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INTRODUCTION

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In arriving at a theme for the 1994 British Mastitis Conference, the organisers were acutely aware of the rapidly changing situation in milk marketing that is taking place at the present time .

New Demands on Mastitis Control was chosen in an attempt to cover not only items requested by delegates at the 1993 Conference but also to discuss the possible effects that market demands may have in relation to mastitis control.

The Conference will cover new developments in mastitis control, the ever popular session on practical methods of control on the farm and then a look at possible future demands on dairy farmers, where we stand at present and how to make improvements and to plan for the future.

There will again be a wide ranging poster display and plenty of time for discussion, which has always proved to be a very lively part of previous Conferences.

The organisers hope that delegates will continue to feed back their views on the Questionnaire provided and very much hope that the 1994 Conference, which is the seventh annual Conference, will prove to be as informative and enjoyable as in the past.

NEW DEVELOPMENTS IN MASTITIS CONTROL

THE USE OF A J-5 BACTERIN TO PREVENT COLIFORM MASTITIS IN THE UNITED STATES

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SUMMARY

An oil adjuvanted J-5 *Escherichia coli* preparation has been utilised successfully in the United States as an aid in the prevention of coliform mastitis in dairy cattle. The reduction in the incidence of clinical coliform mastitis ranged from 65% to 80% in four large field studies. In a large well monitored safety study the bacterin did not cause adverse systemic reactions and it caused only minimal injection-site reactions. Increased profits of $32-$57/cow/lactation have been projected when this oil adjuvant *E. coli* J-5 bacterin is used as indicated.

OVERVIEW OF COLIFORM MASTITIS

The coliforms are an important group of "environmental" organisms that can cause mastitis. The most frequently isolated intramammary pathogens from this group include *E. coli, Enterobacter aerogenes and Klebsiella pneumoniae. Pseudomonas aeruginosa, Pasteurella multocida* and *Serratia marcescens* are less commonly isolated Gram-negative bacteria. Since most of these organisms are either normal gut inhabitants or are found in bedding materials there is almost constant exposure of the mammary gland to these environmental bacteria (1).

THE DISEASE

The most commonly recognised manifestations of the disease are the acute or peracute forms which are characterized by various systemic signs including high fever (41°C or higher), toxaemia and occasionally death. The cow may suddenly develop a very swollen, red and hard udder that yields a yellow, watery milk. As the disease develops, the affected cow often refuses food and dehydrates rapidly. Heroic efforts may be required to save a cow in this peracute to acute condition. Chronic active forms of the disease with periods of quiescence, punctuated with acute flare-ups, may also occur but are much less common. The clinical signs of the disease result from the release of endotoxins contained in the cell wall of these Gram-negative coliforms. The endotoxins are only released upon the death of the organisms. If the infection is severe and enough endotoxin is released, local quarter inflammation occurs. Absorption of endotoxins into the blood (endotoxaemia) causes the systemic clinical signs. According to the National Mastitis Council's "Current Concepts of Bovine Mastitis", "About 80% of all coliform infections present during lactation will result in clinical mastitis, and about 10% will cause peracute mastitis requiring intensive therapy and veterinary services" (2). Many cases of clinical coliform mastitis have visibly abnormal milk and mild to moderate swelling of the infected quarter but little or no systemic involvement. When chronic coliform infections occur they are usually caused by bacteria other than *E. coli*.

CONTROL METHODS

Even though clinical coliform cases are treated with antibiotics and supportive therapies, the focus for this disease should be on prevention since there is at present no method of reversing the effects of the released endotoxin. Therefore, a complete preventive management program should be implemented to enhance general environmental, to prevent contamination of the teat when the streak canal remains open following milking and to enhance the general immunity of the cow to Gram-negative organisms. Unlike mastitis caused by *Staphylococcus aureus* or *Streptococcus agalactiae*, cow to cow transmission at the time of milking has minimal impact upon the spread of coliform mastitis since the source of infection is the environment. Infection may be introduced at milking, between milkings, or during the dry period.

Specific management procedures, and their rationale, that can be utilised to prevent clinical infections are recognised (1).

1) The proper washing of the teats and mammary gland is essential in order to reduce the exposure of the teat ends with contaminated wash water dripping from the udder or back of the cow.

2) Dipping of the teats prior to milking has been utilised to reduce the population of skin contaminants and consequently should reduce the incidence of infections of environmental origin.

3) Providing ideal bedding is another method of reducing the risk to coliform mastitis. Bedding contamination rates in excess of 1×10^6 bacteria/g have been associated with increased incidence of clinical coliform mastitis.

4) Keeping the cows standing after milking will help to ensure that the streak canal has closed thus reducing the incidence of coliform mastitis.

5) Enhancing the immunity of the cow to Gram-negative pathogens through an effective vaccination programme is apparent. The vaccination programme must be designed to reduce the initial challenge by the pathogen early in the disease pathogenesis.

THE CORE ANTIGEN (J-5) VACCINE CONCEPT AND ITS USES

The vaccine

The morbidity and mortality associated with Gram-negative infections are a consequence of the host animal's reaction to endotoxin, the lipopolysaccharide (LPS) cell wall component of Gram-negative bacteria. Sometimes these endotoxins can produce profound effects by initiating a cascade of events that affect many different tissues including the cardio-pulmonary system, inflammatory mediators, macrophages, neutrophils, platelets etc. Occasionally animals will die from "mediator shock" which is the systemic manifestation observed after a stimulatory factor, such as endotoxin, causes the release of preformed inflammatory compounds or mediators from cytoplasmic granules and causes production of newly synthesized inflammatory compounds. If sufficient concentrations of these mediators are present vascular collapse and death will occur. Since it is very difficult to treat the affected

animal at or before the cascade of events start, it is important to assist the animal in preventing disease by stimulating its own immune system against these Gram-negative organisms and their toxins (1).

There are three different regions of the Gram-negative bacteria LPS that are theoretical targets for stimulating protective antibodies. These are the O polysaccharides, the core polysaccharides and Lipid A. The O polysaccharides are found on the outermost portion of the LPS and vary with each species and serotype of Gram-negative bacteria. Therefore the vaccines containing only O polysaccharide side chains and the pilus antigens, such as K88 or K99, are very strain specific and provide little or no cross protection against other Gram-negative bacteria. Lipid A is a common toxic component of the Gram-negative bacteria but is located deep in the bacterial cell wall and is considered a poor immunogen. The core polysaccharide region of the LPS lies between the Lipid A and the O polysaccharide. It is highly antigenic and shows considerable homology among various gram-negative endotoxins making it a good choice for use in cross-protective vaccines.

Several mutant strains of Gram-negative bacteria are devoid of the outer O polysaccharide side chains. This allows exposure of the cross-reactive core antigens to the animals immune system producing cross-protective antibodies against various Gram-negative bacteria. One of these strains is a genetically stable, rough mutant of *E. coli* O111:B_4 which is commonly called J-5. It lacks an enzyme, udg-4-epimerase, which is necessary for completion of the LPS core and subsequent attachment of the O polysaccharides. When used in properly designed bacterins, this incomplete core of the J-5 LPS antigen induces antibodies which cross-react with various other Gram-negative organisms. This provides cross-reactive immunoprophylaxis against the Gram-negative environmental coliform bacteria. This prophylaxis, along with management practices that reduce the environmental load of these bacteria and prevent their entry into the teat after milking, can work in tandem to reduce the incidence and severity of coliform mastitis in dairy cattle herds.

The use of this *E. coli* core-antigen vaccine in late gestating dairy cows has the added advantage of allowing passive antibody transfer via the colostrum to neonatal calves. These passively acquired antibodies against the Gram-negative bacterial core antigens can be utilised along with proper management practices to reduce the incidence and severity of Gram-negative infections in the neonatal calves of the herd.

Use of a J-5 vaccine to prevent clinical coliform mastitis

In the 1980's J S Cullor and other researchers at the University of California-Davis (UCD) found that clinical signs of endotoxic shock were less severe in calves vaccinated with an *E. coli* J-5 bacterin. Serum IgG_1 ELISA titres against *E. coli* J-5 decreased during the course of endotoxin infusion, suggesting the clearance of cross-reactive IgG lipopolysaccharide immune complexes by the reticuloendothelial system (1). Similar protection was noted in vaccinated calves that were challenged with live *Salmonella typhimurium.* In addition the UCD researchers documented that cows with naturally low titres to *E. coli* J-5 experienced a five-fold increase in the risk for clinical coliform mastitis when compared to cows with higher anti-(J-5) titres (3). To further the research effort experiments were conducted to investigate the efficacy of an *E. coli* J-5 bacterin in reducing the incidence of coliform mastitis. The bacterin and the dosage regime used in these studies were developed by the researchers at UCD to maximize the protection of the cow during the time that she is most

susceptible to coliform mastitis. In order to overcome the immune suppression that the cow experiences before and after parturition a three shot dosage regime is utilised. The UCD researchers also selected an oil adjuvant in order to maximize the immune response to the J-5 antigen.

This successful vaccination program includes administering the product twice during the dry period (at drying-off and mid-way through the dry period) and within 2 weeks post-parturition.

A commercial product, based on the UCD research, was first produced and commercialised in the State of California by Poultry Health Laboratories, Davis, California in a collaborative venture with the California Milk Advisory Board and the University of California-Davis. A California conditional licence was issued in 1989 and the full California licence was obtained in 1991. The product received national (USDA) approval in August of 1993 and is sold by The Upjohn Company. Over 4 million doses of the product have been utilised in the United States.

Field Efficacy Trials

All studies reported below utilised an oil adjuvant as described above. The dosage regime for the studies was the same as that described above except for the VMTRC study where the last injection was administered within the last two weeks prior to parturition.

University of California-Davis J-5 Field Trial 1: In a 17-month double-blind trial involving 486 cows in two herds, the incidence of clinical coliform mastitis in vaccinated cows was only 2.57% (6/246) compared with 12.77% (29/240) in the controls. Thus the incidence of clinical coliform mastitis in the vaccinated group was 80% less than in the control group. In addition, three cows in the control group were culled due to chronic coliform mastitis and a fourth cow was culled because of post coliform agalactia. No cows were culled in the vaccinated group (4).

University of California-Davis J-5 Field Trial 2: This trial, which supported the efficacy for the Upjohn J-5 bacterin licence, involved 845 cows. The incidence of clinical coliform mastitis was only 3.77% (16/424) for the vaccinated group compared with 10.69% (45/421) for the control group. The incidence of clinical coliform mastitis in the vaccinated group was approximately 65% less than in the control group (5).

UCD Veterinary Medical Teaching Research Center J-5 Vaccine Field Trial: In a trial involving 441 cows on a dairy in central California the incidence of clinical coliform mastitis was only 3.3% (7/212) for the vaccinated group compared with 10.9% (25/229) for the control group. The incidence of clinical coliform mastitis in the vaccinated group was approximately 70% less than in the control group (6).

Ohio Agricultural Research and Development Center J-5 Field Trial: In this 2.5 year study involving 225 cows in an Ohio dairy herd there was a 75% reduction in the incidence of clinical coliform mastitis in the vaccinated group compared to the control group. It should be noted that the percentage of the quarters infected with Gram-negative bacteria at calving, as measured by sampling all quarters, did not differ between groups. Interestingly a total of 67% of the quarters infected with Gram-negative bacteria at calving became clinical during

the first 90 days post-parturition for the control group. In contrast, only 20% of the quarters from which Gram-negative bacteria were recovered at calving in the J-5 vaccinated group had clinical coliform mastitis during the first 90 days of lactation (7).

Safety of the Upjohn J-5 Bacterin

Safety Testing of the Formulation: Gram-negative vaccine products have traditionally been troubled with adverse reactions. These products often contain high levels of endotoxin which can affect the animal especially if several Gram-negative products are used at the same time and/or if the animals are stressed. When high levels of endotoxin are introduced into animals there should be concern as to whether the vaccination program could create more harm than good.

When the UCD researchers established the J-5 formulation the level of endotoxin was kept at a minimum. First they demonstrated that the *E. coli* J-5 strain naturally produces less endotoxin when compared to *Salmonella dublin* grown under identical conditions. They then demonstrated that multiple washings significantly decreased the amount of free endotoxin in the J-5 antigen (6). As a result a triple wash procedure was instituted and has continued to be used in the Upjohn J-5 bacterin that is produced today. The Limulus amoebocyte lysate (LAL) test is used to measure free endotoxin. The Upjohn J-5 product contains less than 200 ng/ml of endotoxin compared with other Gram-negative bacterial products that contain milligram quantities of free endotoxin as measured by the LAL test.

Field Safety Evaluation of the Upjohn J-5 Bacterin: A five state field study was conducted to determine the field safety of the Upjohn J-5 bacterin. A total of 1079 cows and heifers were used. There were 541 vaccinated and 538 non-injected negative controls including 87 vaccinated and 86 control heifers. Cows that were assigned randomly to the vaccination group received their first 5 ml dose at the time of drying off. Heifers assigned randomly to the vaccination group received their first 5 ml dose approximately two months prior to calving. The second injection was administered approximately 30 days later for both the cows and heifers. The third injection was administered within one week post calving. Five ml of bacterin was injected subcutaneously four inches (approximately one hand-width) cranial to the shoulder of the cow or heifer. This schedule and procedure are the label recommendations.

The results of this study demonstrate that the administration of the per label recommended three injection dosage regime for the Upjohn J-5 bacterin does not cause adverse systemic effects. There were no systemic reactions caused by the injection of the bacterin. There was only one abortion that occurred within 72 hours of an injection and no adverse clinical signs were observed in the animal that would attribute the abortion to the injection. There were 1070 injections administered to pregnant cows/heifers at their first and second injections. There was a very high rate of resolution of injection site lesions by day 60 post injection. The injection site resolution rate was 98.6%. The results of this study demonstrate that the Upjohn J-5 Bacterin is safe and causes minimal injection site reactions (8).

Economics of the Upjohn J-5 bacterin

Two studies have been reported that address the economics of using the J-5 bacterin for prevention of clinical coliform mastitis. In the first, Gonzales (9) based his analysis on the

incidence of clinical coliform mastitis in California dairies. It was concluded that there was a $32.00/cow benefit from the three time administration of J-5. In another study the analysis based on a United States database concluded that the use of J-5 would increase profit by $57.00/cow in a typical herd (10). In addition it was calculated that it would be profitable to use the J-5 bacterin even when the incidence of clinical coliform mastitis was only 1%.

CONCLUSIONS

An oil adjuvanted J-5 *E. coli* formulation has been used successfully in the United States as an aid in the prevention of clinical coliform mastitis in dairy cattle achieving a reduction in the incidence of clinical coliform mastitis from 65% to 80% in four large field studies. The bacterin was formulated to have as low levels of endotoxin as possible to help assure its safety. In a large, well monitored, safety study, the bacterin did not cause adverse systemic reactions and it caused only minimal injection site reactions. Now over 4 million doses of the bacterin have been utilised in the United States dairy industry. The increased profit from using this vaccine has been projected to be from $32 to $57/cow.

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VACCINES - HAVE THEY A FUTURE IN THE UK?

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SUMMARY

Vaccine preparations intended to help in the control of bovine mastitis are available in some countries and several new products are under development. These preparations are specific to particular types of mastitis. The likely means of use of these products and their possible impact in the UK is considered. It is essential as the products are type-specific to identify the particular mastitis problem prevalent in the herd to ensure suitability. The value of use includes animal welfare, milk quality and a financial calculation.

The requirements of a vaccine in deciding the advantages in using any particular product in any herd are considered including selective versus blanket use. It is concluded that vaccines will be useful in the UK but that by the time products are available it will be possible to optimise their use. They will always remain additional to conventional mastitis control practices.

Much of the drive for further improvements in disease control comes from implementing health management programmes. This includes moving from a therapeutic approach i.e. cure the disease to prophylaxis, not the chemical prophylaxis as found in dry cow therapy, but by improvement of biological systems. These improvements can include breeding for resistance, when we know what the resistance mechanisms are, and improving immune defence systems by vaccination. Use of such systems in control of mastitis has been considered and tried with no obvious success over a number of years but we may now be on the threshold of producing effective vaccines largely from developments in understanding the chemistry of the pathogens causing disease, the pathology (mechanisms of infection) and the special immunological conditions in the mammary gland. The success of such vaccines will vary with the quality of the product but it is also important to manage use by matching the ambitions to the problem and by realistic assessment of the cost/benefit. The potential application of vaccines to help control mastitis will be considered.

