count allowing this data set to be cleaned and remove erroneous data points due to laboratory errors; however, this was not possible for the milk amyloid results. Results were categorised either as no inflammatory response or as a potential inflammatory response by selecting cut-levels of 200,000 cells/ml for somatic cell count response and 800 ng/ml for milk amyloid A. This was at the cow level and not the quarter.

Using the results of the bacteriological analysis and clinical detection, cows were classified as uninfected, infected with coagulase negative staphylococci, *Corynebacterium bovis* and streptococci, or as having a clinical case of mastitis during the trial period.

RESULTS

Trial one

Using the Bayesian interpretation at the optimum cut off to optimise test performance the probability that the specificity of milk amyloid A was better than that of somatic cell count was 40%. Likewise the probability that the sensitivity of milk amyloid A was higher than somatic cell count was approximately 94%. The median specificity and sensitivity of all 4 tests at their optimum cut off values together with their 95% credibility intervals is given in Table 1.

Table 1 Specificity and sensitivity with credibility intervals at optimum cut off levels of 700 ng/ml of milk amyloid A and 150,000 cells/ml somatic cell count

Parameter	Specificity (credibility intervals)	Sensitivity (credibility intervals)
Milk amyloid A	0.97 (0.91-1)	0.93 (0.72-1)
Somatic cell count	0.99 (0.95-1)	0.69 (0.42-0.94)
Bacteriology	0.81 (0.71-0.88)	0.73 (0.49-0.91)
Conductivity	0.53 (0.30-0.77)	0.55 (0.45-0.66)

Trial two

Seven cows had persistent minor bacterial infections in at least one quarter affecting cell count and milk amyloid A throughout the sampling period. Five cows had an infection with either a streptococcus species or a coliform species either for the whole period or just detected at one sample point. Clinical mastitis developed in three of the cows due to these infections.

Transient rises in cell count and milk amyloid A were observed in the uninfected cows lasting on average 1.8 days

The cell count of milk correlated with the concentration of milk amyloid A. On 24 cow-days both somatic cell count and milk amyloid A were increased whilst on five episodes of these occasions the milk amyloid A response was one day later than SCC, the preceding milk amyloid A result was increased, but was in the range 500-800 ng/ml. On five occasions the milk amyloid A response was sustained for at least one day longer than the somatic cell count response.

On four cow-days the somatic cell count exceeded the threshold, but milk amyloid A was only 700 to 800 ng/ml (milk amyloid A 14% false negative results). For 12 cow-days somatic cell count exceeded the 200,000 cells/ml threshold, but no milk amyloid A rise was found (somatic cell count 33% false positive results).

DISCUSSION

The use of milk amyloid A as a marker of inflammation of both sub-clinical and clinical mastitis has been reported previously (2, 3). Differing values have been used for the cut-off level and this is also dependant on whether the cow or the quarter is the unit of analysis (4). Previously, interpretations used a cut-off value for milk amyloid A between 500-900 ng/ml as indicative of an inflammatory response. This cut-off is within the range indicated by the Bayesian analysis at the quarter level (Trial one) and the value used for the whole cow values (Trial two). Good sensitivities and specificities have been reported before (2). This analysis assumed all 4 tests were conditionally independent. This is reasonable as milk amyloid A is believed to be produced locally, inflammatory cells traffic into inflamed tissue although there may be subsequent proliferation of neutrophils. Bacteriology depends on the detection of bacteria in the milk whilst electrical conductivity depends on changes in the acid-base balance of mastitic milk.

Previous work has tended to focus on previously selected populations of cows either with sub-clinical or clinical mastitis, or at one time point (6). These two trials used groups of cows with no previous selection for cell count or previous mastitis history. While the sample numbers are small, the results are in agreement with other published work. Further work is necessary on a larger sample size of cows.

Variations in the levels of milk amyloid A in sub-clinically infected cows have also been reported. Some interpretative problems remain including using composite samples and levels of detection if only one quarter is sub-clinically infected. This did not appear to be an issue using the whole cow data in trial two, but this was on a small sample.

The effects of other factors such as stage of lactation, milk production or oestrous cycle on milk amyloid A have still not been fully elucidated.

Currently the milk amyloid A test is only available as an ELISA and is relatively expensive in comparison to somatic cell counts. However, it does merit further investigation and may be appropriate as a confirmatory test to be used following routine cell counting to investigate high cell counts cows. As it becomes used more widely within the animal field there may be further developments in the type of test available.

CONCLUSION

Both milk amyloid A and cell counts are non-specific markers of inflammation, hence a rise in either may not necessarily be accompanied by detection of bacteria, either due to limitations of bacteriology, or non-infectious inflammation or after successful treatment, but without incomplete resolution of all inflammation. However, a low milk amyloid A is generally indicative of a healthy uninfected quarter. An increased level of milk amyloid A indicates an inflammatory response and this is usually for a cut-off point greater than 500 ng/ml. Some interpretation issues remain including whether the analysis is carried out at the quarter or cow level. Sensitivity and specificity of milk amyloid A are comparable if not better than somatic cell count.

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ADDING VALUE TO THE MILK SAMPLE

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SUMMARY

This paper reviews the traditional use of the milk sample and the information that can be gained from the testing of the sample. A review of the trends in performance of the recorded herd developed from the milk sample is displayed along with some of the shortfalls. However, the sample gives a whole range of other information regarding animal health, breeding, milk quality and parlour hygiene.

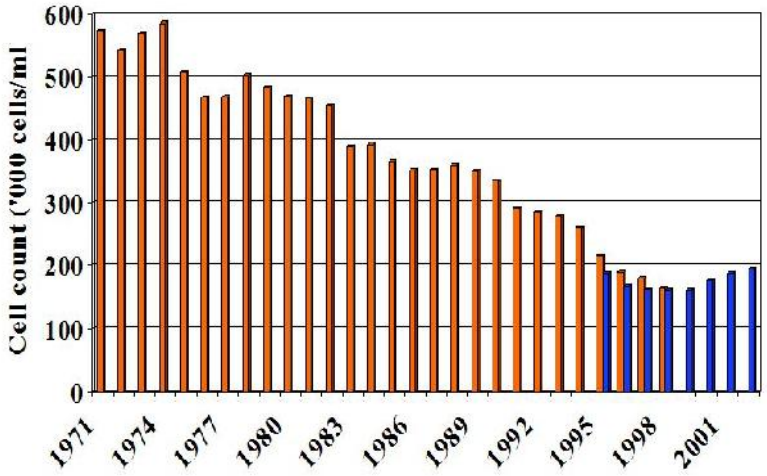
The traditional value of the sample has been available to the farmer to help him in the breeding and management of his herd. As the testing capabilities have developed the additional value of the sample has become available to the veterinary and consultants in the interpretation and analysis of the result and the knowledge of the herds with which they are involved.

INTRODUCTION

Dairy farmers have traditionally received a weekly, bulk milk, test result from their buyer showing fat %, protein %, lactose %, bulk cell count and total bacteria count (TBC). The TBC count has been superseded by a bactoscan count measuring Total Viable Count of all bacteria in the milk, both harmful or otherwise. More recently, this sample has been used to test for antibiotics in the milk, added water and the urea content of milk. These data are used by the farmer as a guide to the quality and hygiene of the milk the herd is supplying. A high bulk milk cell count will often prompt the farmer to test individual cows as opposed to the bulk milk.

As legislation has demanded lower average milk cell counts the hygiene of UK milk has improved progressively (Figure 1). Since the demise of the MMB, farm gate, milk prices have partly been based on milk hygienic quality, so farmers have an incentive to produce cleaner milk to achieve a better price. However, a slight rise in cell count has been seen in the past five years to a current level just over 200,000 cells/ml.

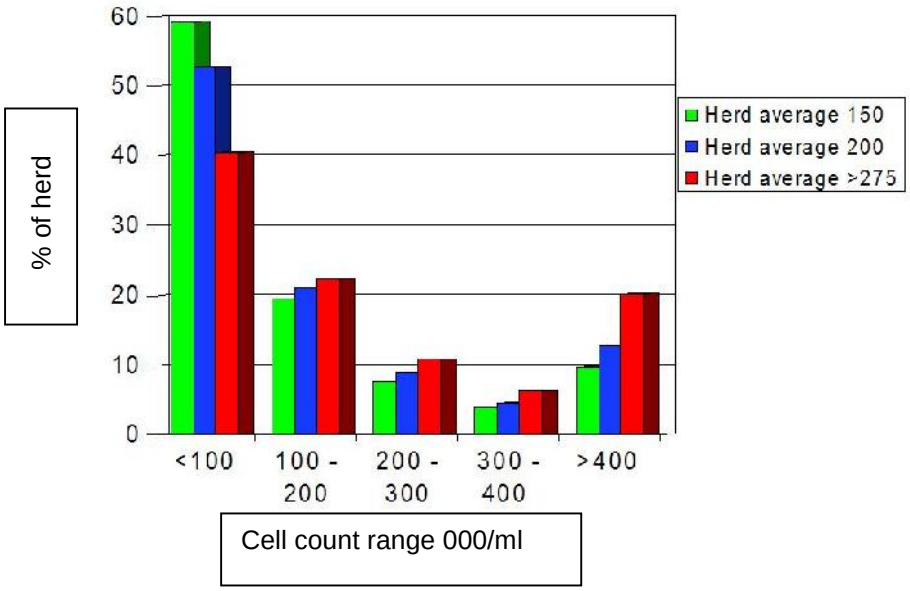
Figure 1 Average herd bulk milk cell count (1971–2004)



Source: - 1971- 1999 MMB and successors, 1996- current NMR

The use of average cell count results disguises that at the extremes in the herd some animals with a very high cell count, increase the average.

