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INDEX

INTRODUCTION

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The 1995 Conference has been designed to tackle some of the many changes that are occurring in the field of mastitis control and the marketing of milk and as in the past, these subjects have been chosen as a result of the feedback from delegates to the previous Conference.

The changes that have taken place and further changes that will inevitably occur in the future marketing of milk through the EU Milk Hygiene Directives and the new marketing arrangements, mean that even greater attention will be paid to milk quality and especially cell count levels. The prospect of farmers who have average cell counts exceeding 400,000 cells/ml being unable to sell their milk for human consumption in 1998, leaves very little time for a number of producers to take the actions required to meet this standard.

At a time when the emphasis is on milk quality and dairy farmers are under increasing pressure, it is extremely disturbing to find that reductions in the availability of technical expertise at Veterinary Investigation Centres, ADAS, research and equipment manufacturers has occurred. These must cause concern to all those involved in the dairy industry.

What may be seen by some as short-term cost cutting could result in longer term problems for disease monitoring and control, safety of food and the welfare of animals. All of these issues will be of concern to consumers and thus it is imperative that this Conference addresses areas where the organisers believe that problems need to be tackled by involving research workers, farmers and the marketing companies. We hope that the programme will go some way towards highlighting these issues and offering some guidance as to what can be done because without reasonable availability of help and advice, some farmers will face increasing difficulty on how to continue to produce the quality of milk that the market demands from healthy animals.

TACKLING MASTITIS PROBLEMS

TACKLING A HIGH SOMATIC CELL COUNT : A CASE STUDY

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SUMMARY

A commercial dairy farm was successful in reducing its bulk milk somatic cell count from approximately 800,000 cells/ml to under 250,000 cells/ml in less than a year. This was partly due to an effective culling policy, as well as attention to the rest of the Five Point Mastitis Control Plan.

INTRODUCTION

This case concerns a Friesian-Holstein herd of about 170 cows located near London. In May 1994 the owner requested assistance in reducing his bulk milk somatic cell count which, that month, was 826,000 cells/ml with a three month rolling geometric mean of 702,000 cells/ml. This placed the herd within the then Milk Marketing Board Band 3 and resulted in a deduction of 1 p per litre from milk price. With a total quota of approximately one million litres this represented a loss of £10,000 per annum in milk sales alone. Our investigations were carried out in conjunction with Mr John Runnalls of ADAS and I am very pleased to acknowledge his contribution to the case, particularly his investigation of the milking machine.

INVESTIGATION

(a) Records

Examination of milk payment records over the previous two years indicated that the herd cell count had been in Band 3 throughout that period. Total Bacterial Counts had generally been in Band A during that period, but in the previous 24 months, Band B had been reached on seven occasions and Band C on one occasion. Problems with high Total Bacterial Counts had occurred during the winter months only.

Although some records of clinical mastitis were kept these were not complete. From these it was estimated that there had been approximately 70 cases during the previous year. corresponding to 41 cases/100 cows/year.

Table 1 Practice records of intramammary antibiotic tube sales for this farm in the previous year

Lactating Cow Preparations	Tubes
cloxacillin/ampicillin	100
clavulanic acid potentiated amoxycillin/prednisolone	84
cefuroxime	60
TOTAL	244

Dry Cow Preparations	Tubes
procaine penicillin/dihydrostreptomycin	480
cephalonium	40
TOTAL	520

The number of lactating tubes purchased (Table 1) would correspond to about 81 courses of 3 tubes each, although it is very likely that some cases would have used more or less tubes. The number of dry cow tubes purchased would be sufficient to treat 130 cows.

Perhaps the most useful records examined were the individual cow cell count reports provided by National Milk Records. At the most recent recording 150 cows had been sampled and 22 cows had been dry.

Table 2 Distribution of cell counts for 150 cows sampled

Cell count (thousand per ml)	No. cows
> 1,000	23
500 - 1,000	32
200 - 500	25
< 200	70

Over one third of the lactating herd had cell counts over 500,000/ml (Table 2); furthermore the NMR records indicated that four cows were responsible for 23% of the bulk milk somatic cell count, the worst offender having a cell count of over 8 million cells/ml.

(b) Testing of cows

It was decided to try to identify the infected quarter or quarters of each cow using the California Mastitis Test. All quarters of all cows in milk were examined, irrespective of the individual cell count recorded by NMR. Any quarter showing a weak positive reaction or stronger (2) was milk sampled aseptically and the milk examined for bacteria by the local Veterinary Investigation Centre.