THE PROBLEM

If a 'mastitis' vaccine were offered now there would be several different responses. Many would be very willing to buy and a few reluctant. Thankfully a large (?) number of buyers would ask what was on offer. It is important to know what the vaccine is intended to do, to what problem it is being applied and, for many people, how much it will cost for what effect. These questions are asked at all levels of producing animal health products, by the researcher in defining the problem, the manufacturer in assessing the potential of the product and by the consumer in entrusting his faith and money. These questions comprise a classical problem of Animal Health Economics where we need to consider the concepts involved, the procedures to be used and the information necessary to decide on a strategy to optimise health management.

Given the assumption that everyone in this industry wishes to maintain, if not improve animal health by controlling disease, any system can be afforded even when input slightly exceeds output. When making the decision for or against any development or new product how we decide 'for' or 'against' depends on three questions.

1. What is our estimate of the financial loss caused by the disease?

2. What are the costs and benefits relative to alternative approaches? (The commercial company might add a question about market opportunity/share here)

3. How do we optimise our decision to maximise the effect for the individual animal or the whole herd, or even the national herd depending on who needs to make the decision?

Mastitis is a complex of controllable diseases any of which may be better controlled by a vaccine. Market economics tend to influence how much control we want and the level of product quality (TBC and SCC mostly) necessary as demand tends to increase with quality. An increase in quality drives a supply need (more quantity) and so controls product value (market price). The summary of this is that disease control is beneficial as it minimises resource use (real cost) and maximises production (real value) whether we make the judgment from the local/farmer level or at a national/strategic level.

Mastitis is different from most of the other production diseases. The product price (quality requirement) has been buffered by the pool approach of selling and the flexibility of the selling market unlike e.g. BSE which has led directly to lost markets. It should be considered how the partial or gradual removal of the pool approach will alter our assessment of quality control and hence adoption of new technologies.

Let us consider the three questions advanced earlier in deciding on the needs, and opportunities, for a vaccine.

FINANCIAL LOSSES

These are not very important, not in the sense of the total loss, as they only convey an overall view. The losses tend to be spread over the whole industry, herd etc. as it is difficult to predict specific areas of loss. The accuracy in assessing loss is suspect as it depends on the amount and usefulness of the data including the precision with which information is collected. The main problems in calculating losses are

(a) they are not always obvious or pronounced e.g. sub-clinical mastitis or some dry period infections

(b) they are influenced by other factors e.g. nutrition, either the quality available or quantity consumed

(c) they are mostly temporal and there are complicated interactions with time such as stage of lactation or age of animal

(d) they are part of a complex with the whole health situation. This may be less so for mastitis than other diseases whilst we might predict that mastitis is commonly related to calving time, perhaps milk fever etc, no clear relationship has been shown (12).

There have been several attempts of differing completeness to model the financial losses (1) but it is difficult to know how accurate these are. This can be shown by the disparity in the estimates of loss occurring with clinical mastitis from £40/case (2) to £186 av. (6). The problem is in the generality of the studies, most deal with a little data from many farms whilst others deal with certain types of farm only. None of them deal with your farm!

For national and marketing needs this general information often works adequately and is appropriate to make decisions. These data show approximately 40 cases of clinical mastitis in every 100 cows every year (3) and this does not appear to have changed much over a number of years. Actually the range is 5-120 cases/100 cows/year (11) and 2-40% quarters are infected at any time depending on the farm. It can be quite important to assess how important mastitis is relatively as there are widely different economic implications for different rates of incidence.

The term mastitis is the biggest generality of all. It has been reported that there are more than 135 agents which cause mastitis (13). It will be some vaccine to control this problem. More typically on the UK farm there will only be half a dozen common agents (Table 1) and the general occurrence of these will vary very widely between farms.

Table 1. Breakdown of 40 cases of mastitis on a farm according to national average incidence

Agent	No. cases	Range
S. uberis	12	2-25
Gram - coli	14	2-30
S. aureus	3	0-30
S. agalactiae	0	0-15
S. dysgalactiae	1	0- 6
OTHERS	10	2-25

Every farmer needs to know his particular mastitis portfolio, even then control of the major pathogens only accounts for 75% of mastitis, 25% may be caused by the other 132 agents. In assessing the losses and defining what we want from a vaccine first the problem has to be defined. A **mastitis** vaccine will never exist but we may be some significant way towards separate vaccines against coliform bacteria (7), streptococci (9) and staphylococci (several studies). Perhaps at some time in the future these might be applied sequentially or in combination, we are even further away from a single multi-valent vaccine.

Given the huge variation possible in the mastitis situation on any one farm it becomes essential to know exactly what that situation is. Some farmers/veterinary advisors may already know and the knowledge becomes more essential as we try to tackle residual mastitis problems given the major advances made in reducing contagious forms of mastitis by

hygienic milking practices. It is difficult to compare expectations with results when it is unknown if the action taken was appropriate.

RELATIVE COST-BENEFIT

When making comparison of effect of using a vaccine with alternative means of control we need to know the specification of the vaccine and the practicabilities of using it. Many of these can only be guesses, at present, although many will be requirements in the design of a commercially acceptable product.

(a) **Specificity** So far it looks as if first generation products will be specific to one type of mastitis only e.g. coliform infections.

(b) **Efficacy** The efficacy will never be 100% but there is no need to be absolutely effective. This is especially so for contagious forms of mastitis. There are very many well developed models of disease transmission and control which show that total eradication is achievable from entire populations using vaccines less than totally effective. It is unlikely that eradication will ever be possible for 'environmental' forms of mastitis as there will always be challenge from sources which cannot be controlled. With products to control coliform and *S. uberis* infections the highest achievable efficacy should be the goal. In these cases a vaccine will only be part of a control plan. Good practice, the five-point mastitis control plan, will always be necessary too but vaccination can be envisaged as making a major impact on the rate of clinical disease. Therefore, trying to estimate a relative cost-benefit is not entirely appropriate for a vaccine applied to an environmental pathogen, coliform or *S. uberis*. These vaccines will be used to obtain an additional effect as conventional techniques achieve very little control.

(c) **Persistency** There will be an obvious desire to maximise the duration of effective protection and probably a minimum of a lactation will be required with boosting each dry period. Greater persistency would be a bonus. It is quite unclear if a vaccine would work in the very different conditions of both the dry and the lactating gland where the conditions are so very different. However, control in the dry period would be extremely useful especially for control of *S. uberis* infections as a significant proportion of the infections have their origin in the dry period. Different periods of cover may be necessary for the different principle pathogens. Most coliform infections are in early lactation, near peak yield/flow rate and of course in winter; *S. uberis* are more frequent in summer except on loose straw beds. *S. aureus* infections are more evenly spread throughout lactation but clinical mastitis is commoner when yield is declining. It will be essential that vaccination does not lead to milk withdrawal, this and the need for dry period protection, suggests vaccination should take place early in the dry period with dry cow therapy still being needed. It is a requirement of such products that the animal will be protected from any adverse effects of vaccination.

(d) **Means of delivery** This could raise some problems. Many of the early studies used local, intramammary presentation and this can cause a mastitis itself. The most likely practical method, as described for the J5 vaccine, will be 2 or 3 sub-cutaneous inoculations 2-3 weeks apart.

Once this type of information starts to become available then it is possible to start considering the cost of vaccination programmes. Simply this may be that

Cost = 3 x (cost of vaccine + cost of vaccination) + cost of vaccination losses

Benefit then has to be measured. This may be done objectively in field trials and could be assessed as

Reduction in number of clinical cases
(no. of cases needing treatment + reduced use of antibiotics + reduced milk discard + maintenance of yield + reduced labour/vet cost + etc.)
+ reduction in number of sub-clinical cases
+ reduced culling rate
+ increased milk value (lower TBC and cell count)

This all starts to sound very attractive especially as good efficacy rates are claimed so far (7) and published estimates of the economic justification for using an 'appropriate' vaccine show considerable advantages for certain US farms (5) but obviously there will be a need to carry out cost-benefit appraisal for <u>your</u> herd given your mastitis situation. The calculation of benefit has to be considered carefully as it is unknown that if clinical disease is prevented so are sub-clinical infections and that infections are not simply suppressed.

There are other ways of looking at the financial aspects. One is to consider not what the theoretical advantage might be but to be practical, as theory is rarely achieved in production and to set a target for the effect to justify the economic investment. It is important in such calculations that there be consideration of more than the financial cost-benefit. There will be a welfare cost-benefit advantage for the herd and a quality cost-benefit for the herdsman/owner and the consumer. The latter are not to be taken lightly in ensuring the market for milk and the success of the industry.

OPTIMISATION OF BENEFIT

The third question is how to make the decisions on what to do and how to use a vaccine. This may include an extension of the risk assessment of any particular form of mastitis which is targeted by a vaccine, beyond the herd problem. This could be made in several ways. If there is a limitation in persistency of effect then both the calving pattern and season become relevant in optimising use and timing of use of a vaccine. If would be more useful to have maximum effect of a vaccine for coliform mastitis over the housing period and probably against *S. uberis* over the grazing and end of dry period through to early lactation.

The greatest advantage in optimising effect might come from a risk assessment for individual animals within the herd. This can start from very basic information. The forty cases/100 cows/year occur in 25% cows, the remainder of the cases are recurrence of the original uncured infection or a subsequent infection in the same cow if not quarter. The recurrence/re-infection rate will vary with herd especially with the type of pathogen dominating the disease. The recurrence rate is highest with *S. aureus* but there is little difference in re-infection rate between the different pathogens that can be ascribed to the pathogens alone. The most basic fact from this consideration is that 50% cows in the herd will NEVER suffer clinical mastitis which gives considerable scope for optimising the control

programme. The low rate of infection is greatest for heifers when 80-90% will not suffer mastitis. The likelihood of infection increases with age increasing by about 50% per lactation (10). This information introduces more questions. Whether only multiparous animals, which might be considered proven !!, should be protected preferentially especially when the target is not a contagious form of mastitis and how to identify the sub-population of the herd most at risk?

That one half of all animals never suffer mastitis and a small proportion suffer repeated infections introduces the concept of different susceptibility and identifying this as a risk factor in targeting mastitis prevention strategies. Several risk factors have been identified. There is a small genetic effect (4) but mostly the effect is environmental related to the husbandry and management used on the farm. One well identified effect is that high flow rate, and these tend to be high yielding, cows suffer more infections (8). There are still a large number of investigations necessary to unravel the complexities to risk of mastitis and such understanding may make a major impact on how vaccination and other mastitis control programmes are used to maximum effect. Investigation of risk factors to infection and development of mathematical models to assess importance of the individual factors along with estimating the impact of control strategies is an area being developed at Compton now. It is becoming of increasing importance to mastitis and other animal disease as less resource is available to carry out classical large scale epidemiological investigations.

CONCLUSIONS

It is extremely likely that even individual pathogen-type vaccines will be economically effective. There is an unarguable case that they will be cost-effective for animal welfare and the image of the industry may well require them too. So far we do not know how we shall use them most effectively. They will probably be used in a 'blanket' fashion initially but more sophisticated use will be possible when we understand risk more. To do this we need useful, high quality data especially on mastitis awareness within each herd, information which should be available now for good disease control. It will be essential to strive towards the highest quality management of udder health, effective vaccines will not be an excuse for lowering standards of husbandry. At the very best a complex vaccine could control 75% cases. The remainder including those already being prevented (mastitis incidence has been reduced by approximately 100 cases/100 cows/year since 1970), will require continued use of the five point control plan.

Yes, vaccines will be useful in the UK although unfortunately for the producers they will not sell 3 x 2.8 million x year x vaccine as by the time licensed products are available in the UK we shall have a good idea on how to make the most efficient use of them.

ACKNOWLEDGEMENT

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TACKLING MASTITIS ON THE FARM

TEAT SKIN LESIONS ON THE DAIRY COW

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SUMMARY

Teat skin lesions occur in every dairy herd from time to time, but since the introduction of teat disinfectants, particularly those with added emollients, lesions are not the problem of earlier decades. An outbreak of teat lesions will increase the risk of intramammary infection and subsequent clinical mastitis. Good management practices, well maintained milking machines and a good parlour routine will normally ensure a trouble-free herd. However if an outbreak occurs, then attention should be paid to the application of a good quality teat disinfectant containing 10% glycerin. Alternatively a teat gel consisting of a high level of glycerin combined with a disinfectant should be applied.

INTRODUCTION

During the development of the mastitis control routine by the research team at the National Institute for Research in Dairying, teat skin condition was a secondary consideration to the overall value of teat disinfection to reduce new intramammary infections. It had been found during those early days that *Staphylococcus aureus, Streptococcus dysgalactiae, Streptococcus uberis* and other pathogens could survive on the teat skin (1,2). Moreover where a skin lesion was present, it became infected and provided a suitable environment for the pathogens to multiply (Table 1), thus being ideally placed to contaminate the teat-cup liner. Infections of the teat skin can be reduced by the application of post-milking teat disinfectant.

On many of the farms in the 1960's, teat lesions were an irritation that were present continuously to some degree and the best way of dealing with the problem was to apply large amounts of ointments, ranging from proprietary brands to goose fat or even engine oil!

Table 1. The frequency of recovering *S. aureus* from teats prior to milking (3)

	Cows with normal teat skin		Cows with teat lesions	
	No. of swabs	% positive	No. of swabs	% positive
No teat dipping	38	37	44	84
Teats dipped	184	11	116	44

Since the widespread introduction of post-milking teat disinfection, teat skin condition has improved enormously, particularly since the addition of emollients to the various disinfectants (Table 2).

Table 2. Prevalence of teat lesions found at the start of various trials

Year	Number of cows	Percentage of teats affected			
		total	chaps	lesions	Misc*
1966	1524	26.0	14.3	10.9	3.2
1972	904	12.9	7.3	6.6	n/r
1983	569	4.6	1.5	1.4	1.7
1989	2129	3.4	1.2	1.8	0.6

* Scratches or damage
n/r not recorded

INFECTIOUS LESIONS

Pseudocowpox

The lesion is caused by a *parapoxvirus* closely related to the orf virus (contagious pustular dermatitis) of sheep (4,5).

The first indication is often that the animal is uncomfortable during milking. On examination reddening of a small area of the teat may be found. This is followed by the formation of a small raised scab a few millimetres across. This develops into the typical lesion which appears as a ring of small scabs with the centre appearing normal. As healing progresses only a partial ring may be visible. Complete healing takes approximately five weeks for each lesion, but various foci at different stages on the same teat prolong the assault. The lesion, once it has scabbed, does not appear to bother the cow and milking continues as normal. There is an immunity period of three to four months before reinfection occurs (6).

Bovine Herpes Mammilitis

This is a highly contagious lesion, caused by bovine herpesvirus 2 (2,7). The lesion is painful and can affect teats, udder and other areas of the cow. Early signs are reddening of the skin (as seen with Pseudocowpox) followed by vesicular lesions which appear as white blisters filled with a serous fluid. A thick scab forms over the affected area, often covering much of the teat. The scab eventually peels off leaving a soft epidermal layer which takes time to recover the natural elasticity of the skin. Complete recovery takes about three weeks. Reoccurrence of the BHM lesion is unusual. Shearer *et al.* (2) reported that the lesion usually occurs in the summer and autumn, and that extensive scab formation over much of the teat surface is indicative of BHM.

Cowpox

This condition is rare and can only be identified with laboratory confirmation of the presence of the cowpox virus. It was described by Francis (8) as a vesicle developing into a pustule which ruptures followed by a thick red scab. The lesions appears similar to the early stages of BHM. The scab is often removed by the milking action giving rise to an ulcerated lesion which normally heals without secondary problems. However once the infection is in the herd the disease is spread by the milker's hands and the milking machine.