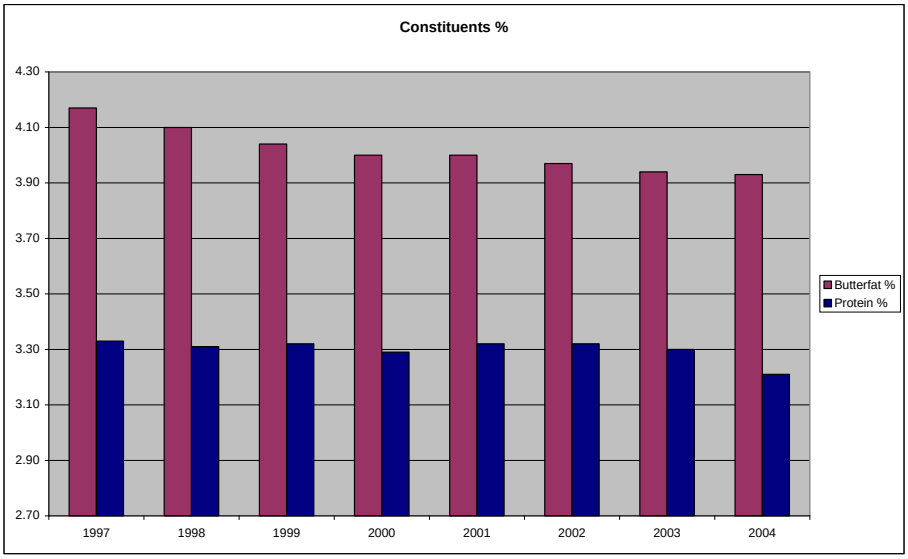
Figure 2 Spread of individual animals within a herd bulk average



A herd with a bulk milk average cell count of 200,000 cells/ml will have approximately 13% of the cows in the herd with a cell count greater than 400,000 cells/ml and another 5% of cows greater than 300,000 cells/ml. Current veterinary thinking suggests that a cow carries an infection when the cell count is above 200,000 cells/ml.

Whilst the cell count has been a major item in the price of milk and used widely by the farmer, the quality of the milk as measured by fat and protein also contributes to the final milk price paid. The recent trend in average percentage fat and protein production is shown in Figure 3.

Figure 3 Change in milk fat and protein composition with time



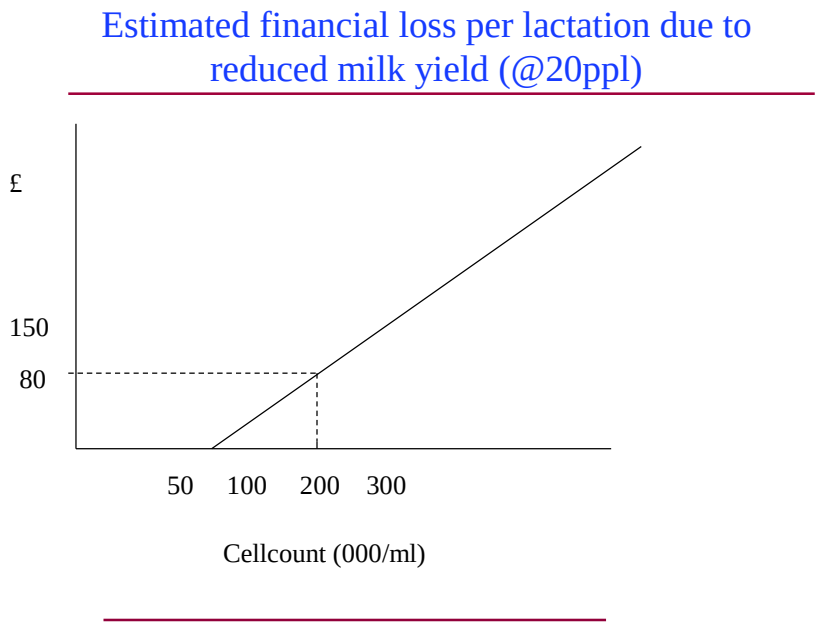
Butterfat production, in response to demands for lower fat from milk buyers, has fallen over the past decade from 4.10% to approaching 3.90%. Protein production had remained at around 3.30%, but has fallen for the past 3 years to around 3.20%.

Traditionally, this was the limit of the use of the milk sample – milk constituents, cell counts and bacterial count and the farmer would look to manage the herd on the basis of these results from the milk buyer.

INDIVIDUAL COW SAMPLES

For many years half the dairy farms in the UK have tested every animal in the herd and not relied just on the result from the milk buyer. The producer gets fat, protein and cell count content for each animal allowing management of the cow not simply the herd. With high cell counts the loss of revenue through discarded milk and through reduced yield can be predicted (Figure 4).

Figure 4 Estimated financial loss due to reduced milk yield



The management of the individual cow is now possible and those cows with greater effect on the herd bulk standard can be identified. Standard cell count reports from milk recording organisations (MRO) highlight the previous three recording results, the contribution of each cow to the bulk total, the cow lactation average and the number of counts greater than 200,000 cells/ml.

Individual cell counts also allow review of cell count performance by lactation and bench marking of the herd performance against the top 25%, average and bottom 25% herds (Figures 5 and 6).

Figure 5 Cell counts for top and bottom 25% and the average national herds

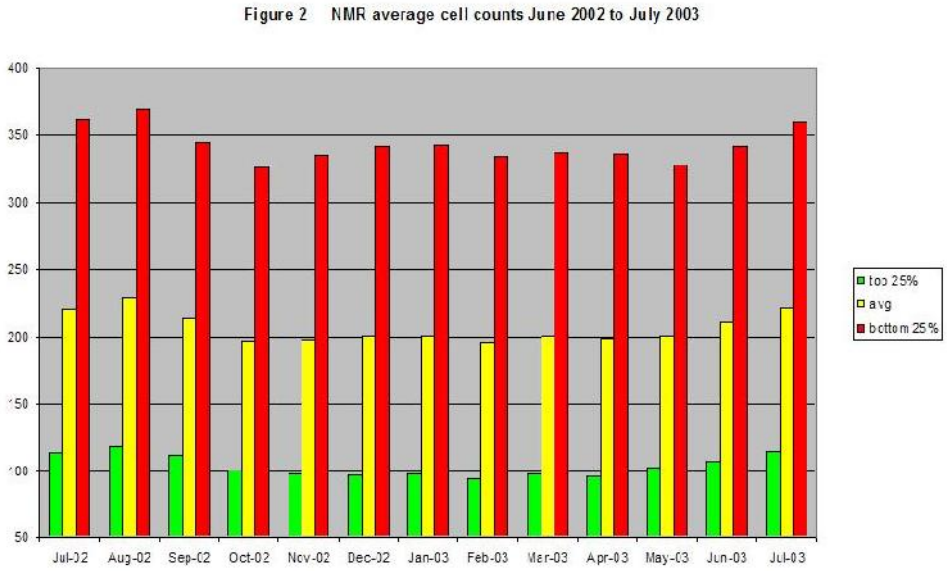
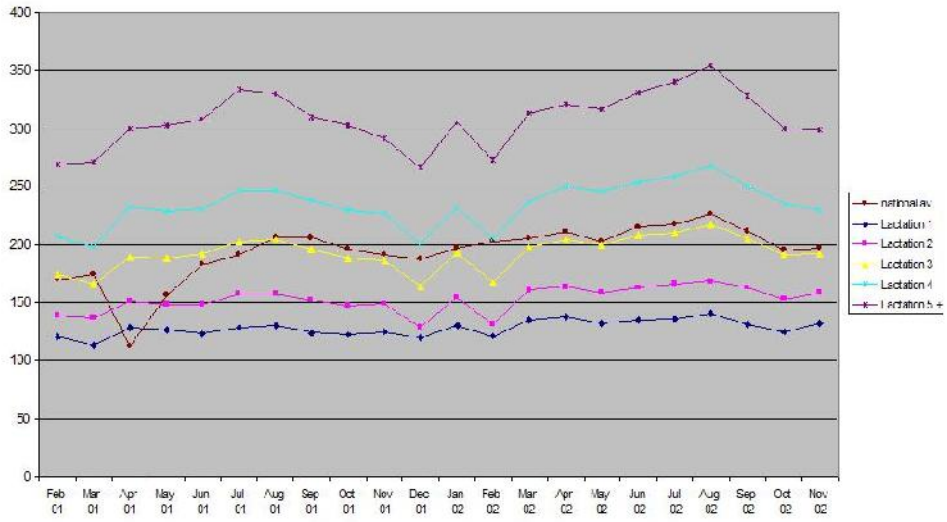