This was a long job and was carried out at a morning milking. Undertaking this task gave an ideal opportunity to observe the herdsman during his milking routine and discuss the problem with him.

At the time of sampling ten cows had already been culled as a result of the previous individual cow cell count records. From the remaining 140 cows a total of 122 milk samples were taken.

Table 3 The bacterial recoveries from 122 milk samples

Bacteria	No. isolates
Streptococcus agalactiae	33
Streptococcus dysgalactiae	3
Streptococcus uberis	24
Staphylococcus aureus	26
Corynebacterium bovis	44
Escherichia coli	1
No significant bacteria	23

In total 154 isolates were made with 24 samples containing growth of two pathogens and four samples showing three species of pathogenic bacteria.

A sample of milk taken from the bulk tank at the end of milking revealed *Staphylococcus aureus*, *Streptococcus uberis* and *Streptococcus agalactiae*.

(c) Observations at milking time

Discussion with the herdsman during milking indicated that he believed the problem had a genetic basis. I believe that after spending five hours in the parlour with him, early in the morning, he was reassured that we were not trying to blame him for the entire problem!

Although our presence in the parlour disrupted the usual milking routine considerably it was very revealing to observe what was happening. None of the cows were being foremilked and the herdsman was very happy to admit to this. That several cows had obvious clinical mastitis came as a complete surprise to the herdsman, suggesting that milk from these cases may have been entering the bulk tank and that they may have remained untreated.

An udder cloth, sitting in bucket of disinfectant was being used on some of the cows. In most cases, however, a piece of paper towel was being used to wipe the teats of several cows before being thrown away.

In-line mastitis detectors were installed but were rarely checked. Postmilking teat disinfection was being carried out using a spray gun. This was not being applied effectively and coverage of the teats was very poor. Teat condition was generally good and there were few cows with teat lesions or everted teat orifices.

(d) Testing of milking machine

This was carried out entirely by ADAS. No major problems were discovered. The vacuum was slightly high and two clusters with excessive air leaks were identified. Perhaps more importantly the pulsation in three units was found to be defective. I am not an engineer and I, personally, found it very reassuring to have the milking plant thoroughly tested by an impartial expert in this way.

SUMMARY OF FINDINGS

A high prevalence of *S. agalactiae* infections were identified and it was considered likely that these would be contributing greatly to the bulk milk somatic cell count. Such infections are amenable to intramammary antibiotic infusion and being largely confined to the udder can, theoretically at least, be eliminated by the simultaneous treatment of all infected quarters. This is commonly called Blitz therapy and it was felt that this would be useful in this case.

The high prevalence of *S. aureus* presented more of a problem. It is known that therapy of these infections is often disappointing, even with appropriate dry cow therapy. Generally these animals need to be milked separately at the end of the session to reduce contamination of other cows. Often culling is the most realistic option.

It was decided that cows identified as having *S. aureus* infections should be isolated into a 'Problem Group' to be housed separately and milked at the end of each session. Some cows were culled at this time.

The high number of isolates of *S. uberis* caused great concern. This organism, as well as acting as a contagious organism and spreading during milking may more usually be acquired from the environment. As the cows were at pasture at the time of sampling it was considered that environmental contamination would be less of a problem at that time of year. Treatment and control of this infection is known to be difficult.

Corynebacterium bovis is not usually considered to be pathogenic, however it may be responsible for a rise in somatic cell count. It was the most commonly isolated bacterial species found in the herd and it was felt that this was a good indicator that parlour hygiene may have been inadequate.

Crucially important was the need to improve the milking routine; especially to ensure that the udder cloth was thrown away and that teats were dry wiped with a separate paper towel for each cow.

ACTION TAKEN

After establishing a problem group and culling a small number of cows an attempt was made to reduce the amount of *S. agalactiae* infection using Blitz Therapy. This was far from straightforward because many of the cows with *S. agalactiae* had mixed infections. Antibiotic sensitivities indicated that in addition to the *S. agalactiae* isolates the *S. uberis* and *C. bovis* were sensitive to erythromycin.

It was decided, therefore that all cows known to be infected with any of these three bacteria should be treated by Blitz therapy. None of the problem group were treated however. We realised that the efficacy against S. *uberis* and *C. bovis* would be far less than that against *S. agalactiae*. A total of 60 cows were treated with intramammary erythromycin in all four quarters at two consecutive milkings (1).

The Blitz Therapy made no impact on the bulk milk somatic cell count. In fact in the subsequent month it increased to over 1 million cells/ml. Individual cow cell counts from animals with *S.agalactiae* infections showed a dramatic reduction but those infected with other organisms or with mixed infections generally showed a disappointing response.