With all the viral infections, close observation of the teats may reveal early signs of an infection. It has been reported that the first sign is the reddening of the skin at the site of the infection. This coincides with tenderness and possible reaction from the cow. Hence, when the scab appears in the case of pseudocowpox, the cow seems unperturbed when being milked, having passed the painful stage. Gibbs *et al.*, (6) include a laboratory diagnosis section in their paper, describing the differences between the various viruses.

Blackspot (Teat End Lesions)

This lesion usually found at the teat orifice is associated with the anaerobic bacterium *Fusobacterium necrophorum* (2), although an earlier lesion or trauma may predispose to the infection. Frequently any lesion at the orifice is termed 'blackspot'. The accepted definition is a scab formed at the orifice often black or black/red in appearance. Milking is prevented by the closure of the orifice by the scab. The lesion affects the lower portion of the streak canal. If the scab is forcibly removed the wound bleeds and the sore is further aggravated by the action of the milking machine. If left to heal the scab will harden and drop off.

ENVIRONMENTALLY ASSOCIATED LESIONS

Chapping

Mild chapping may be seen as small splits in the epidermis following a fold of the skin. They can be horizontal, vertical or diagonal. In severe cases the split may encompass most of the teat, in particular at its base (junction of teat and udder). The latter occurs where there has been little or no treatment to the affected area. Occasionally the chapping will occur as a secondary lesion formed across another.

Chaps can be found on any part of the teat although they are more common at the base. Chaps occasionally affect the orifice. In these cases the problem may exist for varying periods throughout the animal's lifetime.

Chapping of the teat occurs when the skin becomes too dry. This may be due to the weather over which there is no control, or other environmental conditions. Draughty buildings and contaminated bedding predispose to poor teat condition. At milking time, inadequate or no udder preparation, leaving teats contaminated with faeces or mud will quickly lead to poor teat condition. Drying of the skin can occur when the cows are allowed to stand in cold windy places and it can also be caused by harsh chemicals.

Mud Sores

Severe ulcerated lesions were observed by the NIRD team, in association with the accumulation of mud and faeces where udder preparation for milking had been poor or non existent. Such lesions have been termed 'mud sores' (unpublished data). Healing could only commence once all of the contamination had been removed.

Raw Areas (Lick or Summer sores)

This condition (known by various names) is seen between August and October, especially on the front teats. The development follows the licking action by the cow in response to irritation by flies. The lesion may start as any small wound which attracts flies which in turn enlarge the wound by feeding. The ulcerated area scabs over and can become quite extensive covering a large area from the base of the teat to three quarters of the outer area of the teat. Without treatment this lesion will heal after the fly population declines. However the application of a sealant or good emollient dip and/or gel will reduce fly irritation.

Frost Sores

Teat end lesions associated with frosty conditions have been reported by Shearer (2). Lesions of a similar description were observed on a farm in Oxfordshire by Kingwill and Shearn (unpublished observations).

Sunburn

During periods of strong sunlight, unpigmented teat skin can be affected. The reaction is usually confined to the teat surface directly exposed to the sunlight. The surface becomes inflamed and dry, as though burnt. If the teat is stretched the skin in the folds appears normal. In extreme cases the udder can be affected.

A severe lesion, can occur. This has an appearance similar to BHM with raw areas and is the result of photosensitization. The lesion occurs when cattle ingest photodynamic substances from plants. The animal's udder and other areas are also affected with the skin sloughing off (9,10).

Petechiae

Small blood spots less than 1 mm in diameter can be observed on teats. These can be from a few to very many and are seen mainly near the tip of the teat. In severe cases the spots can coalesce and form a scab. These are usually associated with machine milking faults (10), including loss or lack of correct pulsation. Slow-milking cows appear to be more liable to this problem. The condition has been seen on freshly calved cows where teats need "re-conditioning" to the action of the milking machine. This should only be considered a problem if many cows are affected persistantly to a considerable degree.

Blood Scabs

This lesion appears as a raised red scab and is most frequently near the tip of the teat. There is usually no accumulation of fluid under the scab and it soon heals. It is most probably associated with friction of the liner on the teat. It is not a problem in most herds although a higher percentage of occurrences have been noted in "hydraulically" milked herds.

CHEMICAL DAMAGE

Chemical damage results from the use of unsuitable chemicals for udder washing and teat disinfection - more important if pre-milking disinfection is practised. One incident known to the author occurred when the milker used the plant cleaning disinfectant as the dip. Within 24 hours the teats were extremely sore with resultant problems at milking time. This can easily occur where the disinfectant is applied by a sprayer. Care must be taken in using the correct material particularly on dark mornings where the teat disinfectant and plant cleaning drums are stored in the same place. When diluting a concentrated disinfectant, ensure the drums are **clearly** labelled to show that the drum contains useable teat disinfectant. All chemicals may cause teat problems if incorrectly diluted. Teat-cup liners contaminated during plant cleaning and not properly rinsed out before use may also cause problems. The type of lesion associated with disinfectants varies in appearance with the chemical involved. Hypochlorite, if applied at 4% available chlorine level without a period of progressive conditioning from lower concentrations, may cause a burn near the tip of the teat. This forms a blood scab, which will heal after a few days. Early iodine-based dips used to cause peeling skin which would be stained brown. The chlorhexidines if too concentrated also cause peeling skin. In this case the teat skin goes very dark, as though it was dirty, peeling off a very thin layer of the dermis. This would then repeat a week or so later.

The descriptions given are based on observations of teats affected on trials where disinfectants were either being tested or environmental conditions changed. Teat disinfectants formulations have been improved significantly and with the addition of emollients many of the original problems have been removed. It does not follow that in every case seen the basic teat disinfectant can be blamed.

It was observed during a commercial trial that a teat disinfectant containing an emollient was not satisfactory during frosty conditions even though the cows were housed. In this situation knowing the level of the emollient in the ready to use (RTU) solution is important. A disinfectant containing a higher level of emollient was introduced to rectify the situation.

TREATMENT OF LESIONS

Healthy teat skin has a smooth appearance neither dry nor silky. Where a reasonable level of emollient is added to the teat disinfectant and the teats are regularly **well** covered, the skin will have a softer feel and a slightly shiny appearance. This appearance is important in checking for regular good post-milking disinfection, particularly when investigating an outbreak of teat lesions.

It is reported by Shearer (2) that halogen disinfectants are likely to inactivate virus. Hypochlorites or iodophors with emollient are recommended for use during outbreaks of viral lesions. Although not all iodophors will control an outbreak, introducing a different brand of iodophor disinfectant with a pH of 2.0 and a 10% glycerin resolved a serious outbreak of pseudocowpox in a dairy herd in Avon in 1990 (personal unpublished data).

Environmental

With mild outbreaks of chapping etc., the use of a teat disinfectant containing a 9 to 12% emollient at the **point of use**, will promote healing, provided that the lesions are well covered with the preparation. In severe cases, glycerin or a preparation containing glycerin as the main ingredient may be smeared onto the affected area of the teat after milking prior to disinfection.

With teat end lesions the removal of the scab to allow milking prolongs the time the lesion is present. Further damage may be caused by mastitis pathogens leading to a severe clinical incident and administration of antibiotics becomes difficult due to the lesion. Continued use of the disinfectant to minimise the level of bacterial contamination is required.

Post milking teat dipping with a quality disinfectant containing an emollient should prevent most of the teat lesions from occurring. The level of emollient required depends on the circumstances on an individual farm. Knowing what amount is in the disinfectant will allow the user to establish the best level to suit their conditions.

Use of udder salves

The regular use of udder salves should not be necessary to treat teat lesions if a good teat disinfectant is applied correctly. It has also been reported that the antibacterial activity of some udder salves although inhibiting some mastitis pathogens *in vitro* had little or no effect on the bacteria on the teat skin (11,12). A chlorhexidine-based salve was no more effective at promoting healing of teat lesions than an iodophor disinfectant with 10% glycerin (11).

TEAT CONDITIONS ATTRIBUTED TO THE MILKING MACHINE

Hyperkeratosis is the frond-like appearance of the orifice. A scoring system for this has been developed and used over many years. The system quantifies the degree of hyperkeratosis from 0 to 5 and as it may be helpful to practitioners involved with teat problems it will be published.

The teat orifice condition can change under stress, particularly when the milking machine is malfunctioning. Persistent overmilking can increase the degree of hyperkeratosis, therefore attention should be paid to the milking routine and checking for faulty automatic cluster removal (M F H Shearn & J E Hillerton unpublished). The genetic make-up of the cow will also influence, to some degree, the response of the teat orifice. Invariably only a proportion of the herd will be affected with moderate to severe levels of hyperkeratosis.

Machine milking can cause other various visible changes to the teat. On removing the machine blueing of the teat end or the whole teat indicates lack of pulsation. Oedema may be seen, either as a swelling or stiffening at the tip or varying degrees of a ring at the base

of the teat, caused by high vacuum in the liner mouthpiece. Other changes are a wedge-like appearance, more easily felt than seen and a white line across the teat-end. These conditions will not be seen after every milking and in almost all the cases the teat will regain its normal condition soon after milking. Although it is known that with hydraulic milking systems these conditions are seen more frequently, they do occur in conventionally-milked herds, sometimes with severe consequences.

CONCLUSIONS

Teat lesions are not such a problem today when compared to the situation in the 1960's. If a problem exists, it is important to check general husbandry as well as the parlour routine. However prevention is better than cure. Maintaining good teat condition is possible with attention to: 1) Good clean housing conditions in the winter, avoid wherever possible muddy tracks. 2) Application of a good milking routine from udder preparation to good coverage of the teat with disinfectant containing emollient. This is the best preventative action that the milker can apply at milking time. 3) If the milking machine is suspected, check the operation of the ACR's, pulsation and vacuum, liner size compared to the teats, and the way the machine is used.

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MY APPROACH TO MASTITIS CONTROL

R W RUSSELL (Partner/Owner), Church Farm, Brinkworth, Chippenham.

SUMMARY

A brief description is presented of changes at Church Farm between 1980 and 1994. After describing the structure of the farm, the initial efforts in mastitis control which reduced cell count from 1,200,000 cells/ml to 360,000 cells/ml are presented. The extra attention needed to reach the present level of 104,000 cells/ml and to stay there are presented.

INTRODUCTION

The following thoughts and information relate to my experiences over the last 14 years in an effort to reduce bulk milk cell counts resulting from episodes of clinical mastitis within my herd over that period. This effort, has resulted in a reduction of cell counts from an initial figure of 1,200,000 cells/ml to our present rolling average of 104,000 cells/ml (Fig. 1).

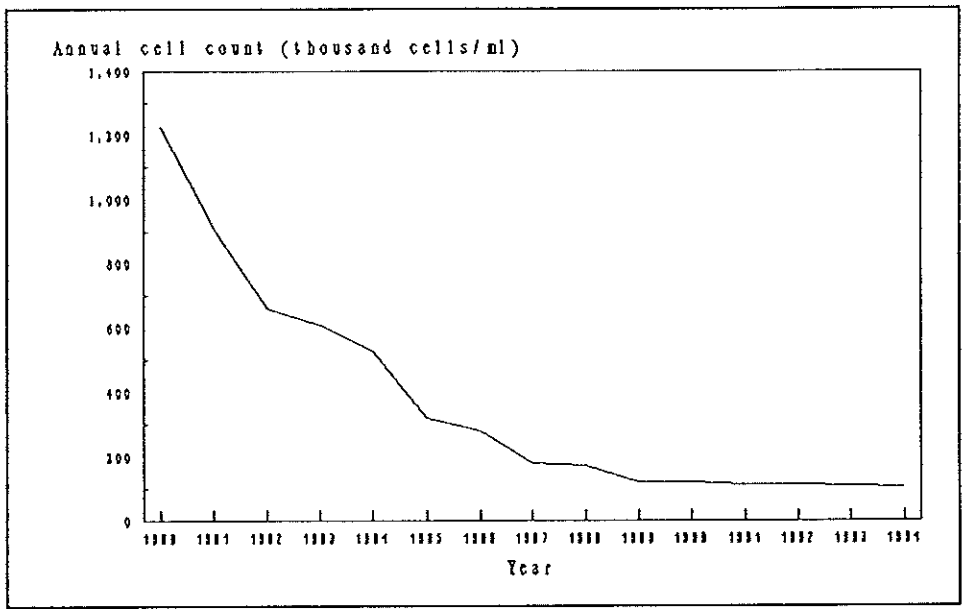

Figure 1 Church Farm bulk tank cell count

THE FARM

Church Farm, Brinkworth, comprises 180 acres of fairly heavy clay soil which is mostly down to permanent pasture. The farm supports a milking herd of 80 pedigree Holstein-Friesian cows, together with 60 young stock to make a total of 140 dairy cattle. The cattle have been home-bred for the last 30 years, and all heifer calves are reared. Nominated bulls have been used throughout this time, and for the last six years these have been predominantly Canadian stock. Cows are housed from the end of September to mid-April, and diet is

block-fed silage with a high energy 18% dairy concentrate in the parlour. Total concentrate usage is at present 2.1 tonnes/cow. The cows are housed in cubicles using dry screened soft sand as bedding. They are milked twice a day through a 6/12 herringbone parlour. Current NMR figures show a herd average of 7,220 litres per cow at 4.21% butterfat and 3.34% protein. The calving index is 381 days. The staff comprise me as principle milker, helped by one son working full-time and one son working part-time.

MASTITIS CONTROL PLAN

Background

A decision was taken in the Spring of 1980 to try and control a chronic mastitis problem that we had within the herd. My father-in-law, Mr. Bob Kennedy, owner of the farm at that time, had remained loyal to a cowman, who had, over a ten year period, grown complacent, and so in conjunction with our vet, Mr. Ray Williams of The George Veterinary Group in Malmesbury, we began a mastitis control programme. From the outset we decided to engage the services of the MMB Mastitis Control Service, our advisor being Mr. Graham Clark. I was given full-time milking responsibility, and vividly recall my first milking on the 14 June 1980. Hygiene standards within the parlour were pretty poor, and milking was a slow, miserable task. Before that first milking, I decided to clear the clutter from the parlour pit. I parked a wheel barrow outside the door and proceeded to remove different sized bricks and blocks that were used as weights to milk-out various cows. On top of this I piled ropes and cords and other gadgets that I was told were essential to milking! I then proceeded to milk 78 cows, of which 16 needed treatment for severe clinical infection. Milking completed, I then decided to tackle the hygiene. I filled a rose sprayer with hypochlorite and sprayed all surfaces, promptly purchased a pressure washer and from that day to this have managed to maintain a fairly clean and considerably more pleasant environment in which to work.

Initial efforts

The next move was to sample all cows individually in conjunction with the MMB Veterinary Lab at Worcester. The results showed 41% (32 cows) with a cell count in excess of 1,000,000 cells/ml and 7.7% (6 cows) with cell counts in excess of 5,000,000 cells/ml (Fig. 2)! We had decided from the outset not to result to drastic culling but, despite this, we did sell 3 cows, 2 of which were only milking on 2 quarters. Udder tissue condition and teat end erosion were a major problem apparently caused by over-milking, poor vacuum stability and bad hygiene. Milking plant testing followed, and we decided to replace the sealed milking units with conventional shells, and Alfa Laval shielded liners. A new vacuum controller was resited in the line, and measures taken to ensure the water used for cleaning with the Acidified Boiling Water System was actually boiling.