Figure 6 Cell counts changes with time by lactation



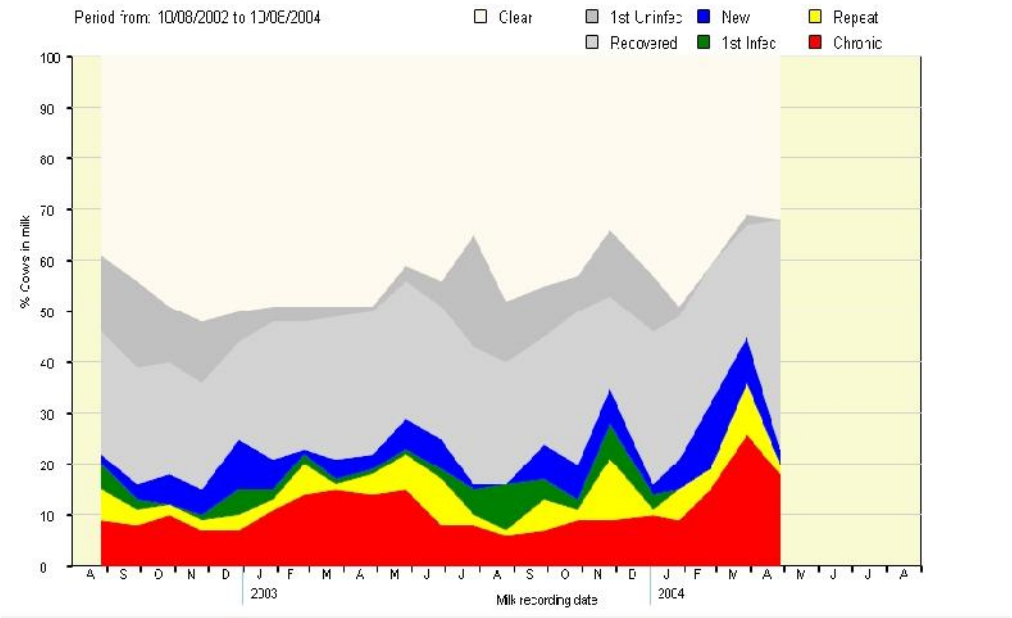
Recent developments have introduced new categories to describe herd cell count (Table 1). These data can be provided in graphical format to show periods of good and bad performance (Figure 7). Clearly, the herd in the example has a high cell count problem in the winter months.

Table 1 Cell count variation by date.

SCC breakdown by milk recording date Threshold SCC: 200 ,000 Change

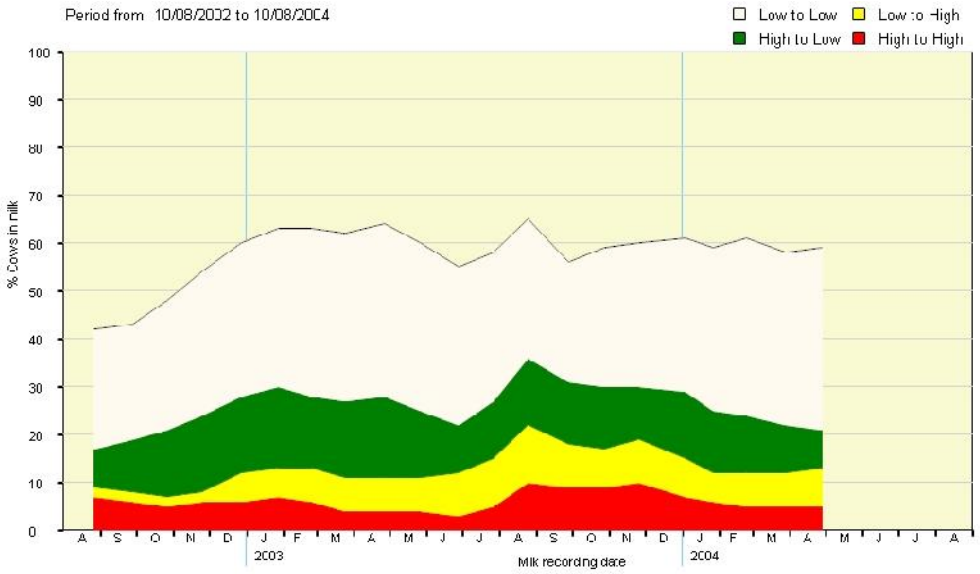
Recording Date	Cows milked	Herd SCC	% >200	Cows >200	New	First	Repeat	Chronic	First uninfected	Recovered	Uninfected
20/04/2004	**128**	**140**	**20 %**	**25**	**9**	**1**	**7**	**8**	**8**	**15**	**80**
16/03/2004	131	222	**15 %**	19	**3**	3	3	10	12	18	82
16/02/2004	144	97	**15 %**	21	**5**	1	3	12	6	24	93
19/01/2004	150	108	**13 %**	19	**4**	1	2	12	17	30	84
15/12/2003	132	176	**18 %**	24	**7**	3	2	12	22	20	66
17/11/2003	131	99	**18 %**	24	**5**	4	2	13	14	25	68
16/10/2003	118	116	**20 %**	24	**8**	2	5	9	21	21	52
18/09/2003	112	112	**13 %**	14	**3**	0	1	10	13	28	57
14/08/2003	118	152	**24 %**	28	**4**	1	1	22	7	24	59
16/07/2003	120	146	**27 %**	32	**8**	1	4	19	6	21	61
17/06/2003	123	143	**19 %**	23	**7**	0	4	12	3	24	73
15/05/2003	133	140	**16 %**	21	**3**	0	6	12	6	26	80
14/04/2003	131	175	**18 %**	23	**7**	4	5	7	7	25	76
18/03/2003	124	127	**15 %**	18	**5**	5	1	7	7	22	77
19/02/2003	124	106	**19 %**	24	**5**	2	7	10	13	16	71
16/01/2003	120	132	**15 %**	18	**4**	3	1	10	13	22	67
12/12/2002	121	133	**21 %**	25	**5**	3	2	15	10	19	67
14/11/2002	115	116	**20 %**	23	**3**	0	2	18	11	19	62
16/10/2002	112	154	**24 %**	27	**6**	0	3	18	5	18	62
16/09/2002	117	145	**25 %**	29	**5**	3	3	18	14	15	59
12/08/2002	122	208	**27 %**	33	**7**	3	2	21	14	16	59

Figure 7 Cell count by time



Analysis of the cell count data before and after the dry period can show the rate of new intramammary infection in the dry period. This is particularly useful as 60% of all infections originate in the dry period.

Figure 8 Performance during the dry period



The herd described in Figure 8 had approximately 5% of cows that finished their lactation with a high cell count and started the new one still with a high cell count. Any dry cow therapy used had not worked well. Further, another 10% of cows had a low cell count at the end of lactation and a high cell count in early lactation indicating new infections.

Bacteriological testing

Having identified the high cell count animals, the milk sample can be used to identify the bacteria causing the infection and the antibiotics that will be most effective.

Table 2 shows an example report from a bacteriology test, a report normally sent to the vet.

Table 2 Bacteriology test report

Cow identification	Somatic cell count x1000/ml	Bacterial pathogen(s) isolated	Antibiotic sensitivity
26	479	*Strep. agalactiae*	Neomycin YES Novobiocin YES Penicillin G YES
558	570	*Staph. aureus*	Streptomycin YES Neomycin YES Cloxacillin YES Erythromycin YES
29	1500	Coagulase - positive staphylococci	Streptomycin YES Penicillin G YES Cloxacillin YES Erythromycin YES
112	5421	*Strep. agalactiae*	Neomycin YES Novobiocin YES Penicillin YES
42	1320	*Strep. uberis*	Amoxycillin YES Cloxacillin YES

A high Bactoscan result can be investigated by testing the bulk milk sample for bacterial composition. Table 3 shows an example report that supplied to the vet.

Table 3 Bactoscan analysis

Lab Number: Sample Type: Sample Description:
22006838 Raw Milk 22051025 1

Determination	Result	Units	Method Ref.
Total viable count 30°C	7100	cfu/ml	Micro/143
Coliforms 30°C	11	cfu/ml	Micro/004
Total Clostridial count	400	cfu/ml	Micro/121
E. coli	<10	cfu/ml	Micro/149
Enterococcus faecalis	150	cfu/ml	Micro/149
Psychrotrophs	<10	cfu/ml	Micro/121
***Staph. aureus* 30°C**	2700	cfu/ml	Micro/128
Strep. agalactiae	<10	cfu/ml	Micro/149
Strep. dysgalactiae	<10	cfu/ml	Micro/149
Strep. Uberis	4200	cfu/ml	Micro/149

Disease surveillance

At one time the payment testing laboratories tested for Brucellosis and EBL as part of a legislative requirement, however, the milk sample can now be used for a much more extensive range of testing to find diseases such as Leptospirosis, Infectious Bovine Rhinotracheitis and Bovine Viral Diarrhoea. The tests these can be carried out on either bulk milk or individual cow milk.