More samples were taken in an effort to isolate the pathogens responsible for the mastitis, in order to treat more effectively. All cows were dried off using Orbenin Dry Cow. Sodium hypochlorite was replaced as teat dip by a high emollient iodophor preparation. These initial steps meant that by March 1982, the cell count had dropped from 1,200,000 to 911,000 cells/ml. The individual samples revealed a reduction of samples containing more than 1,000,000 cells/ml from 41% to 21.6% cows. The five-point-plan was fully operational by now, strict records were being maintained and increasing hygiene standards meant that

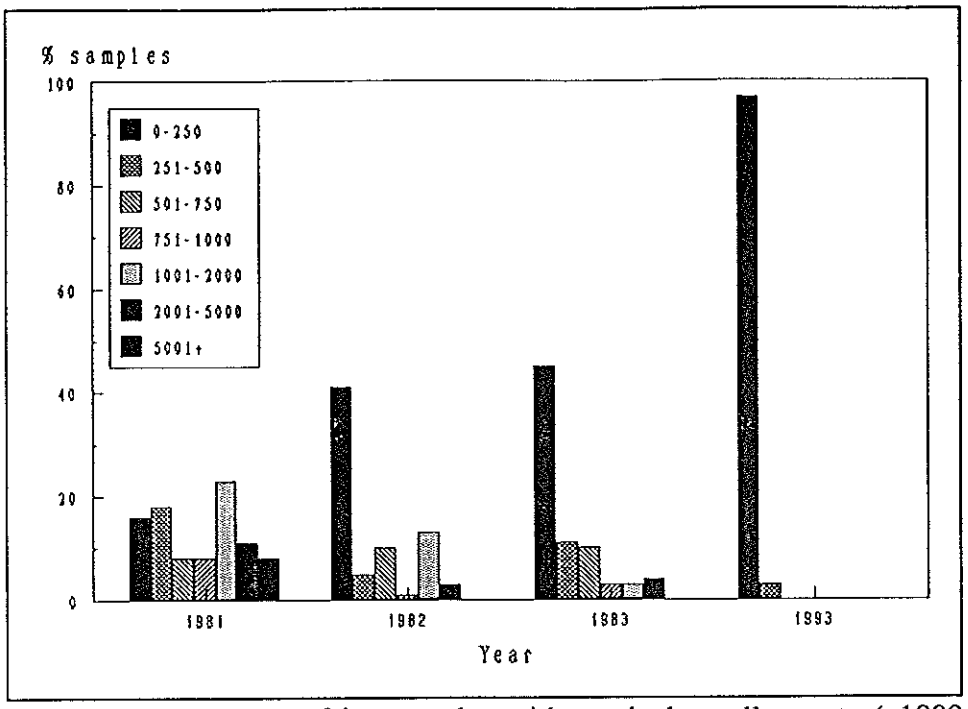

Figure 2 Percentage of jar samples with particular cell counts (x1000 cells/ml) from 1981-1993

milking was becoming more enjoyable. Good progress was being made and by 24 September 1982 the cell count had fallen by another 384,000 to 606,000 cells/ml. The incidence of clinical cases of mastitis had been fairly low and nearly all cases confined to recurrent offenders. By August 1984 samples showed a further reduction in bulk tank cell count to 366,000 cells/ml, with 9.2% of cow samples having over 1,000,000 cells/ml. Cows treated for clinical mastitis had been reduced to 23 (65% of cases occurring in 3 cows). This initial reduction from 1,200,000 to 360,000 cells/ml in the first 4 years was relatively easily achieved. However, the drop from 360,000 to our present level of 104,000 cells/ml with only 2 clinical cases treated in the last twelve months has demanded absolute attention to detail.

The current situation

In 1990 I decided to update the 20 year old parlour, and opted for a Surge re-fit that included a large bore vacuum line and a change to Surge electronic pulsation with a 55/45 ratio split. I also replaced the Alfa Laval claw pieces with Surge large capacity Eclipse claws. Since then I change the liners every six months, irrespective of wear. I have the whole plant tested twice a year, once by the installers and once by the MMB. One of the major reasons for the improvement in cell count has been my obsession with clean cows. Each member of the farm team is fortunate enough to realise the advantages that exist from presenting clean cows to the parlour. We are all fully aware that clean, healthy cows produce clean, healthy milk, and, as we all know, this is becoming increasingly important. Starting at calving, each cow has its own box that is kept scrupulously clean and disinfected after each calving. Cows are washed after calving if necessary, and tails and udders clipped. The cubicle passageways are scraped thoroughly by tractor at each milking and the tractor drivers know that each cubicle

bed must be prepared properly before cows re-enter. The whole cleaning-out operation takes 10 minutes, twice a day, and the cubicles are only re-filled with sand once a week. This takes about 1 hour, and uses 4 tonnes of sand for 80 cubicles. Total cubicle management time equals 3 hours and 20 minutes per week and costs £30. In 1989 I replaced the Newton Rig cubicles with those of a Dorstun space sharing design. All yards are kept as clean as possible, and collecting yards are pressure washed daily. Cows are never left to stand in slurry.

Cows are held for about one hour after milking to ensure teat ends close fully. Teat dipping, using an iodophor from a cup, is carried out on every cow at every milking. This is not a hit and miss occasion. The whole teat is totally immersed immediately after milking, and teat dip changed every milking. Dry cow therapy is also used on every cow at drying-off. I batch cows to dry-off at the end of afternoon milking, after washing the parlour through. Orbenin Dry Cow has been used for a number of years and I adopt a policy of cleaning the teat ends thoroughly before inserting tubes. Tubes have never been fully inserted into teats and I find that insertion is easier at afternoon milking because the higher day time temperatures make the carrier more viscous

CONCLUDING REMARKS

I am sure that the points I have mentioned are familiar to all milk producers and there is nothing new or radical. However, I realise I have a duty to provide the type of foodstuff the market demands, and I believe I realise the true costs of high cell count milk. Not only the tangible costs that include antibiotics, lost milk and lost bonuses, but more importantly the invisible costs such as a disrupted milking routine caused by dumping of milk and inserting intramammary tubes, and above all the lost potential. The occurrence of only 3 cases of clinical mastitis in the last 2 years have helped to achieve an increase in yield of my cows from 6,600 litres to 7,220 litres, as well as composition improvements. The real reason I have been able to make the reduction in cell count, is because I have a real pride in my cows. Humble though they may be, I love each one and treat them as my best friends. I look forward to each milking and feel privileged to be able to share 5 hours every day in their company. For me, milking is not a black and white blur, instead it is a special chance to appraise and continually re-assess my herd. Every milking I am lucky enough to have the chance to monitor their condition, to plan their next calf and to check on their udder texture and teat end condition. I do not want this special time to be interrupted by having to treat mastitis, or to see a beautiful heifer become ugly because she has lost a quarter. All the cows I milk now have level, even udders, they are a joy to milk and I do not intend to have this joy marred by allowing mastitis to interfere again.

HIGH CELL COUNT PROBLEM HERDS - A PRACTICE APPROACH

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SUMMARY

This paper details a practice approach to mastitis control in high cell count "problem herds" within a mixed practice on the Wilts/Glos borders. Individual cow cell counts combined with selective sampling of quarter milk samples for bacteriology together with the standard five point mastitis control plan forms the basis for sorting these herds. The value of regular cell count league tables together with target levels will be discussed, whilst the roll of an alternative approach to dry cow therapy for persistently high cell count cows is highlighted. As the standards set for bulk tank supplies tighten with some companies awarding bonuses, the future of "problem herds" is debatable.

INTRODUCTION

The success of any mastitis control plan is dependent on many factors of which, as highlighted by Bob Russell's paper, the conviction and commitment of the farmer and herdsman is paramount. The results within the UK national herd for these committed individuals can be seen readily from bulk milk cell count figures (Fig. 1).

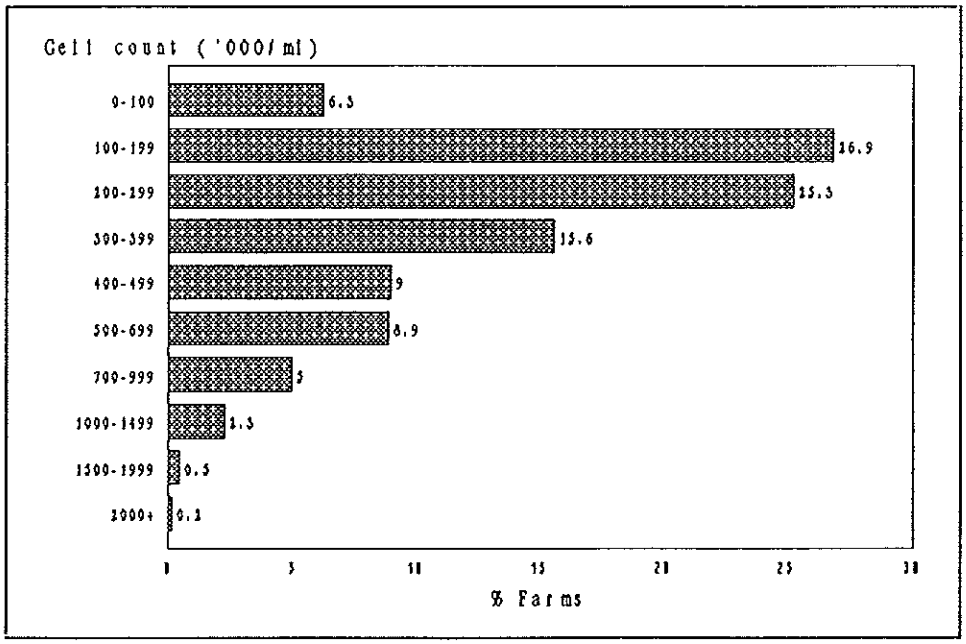

Figure 1 Proportions of UK national herds producing different bulk milk cell count levels

The EC Health and Hygiene Directive (92/46/EEC) set new parameters for raw milk which have to be met if it continues to be sold (Table 1).

Table 1. Requirements for somatic cell count

Raw Milk	Three Month Geometric Mean Somatic Cell Count (cell/ml^{-1})	
a. Liquid Market	≤ 400,000	
b. Manufacture	from 1.1.1994 ≤ 500,000	from 1.1.1998 ≤ 400,000

Standards are therefore set to become stricter but, for the moment at least, do not strictly apply to individual ex-farm supply. With the advent of the open market next month, the requirement of the individual purchaser will vary, with narrower bands in some cases, attracting bonuses and penalties. Several companies now require suppliers with somatic cell counts below 250,000 cells/ml with one company even offering a bonus at this level. One company is also setting a penalty of 0.1 ppl for milk in somatic cell band 250,000-400,000 cells/ml. So the market place is set to become more competitive and at the same time, the demand for hygienic, low cell count ex-farm suppliers will become even greater.

The high cell count herd has therefore a limited amount of time in which to try and achieve these higher targets. How long will individual bulk milk transport vehicles be allowed to call at two neighbouring farms with bulk milk cell counts below 250,000 and above 400,000 cells/ml?

PRACTICE EXPERIENCE

We have been working, like most practitioners, to improve the position of our practice herds in terms of mastitis control for many years. It is however only in the last 16 years that we have specifically used individual cow cell counts within the mastitis control strategy to:

1. produce regular twice yearly league tables of client's bulk milk counts to stimulate discussion and provide incentives for individual farms (Table 2).

2. investigate high cell counts herds using individual results to target a sample 6-8 cows for individual quarter milk samples for bacteriology.

3. monitor progress of a mastitis control programme in conjunction with accurate records of clinical cases of mastitis.

4. provide data together with clinical records in preparing a list of chronically infected cows for culling following a set treatment routine.

Historically the success of this approach together with other trials by the MMB, ADAS, NIRD etc can be seen from Figure 2, demonstrating the geometric mean somatic cell count for all practice herds in the Veterinary Cell Count Information Service (VCCIS) in 1978-1994.

Table 2. Practice league table of herd cell counts

Farm Number	Rolling Mean	Change in Mean Over 12 months	Farm Number	Rolling Mean	Change in Mean Over 12 months
1	87	-13	49	238	-31
2	93	7	50	240	46
3	101	-15	51	247	-2
4	115	1	52	249	2
5	118	3	53	253	-121
6	119	-20	54	258	66
7	123	-95	55	260	NEW
8	131	17	56	263	HERD
9	138	28	57	263	119
10	138	11	58	266	-83
11	143	-6	59	269	-17
12	145	13	60	277	48
13	148	1	61	277	-69
14	149	-3	62	280	32
15	149	8	63	293	-37
16	150	-35	64	295	64
17	152	-69	65	295	-25
18	162	21	66	300	-202
19	165	21	67	303	17
20	170	5	68	303	-50
21	171	-90	69	304	45
22	176	5	70	312	-4
23	179	-28	71	313	63
24	180	21	72	318	65
25	181	-43	73	321	3
26	182	-23	74	324	-29
27	185	-13	75	326	-130
28	189	26	76	327	-82
29	189	-83	77	329	19
30	190	30	78	330	-164
31	193	-26	79	345	-54
32	193	-45	80	364	-33
33	195	3	81	374	109
34	199	-67	82	376	-20
35	200	35	83	405	106
36	206	-12	84	423	-109
37	211	32	85	437	-3
38	211	-1	86	474	-271
39	212	-9	87	518	80
40	220	40	88	538	-103
41	224	-63	89	575	29
42	225	42	90	580	-34
43	226	-48	91	613	-99
44	227	-48	92	775	49
45	228	-5	93	778	96
46	229	-6	94	794	-445
47	229	15	95	1,627	-159
48	232	-4			

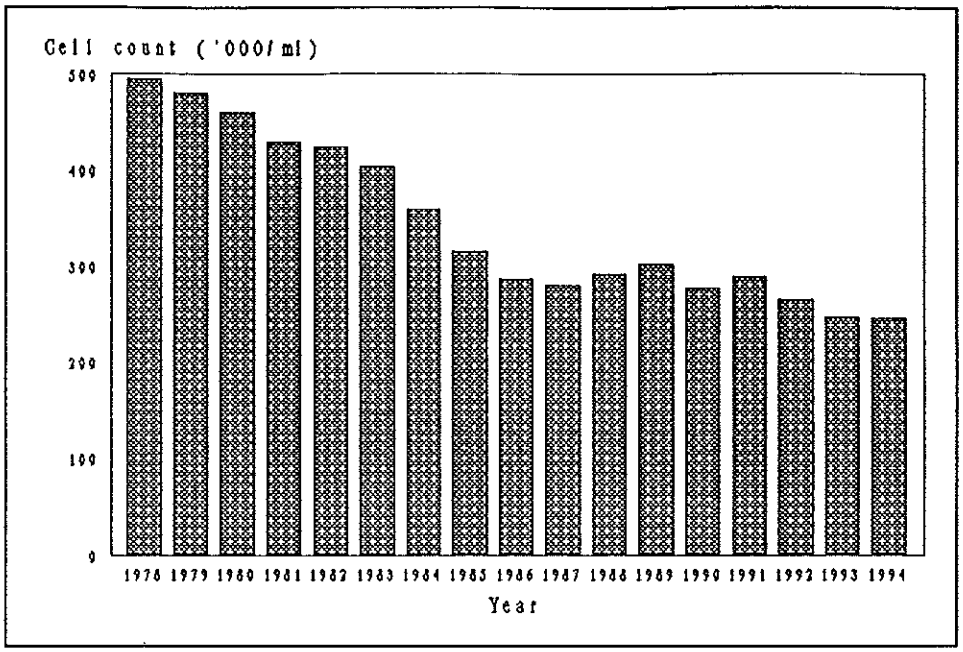

Figure 2 Geometric mean somatic cell counts for practice herds 1978-1994

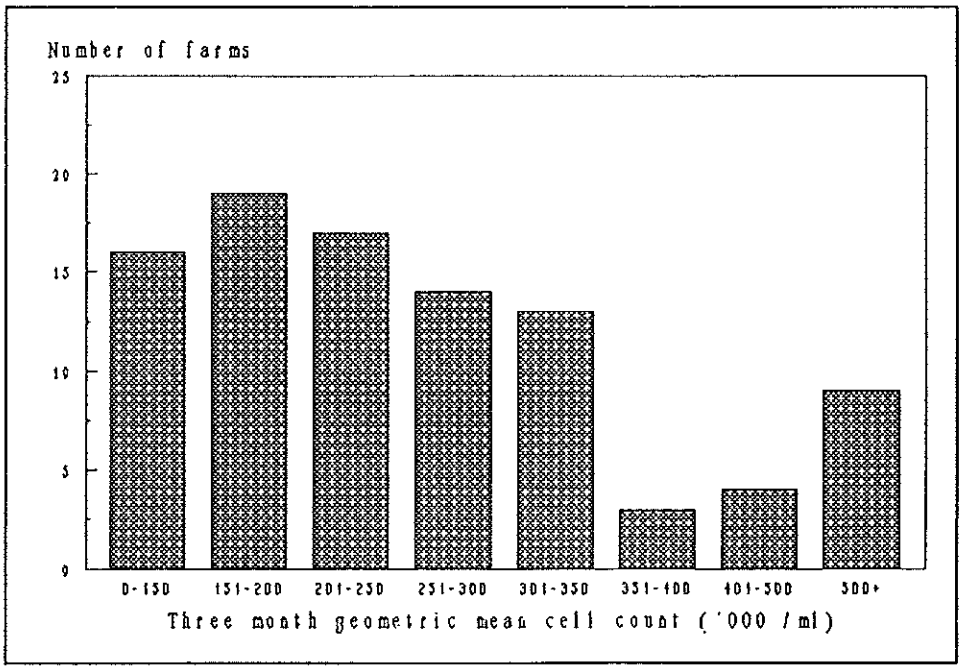

Figure 3 Distribution of somatic cell counts for practice farms

In general the trend has shown a gradual improvement over the years with the exception of the period 1988-1989 when the effects of culling due to BSE meant that a lower proportion of cows were culled for other reasons including chronic mastitis. As the increased number of inseminations to Friesian dairy bulls worked through to a greater number of heifer replacements the improvement in cell counts has continued.