Pregnancy diagnosis

Milk progesterone can indicate the pregnancy status of the cow. Progesterone is produced by the ovary and is high at mid-cycle and in pregnancy. It is low at heat or if the cow is anoestrus

Progesterone assays may be used in a number of ways to investigate or improve reduced fertility in a dairy herd.

Sampling at day of observed heat. All samples taken on this day should show low progesterone. Samples taken from 20 cows would show, if the levels of progesterone were not consistently low, that heat were being wrongly predicted.

Sampling at day 19 after service. A low level of progesterone in the sample on day 19 will give an early warning of return to service in 80% of cows, such cows should be carefully observed for heat.

Sampling at day 24 after service. If the progesterone level appears to be rising at day 19 then a re-test should be made at day 24. This is statistically the best day for determining pregnancy. However, a sample at day 24 alone will identify most non-pregnant cows too late for to be served at that heat.

Weekly sampling. Samples taken on a weekly basis can give information on whether cows are showing regular heat cycles:

- Cows should show a low level of progesterone (indicative of heat) at least once in 3 - 4 samples.
- If all samples are low the vet should be consulted about anoestrus. This will occur in some cows after calving.
- If all samples are high the vet may conclude that there is a cystic *corpus luteum* (but beware, the animal may be pregnant!).
- If evidence of normal heat cycles is seen, then heat detection may be poor.

Heat prediction. Samples are taken on alternate days from day 15 or 16 after a previous heat (low level of progesterone during weekly sampling). When a new low level of progesterone is seen, cows are observed closely for signs of heat. Alternatively the cow is served "blind" on the day after the low value. This may result in increased double services if the cow is bulling on a later day.

Individual cow results for breeding analysis

With the widespread testing of individual cows for milk quality the data have been used to provide genetic evaluations both for sires and individual cows. This is then used to generate predictive transmitting abilities (PTA) to aid in selection to change the genetic status of the herd.

THE FUTURE

The milk sample has a wide variety of uses and with the development of National Milk Laboratories allowing the seven day collection of samples, last seen in the days of the MMB, the ability to add value to the sample continues to increase.

Seven-day collection of samples means the bulk milk samples from 70% of the UK dairy herds are available for testing. The DEFRA Animal Health and Welfare Strategy requires farmers to have a plan for animal health and this

will inevitably lead to additional requirements for testing of the milk samples.

Many milk buyers structure their pricing to obtain milk from the quality end of the market and require their suppliers to take note of the test results. If the quality of the milk supplied is too poor, then heavy price penalties may result along with some compulsion for suppliers to use the services of vets, recording organisations and consultants to improve output quality.

Two examples of the new testing of samples can be seen with the development of individual cow urea results from the recording sample and the testing at herd or cow level for Johnes' disease.

The value of the sample lies in the infrastructure created to allow the variety of testing and analysis required from whole range of different interested parties – the farmer, the breeding companies, consultants, milk buyer, vets and Government. As the testing services develop the opportunities to ensure the right quality milk is used in the appropriate markets from healthy cows will pay benefits to the farmer and his cows.

A NATIONAL MASTITIS INTERVENTION STUDY – PRELIMINARY RESULTS

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SUMMARY

Bovine mastitis remains as a significant cause of financial loss to the UK dairy industry. Despite the fact that mastitis is considered to have a substantial deleterious impact on animal welfare and farm finances, there is little evidence of any significant improvements during the last 20 years. In fact, the situation may be worsening. This paper discusses the possible reasons for the current situation and aims to promote the discussion of possible solutions. The methods and preliminary results from a national mastitis project are presented including an assessment of the current UK situation and initial findings from an intervention study on mastitis control.

INTRODUCTION

Mastitis in UK dairy herds remains a major problem with clinical episodes alone considered to cost the industry in excess of 160 million pounds each year (2). Further losses are caused by sub-clinical infections through loss of milk yield and a potential reduction in milk price if bonuses are lost. Mastitis has further ramifications, namely an important impact on cattle welfare and potential influences on public health (2).

Yet despite the clear importance of bovine mastitis and an abundance of peer-reviewed literature on the subject, it is hard to be convinced that the UK has made any significant progress in preventing the disease for the last 15-20 years. The incidence rate of clinical mastitis is difficult to determine precisely, but estimates place the figure at around 35-50 cases per 100 cows per annum (1, 3, 6), and this is probably not dissimilar to that twenty years ago (7). It appears that during this period we have seen a change in aetiology with a relative increase in environmental organisms at the expense of contagious pathogens (4). Bulk milk somatic cell counts (SCC) have decreased since the implementation of financial penalties in 1991, although recently bulk milk SCC have started to increase slightly (www.mdcdatum.org.uk). No evidence appears to exist to show that the reduction in SCC is a result of reduced infection rates, it may simply be due to increased culling of high SCC cows and/or manipulation of bulk milk SCC by the exclusion of milk from infected cows.

An update of the current situation

A nationwide postal survey of clinical and sub-clinical mastitis, funded by the Milk Development Council, has recently been completed. One hundred and twenty dairy herds, were selected at random from National Milk Records (NMR) recorded herds. Herds were enrolled on a monthly basis throughout a 12 month period. Each herd was then asked to identify and sample the next five cases of clinical mastitis and to then take milk samples from 5 high cell count cows, randomly selected by following a standard operating procedure.

At the time of writing, data have been collected from around 70 farms, data from a further 30 farms are expected to be available by the end of August. From this the incidence rate of clinical mastitis will be calculated and the pathogen-specific causes of clinical and sub-clinical mastitis identified. Preliminary results will be presented at the conference, though initial analysis of current data would suggest that the aetiology of clinical mastitis in the UK is much as expected (*i.e.* primarily environmental in origin), though the incidence of clinical mastitis may be somewhat higher than previously thought.

The current dilemma

So why have we failed to make significant progress with mastitis in the UK over the last 15-20 years? At best, all we can boast is that mastitis may not have got much worse despite a large increase in cow yield and a possible increase in cow susceptibility to the disease (5). Surely a situation where approximately 30% of the national herd having at least one case of clinical mastitis each year and probably over 10% cows having a chronic infection at any time is not a sustainable position?

Arguably there is a widening gulf between research findings and what is occurring at grass roots level. We have literature on vaccine candidates, manipulating genetic resistance to disease, novel treatments such as immune modulation and so forth, and yet you can't help but feel that we are searching for the ultimate answer before ensuring that all current knowledge is thoroughly applied. So it is from this background that we raise the question:

"If existing knowledge of mastitis control was applied on all UK dairy farms, by how much would we change the current disease situation?"

In an attempt to address this question the authors have undertaken a 12 month mastitis intervention study, funded by the Milk Development Council. The aims of this project were to attempt to answer the following questions:

- If dairy herds with above average incidence of clinical mastitis are advised on mastitis control and implement accepted current methods wherever possible, what would be the outcome on the incidence of clinical mastitis in a 12 month period?
- What are the most important elements of mastitis control that result in the biggest improvements in mastitis incidence and prevalence in different situations.

MATERIALS AND METHODS

The salient aspects of the materials and methods are outlined below:

- Fifty two herds were selected at random from herds that undertook milk recording with NMR and that had an incidence rate of clinical mastitis above the national average (taken to be 35 cases per 100 cows per year)
- The average herd size in the study was around 150 cows and the average incidence of clinical mastitis before the study commenced was approximately 85 quarter cases per 100 cows per year.
- Herds were split into two groups of 26 (intervention and control) and matched on bulk milk cell counts and geographical location.
- A whole herd mastitis control scheme, "The Mastitis Diagnosis and Control Plan" (MDC Plan) was developed using current literature as its basis. Steps in the Plan were

    i. Define the herd situation using appropriate clinical mastitis and cell count indices.
    ii. Use strategic milk samples to identify pathogens.
    iii. Assess current herd control measures against the Plan 'gold standard'.
    iv. Define areas of control that need to be addressed, but prioritise them according to the patterns of mastitis identified on the unit.
    v. Confer with the farmer every four months to re-appraise the data and re-assess the targeted control plan.

- A critical part of the plan was to select from over 300 control measures (the 300 point plan?) and to target the 6-12 measures most likely to give the biggest improvements on any particular unit. We believe that farmers have limited resources and most benefit will be gained from focusing on relatively few 'big win' issues at a time. Therefore, a strategy to achieve this was developed and tested. The whole scheme will become available through the Milk Development Council (www.mdc.org.uk) in February 2007.

- The mastitis control plan was carried out on the 26 intervention farms, but not on the 26 controls. All farms were closely monitored throughout the study period.
- The change in incidence of clinical mastitis over a 24 month period was assessed between intervention and control farms.
- Main causal factors that influenced the success of the control strategy were investigated.