Looking at the distribution of farms within a number of somatic cell counts bands (Fig. 3) the success of the majority can be seen with >50% farms below 250,000 cells/ml. However a worrying proportion, 13 farms out of a total 95, still have cell counts in excess of 400,000 cells/ml. It is these farms that require further help and advice if increasing penalties and possible loss of an outlet for raw milk are to be avoided.

Within the practice, we have set ourselves a target of getting all herds below 400,000 cells/ml and >60% of herds below 200,000 cells/ml in the next two years. Targets stimulate both client and clinician!

INVESTIGATING HIGH CELL COUNT HERDS

Prior to 1991 individual cell counts were only available on request to the MMB but were recognised within the practice as a very useful tool to start an investigation into a herd problem. All cows calved less than a month or due to dry off within a month were <u>not</u> sampled and therefore, except in a herd with a tight seasonal calving pattern, a second set of samples 4-6 months later was necessary.

Whilst these samples are being organised and processed a comprehensive mastitis questionnaire is completed and clinical mastitis and culling records examined (if applicable!). The adoption of the <u>full</u> five point mastitis control plan is essential by <u>all</u> persons involved in milking if success is to be achieved. The enthusiasm of <u>all</u> concerned should be assessed at this stage and realistic targets set.

As demonstrated in Table 3 the results obtained on 78 samples from Mr Russell's herd in May 1980, showed 41% of cows with cell counts 1,000,000 cells/ml. From any such listing identifying 5-10% of cows for routine bacteriology on individually drawn quarter milk samples presents little problem.

Table 3. Proportion of herd with different levels of somatic cell count and change over 3 years

	May 1980	March 1982	April 1983
less than 250,000 cells/ml	20.5%	56.8%	59.2%
250,000-500,000 cells/ml	23.1%	6.8%	14.5%
500,000-750,000 cells/ml	7.7%	13.5%	13.2%
750,000-1,000,000 cells/ml	7.7%	1.3%	3.9%
1-2 million cells/ml	23.1%	17.6%	3.9%
2-5 million cells/ml	10.3%	4.0%	5.3%
More than 5 million cells/ml	7.7%	0%	0%
Number of cows	78	74	76

The predominance of *Staphylococcus aureus* infection (Table 4) suggested a parlour related problem and sensitivity testing highlighted that most isolates were resistant to penicillin - the antibiotic used during lactation and drying off. The obvious change to a cloxacillin product was made and accurate clinical records kept.

Table 4. Results from bacteriology of individual quarters samples taken from high cell count cows

Cow No	Cell Count ('000/ml)	Bacteria Isolated			
		RF	RH	LF	LH
3	1,987	*S. aureus*	*S. aureus*	*S. aureus*	-
16	1,858	*S. aureus*	*S. aureus*	Blind	*S. aureus*
18	6,641	*S. aureus*	-	*S. aureus*	*S. aureus*
32	3,178	*S. aureus*	-	*S. aureus*	-
47	3,118	*S. aureus*	*S. aureus*	*S. aureus*	*S. aureus*
60	5,098	*S. aureus*	*S. aureus*	*S. aureus*	*S. aureus*
69	1,728	*S. aureus*	Blind	-	*S. aureus*

The success of treatment of staphylococcal infected quarters during lactation is at best variable, often disappointing, and is considered by many to be uneconomic. It has therefore been a policy in the practice in the last 10-12 years that such quarters and persistently high

cell count cows be dried off twice with two different antibiotic based dry cow preparations at three week intervals. The first using a cloxacillin product is followed three weeks later by a novobiocin preparation. The monitoring of clinical incidence and lately monthly cell counts can be used to assess the success of this and all other aspects of a mastitis control programme. If individual animals fail to respond to this routine, segregation during milking (milk last or flush clusters) and early culling is the only policy.

The advent of monthly cell counts, linked to NMR records, in October 1991 has allowed us to monitor the success or otherwise of double, dry cow treatment more closely. Table 5 summarises the result for 19 cows treated with double dry cow therapy in a well managed 240 cow unit in 1993. The rolling mean cell count for the unit was 260,000 cells/ml and the infected animals had average lactation cell counts $\leq$250,000/ml or $>$3 cases mastitis in their last lactation. Following the double dry cow regime and a full control programme the annual mean cell count now stands at 173,000 cells/ml. However some cows have not responded (235, 256) and/or have had $\geq$3 cases of clinical mastitis during the current lactation and have therefore been added to the list of cows due for culling.

Table 5. Individual cow cell counts on a 240 cow dairy unit

Cow No	Average Cell Count Last Lactation	Cell Count One Month After Calving	Average Cell Count This Lactation
17	282	15	30
60	658	767	419
124	407	134	497
153	473	27	284
161	755	188	577
162	371	531	262
191	1,246	362	424
218	651	306	368
233	259	235	194
235	1,318	6,054*	642
236	267	505	514
256	3,696	630	2,001
277	261	278	132
442	753	223	836
664	306	56	194
678	482	194	395
688	136	17	67
716	193	188	228
760	484	44	432

* Clinical mastitis at sampling

Prior to October 1991, the success of a given policy was based primarily on the lack of clinical mastitis in the subsequent lactation. Now however, successful control of sub-clinical mastitis monitored by a persistently low cell count is also required.

The possibility of culling chronically infected cows is influenced by many factors including the number of heifer replacements available; other health (BSE, tuberculosis etc) and fertility problems. Each farm must be advised on its own particular situation taking into account the penalties being incurred. The success of using individual cow somatic cell counts does not have to be linked to NMR or any yield recording system. An opportunity presents itself to an organisation to supply a monthly cell count monitoring service not dependent on individual yield and quality records too.

CONCLUSION

The value of individual cow somatic cell counts in monitoring a mastitis control programme has become widely accepted, but "problem herds" persist despite incentives and penalties. The future for such herds is very debatable as the demand for increasingly higher hygiene ex-farm supplies of raw milk increases. Producers may find themselves with a more demanding set of potential purchasers for the raw milk and conditions of contract that are increasingly hard to maintain. Any mastitis control programme is at best one of control not eradication because of the very nature of the disease so even well managed units must never become complacent. Individual cow somatic cell counts provide this constant reassessment.

MASTITIS AND QUALITY MILK PRODUCTION

MASTITIS AND QUALITY MILK PRODUCTION: THE PROGRESS WE HAVE MADE

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SUMMARY

Milk quality schemes have been operated by the UK Milk Marketing Boards for over 40 years. The earliest schemes concentrated on butterfat content and the keeping quality of milk. Continuous testing and associated payment schemes have brought about improvements in all measures of the quality of milk, and these improvements have been especially marked since the introduction of centralised testing. The control of mastitis, reinforced latterly by the payment schemes for hygienic quality as determined by TBC and cell count, has been a major factor in improving the quality of milk produced, although mastitis still costs the country £80 million per year.

INTRODUCTION

The quality of British milk has been measured for many years. Payment schemes have been introduced and refined with the general objective of improving the quality of milk produced.

The initiative for the improvement in milk quality has come from farmers' organisations rather than directly from the market. This system is likely to change now with the abolition of the Milk Marketing Boards.

However, substantial progress in the production of quality milk has been made by British dairy farmers over the years, and it is worth recording just what has been achieved.

In this paper I will be looking briefly at all aspects of the quality of milk although concentrating on those affected by mastitis. The schemes and statistics relate specifically to England and Wales, and similar progress has been achieved in Scotland and Northern Ireland.

SCHEMES

The first milk quality schemes were introduced by the Milk Marketing Board. Responsibility for the operation of the schemes was taken over subsequently by the Joint Milk Quality Committee of the Milk Marketing Board and the Dairy Trade Federation. The first schemes, all of which have been revised at regular intervals, were introduced more than 40 years ago, and the last just three years ago. The main schemes have been:

1952	Hygienic Quality Scheme Butterfat Testing Scheme
1956	Butterfat and Solids-not-fat Testing Scheme
1966	Antibiotic Price Deduction Scheme

1977 Cell Counting - advisory

1982 Total Bacterial Count

1983 Protein and Lactose Testing

1991 Cell Count Payment Scheme

Most of the results given in subsequent tables have been drawn from the annual reports of the Joint Milk Quality Committee. The first report was issued in 1964 so progress for up to 30 years can be quantified.

The various milk quality schemes have operated generally by providing incentives for producers of the better quality milk whilst penalising those producing milk of lower quality. Thus it is relevant to note that the number of milk producers has declined substantially throughout the period under review. In 1963-64 there were 105,576 milk producers in England and Wales. By 1992-93 almost three-quarters (73%) had left dairy farming and the total was down to 28,729. The trends were similar for Scotland and Northern Ireland with 66% and 64% reductions in milk producers respectively. How much the improvements in quality milk production have been as a result of this exodus is a moot point.

COMPOSITIONAL QUALITY

There has been a consistent increase in the butterfat content of milk (Table 1). The overall increase is approximately 0.3% with the greater part of this increase occurring in the past five years.

Table 1. Changes in compositional quality of milk

	Percentage (weighted averages)			
Year	**Butterfat**	**Solids-not-fat**	**Protein**	**Lactose**
1963-64*	3.81	8.70	-	-
1973-74	3.81	8.69	-	-
1983-84	3.90	8.75	3.23	4.62
1988-89	3.94	-	3.23	4.56
1992-93	4.10	-	3.29	4.56
1993-94	4.11	-	3.26	-

* Simple averages in 1963-64

The same cannot be said of the other major constituents. The solids-not-fat has increased slightly and, at least over the last ten years, there has been a small increase in protein. In view of the improvement in the cell count of milk (see later), it is surprising to note a fall in the percentage of lactose, albeit this was the lowest value constituent when it was paid for separately.

HYGIENIC QUALITY

The resazurin test for hygienic quality was used from 1952 until the introduction of total bacterial counts (TBCs) in 1982. Substantial progress was made during the later years in reducing the number of supplies failing this test (Table 2).

Table 2. Changes in hygienic quality of milk

Year	% Failures	TBC*
1966-67	3.8	-
1974-75	3.2	-
1981-82	0.4	-
1982-83	-	23
1987-88	-	16
1991-92	-	14
1992-93	-	13
1993-94	-	13

*National weighted average - thousands bacteria/ml

Table 3. Hygienic quality of milk: distribution of herds by TBC Band

		% supply by band*		
Year	Average TBC	A	B	C(+D)
1983-84	22	65.0	30.8	4.2
1987-88	16	75.5	22.6	1.9
1991-92	14	79.2	20.2	0.6
1992-93	13	81.1	18.3	0.6
1993-94	13	86.3	13.6	0.1

* Band A: 20,000 bacteria/ml or less
Band B: 21,000 to 100,000 bacteria/ml
Band C: Over 100,000 bacteria/ml
Band D (until 1987): Over 250,000 bacteria/ml

Understandably greater progress in reducing TBCs was made during the first year after introduction of the payment scheme. However, the average disguises and tends to minimise the progress made in improving the overall hygienic quality of milk, especially over the last few years (Table 3).

The table shows clearly that there has been continuing improvement in the proportion of herds producing band A milk, even in 1993/94 when the average TBC did not change. More impressive still is the reduction in number of herds with TBCs over 100,000 bacteria/ml. These have now declined to an average of fewer than 40 per month producing less than 0.03% of all milk. By comparison, 92.1% of all milk is now of band A quality.

MASTITIS CELL COUNTS

In EU legislation the cell count of milk is considered to be a measure of its hygienic quality.

Regular monthly bulk milk cell counts were first made available to farmers in England and Wales on a paid-for basis in 1971 as an aid to the control of mastitis. In 1977 this service was extended to all dairy farmers on a free, advisory basis. In 1990 the MMB Quality Testing Service incorporated weekly cell counts into its range of testing and, after a one year advisory period, the payment scheme was implemented in 1991.

The national average cell count has been halved over the past 23 years, although progress has tended to be spasmodic and frequently influenced by factors other than disease control (Table 4).

Table 4. Somatic cell counts of herd milk

Year	Average ('000/ml)	% Supply by Band# 1	2	3	4
1971[a]	573	-	-	-	-
1976[a]	467	-	-	-	-
1981[b]	465	39.2	41.2	13.4	6.2
1986[b]	352	62.7	27.4	6.9	3.0
1991-92[c]	289	69.9	21.2	5.8	3.0
1992-93[c]	282	71.8	19.9	5.3	2.9
1993-94[c]	268	75.2	9.2*	13.0*	2.6

\# Band limits (thousand cells/ml):
1: 0-400, 2: 401-700, 3:701-1,000, 4: 1,000 +

* Band 2: 401-500, 3: 501-1,000

[a] Survey data

[b] MMB Veterinary Services

[c] MMB Quality Testing Service

Despite the fact that reducing mastitis cell counts is a longer and more difficult process than reducing TBCs, good progress has been made in increasing the proportion of herds with cell counts of 400 thousand cells/ml or less, the present limit for band 1. These herds now produce 85.0% of all milk. The situation is similar to that with TBCs six years ago, and the more severe penalties introduced in April 1993 appear to be having an effect on all but those herds with the highest cell counts.

However, at least three dairy companies have stated that they will be seeking milk with a cell count below 250 thousand cells/ml. Only about 40% of herds currently meet this criterion so further pressure for improvement in cell count is to be expected in the future.

ANTIBIOTIC RESIDUES

Great progress has also been made in reducing antibiotic residues in milk. The need to keep residues at minimal levels is an accepted part of good practice, but the risk to human health posed by antibiotic residues in milk is frequently over-stated. Compared to the risks from the medical prescription of antibiotics it is almost infinitesimal. The sensitivity of the testing is now very high and there would appear to be little logic in increasing this further, at least insofar as the penicillins are concerned.

Progress made since the first national survey over 30 years ago is shown in Table 5.

Table 5. Antibiotic residues in milk

	% Failures by sensitivity of test (iu penicillin/ml)			
YEAR	.05	.02	.01	.006
1961*	6.1			
1966-67	1.2			
1975-76	1.1			
1976-77		1.6		
1984-85		0.37		
1986-87			0.35	
1989-90			0.24	
1991-92				0.45
1992-93				0.42
1993-94				0.36

* National survey

The instigation of national testing by the MMB in 1966 had an immediate effect in reducing antibiotic residues in milk. However, it required testing to a lower level and greater financial penalties in the period 1976-85 before significant further progress was made. After each increase in sensitivity of the test the number of failures increased by about half.

Increased frequency of testing also resulted in a higher number of test failures. Nevertheless the present test failure level is extremely low and is declining.

MASTITIS

So far in this paper I have concentrated on the results of tests carried out on milk delivered for sale. However the level of mastitis on the farm will naturally have a major effect upon the production of quality milk.

The milk from clinically affected cows is of course withheld from sale, but there is a period both before and after a clinical episode when the quality of the milk produced will be affected. There is good evidence that the incidence of clinical mastitis has declined substantially over the last 30 years, from an annual rate of about 150 cases per 100 cows to about 40 cases per 100 cows in the 1980s. Unfortunately no surveys have been carried out to monitor whether this progress has continued more recently.