PRELIMINARY RESULTS AND DISCUSSION

Data analysis is currently ongoing as this paper is being written.

Initial findings would suggest that the aetiology of clinical mastitis in the UK is much as expected (*i.e.* primarily environmental in origin), though the incidence of clinical mastitis may be somewhat higher than previously thought.

However, on a brighter note, it would appear that there is significant scope to improve the mastitis situation in the UK through the application of current knowledge.

Arguably the question is 'whose responsibility is it and can we find a better model for mastitis prevention in the UK?'. Implementing a national mastitis control plan that works would benefit the producer (financial), the milk purchaser (milk quality, residues), the veterinary surgeon (more fee paying time less reliance on medicines), the 'dairy industry' (better image for milk quality and welfare), the consumer (happy with reduced disease in farmed animals), the politicians (lower endemic disease, better welfare, better structure for farm vets to work in).

Just imagine...
...a reduction of around 15-25% in clinical cases in a 12 month period appears realistic... that would make a huge difference to the dairy industry in the UK ...or shall we leave it another 20 years...?

A more detailed presentation and discussion of the preliminary findings of this research will be made at the conference.

ACKNOWLEDGEMENTS

The authors would like to take this opportunity to thank both the dairy farmers and herdspersons directly involved in the research presented in this paper, especially those that agreed to act as control farms as well as NMR for their cooperation. This research was funded by the Milk Development Council (Project Number: 03/T2/07-1)

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MASTITIS MANAGEMENT – EAST EUROPEAN EXPERIENCES

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No written paper has been submitted.

KEEPING CONTROL OF A LARGE HERD AND ROTARY

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BACKGROUND

J.F. Cobb and Sons have been farming at West Chaldon since 1928. The business now totals 2250 acres with its main enterprise being the 600 cow Chalclyffe Holstein herd.

In 1997 the business was selling 3 million litres of milk from 500 cows on three different sites. Four hundred of these cows were housed on a straw yard system as we had plenty of straw from the 1000 acres of arable on the farm. One of the sites, Northground Dairy, needed to be updated, so an eight abreast parlour was replaced with a 24-internal Westfalia Rotary parlour. Naively, we thought that this would solve all of our problems at this site by speeding up our milking time. At the same time dairy companies were placing more and more emphasis on milk quality. In the first year the 200 cows at Northground produced milk with an average cell count of 250,000 cells/ml, Bactoscan of 25,000/ml and the number of clinical cases of mastitis seemed to get out of control, often with more than 20 cows under treatment at any one time. After investing a significant amount of money in the new milking facility we had to get on top of these problems and start seeing some returns. The partners have never had a problem with taking advice and our newly appointed business advisor encouraged us to change vets to a more proactive practice and employ the help of a milking technology specialist, at least until we were on top of our problems.

This new team approach found:

- *Staphylococcus aureus* and *Streptococcus uberis* were the main pathogens causing the mastitis.
- Milking preparation in the new parlour was not adequate, especially as the cows were housed on straw yards.
- Milking cows into a dump bucket only confused the milking routine and was a possible source of cross contamination.
- The lying area for cows in the straw yards was probably not enough with larger cows and increasing yields.
- Regular cleaning out of straw yards was difficult especially during busy periods of field work in the summer and autumn.
- A better system of recording clinical and sub-clinical cases was necessary to monitor performance.
- Culling a number of problem cows was really the only way to get on top of the problem.

THE NEW PLAN

That year the decision was made to cull the problem cows and reduce the stocking density of the straw yards from 200 cows to 160. The herd moved to 3x milking in an attempt to compensate for some of the wasted milk and a hospital area was made for cows whose milk had to be withheld. The herd was turned into loafing fields with feed trailers during the summer months to give the yards a break. An udder preparation routine was started in the parlour with a pre-milking spray, wipe and post-milking spray. All yards were cleaned out every three weeks during the housed months and bedded twice a day. Interherd was used to help build a picture of herd health, especially mastitis and cell count. These initial changes made a vast improvement. In 2000, cell count was 150,000 cells/ml, the Bactoscan was 18,000/ml and number of clinical cases was significantly reduced, but there were still a number of problems.

- With one man milking on the rotary it was difficult to maintain the new routine for any length of time.
- Cleaning out the straw yards every three weeks was becoming extremely labour intensive. With cows giving improved yields it was becoming obvious that we were going to have to house at least some of the herd in the summer months.
- We also had two other units that were in need of improvement
- Milk produced for sale could not exceed 2 million litres with 160 cows.

THE NEXT PHASE

A visit to the USA enabled me to see how they ran more intensive dairy systems and it was the first time I had seen cows housed on sand. A number of our advisors had also had good experiences with other clients using sand bedded cubicles with obvious benefits in cow comfort. Our local sand quarry is only two miles from the farm and it seemed an obvious choice to cost-out using sand, especially after looking at the capital cost savings over a mattress system.

In 2001 a budget was created to build a 300-cow, sand-based cubicle system and, after careful planning with the new team, the decision was made to shut one of the other dairies that was most in need of a new parlour and invest the money at the Northground site. In the first year we moved 250 cows into cubicles for the first time, we only lost 10 animals that could not be trained to lie in. This in itself sold the comfort element to us and, as well as other health benefits, we saw an immediate reduction in cell count and clinical cases of mastitis. Continuing 3x milking and with less milk going down the drain, the milk sales rose on this one unit to more than 3 million litres. A year later, in 2003/4, after a change in herdsman, the herd

was runner up in the Pfizer AH milk quality awards. The health of the cows had never been better, but we still had another dairy in need of upgrading struggling with cell count on straw yards. Following the success at Northground, we decided to go back to the USA with our milk technology consultant to look at the feasibility of putting 600 cows on to a single site. Staffing this large unit would also need to be addressed. We took the decision to try and take a simple chain of command, with myself and one herdsman in charge of day-to-day work.

Feeding was taken care of, but we also needed general dairy staff to do a lot of the routine dairy work. We were finding it increasingly difficult to find local people prepared to do the type of work required and travelling workers always seemed to be moving on so we looked for a more permanent solution. We initially took on two, unskilled, Polish lads and are now employing five Polish dairy staff, none of whom had any previous farming experience. After a number of training sessions, with our vet and milking technician, they are now very competent and do the majority of the milking and yard work.

THE RESULTS

Today we are running one herd of 600 cows, increasing to 700 cows by the summer of 2006. The new unit has sand cubicles for all cows unless they are in the hospital area or a calving box. A flush system does away with the need for tractor scraping. Our current results from 600 cows yielding 10,200m litres/cow are, 100,000 cells/ml cell count, 14,000/ml Bactoscan, 4.2% butterfat, 3.2% protein and 22 cases of mastitis per 100 cows. The parlour runs for 15 hours a day, three shifts of 5 hours, and next year will be running up to 18 hours a day. We try and keep two people in the parlour at all times and up until this summer the routine has been to dry wipe, pre-strip with a 60 second lag time until cup attachment and then post-spraying.

Clinical incidence of mastitis has been under control with the most common pathogens being *Streptococcus uberis* and *Escherichia coli*; we still have a level of *Staphylococcus aureus* in the herd although we cull these cows as soon as we can. This has been the first summer that we have been totally housed and in the worst of the heat we experienced a significant increase in *E. coli* mastitis, both cases and sick cows. We have adapted our milking routine to include a pre-milking disinfectant teat spray and we will be looking at cooling the cows in the collecting yard next summer.

Clinical case protocol:

- Milk into a bucket and put the cow in the hospital area.
- Sample the quarter and freeze the sample.
- 15 ml of Metacam, (injectable antibiotics given if cow has no milk).
- Inject oxytocin and reattach the cluster (4x in 24 h).

- Leo Yellow or Cephaguard tube next morning milking and repeat every 24 h.
- Sick cows are given 30 to 50 litres of fluids daily and boxed until an increase in appetite is observed.
- All affected cows are milked last to reduce the risk of cross-contamination, contamination of the bulk milk and so dedicated time can be spent on treatment, by skilled staff.

Dry cow protocol:

- One month before the planned dry-off, milk quality is assessed with our vet, we sample and test any potential problem cows and use one of three treatments - Orbeseal tube (approx. 80% of herd) OR Cepravin dry tube and Orbeseal (approx. 15% of herd) OR Cepravin dry tube Orbeseal and Tylan injection (approx. 5% of herd).
- Cows are drafted on Wednesday afternoon to housing on sand cubicles in a drying-off area away from the parlour and fed barley straw.
- On Thursday afternoon, before milking, the cows are brought to the clean parlour, milked, teats cleaned meticulously using cotton wool and surgical spirit, infused as above and teats sprayed, all carried out by a skilled member off staff.
- Cows go on to a dry ration and are observed to ensure no problems.

Current targets:

- To sell more than 7.7 million litres of milk.
- To maintain milk quality.
- To reduce clinical cases especially during the summer months.
- To reduce heat stress next summer.
- To breed a better, healthier Holstein cow.

CONTROLLING COLIFORM MASTITIS

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SUMMARY

Coliform bacteria are a primary cause of mastitis in most herds. Coliforms reside virtually everywhere in the cows' environment, with bedding and manure the primary point sources of these bacteria for causing mastitis. Rates of new infection caused by coliforms are greatest during the dry period and early lactation. The thrust of herd management strategies for controlling coliform mastitis should focus on reducing IMI during the dry period and early lactation by reducing the exposure of cows to the pathogens and enhancing the ability of cows to combat the infections.

INTRODUCTION

Gram-negative bacteria are the aetiological agents most often isolated from severe clinical cases of mastitis. The term *"coliform mastitis"* is used frequently incorrectly to identify mammary disease caused by all Gram-negative bacteria. Genera classified as coliforms are *Escherichia* spp., *Klebsiella* spp., and *Enterobacter* spp.. Other Gram-negative bacteria frequently isolated from intramammary infections (IMI) include species of *Serratia* spp., *Pseudomonas* spp., and *Proteus* spp..

Gram-negative bacteria are considered environmental mastitis pathogens. The importance of transfer of Gram-negative bacteria from the mammary glands of infected cows to uninfected cows appears minimal compared with the constant environmental exposure. Coliform bacteria occupy many habitats in the cow's environment. *Escherichia coli* are normal inhabitants of the gastrointestinal tract of warm blooded animals. Both *Klebsiella* spp. and *Enterobacter* spp. populate soils, grains, water, and intestinal tracts of animals. *Serratia marcesens* share many environmental sources with *Klebsiella* spp. and *Enterobacter* spp. *Pseudomonas* spp. and *Proteus* spp. commonly contaminate drop hoses used to wash teats before milking. Gram-negative bacteria may be isolated from virtually any surface area of the cow or her surroundings and cause a host of diseases other than mastitis. Coliform bacteria are among the aetiological agents commonly responsible for infectious respiratory and urogenital diseases in dairy cows. However, the spread of Gram-negative bacteria from other regions of the body to the mammary gland via the vascular or lymphatic systems appears minimal. Intramammary infections caused by Gram-negative bacteria typically result from the bacteria traversing the teat canal and multiplying in the gland. Although the mammary gland is not considered a natural habitat for coliform bacteria, many strains are capable of surviving and multiplying in the mammary gland.

The key to controlling coliform mastitis to an economically acceptable level within a herd is to reduce the exposure of cows to the pathogens and enhance the ability of cows to combat the infections when they occur. Climatic factors that affect the risk to coliform mastitis are temperature and humidity. As the ambient temperature and moisture increases, populations of pathogens increase in the cow's environment and the mammary defence systems to combat infections become compromised.

MANAGEMENT FACTORS

Monitoring coliform mastitis

The prevalence of IMI caused by coliform pathogens is seldom great enough to cause bulk tank somatic cell counts (SCC) greater than 400,000 cells/ml, but approximately 85% of coliform infections will cause clinical mastitis. Therefore, even low SCC herds can still have mastitis problems and these problems generally involve clinical cases of mastitis. A survey of herds with bulk tank SCC of less than 250,000 cells/ml showed the average rate of clinical mastitis to be 46 cases per year in a 100 cow herd. The high frequency of clinical cases and relatively short duration of Gram-negative bacterial IMI render the use of individual cow SCC and bulk tank SCC as poor indicators of the prevalence of disease caused by these bacteria. The prevalence of IMI caused by Gram-negative bacteria seldom exceeds 5% of quarters in a herd, however greater than 25% of cows in well-managed herds are annually diagnosed with clinical mastitis caused by coliforms.

Recording the number of clinical cases and documenting the seasons and stage of lactation when they occur will aid in determining when cows are at greatest risk to clinical coliform mastitis. Management practices can then be altered to reduce exposure of teat-ends to pathogens and enhance the ability of cows to fight infections. Gram-negative bacteria are the bacterial group most commonly isolated from clinical cases of mastitis in many surveys. The percentage distribution of Gram-negative bacteria causing clinical mastitis is herd dependant, but studies in the United States and Europe consistently report that appropriately 40% of clinical cases are the result of Gram-negative bacteria. The rate of clinical cases caused by Gram-negative bacteria averaged approximately 20 cases per 100 cows per year in these studies. The severity of clinical coliform cases ranged from mild, local signs, to severe systemic involvement. The vast majority of clinical coliform cases are characterised by abnormal milk and a swollen gland. Only about 10% of clinical coliform cases result in systemic signs including fever, anorexia, and altered respiration. Despite the relatively low percentage of clinical coliform cases yielding systemic signs, coliform bacteria have an exaggerated reputation for causing peracute mastitis. The basis for this distinction originates from the point that the coliforms are the most common cause of systemic illness resulting from mastitis. Survey averages suggest that coliform bacteria are the culprits of 60 to 70% of peracute clinical cases.

Therefore, the general conclusions concerning severity of clinical coliform cases are that few coliform IMI cause systemic clinical signs, but the majority of clinical cases resulting in systemic signs are caused by coliform bacteria.

Although clinical mastitis caused by species of *Serratia, Pseudomonas,* and *Proteus* tend to occur much less frequently than clinical coliform mastitis, sporadic herd outbreaks involving Gram-negative bacteria other than the coliforms have been reported. Intramammary infections caused by these bacteria develop into clinical disease less often and clinical cases tend to be less severe than coliform clinical cases.

Stage of lactation

Rates of new IMI caused by coliforms are greater during the dry period than during lactation. Therefore, the thrust of herd management strategies for controlling coliform mastitis should focus on reducing IMI during the dry period and early lactation. During the dry period, susceptibility to IMI is greatest in the two weeks after drying off and the two weeks prior to calving. Many infections acquired during the dry period persist to lactation and become clinical cases. Research has shown that 65% of coliform clinical cases that occur in the first two months of lactation are IMI that originated during the dry period. Coliforms are adept at infecting the mammary gland during the transitional phase from lactating to fully involuted mammary gland. However, *Klebsiella pneumoniae, Serratia* spp., and *Pseudomonas* spp. are more capable than *E. coli* at surviving in the mammary gland from the onset of involution until calving. Distribution of infections reveals that the greatest proportion of *K. pneumoniae* infections present at calving originated in the first half of the dry period. *E. coli* infections present at calving and early lactation originate most often during the last two weeks of the dry period.

The rate of IMI during lactation is highest at calving and decreases as days in milk advances. The prevalence of coliform mastitis in a herd seldom exceeds 5% of quarters, because coliform infections tend to be of short duration during lactation. The average duration of an *E. coli* IMI during lactation is less than ten days. Duration of IMI caused by *K. pneumoniae* averages about 21 days. Chronic infections of greater than 90 days caused by *E. coli* or *K. pneumoniae* are relatively rare. A major difference between IMI caused by coliform bacteria and those caused by other Gram-negative bacteria is the duration that bacteria persist in the mammary gland. Intramammary infections caused by *Serratia* spp and *Pseudomonas* spp often are chronic infections that may persist over multiple lactations.

Treating clinical cases

Currently available antibiotics have minimal effect on shortening the duration of IMI caused by coliform bacteria. The use of antibiotics administered by intramammary or systemic routes for treating *E. coli* clinical cases is virtually useless because of the short duration of infections and the high spontaneous cure rate. Treatment of peracute clinical coliform mastitis often involves supportive therapy including oral or intravenous fluids and anti-inflammatory agents.

Dry cow therapy

The purpose of dry cow therapy is to have the herd calve with fewer infections than were present at drying off. This is accomplished primarily by eliminating contagious pathogens present at drying off and preventing new environmental streptococcal IMI from establishing during the early dry period. However, dry cow products fail to reduce the incidence of coliform mastitis at calving. Efficacy of dry cow products against Gram-negative bacteria is minimal.

Teat dips

Teat dip efficacy is dependant upon the time of application relative to milking and the pathogens causing mastitis. Most germicidal teat dips effectively and rapidly destroy microbes on teat skin by chemical or biological action. However, the persistency of germicidal activity is limited and neutralised by organic materials such as milk and manure. Therefore, the use of germicidal teat dips post-milking will effectively reduce the incidence of new IMI caused by the contagious mastitis pathogens transferred between cows at milking, but post-milking teat dipping has no effect on incidence of new IMI caused by coliform pathogens. Although most germicidal products will kill coliforms on teat skin, exposure to these pathogens occurs primarily between milkings, long after the killing activity of the dips has diminished.

Dipping teats in a disinfectant prior to milking (pre-dipping) reduces new IMI caused by coliforms during lactation. Field trials have shown pre-dipping reduces the incidence of clinical mastitis by 50% in herds with low levels of contagious mastitis. Current recommendations in the US for pre-dipping include fore-stripping the first few streams of milk, removing excess manure and dirt from teats, dipping teats in the germicidal teat dip, allowing teat dip to contact teat skin at least 30 seconds, and manual drying of teats with either individual paper towels or freshly laundered cloth towels. Although this is potentially a very valuable milking hygiene procedure, extreme care must be taken to assure that teats are thoroughly dry of disinfectant before the milking machine is attached to prevent milk contamination.