Cows with subclinical mastitis also produce milk of a lower quality, both compositional and hygienic. Thirty years ago over 50% of cows in the average herd were infected with subclinical mastitis. Today it is estimated that the figure is approximately 15%, although again no recent surveys have been carried out. The evidence of the national cell count would indicate that the improvement has continued in recent years.

However, it has to be said that mastitis continues to be a major source of loss to dairy farmers. At current 1994 prices we calculate the UK loss to be at least £80 million - an average of £30 for every cow in the national herd - with substantial associated consequential losses. There is thus every incentive for tightening the control of mastitis at the national level.

CONCLUSIONS

Milk quality schemes have operated in the UK for more than 40 years.

Over that period there have been substantial and continuing improvements in all measures of milk quality:

- Butterfat and protein percentages have been increased
- Total bacteria counts have been reduced
- Cell counts have been halved
- Antibiotic test failures have been reduced hugely
- Clinical and subclinical mastitis have been reduced by two-thirds

The fact remains however that the retail sellers of milk will use quality as their watchword. Dairy farmers can be proud of the quality of the product they produce, but they can also be sure that the market will continue to demand ever higher standards.

ACKNOWLEDGEMENTS

The annual progress reports issued by the Joint Milk Quality Committee and statistics from the Milk Marketing Board Central Testing Laboratories have been invaluable sources of information and data in the preparation of this paper.

REDUCING CELL COUNTS

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SUMMARY

Milk is a food and must be produced as hygienically as possible, so that a quality product can be supplied. It is often necessary for veterinary assistance to be sought to help reduce milk cell count and total bacterial count. My approach is fairly conventional, although methods of enhancing dry period cures are being tried. It is necessary to adopt a whole system and integrated approach to producing the best quality milk now and in the future.

The current changes in milk marketing, together with tighter milk quality standards, has put pressure on the industry and particularly on those dairy farmers whose milk quality is below standard.

Veterinary surgeons are often consulted to give advice on herds with high bulk milk cell counts and high total bacterial counts.

BULK MILK CELL COUNTS

The somatic cell count is a good guide to the incidence of sub-clinical mastitis. This type of mastitis is caused by the pathogens responsible for contagious mastitis. The bacteria commonly involved are *Staphylococcus aureus*, *Streptococcus agalactiae* and *Streptococcus dysgalactiae*. The cow is the reservoir for these organisms and they are spread from cow to cow via the milking machine.

High somatic cell counts occur when these bacteria invade the alveoli of the mammary gland. The body, in defence of this invasion, produces large numbers of white blood cells (neutrophils) to combat the infection. It is these cells which make up the majority of the somatic cell count. Some cells are produced from the udder itself as they are shed from the alveolar lining as a result of the infection.

The bacteria not only produce a clinical mastitis, but also form small pockets of micro-infection which continue to stimulate white blood cell production. These pockets of infection are deep in the tissues of the udder. They can produce no visible effect in the milk and are often resistant to normal treatment.

In a normal cow the cell count usually rises at the end of lactation due to a natural increase in the shedding of cells from within the udder.

In recent years the greatest advance in the control of herd cell counts has been the introduction of national individual cow cell counts in October 1991 which meant that the chronic persistent offenders could be identified.

APPROACH TO REDUCING THE LEVEL OF BULK CELL COUNTS

1. Initially I refer to the herd mastitis records. From these it is possible to discover the incidence of mastitis in the herd, the individual cow case histories, the number of cases and the herd intramammary tube usage.

2. The bulk milk cell count should then be examined to identify the trend over the previous 9-12 months.

3. The individual cow cell counts (ICCS) over the previous 3-6 months will again indicate trends and identify persistent problem animals. The individual with an odd ICC is noted for future reference.

METHODS USED TO REDUCE AND CONTROL BULK MILK CELL COUNTS

1. Dry cow therapy (DCT)

This is essential. I recommend the use of the longer acting products where the dry period is long enough. It is important that the cows are treated in a place where strict cleanliness can be observed. The parlour is ideal. It is worth remembering that DCT will only cure 50% of staphylococcal infections and that only 25% are cured by treatment during lactation.

During the last year I have been experimenting using two tubes simultaneously at drying off. It is successful in reducing cell counts, but it is too soon to assess whether the success rate is a significant improvement on the standard treatment with one tube.

I had no knowledge of the affect on milk withdrawal times of this treatment, but there have been no problems thus far.

2. Treatment during lactation

In theory, following a bacteriological examination and organism sensitivity, it should be possible to reduce the number of sub-clinical udder infections and individual cow cell counts during lactation using the correct antibiotic. In my experience this treatment has been very unsuccessful and is not cost effective.

3. Treatment prior to drying off

The treatment of infected quarters or individuals with high cell counts immediately prior to using DCT, can be used in an attempt to reduce the cell count level. The quicker release products are used and cows are treated for 3-5 milkings. It adds considerably to the cost of drying off, but the benefits can be a cow removed from the cull list.

4. Bacteriology

The bacteriological examination of milk has a place, as an adjunct to the treatment and control of mastitis and high bulk milk cell counts, but can be overused and over emphasised.

Bacteriology of the clinical case of mastitis, prior to treatment, is useful in identifying the organisms involved and their antibiotic sensitivity. From this we can ascertain the type of mastitis involved - Contagious or Environmental and the correct intramammary treatment.

Milk samples must be taken correctly, stored correctly, at 4°C or less in the fridge, and taken to the laboratory quickly. I sometimes doubt the value of a milk sample sent through the post. There is no advantage in taking a sample from a cow under treatment, the antibiotic may well have killed the causative bacteria or at least inhibited growth.

Bacteriology on milk from the individual cow with a high cell count can be useful, but very often samples show no significant growth. In theory the bacteriological examination and antibiotic sensitivity of the milk of all cows prior to drying off would be beneficial. However in my experience it is unnecessary since the results show that the dry cow intramammaries available are so broad spectrum they cover all the organisms involved. However it is useful with individuals that have very high counts.

5. Culling

Removing chronic high cell count cows from the herd remains the most effective way of lowering the herd bulk milk cell count. The individual cow cell count indicates the percentage influence each cow has on the overall herd figure so it is possible to calculate the improvement to be made by the culling of individual cows.

After applying control measures, it can take several months for the rolling mean bulk cell count to fall to a satisfactory level. For example if the herd rolling mean cell count is 600, in order for the count to fall to below 400 in three months, there must be two successive monthly counts of less than 300.

Also high cell count cows may be culled only to be replaced by fresh calved cows that also have a high cell count. So attempts to lower a herd bulk milk cell count can be very frustrating in the beginning.

TOTAL BACTERIAL COUNT (TBC)

The TBC is a measure of the total number of bacteria in the milk. The bacteria concerned with a high TBC are usually associated with the environment and contaminate the milk. These organisms are also often associated with Environmental Mastitis. High TBC's are caused by bacterial contamination of the milk from four sources

Mastitis pathogens
Contaminated teats
Dirty milking equipment
Faulty refrigeration

MASTITIS PATHOGENS

A cow will shed many thousands of bacteria both immediately prior to, and during a clinical phase of mastitis. Immediately prior to a clinical case there are no obvious signs in the milk - it appears normal. However, if a sample is taken for a TBC at this time then there will be a very high value. It is therefore very important to detect mastitis early.

CONTAMINATED TEATS

A cows teats become contaminated from the environment in which she lives, particularly from cubicle houses and straw yards. Teat chaps and sores also harbour bacteria which can contaminate milk. Poor teat preparation for milking will influence the TBC. It is essential that teats should be dry prior to milking. Wet udders will have a watery solution of bacteria dripping from the end of the teats. Usually a dry wipe with a paper towel is all that is necessary.

DIRTY MILKING EQUIPMENT

Inadequate cleaning of the milking machine will lead to a build up of bacterial contamination. Many machines have blind ends and old worn rubber piping which is difficult to clean. The tank itself needs regular and thorough cleaning.

REFRIGERATION

Milk should be cooled to 4°C, warm milk is an ideal medium for bacterial growth. The advent of two day milk collection will probably help cool milk faster and lower TBC.

An examination of a bulk milk sample is useful in identifying the cause of the high TBC. Simple tests can be carried out in most laboratories and from these tests the source of the contamination can be identified.

CONCLUDING REMARK

We must all remember that milk is a food and it is vitally important that it is produced as hygienically as possible and that a quality product is supplied to the consumer.

HOW AM I PLANNING FOR THE FUTURE?

IAN S WATSON, Newlands Farm, Carleton, Cumbria

SUMMARY

Although extremely good quality milk can be produced by rigorous attention to details and application of well rehearsed mastitis control this may not be enough on a changing playing field to ensure maximum milk value in future. The UK farmers must provide what the market wants and what legislation demands. It is considered that application of the legislation and the means of deciding on cell count standards underestimate the performance of the UK. The levels of cell count and their significance to human health and product quality are also challenged.

In this description of how I am planning for the future, with particular reference to somatic cells, it will become clear to you that my planning is quite simply a continuation of my present management policy. A policy that has served me well in the past. I will however touch on some political points and comment on the situation in other parts of the world as well.

EU DIRECTIVE ON MILK HYGIENE

We must accept at the outset that it is in our interests as milk producers to produce top quality milk. I also believe that we need to provide what the market requires. Statistics show that we have achieved considerable progress over the past three years in improving somatic cell count of milk but sadly a number of producers still let the side down. I hesitate to say it but these 'culprits' seem to be the smaller volume producer. Some 78.6% of suppliers produce 88.3% of Band 1 milk volume and conversely the 2.6% of suppliers producing Band 4 milk account for only 0.7% of the volume. This indicates that, on average, larger volume suppliers produce better cell counts and vice versa. However, the market and the legislators must not put unreasonable restrictions on us as farmers and one could argue that the Health and Hygiene Directive introduced from 1 January this year does exactly that in two ways.

First, the standards are set at farm level and not at processing establishments. Whilst, in fairness to MAFF they remain supportive of our decision that the standards ought to be set at processing establishment, it is obvious that the government are coming under increasing pressure from other parts of the Union to adopt a standard at farm level and not at the processing establishment. I hope that they continue to support us and hopefully will try to persuade, within the Commission, other European countries that there is a need to adopt more sensible cell count standards. This issue is a long way from being resolved and it is unlikely that we will see legislation much before the end of the year.

Secondly, the EU Milk Hygiene Directive sets standards of 400,000 cells/ml at farm level above which milk must be taken out of the liquid market and 500,000 cells/ml above which milk is unmarketable. Based on the lower cell count figure 10.7% of milk in Europe would, on average, have to be moved out of the liquid market (Table 1). This would be an

extremely costly exercise. The milk diverted would find its way into milk products such as cheese, where there would be greatest negative impact, if somatic cell counts have any impact at all on product quality at a level of 400,000 cells/ml.

Table 1. Amount of milk supply failing to meet EU Milk Hygiene Directive 92/42 on cell count

Country	% supply 4-500,000 cells ml^{-1}	% supply >500,000 cells ml^{-1}
Belgium	14	16
Germany	6.9	9.4
Denmark	12	7.0
Great Britain	11.2	21.5
The Netherlands	9.2	11.0
EU average	10.7	13.0

(IDF Mastitis Newsletter No. 18)

Furthermore, a further 13% of milk on average would be deemed to be unmarketable (Table 1). There is no justification in human health or product quality terms for discarding milk with a somatic cell count over 500,000 cells/ml yet the proposed standard would require 13% of milk to be wasted.

INTERNATIONAL TRADE

The GATT treaty signed earlier this year will give increased access for dairy products into the European Union and the United Kingdom in particular. Some of these products will no doubt have come from outside of the Union where the specification on cell count may not be as rigid as our own. Can these products be tested and will the World Health Organisation who will monitor this in the future be able to assure British dairy farmers that we are competing on a level playing field and the rules are the same for all ? If so, then I and other dairy farmers will accept the continuing challenge to be better than anyone else.

LIKELY NEEDS

There are at present many thousands of UK dairy farmers who are achieving the desired target of milk below 250,000 somatic cells/ml but there may be reluctance and there may be difficulty for some of those who are running at a figure higher than that to reduce to the target figure. No doubt the question they are going to ask is what they are going to be paid to meet that extra requirement. My concern is that it will not be worth anything and that the buyers will simply pay less for milk that fails to achieve that standard. I can still remember the supermarket meat buyer who, talking to an audience of farmers in Cumbria a year or so ago, said that he wanted lambs all of the same weight, all of the same shape and all with the same killing out percentage. When asked how much more he would pay for those lambs his reply was that there would be no more and that he would pay less for those which did not meet the specification. I am concerned that is exactly what will happen in milk in the future.

There is no doubt that financial losses are incurred by keeping cows that have continually high somatic cell counts. When a financial loss actually begins to happen, I am not too sure. Some would argue, indeed the International Dairy Federation do, that those losses begin to occur above this 250,000 somatic cells/ml and therefore it goes without saying that it is necessary for dairy farmers to reduce the level of intramammary infection in the dairy herd.

AT HOME

In my own circumstances at home, where we have a dairy herd of 140 cows, we have achieved our results quite simply by good management and I might add or boast, without difficulty so it must be achievable by many more. In October 1991, when testing started, we had a somatic cell count of 142. It is now, some three years later, down to 96. Our management practices are, I believe, quite simple. We have, for as long as I can remember, used dry cow therapy on all cows and we do not tolerate keeping a cow that has had mastitis that required treatment more than three times in any one quarter. In winter, our cows lie on mats in cubicles, we bed down with sawdust three times a week, and we have automatic scrapers running on the hour. We dry wipe in the winter and only touch those that may be dirty in the summer. We teat dip with an iodine based solution all year round and I insist on dipping rather than spraying for two reasons. First, I am convinced that you get a better coverage of the teat and secondly, as the teat cup touches the udder you can get a feel of whether that cow was milked out properly, or more importantly, if she has a hard mastitic quarter.

We all continue to strive for better genetics and this means that our herd is young with an average lactation of only four. This helps to remove the problem from old cows and therefore the risk of mastitis. We have also since National Milk Records introduced individual cell counting, been using that service and indeed it has identified the problem cows. It does not need many of these cows to give a high herd average and their removal can have a dramatic effect on the total.

Indeed one should take note of the percentage contribution to the herd of any one individual cow which highlighted in the Cell Count Report. In June this year I had two cows contributing 25% and 18% to my herd. One of these cows I was aware of; she was old but gives a lot of milk so I tolerate her; the other result came out of the blue with no clinical signs of mastitis but it does beg the next question of how to use those results.

Having got the results of the individual samples back from NMR we must consider taking the testing a stage further and certainly those with problem herds I believe have no choice. The next step would be to try and identify the infected quarters on those cows with high counts to establish the organism and hopefully the sensitivity that in turn will allow the right choice of antibiotic treatment to be found but before we automatically treat these cows in the cool light of day one has to decide what to do. Treat the infected quarter with the appropriate antibiotic? Dry off the cow early with a dry cow treatment appropriate to the cause of infection? Dry off only that quarter and continue to milk the other three or continue to wait until the normal drying off time and treat it then or throw the cow out of the parlour and suckle it with calves or is the more drastic approach to kill the cow or sack the cowman?

The overall aim is two fold. First to lower the herd cell count but also and most importantly to reduce the risk to clean cows in the herd, particularly the younger ones. There is no doubt that healthy cows have a lower cell count but are more prone to new infection and therefore it does mean that we have to monitor continually what we are doing. I believe that a herd with a bulk tank cell count of 100,000 cells/ml will have one cow in ten with an infection. A bulk tank sample of 250,000 cells/ml would suggest that one in six cows have an infection. It goes without saying therefore that a count of 400,000 cells/ml must indicate poor milk quality and a large number of cows infected. We must also look to the geneticists in the world when considering bull evaluation in the future to try and help us. Are high yields and mastitis rates linked together and is this a trait that is passed on by the bull because although we may be using bulls at the moment for high yields we must also be concerned as to whether or not their daughters have high cell counts ? I think it is essential that we start to gather up this data as rapidly as possible.