Barrier post-milking, dips form a physical obstruction between teat skin and the environment. Latex, acrylic and polymer based products form a physical seal over the teat end to impede entrance into the udder between milkings. The use of latex barrier teat dips may reduce the incidence of coliform mastitis, but efficacy of physical barrier teat dips against other pathogens is minimal. A large number of barrier teat dips containing germicides are available. Barrier teat dips containing germicides have not been shown to be more effective than conventional germicidal dips in reducing coliform mastitis in controlled studies.

Vaccines

Vaccination against coliform mastitis has become an accepted management tool to reduce the severity of clinical signs. *E. coli* J5, and other rough mutants, are naturally occurring structurally modified strains that have unique antigenic properties that may cause enhanced immunity to not only themselves, but also other coliform strains. Use of *E. coli* J5 bacterin does not prevent IMI. However, the use of an *E. coli* J5 bacterin reduces the severity and duration of mastitis. Most immunisation schemes include vaccination at drying off, mid-dry period, and calving to maximise protection during the late dry period and the first month of lactation. Immunising cows during lactation may have little value because the risk to IMI decreases significantly as lactation progresses. Using coliform vaccines to treat clinical cases will not influence the course of the infection in most cases.

Vitamin E and selenium

Deficiencies in a number of essential micronutrients have been shown to cause cows to be more susceptible to disease. Among this list are vitamins A, D, and E, zinc, selenium and copper. One can easily rationalise feeding diets deficient in essential micronutrients will eventually result in decreased resistance to mastitis. The two micronutrients that have been shown linked to bovine mastitis most often are vitamin E and selenium. Animals deficient in either or both of vitamin E or selenium have had higher rates of infections, more frequent cases of clinical mastitis, infections of longer duration, and more severe clinical signs that cows fed supplemented diets.

The recommended limit for selenium concentration in dairy cow rations is 0.3 ppm, corresponding to an approximate intake of 3 mg/day for dry and 6 mg/day for lactating Holsteins. Little data exist to suggest that dietary selenium greater than 0.3 ppm results in additional enhancement of host defenses against mastitis. Dry and lactating cows should consume 1000 IU/d of vitamin E. For cows fed stored forages, vitamin E may need to be supplemented at 1000 IU/day for dry cows and at 500 IU/day for lactating cows, dependent on forage quality and dry matter intake.

The recommended dietary and blood concentrations of vitamin E and selenium relate to maintenance of host defenses to protect against infections. Optimal blood concentrations may be greater during periods of

stress. One such period of stress is calving. Plasma vitamin E concentrations in dairy cows are normally lowest when rates of mastitis are highest and when white blood cell functions are depressed around calving. Subcutaneous injection of vitamin E successfully elevates vitamin E concentrations in plasma and white blood cells during late gestation and early lactation periods. Cows injected subcutaneously with 3000 IU of vitamin E, 10 and 5 days prior to predicted calving have elevated plasma vitamin E concentrations and maintain intracellular killing of bacteria by white blood cells when dietary supplementation cannot support these defenses.

Cow lots

Populations of the bacteria in bedding are related to the number of bacteria on teat ends and rates of clinical mastitis. Reducing the number of bacteria in bedding generally resulted in a decrease in the occurrence of coliform mastitis. Coliforms do not live on teat skin for a long period of time. If these bacteria are present in large numbers on teat skin, it is the result of recent contamination from a source such as bedding. Therefore, the number of these bacteria on teat skin is a reflection of the cow's exposure to the contaminating environment. Hygiene should be as important in the lots and cubicles as the milking parlour.

Ideally, bedding should be inorganic materials that are low in moisture content and contain few nutrients for bacteria to utilise. The bedding material most recommended for controlling environmental mastitis is washed sand. Compared to organic materials such as sawdust, recycled manure, straw, and dirt, washed sand consistently contains fewer mastitis pathogens. Many farms are forced to use organic bedding materials that are compatible with liquid manure handling systems. Little advantage exists in using one organic material over the use of another. For example, straw tends to have highest streptococcal counts, while sawdust and recycled manure have highest coliform counts in comparisons among these bedding materials.

Any material to be used as bedding should be stored in a dry area to prevent wetting by rain and ground moisture. Composting organic materials such as manure is an effective way to reduce bacterial counts before use as bedding. However, although many organic bedding materials have relatively few mastitis pathogens prior to use, the pathogen populations often increase 10,000-fold within a few hours when used as bedding. Fresh bedding tends to absorb moisture from the cows' environment for use by the great number of bacteria that are constantly present in manure and soiled bedding. Regardless of the bedding used, removing wet and soiled material from the back one-third of stalls will significantly reduce the bacterial counts. Stalls should be raked a minimum of twice daily when animals are moved to be milked. Spraying bedding with disinfectant and adding powdered lime to bedding has met with little practical success in reducing bacterial counts. These practices cause an initial decline in bacterial populations, but

pathogen numbers quickly recover. Twice a day application of powdered lime may be necessary to sustain an advantage in lowering bacterial numbers. Avoid standing water and mud in cubicles, yards and holding areas. Outbreaks of coliform mastitis are common during rainy seasons when cows are exposed to manure contaminated yards and races leading to the milking parlour.

Maternity and dry cow lots

Providing cows with a clean and dry environment is not limited to during lactation. Dry cow and maternity facilities also should be managed similar to lactating cow housing. Dry cow areas should be well drained and free of excess manure. Dirt covered areas can expose cows to pathogen levels comparable to those in cubicles. Box stalls and loose housing areas should be cleaned to the foundation base regularly. Manure packs are to be avoided because they generally contain extremely high counts of pathogens dangerous to both dam and calf.

CONCLUSIONS

Management practices to both reduce pathogen loads and enhance resistance are most practical and economically justified when directed at the most susceptible population of cows. The cows on most dairies at greatest risk to environmental mastitis are cows calving during warm and rainy weather. Hygiene, vaccination, nutrition, and housing management practices to control environmental mastitis during the dry period and at calving will pay benefits during lactation.

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IMPROVING MILK HYGIENE - A FIRST STAGE IN CUTTING MASTITIS LOSSES AND BOOSTING RETURNS

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Mastitis is still costing UK dairy farmers around £138M in lost production and treatment costs. Treating and handling mastitic animals is a major inconvenience during milking with herdsmen estimating each mastitic cow adds 20 minutes to each milking.

In addition, dairy farmers are still experiencing losses as a result of sub-clinical infections.

Many farmers also fail to achieve all the available hygiene (Bactoscan and cell count) bonuses on offer within milk pricing schemes. It is estimated that an additional £54M is available within current pricing schemes – equivalent to 0.48 ppl.

New research by Johnson Diversey indicates that few farmers review their hygiene routines on a regular basis with only 50% checking the routine on a weekly basis.

Better attention to hygiene can have a significant impact on profitability. Johnson Diversey has developed the Deosan Hygiene Score® to allow farmers to benchmark hygiene management and identify areas for improvement. Initial results show that by making just small changes to their routine, most farmers could improve hygiene management, reduce losses associated with mastitis and improve the attainment of hygiene bonuses.

Milk hygiene management costs around 1.08 ppl, taking into account labour, water, electricity and dairy chemicals. Most farmers would see a positive contribution to profit through improved hygiene management with returns outweighing the additional costs.

MANAGING BULK MILK CELL COUNT USING THE DCC

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The successful dairy farmer manages bulk milk cell count to maximise milk price especially when milk price is affected by quality bonuses and penalties. Crucial information comes from detecting high cell count cows and then withholding their milk. Cell count can be high at predictable times including immediately post-calving, post-treatment and in late lactation.

Milk is considered abnormal and must be excluded from sale in the EU for the first four days after calving. Bulk milk is required to have a cell count less than 400,000 cells/ml.

The Direct Cell Counter or DCC (DeLaval, Sweden) allows cell counts to be measured directly cow-side in real time. It is possible to obtain a cell count in little more than one minute, including taking the sample and making the measurement. Thus high cell count cows can be identified rapidly and easily. The DCC has been used to show the decline in cell count *post partum* and the impact recent calved cows may have on bulk milk quality.

Table 1 Changes in quarter milk cell count for morning milk after calving ('000 cells/ml)

Day	Geometric mean	Median	% quarters <200	% quarters >1000
1	505	410	15	25
2	516	431	27	33
3	281	253	45	25
4	130	119	63	10
5	68	47	73	7

The decline in quarter milk cell count for uninfected quarters after calving is progressive. Cell counts average more than 500,000 cells/ml for the first two days and then halve daily. On day 5, when milk can be legally sold, the average quarter cell count was 68,000 cells/ml, yet only 73% of quarters had a cell count less than 200,000 cells/ml, the level considered the threshold indicative of infection, although all quarters were free of pathogenic bacteria. Some 40% cows had one or more quarter cell counts greater than 200,000 cells/ml and 5% of cows had 3 or 4 quarters with a high cell count indicating that attention should be given to individual cow

cell counts when milk is first to be consigned sale. The impact on bulk milk may be high in small herds or when many cows calve in a short period.

THE HIDDEN DANGER IN ROUTINE SOMATIC CELL COUNTS: LONG-TERM INFECTIONS START AT LEVELS THAT ARE REGULARLY IGNORED

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Analysis of individual cow milk samples collected routinely by milk recording organisations is the most common method for monitoring somatic cell count (SCC) within dairy herds. Each cow's sample is comprised of milk commingled from all four quarters of the animal.

SCC reports typically list cows in descending order of SCC, emphasising individual contributions to the bulk tank. Many of the animals at the top of these lists are known problem cows that have been "high" for a number of consecutive months.

A study of monthly milk recording data for March/April 2005 from 110 herds associated with veterinary practices in Devon, Gloucestershire and Cheshire, identified 1,948 cows with persistent high cell count measures. Each of these animals had recorded "high" SCC levels (above a threshold of 200,000 cells/ml) for a minimum of two consecutive milk recordings. Tracing back to the initial SCC above the threshold level showed that in 62% of cases the initial high SCC measure was below 500,000 cells/ml.

A new infection in one quarter is diluted by clean milk from the other three uninfected quarters, limiting the impact on the overall SCC measure. Cows which become persistently infected in a single quarter may go undetected. This is particularly the case where the remaining quarters have low SCC levels. One quarter yielding milk at 2 million cells/ml mixed with equal volumes of milk from three quarters at 50,000 cells/ml will have a commingled SCC reading in the region of 500,000 cells/ml.

This dilution effect on SCC measures is causing delay in recognising new infections. Consequently infections are frequently well established by the time that the SCC count reaches a level that is noticed as a problem by the farmer.

TREATMENT PROTOCOLS FOR CLINICAL MASTITIS IN 8 DAIRY HERDS.

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INTRODUCTION

Approaches to the treatment of a clinical case of mastitis include the use of intramammary (IMM) antibiotics every milking or once daily; for several days or up to 1 week and in combination with systemic antibiotics and anti-inflammatory drugs depending upon the severity of the case. This poster presents preliminary data from a sub-set of almost 7500 cases on 52 dairy herds during a national mastitis intervention and control study.

MATERIALS AND METHODS

Eight dairy herds having an Incidence Rate of Clinical Mastitis (ICRM) of between 44 and 131 cases per 100 cows/year were involved in a national, 52-herd, intervention and control study that recorded all cases of mastitis for one year. Case details included cow identification; quarter affected; date; grade of mastitis (1=clots only, 2=swollen quarter, 3=sick cow); whether veterinary attention was required; intramammary antibiotic product used (if applicable) along with dose frequency and duration of treatment; systemic antibiotic product used (if applicable) along with dose frequency and duration of treatment and any additional treatment administered such as a separate anti-inflammatory product or oxytocin analogue.

RESULTS

Results are summarised in Tables 1 and 2. Across the 8 farms, a total of 1029 cases (98%) had full treatment information recorded. A total of 709 cases (68.9%) were treated with an IMM antibiotic product, only whilst 215 cases (20.9%) received combined IMM therapy with systemic antibiotic therapy. Non-steroidal anti-inflammatory drugs (NSAID) were given in a total of 89 cases (8.6%) and 9 cases (0.9%) were not treated. Veterinary advice was sought for just 5 of all grade 3 cases (3%). Duration of treatment with IMM antibiotic therapy and systemic antibiotic therapy was looked at with a cut-off point of less than or 3 or more days. The approach to treatment varied widely between the 8 farms.

Table 1 Treatment of clinical mastitis depending on grade

	Grade 1	%	Grade 2	%	Grade 3	%
TOTAL CASES	450	43.7	414	40.2	165	16.1
IMM antibiotic only	389	86.4	306	73.9	14	8.5
IMM antibiotic plus systemic antibiotic only	49	10.9	87	21.0	79	47.9
IMM antibiotic plus NSAID only	0	0	12	2.9	0	0
IMM antibiotic plus systemic antibiotic plus NSAID	0	0	7	1.7	69	41.8
Systemic antibiotic only	3	0.7	1	0.2	3	1.8
Systemic antibiotic plus NSAID only	0	0	1	0.2	0	0
Not treated	9	2	0	0	0	0
Total cases receiving an IMM Antibiotic	438	97.3	412	99.5	162	98.2
Total cases receiving a systemic antibiotic	52	11.6	96	23.2	151	91.5

Table 2 Duration of treatment of clinical mastitis

	Grade 1	%	Grade 2	%	Grade 3	%
IMM < 3 DAYS	204	46.6	160	38.8	39	24.1
IMM 3 days or more	232	53.0	252	61.2	123	75.9
Systemic antibiotic < 3 days	36	69.2	57	59.4	118	78.2
Systemic antibiotic 3 days or more	16	30.8	39	40.6	33	21.9

CONCLUSIONS

There was a high incidence of grade 2 cases within the data set (40.2%). Treatment of grade 1 cases was largely by IMM antibiotic therapy as expected, but for grade 2 cases only 4.8% received an NSAID. More than 90% of grade 3 cases received systemic antibiotic therapy, but only 42% received an NSAID. The duration of treatment with IMM antibiotic was more likely to be extended for grade 2 and 3 cases. The majority of systemic antibiotic treatments were administered for less than 3 days.

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FARMER ATTITUDES AND "SPECIALIST" ADVICE ON FARMS WITH ABOVE AVERAGE MASTITIS INCIDENCE

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SUMMARY

Fifty two dairy farmers randomly recruited for a national mastitis intervention study, from farms with over 35 cases of mastitis/100 cows/year, were asked about their perception of mastitis and any advice received on mastitis control in the past year. Ninety percent ranked mastitis as their first priority for disease control and 77% considered mastitis cost them more than lameness, infertility or other diseases. Twenty seven percent had had a specialist investigation for mastitis carried out. Veterinary surgeons were involved in about two thirds of these. In 40% of the herds, any regular veterinary involvement in mastitis control was solely advice on treatments. Twenty one percent of farmers said they received no advice on mastitis control from their veterinary surgeons.

INTRODUCTION

Despite extensive research into mastitis, the national incidence of clinical cases has not decreased in the last 20 years. A national mastitis research project provided the opportunity to assess farmers' perception of mastitis problems, and the advice they had been given.

MATERIALS AND METHODS

Fifty two dairy herds with an incidence rate of clinical mastitis (IRCM) above 35 cases/100 cows/year in 2003 (as recorded on National Milk Records) were recruited for an intervention study. Interview questionnaires were used to discover the farmers' perception of their mastitis problems and the source and type of support and advice they had received in the year prior to recruitment in April 2004.

RESULTS

Eight percent of farmers (with IRCM of 47 to 94 cases per hundred cows per year) did not consider they had a mastitis problem. Ninety percent ranked mastitis top of their disease control priorities, and 77% perceived that mastitis cost them more than any other disease. The involvement of the farmer's regular veterinary surgeon in mastitis control ranged from no help with mastitis control (21%) through treatment advice only (40%) to various

combinations of bacteriology, regular discussion, advice on mastitis prevention and dealing with high cell counts.

A specialist mastitis investigation had been carried out on 27% of the farms. About two thirds of these involved a veterinary surgeon – either a consultant or the farm's own vet. Farm business consultants, milking machine specialists and nutritionists were also involved. The areas in which advice was given were polarised towards parlour issues and are listed in Table 1.

Table 1 Areas of advice provided in 14 mastitis investigations

Area of advice	No. of farms receiving advice
Milking machine	4
Parlour routine	3
Parlour chemicals	2
Grouping cows	2
Yard management	2
Dry cow treatments	2
Transition cow management	1
Treating clinical cases	1
Youngstock	1

CONCLUSIONS

The majority of farmers recruited to this study ranked mastitis above other diseases in terms of control priorities and perceived cost. Nevertheless, only 27% had undertaken a specific investigation of their herd problem in the last year. Veterinary input to many of these problem herds was minimal. The lack of a sensible well specified whole farm approach may be contributing to the poor rate of progress in mastitis control in the UK.

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SCRAPBOOK OF MACHINE MILKING – WHERE ARE THEY NOW?

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The history of the milking machine has been thoroughly described, with entertaining illustrations, by the late Frank Dodd and Harry Hall (1) in a concise and readable story that every serious student of dairying should know. Part of Frank's material was a collection of marketing and technical brochures, wall charts and pamphlets from before to after World War II. A selection from this material shows the variety and enterprise, conservatism and conformity of milking systems. More than 50 years later the principles and many of the methods remain the same even if the suppliers have evolved, merged, exchanged or simply faded away. The material from the austerity period shows a romanticism, style and culture probably confined forever to the scrap-yard, like probably all of the equipment.

REFERENCES

1. Dodd, F.H. and Hall, H.S. (1992) Chapter 1 History and development. In: *Machine milking and lactation*, Eds A.J. Bramley, F.H. Dodd, G.A. Mein and J.A. Bramley, Insight Books, Berkshire UK and Vermont, USA.