COMPARISONS

I think if you consider the position of the United Kingdom milk producers in comparison with the rest of the world with respect to somatic cell counts we can, I believe, be quite proud of our achievements. Data in the April 1993 IDF Mastitis Newsletter show that, of the 23 countries that responded to a questionnaire at that time, the UK was the only country testing milk for somatic cell count on a weekly basis. Many other countries only sample once a month and our cell count on average is as good as and better than most of the countries that responded.

Looking closer to home and to the situation in Europe, not all of the European countries test more often than once a month. We have larger farms and a higher herd size on average and again our cell counts are as good as the other countries in the European Union.

CONCLUSIONS

I do not think that the UK dairy farmer should be concerned about the request that is being made by dairy companies at this moment in time. As I said earlier we have to supply what the market requires and to make sure that we compete on that level playing field. We have as farmers available to us the services that will help us with the management of our herds and so allow us to meet those targets. I remember that when the Milk Marketing Board introduced its total bacterial count scheme many years ago there was a hue and cry among farmers as to whether the targets were achievable. Again the majority have been successful and we only have that small minority that let the side down. We will face up to the challenge as producers; we will provide what the market requires, all I ask is that we are properly rewarded for the efforts that we will put in.

A VIEW OF MARKET DEMANDS ON DAIRY FARMERS

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SUMMARY

The freeing of the milk market in the UK will result in significant changes for all sectors of the dairy industry. Milk producers and milk buyers will for the first time be able to choose their markets and supply sources. In parallel will be the continuing pressure from retailers and consumers for high quality, good value product. That will require high standards of hygienic production, including mastitis control and cell count management, in order that milk buyers receive even better quality raw milk supplies than at present. Economic pressures due to GATT and internal milk market changes will result in downward pressure on milk price in the short to medium term. Political, market and structural changes are reviewed in relation to the requirement of the market place for high quality milk and the implications for mastitis control programmes are considered.

INTRODUCTION

Mastitis continues to be a major disease of dairy cows but it does so amidst considerable changes taking place within the dairy industry and without doubt, much turmoil. The freeing of the milk market will be the biggest change to the UK dairy industry for 60 years. At the same time farmers must face up to the changes taking place in the CAP regime following the settlement of GATT. All these changes will have direct or indirect effects on mastitis in dairy cows.

The last eighteen months have witnessed a whole range of farmer reactions to the forthcoming changes. Reactions from apathy, "nothing will really change" to almost vociferous positioning for or against the establishment of a large co-operative, Milk Marque. It has been, and will remain for some time, a difficult period for dairy farmers in deciding where to sell their milk.

Sixty years of a monopoly buyer, a 'mother' body which did much more for dairy farmers than just buy their milk, created much loyalty. Up until ten years ago, there was no constraint on production and marketing was a concept about which most had no need for concern. The UK MMBs (there are five) managed quota in the last decade providing valuable information on national output against quota; provided technical support, including mastitis control; carried out R and D; sorted out disputes and so on and so on and, most importantly, negotiated the best selling price for milk with the many milk buyers. Understandably, many farmers have been reluctant to see all that come to an end.

On the other hand, there are others who have argued that a monopoly buyer, inevitably a large organisation, cannot achieve the best milk price for its farmers. Although the CAP has as its stated objective 'the management of the market for dairy products within the Community such that on average dairy producers may obtain the target price for their milk',

not all countries, including the UK, have achieved that goal. Detailed comparisons of milk price between member states are difficult because of differences in the structure of dairy industries in each country. Historically, the UK has been in the bottom half of the EC league for producer prices (1).

Table 1. Milk prices in the EC, 1992

Country	% Target Price	Producer Prices ECU/100 kg
Denmark	105.8	27.12
Holland	101.4	26.03
Germany	100.2	25.71
UK	93.1	23.72
France	93.0	23.65
Belgium	91.4	22.76
Ireland	91.3	22.54

Source: EC Dairy Facts & Figures 1993 MMB

As a result there has been an increasing measure of frustration felt by a growing part of the dairy farming industry that they could do better in a less restrictive framework.

Milk buyers see life in the dairy industry from quite a different perspective, although they too (there have been about three hundred first hand milk buyers) have operated within a rigid framework. The Joint Committee arrangements are well known, as is the pricing of raw milk according to end-use. Whilst some buyers have been relatively comfortable with the arrangements, others have argued against the restrictiveness and economists have pointed out that the arrangements hindered new product development.

For a variety of reasons, only a few of which have been referred to previously, the UK dairy industry is about to be set free. Dairy farmers will be free to sell their milk to whomever they choose, milk buyers will be free to buy their supplies from a variety of sources.

The buyers not only need to secure their requirements in terms of volume and seasonality, they also require milk of good quality in order to satisfy the needs of their market place. Buyers must also consider in detail the changes taking place in the milk market here and elsewhere on the basis that the milk market is global, that production and consumption patterns are international, and that world trade is being liberalised, all of which will influence their milk buying decisions.

THE WORLD MARKET

The steady rise in world milk production came to a halt in 1991 when for the first time in a decade it fell by 2%. The decline has since continued at a slightly reduced rate. All will be aware that the most significant decline occurred in Eastern Europe largely as a result of the removal of price support and subsidies coupled with increasing feed costs. At the same time a 2% decline occurred in the EU primarily as a result of quota cuts and reduced

production in the European Free Trade Association (EFTA) countries (2). But in other countries milk production continued to rise, with the US, New Zealand and Australia all continuing to increase their output.

World trade in dairy products is relatively small compared to world milk production. The market therefore is very sensitive to changes in dairy policy which affects milk production, particularly in those countries which dominate the export market.

New Zealand, Australia and the EU dominate the world market being responsible for almost 80% of the products traded. The EU is responsible for about one half of the entire world trade and so its farmers have been very dependent on the associated subsidies.

Interestingly, consumption patterns are very similar in all the thirty-odd developed dairying nations. Butterfat consumption is declining everywhere whilst the market for yogurt and fresh chilled dairy products grows strongly.

POLITICAL CHANGES

The GATT agreement gives a binding arrangement to an international framework for future trading relationships. It will, in time, reduce the distorting effects of support for agricultural production on internal commodity markets. The world dairy industry will be considerably affected but not all countries will be affected equally due to many different dairy policies and export arrangements (3). As the agreement focuses on the regulation of subsidised exports, certain areas of the world - USA, EFTA, Canada and the EU - will be most affected. Countries such as Australia and New Zealand will have no restrictions and indeed positive incentives to expand.

The changes will be implemented over a six year period beginning in 1995.

The effect will be significant on the EU dairy industry where, in addition to having to accept increased imports (under GATT) there is also at present an internal milk production surplus of about 18%. The likely short term result will be increased internal competition for product markets and to compensate we can expect to see intervention price cuts and/or quota reductions of four to five percent by the end of the decade. Although it can be argued that the UK will be less affected as it has an undersupplied internal market, it would be negligent to ignore these developments. (The 18% surplus referred to earlier is more than the entire UK annual output).

These political changes alone are likely to put downward pressure on milk price.

STRUCTURAL CHANGES IN ENGLAND AND WALES

During the last sixty years the industry has changed considerably. Dairy farmer numbers have reduced steadily, cow numbers have been falling while milk yields have been rising. The effect of the milk marketing changes set against a background of improving technology and genetics, including mastitis control, will continue if not increase the speed of structural change.

Table 2. Structural Changes to Dairy Production in England and Wales

	1983	1992	2000	Forecast % change (1992-2000)
No. of producers	39703	29439	23000	-22
Average herd size	67	71	74	+4.2
Average yield (litres/cow)	5085	5220	6000	+15
Milk output per producer	340695	370620	444000	+19.8

Source: ADAS

The milk buying part of the industry also has seen change and expects to witness more. In recent years mergers and acquisitions have resulted in around a dozen companies handling about three quarters of the UK milk supply. Yet there are somewhere between 300 and 400 'primary' milk processors in the UK most of which are small liquid milk dairies. It is also noteworthy that of the major players seven are controlled by non-UK interests (4).

The reduction in milk production (almost 20% since the introduction of quotas) has left parts of the processing industry with under-utilised equipment damaging the efficiency of the business. One effect of the free UK milk market will be increased competition for milk supplies. Less efficient processors and perhaps smaller businesses may find life hard in the free market. Further and greater rationalisation of the processing industry can be expected in the years ahead. There will be, as with dairy farmers, winners and losers.

BUYERS PERSPECTIVES

Milk buyers clearly have to secure milk supplies over the longer term and in doing so will be influenced by these international and national policies and trends. Dairy farmers too must increasingly be aware of the market forces of supply and demand.

Buyers are well aware that farmers when considering their marketing options have more than one route on offer (5). They can become a:-

- co-operator by joining a (voluntary) co-op such as Milk Marque
- contractor by supplying directly to a dairy processing company under contract
- processor by adding value to milk on farm through some means of processing and packaging

- networker by pooling resources with other farms either to add value on farm collectively or jointly to market their wholesale milk to others for processing

An alternative buyer must therefore be seen as an attractive option and not as someone looking to exploit the free market in the short term.

Analysis of dairy market changes in recent years shows substantial change which will shape the market in the years ahead. Quality features highly.

The liquid market has been in gentle decline for some years but has stabilised recently. Consumer trends show a marked requirement for low fat milk and products. Just over half of the milk drunk is now of low fat (6). In parts of Scandinavia and the USA consumption of low fat milks has risen to 70% of total milk drunk. I predict that the UK will follow that trend.

There has also been a significant change in source of purchase. Of all milk consumed in 1992 only 57% was through doorstep delivery compared to 78% in 1986. The decline continues at 2-3% per year which may well result in retailers putting increased pressure on processors to reduce price which will impact on producer milk prices. Whilst butter consumption falls and cheese shows a slight increase the real growth market is in short shelf life products such as yogurt and soft cheese.

Key to many of these developing market areas is continued improvement in milk quality of the raw material, that is total bacterial and somatic cell counts.

Production of natural reduced-fat milk and increased protein is attracting considerable R and D interest. In the longer term genetic changes to the dairy cow may result in milk constituent alteration. If that proves possible it will compete for priority in breeders planning programmes with other selection criteria such as udder shape and resistance to mastitis. In the short term milk manipulation will be possible by feeding and management strategies which are already being demonstrated at ADAS Bridgets Research Centre. These too may have a bearing on mastitis control programmes.

Milk buyers also have market needs to be met. Consumers expectation are high and increasing. They demand improved quality and value, freedom of choice. They are well educated on diet and health needs and respond quickly to health scares. In reality the supermarkets (the UK has the most powerful in Europe) and high street retailers lead the shape of the market and have been demanding product of higher quality. It is often argued by dairy companies that the retailers quality standards are unreasonably and insupportably high. Now the supermarkets are increasing their interest in not only the quality of the raw product but also the conditions under which it is produced. They have already adopted similar approaches with other food products.

LEGISLATION AND MILK QUALITY

Prior to January 1994, which saw the introduction of a new Milk Directive, the UK, alongside Denmark, could rightly boast that it had the best quality milk in Europe. The two countries were the only ones able to demonstrate that ex-farm supplies had an average TBC of less than 100,000 bacteria/ml. That achievement effectively kept liquid milk out and provided a measure of protection for milk price. Undoubtedly the UK's position had been aided by high hygiene standards on farms which had in part developed due to improved mastitis control methods.

The new Directive, to be put in place by domestic legislation later this year, will demand higher standards and will affect all segments of the producing and processing industries.

Council Directive 92/46/EEC will apply to milk produced from cows, sheep, goats and buffaloes and will cover milk used for all purposes. There will however be different standards initially for milk used for drinking and milk used for manufacture.

Basically raw milk must have a TBC of less than 100,000 bacteria/ml and a cell count of under 400,000 cells/ml if milk is to be used as drinking milks. Milk not reaching that standard will have to be diverted to the manufacturing market. In the UK, unlike most other EU countries, milk collected from the farm is not necessarily directed to either liquid or manufacture outlets. To do so will be expensive and yet it is estimated that a significant proportion of supplies will not reach the liquid milk standard and some with cell counts of over 500,000 cells/ml, will even be unmarketable.

I cannot speak for either Milk Marque or dairy companies but I can only assume that no one will want to engage in establishing a costly, nationwide, second milk collection system. Milk buyers are therefore likely to 'encourage' cell counts lower than required by the law and indeed, TBCs substantially lower than those required by the Directive. That is already evident in some of the milk buying contracts issued by dairy companies. In any event, by January 1998, all raw cows milk regardless of its end use will have to have a TBC of less than 100,000 bacteria/ml and a cell count of less than 400,000 cells/ml.

Pressure to reduce antibiotic levels will continue but milk processors will also have to ensure that their products are free from certain pathogens. For example heat treated drinking milk shall not contain specified numbers of 'pathogenic micro-organisms' and coliforms. Milk-based products shall not contain 'pathogenic micro-organisms and toxins from pathogenic micro-organisms in such quantity as to affect the health of the ultimate consumer'. *Listeria sp., Salmonella sp., Staphylococcus aureus* and *Escherichia coli* are named amongst pathogens to be tested for. Whilst effective pasteurisation should minimise such problems these are clear messages that retailers and buyers will expect that milk should be 'free' from mastitis organisms.

In general terms milk buyers, and I include Milk Marque in this terminology, recognise the considerable improvements dairy farmers have made in improving the hygienic quality of raw cows milk. The pressure for continued improvement will come from retailers. In the next few years milk will be expected to have TBCs of less than 20,000 bacteria/ml and cell counts

of under 250,000 cells/ml. There will be a danger of some retailers setting even lower thresholds to gain a marketing edge but thresholds which will be difficult to support and prove scientifically. Nevertheless, on the basis that 'the customer is always right', dairy farmers may find themselves having to further tighten their clean milk and mastitis control programmes.

CONCLUSION

The reason for my describing in this paper world political and market trends in addition to discussing the domestic changes to milk marketing, was to draw attention to the likelihood that milk price will come under pressure in two to three years time. Dairy farming profitability is currently high - even dairy farmers are prepared to admit to that - but the effects of GATT and the CAP alone will increase price pressure. The changes in milk marketing arrangements will mean a short-term milk price boost for some, giving way to downward pressure in the long term as prices are driven by the lowest value manufacturing price.

Improved feeding strategies and in particular the availability of better genetics will see a steeper rise in individual cow yields to accompany the forecast of fewer milk producers.

Price pressure and higher yields will have a direct bearing on investment in, and the practices of, mastitis control. Milk buyers are most certainly aware of the challenges facing dairy farmers. They, however, have to meet the needs of their customers and in doing so will require a raw material of ever improving hygienic quality.

In the brave, new, free, milk market the dairy industry, producers, buyers and processors, should more than ever before, see themselves as one industry. Of course, there will be competition between producers and buyers, between buyers and buyers, and increasingly in the future, between producer and producer. The big competition will come from overseas. It must, therefore, be in the best interests of all to produce a high quality, high value product, which will allow the UK industry to be competitive and in doing so, provide the best possible returns from the market place for all including the dairy farmer.

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ABSTRACTS OF POSTERS

EARLY DETECTION OF MASTITIS - SOME POSSIBILITIES AND PROBLEMS

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Mastitis of the bovine mammary gland caused by bacterial infection may be identified, prior to overt signs, by any of a number of events early in the pathogenesis of the infection. These include actions of the infectious agent, determination of its growth or measurement of bacterial metabolites; immediate local reaction of tissue to the bacteria or its virulence factors including changes in milk Na^+ or Cl^- concentration, and electrical conductivity; or by local reactions of sentinel T cells including production of NAGase or cytokines.

These indicators can be precise and identify early stages in developing clinical mastitis of sub-clinical infections. However, the changes in any individual parameter vary according to the species, and often strain, of pathogen; with pathogenesis; and with individual animals, either related to experience or innate immunological competence.

Accuracy in the diagnosis of infection requires that the specificity and sensitivity of the different changes are understood. This is being undertaken for changes in milk electrical conductivity, as sensors are available for this parameter only, relative to less immediately determinable changes, bacterial number, cell count and other more recognisable changes, including animal or milk temperature, clinical score, milk yield, let down efficiency and animal activity. The information is being assessed to determine when prior to clinical signs and with what confidence incipient clinical mastitis can be diagnosed, and if this varies between the cause of mastitis.

Preliminary results suggest that it is possible to identify experimental infection by *Streptococcus uberis* up to 36 hours prior to clinical signs, but that this varies greatly between animals. On the contrary changes in milk electrical conductivity do not appear to accompany chronic sub-clinical mastitis caused by *S. uberis*. Established sub-clinical infection by *Staphylococcus aureus* is readily identified but can be confused by changes in adjacent quarters.

When accurate diagnosis prior to clinical disease is possible it will be followed by investigation of the efficacy of different and differently timed therapeutic strategies including different uses of intramammary antibiotics, withholding therapy and early application of management approaches such as frequent milking and milk stripping with and without use of intravenous oxytocin.

EFFECT OF MILKING SYSTEMS ON TEAT DUCT DEFENCES

S JANE LACY-HULBERT and J ERIC HILLERTON, Institute for Animal Health, Compton, Newbury, Berkshire RG16 0NN

The rate of intramammary infection caused by *Streptococcus agalactiae* or *Staphylococcus aureus* is greater, following experimental exposure of the teats by dipping with or inoculation bacteria, in quarters with higher milk flow rate. The rate of infection is also increased by impaired pulsation.

An infection model, based on experimental exposure to *S. agalactiae*, shows that quarter flow rate was of primary importance and the inverse of teat canal length less so when the rate of infection is considered. The effect of flow rate and teat canal length were abolished when keratin was reamed from the duct 8 hours prior to bacterial inoculation.

Milking without pulsation resulted in significant accumulation of reamable keratin in the teat duct and an increased rate of infection. It appears likely that the rate of removal of 'loose' keratin is less from reamed teat ducts and teats milked in the absence of pulsation. *S. agalactiae* can colonise these ducts more readily and a higher rate of intramammary infection occurs as bacteria colonising the keratin are not removed from the teat duct naturally by pulsing milk flow.

MASTERMIND III

A H ANDREWS and J FISHWICK, Royal Veterinary College

The third competition in this successful series, which tests anonymously your knowledge on mastitis and milk quality. All delegates are encouraged to enter the competition and prizes will be awarded for the most correct entries, with the winners identified by their marked entry forms.

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The Contracts Group at the Institute for Animal Health, Compton has an international reputation for commercial research. Clients extend from UK animal health companies to multi-national pharmaceutical organisations.

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For more information about the IAH Contracts Group contact:

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HYPERKERATOSIS OF THE TEAT ORIFICE

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The frond-like eversion from the teat orifice (hyperkeratosis) can cause concern amongst veterinary surgeons, advisors and observant herdsmen. However there are wide differences of opinion on what the score (level) should be in a herd. It is only when there is a serious deterioration from the herd normal level that there is major cause for concern.

The degree of hyperkeratosis is influenced by many factors, most of which are inter-related:-

Age and stage of lactation,
Overmilking,
Genetic influence,
Milk flow rate and yield
Faulty milking machine
Environment

A scoring system 0 to 5 has been developed to allow comparison within and between herds. In examining the teats in a herd, it is important to see most if not all the cows in milk, as a few may give a very false impression.

Shown on the display are examples of the different scoring conditions.

Table 1: Assessment of several herds at different times of the year all on various conventional milking systems. All carrying out post-milking teat disinfection either by dipping or spraying. Annual milking machines teats were carried out.

Farms	Total No. Teats	% Teat Orifice Scores*					
		0	1	2	3	4	5
A	456	50.7	22.8	23.7	1.8	0.9	0.2
B	628	50.6	18.8	28.5	2.1	0	0
C	532	**89.1**	6.8	4.1	0	0	0
D	288	55.6	12.5	27.8	3.8	0.3	0
E	588	79.4	10.7	9.4	0.5	0	0
F	628	41.9	**23.6**	29.3	3.8	**1.3**	0.2
G	226	59.3	23.4	17.3	0	0	0
H	244	7.0	2.5	**64.3**	**9.8**	0	0

* 0 - normal, 1 - slight fronds, 2 - fronds visible seen lateral view (moderate), 3 - moderate to severe 4 - severe, 5 - very severe

Highlighted in bold are the highest percentage for any score in the herds listed.

E C HYGIENE DIRECTIVE - SOMATIC CELL COUNTS

J R BAINES, ADAS National Milking Technology Specialist

EC Hygiene Directive 92/46, due to come into force in January 1994, is likely to be implemented in the UK sometime during 1994. The legislation requires that milk for liquid consumption should have a cell count below 400,000 cells/ml. Milk for manufacture must have a cell count below 500,000 cells/ml. From January 1998, all milk will be required to have a cell count below 400,000 cells/ml.

During 1993/94, more than 15% of supplies, accounting for 8% of milk volume, had cell counts in excess of 500,000 cells/ml. This milk will be regarded as unmarketable under the terms of the Hygiene Directive.

A further 9.2% of supplies, accounting for 7% of volume, had cell counts between 400,000 and 500,000 cells/ml. This milk will be regarded as only suitable for manufacturing. It is questionable whether manufacturers will want this milk. After 1998, this milk also will be regarded as unmarketable.

As an incentive for producers to improve cell counts, the MMB has penalised producers of high cell count milk. The contracts for milk purchase from future direct buyers will include similar or more stringent penalties. In future, producers of milk with cell counts above 400,000 cells/ml are likely to go out of business.

Research and practical experience show that there is no reason why every herd should not produce milk with cell counts below 400,000 cells/ml. Rigid adherence to the **FIVE POINT PLAN** keeps mastitis and cell counts under control.

The **FIVE POINT PLAN** is summarised as follows:

- Hygienic Teat Management - clean and dry housing management combined with adequate teat preparation and post milking teat disinfection.
- Identification, Treatment and Recording of all Clinical Mastitis Cases.
- Dry Cow Therapy administered routinely to all cows in the herd.
- Culling of any Cow with a History of Mastitis.
- Using a Milking Machine capable of meeting modern hygienic milking requirements, which is Maintained in Good Working Order.

Farms which are failing to meet the cell count standards in EC Hygiene Directive 92/46 are invariably failing to implement these measures completely and thoroughly. ADAS consultancy will help to ensure that any producer will meet these standards.

STREPTOCOCCUS UBERIS RESISTS PHAGOCYTOSIS AND KILLING BY BOVINE NEUTROPHILS

T R FIELD and J A LEIGH, Institute for Animal Health, Compton Laboratory, Newbury, Berkshire RG16 0NN

Streptococcus uberis is a common cause of bovine mastitis and is responsible for 30-40% of all clinical cases worldwide. Infective strains of this bacterium are capable of resisting the bactericidal action of bovine neutrophils within the mammary gland. This property can now be reproduced by culture of the bacteria in the laboratory and has been shown to correlate with the production of a capsular layer on the bacterium. The capsular layer, which is composed largely of hyaluronic acid, does not deter antibody from binding to the bacterium and so does not prevent opsonisation, a process essential for efficient uptake and killing of bacteria by phagocytic cells. However, the capsule is capable of inhibiting the phagocytic action of bovine neutrophils directly. The component within the capsule responsible for this effect has not yet been identified.

INDUCTION OF IMMUNE RESPONSES IN THE BOVINE MAMMARY GLAND

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Normal milk from dairy cows contains approximately 2 x 10^5 cells/ml usually cells of the monocyte/macrophage series are the major type in mammary secretions for most of the lactational cycle. Mammary macrophages play a phagocytic role in the mammary gland, express Major Histocompatibility Complex (MHC) class II antigens and may be involved in local presentation of antigen to T lymphocytes.

Differences have been found in the activation of primed and unprimed T lymphocytes, especially in the requirements for antigen presentation. The proliferative response of unprimed bovine T lymphocytes to soluble protein and bacterial antigens *in vitro* was measured and the accessory cell function of peripheral blood mononuclear cells and milk mononuclear cells compared.

Differences in the kinetics of the proliferative response were seen in the presence of peripheral blood mononuclear (PBM) antigen presenting cells (APC) or milk APC. The proliferative response in the presence of PBM APC and soluble protein or *Streptococcus uberis* antigen was maximal by day six of culture, whereas the response in the presence of milk APC and the same antigens, reached a peak at day eight of culture.

The addition of PBM APC to T lymphocytes resulted in statistically significant proliferation for soluble protein or *S. uberis* antigen. Similarly, significant proliferation was observed in the presence of milk APC and the same antigens. However, a higher concentration of milk cells, relative to PBM, was required to provide sufficient APC to initiate T cell proliferation.

The bovine mammary gland is considered to be immunologically compromised compared to the rest of the body. The later peak proliferative T cell response in the presence of milk APC compared to PBM APC may be a true reflection of delayed ability of milk cells to present antigen. It is possible that milk APC may phagocytose and process the antigen for display on the cell surface at a slower rate than PBM. The presence of fat and casein in mammary macrophages may play a role in impairing the speed of the macrophage processing activity.

In conclusion, we have developed a reliable technique for measuring primary, antigen specific responses of unprimed bovine peripheral T lymphocytes. Results have shown that cells derived from bovine milk are capable of priming naive peripheral T lymphocytes to proliferate in the presence of a number of different antigens. A 2-3 day delay in peak proliferation occurs, when compared to peripheral blood APC, which may be due to differences in rates of antigen uptake, processing and presentation by milk cells. This observation may explain the previously reported hyporesponsiveness of milk cells in secondary immune responses to antigen. Our results suggest that cells capable of presenting antigen to unprimed T lymphocytes exist within the local mammary gland environment.

COST OF MASTITIS

ELIZABETH BERRY, Genus Animal Health, Westmere Drive, Crewe.

Mastitis is estimated to cost the British dairy industry over £90 million.

The losses can be attributed both to clinical and subclinical cases.

Mastitis costs for clinical cases range from £60.46 for a mild case to £185.16 for an acute case to £2,248 for a fatal case. There are additional losses for clinical mastitis - reduced milk yield and compositional quality, possible TBC, cell count or antibiotic penalties, and early culling with loss of genetic potential.

A cow with a cell count over 200,000 cells/ml can be regarded as subclinically infected. Although she may never show clinical signs, she can be a potential source of infection to other cows and can result in considerable losses. As the bulk milk cell count rises, so does the proportion of subclinically infected cows.

The cell count of the cow can be related to percentage loss in yield and this rises from 3-4% at 250,000-500,000 cells/ml to over 17% at over 1,000,000 cells/ml. Additional losses for subclinical mastitis include the possibility of increased clinical cases, early culling and loss of genetic potential and reduced compositional qualities.

GENETIC FINGERPRINTING OF *STAPHYLOCOCCUS AUREUS* FROM MASTITIC MILK SAMPLES

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Staphylococcal mastitis is a major problem in that it is common, often subclinical and difficult to eradicate. A better understanding of the organism and its epidemiology will be required before optimally effective control measures can be implemented.

The application of DNA based methods of strain discrimination provides not only valuable epidemiological data, but also the means to rationally select strains for further investigation of pathogenic mechanisms.

Strains of *Staphylococcus aureus* were isolated from herds with high bulk tank somatic cell counts (problem herds) in different parts of Scotland. DNA fingerprints were generated using Hha 1 and Sau 3A digestion of *S. aureus* genomic DNA. Toxin and haemolysin production were also determined. In most individual herds there was more than one strain of *S. aureus*, but also evidence of numerous cows infected with the same strain. Different strains were associated with different geographical regions. Examples of 'in herd' and 'herd to herd' analysis of strain variation on the basis of DNA fingerprints will be presented to illustrate the epidemiology together with correlation of toxin, haemolysin and somatic cell count data.

THE EFFECT OF MASTITIS CONTROL PROCEDURES ON BULK TANK SOMATIC CELL COUNTS IN SCOTTISH HERDS

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There is considerable scepticism amongst producers about the value of the major elements of mastitis control. This is especially the case for those producers in penalty wishing advice about how they might improve their SCC figures. Recently we were able to combine the raw Scottish Milk Marketing Board (SMMB) census data for Dairy Facts and Figures 1993 relating to mastitis control for all 2187 SMMB producers with the actual performance of the herds. In addition further data was acquired from the Aberdeen and District MMB comparing high SCC herds, borderline herds and low SCC herds.

The objective of the study was to compare and combine this information from a large number of herds in Scotland (total 2216) with the more limited data obtained by the two smaller but more in depth studies of subclinical mastitis in Scotland. In order to reinforce the importance and value of the major elements of mastitis control enshrined in the five point plan and thus give producers further evidence that infection with *Streptococcus agalactiae* was an important factor in many of those herds with a high SCC.

In summary SMMB herds which post-milk teat dipped had an average bulk tank SCC of 255,000 cells/ml compared to 298,000 cells/ml for those which did not. Similarly those which used dry cow therapy had an average SCC of 260,000 cells/ml compared to 339,000 cells/ml for those which did not. Other factors correlated with a lowered SCC figure from these studies were the importance of udder preparation before milking, in particular the use of individual paper towel, regular milking machine testing, breeding all replacements on the farm, and the general standard of management of the herd including culling policy.

TIME FOR THE HERDSPERSON

ROSEMARY BARFOOT, ATB Landbase, NAC, Stoneleigh, Warwickshire.

The success of any mastitis control programme rests mainly with the person milking the cows. Even with the best management intentions, facilities and cows, unless the herdsperson has the commitment to carry out the right routine every day, problems can occur. In contrast, where facilities are not ideal, major problems are avoided, in some cases, by the dedication of the herdsperson.

To ensure commitment on a daily basis requires time and effort by everyone involved with mastitis control. Whether you are an employer, veterinary surgeon or consultant, it is important to allow time for the herdsperson. (This includes the relief milker). Time spent in ensuring commitment and understanding may make more effective use of the financial investment in mastitis control measures.

TIME TO:

1. **Discuss the mastitis management policy**

   - this has to be a two way process to ensure everyone has all the facts and facilities needed to carry out effective control.

2. **Listen, advise and encourage**

   - so that mastitis control is a joint venture with everyone involved and motivated to achieving the long-term aims.

3. **Carry out what is required in management and milking aspects**

   - ensuring there is enough time to do the things which should be done, and being aware of the pressure everyone is under to get their work done in the minimal time.

4. **Train**

   - to ensure everyone fully understands why things need to be done, which can lead to greater commitment on a daily basis.

     Training may help in achieving the above with respect to staff management, communication skills, understanding mastitis and improving work methods.

THE RELATIONSHIP BETWEEN REGULAR MILKING MACHINE TESTING AND TOTAL BACTERIA COUNTS

ALISTAIR McIVOR, Genus Animal Health, Hallow Park, Hallow, Worcester WR2 6PG

A survey was carried out to study the effect of regular milking machine testing on total bacteria count (TBC). A random sample of 136 herds was used and their two month average TBCs in December 1993 were analysed. The average TBC for the whole group was 14,800 bacteria/ml which compares closely to the national weighted average of 15,000 bacteria/ml in the same month.

The data for the herds were split into two groups: Group A from 64 herds which were members of the Genus milking machine testing service, each receiving a regular milking machine test at least once a year. Group B from 72 herds which were not members of the service, and most were unlikely to be receiving regular milking machine tests according to market research.

The mean TBC for each group was very similar; Group A averaged 14,600 bacteria/ml and Group B 15,000 bacteria/ml, a 3% difference. The proportion of herds with TBCs of 20,000 bacteria/ml or less was also similar, 81% in Group A and 82% in Group B.

A study of the TBCs for the same herds a year earlier in December 1992 revealed that, although both groups had considerably higher average TBCs at 19,800 bacteria/ml for Group A and 20,400 bacteria/ml for Group B, the difference was still only 3%.

An investigation of the average mastitis cell counts of the two groups was also carried out. Group B had an annual average cell count of 441,000 cells/ml whereas Group A averaged 208,000 cells/ml, 53% lower. This compared to a 26% difference a year earlier.

It is concluded that, whilst regular milking machine testing is not associated with low TBCs, it is highly effective in helping to ensure a low mastitis cell count.

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