It was decided that more extensive culling would be advantageous. This decision was greatly aided by ADAS advice that it would be a good time to lease out quota as the price was rising, and they also advised how much of the herd could be sold without the owner experiencing problems with Capital Gains Tax.

A total of 51 cows were culled during the late summer and autumn of 1994. Cows were selected for culling if they were in the problem group, if they were known to have had repeated cases of mastitis, or if their individual cell counts remained high.

Culling was very successful. By November the monthly bulk somatic cell count was 286,000 cells/ml, and although there was a dramatic increase in February, the downward trend has been continued.

Table 4 Changes in monthly and 3 monthly geometric mean cell count ('000 cells/ml)

Month	Monthly average	Geometric 3 month average
May 94	739	695
June 94	1088	741
July 94	821	756
Aug 94	1026	809
Sept 94	638	702
Oct 94	335	541
Nov 94	286	404
Dec 94	257	302
Jan 95	124	254
Feb 95	626	293
March 95	293	299
April 95	281	317
May 95	180	208
June 95	197	N/A

The most difficult aspect of the case was to attempt to improve parlour hygiene. The herdsman was not prepared to undertake fore-milking, and, although the udder cloth was confiscated, he continued to use a paper towel to clean several cows before disposal. Teat spraying continued to be inadequate.

The herdsman at this farm has now retired, and it has been easier to establish a better milking routine with new staff. I have tried to establish a more controlled and effective use of lactating intramammary preparations on the farm. However, many problems still remain, for example Total Bacterial Counts have extended into Band B on four occasions this year.

The human element in mastitis is probably the most important and certainly the most difficult component to deal with. To expect someone to milk 150 cows to a high standard being dictated by a veterinary surgeon who has not milked a cow for over 10 years is probably asking a lot! We need to think very carefully about how herdsmen and milkers can be encouraged to work to the highest standards. A well designed parlour is certainly important, but perhaps even more important is communication between herdsmen, owners and advisors. I do believe that by testing the entire herd with the California Mastitis Test and by attending two milkings to administer Blitz Therapy I was able to establish some line of communication with the herdsman.

CONCLUSION

There are many aspects to this case which warrant further discussion, for example housing and the use of dry cow therapy. I believe that the case may be of interest for two main reasons. Firstly the Blitz Therapy was completely ineffective in reducing bulk milk somatic cell counts, probably because it was used inappropriately. Secondly because it is unusual to achieve quite such a dramatic drop in bulk milk cell counts the case illustrates how valuable strategic culling may be. Many criticisms could be directed at our investigation and approach to this herd. It was by no means exhaustive and much work remains to be done.

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REFERENCES

1. EDMONDSON P W (1989) Veterinary Record 125 591.

2. SCHALM O W & NOORLANDER D O (1957) Journal of the American Veterinary Medical Association 130 199.

TREATMENT AND PREVENTION OF COLIFORM MASTITIS

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SUMMARY

1. Coliform organisms are often the most common cause of mastitis in well-managed low cell count dairy herds.

2. The efficacy of many common treatments for coliform mastitis is poor, and severe cases have a grave prognosis. A treatment protocol is suggested.

3. Reliable methods of preventing the disease have not been proven in field conditions.

4. "Cow-associated" factors may be important in determining when the disease occurs.

INTRODUCTION

Coliform organisms (Gram negative Enterobacteriaceae of which *Escherichia coli* and Klebsiella spp. are the most commonly isolated) are currently some of the most important causes of clinical bovine mastitis. Over one quarter of bacterial isolates from clinical mastitis have recently been reported to be coliforms (4) and in well managed, low cell count herds, coliforms are often the commonest cause of clinical mastitis (11,16,18,27). Some 80-90% of coliform infections result in clinical cases and they are relatively brief in duration (31,32). The clinical manifestations of the disease have been classified (3) as :

TYPE I (Local): Acute quarter infection only

TYPE II (Systemic): Acute quarter infection with systemic signs (eg pyrexia, loss of appetite, reduced milk yield).

TYPE III (Toxic): Acute quarter infection with systemic signs and the onset of endotoxin-associated (eg sunken eyes, severe weakness/ recumbency, low body temperature)

Around 30% of clinical coliform cases have been associated with systemic signs (18) and 10-15% infections are thought to result in toxic shock (3). Coliform mastitis is, therefore, a common and serious disease. Yet, despite first being recorded in 1896, there have been few controlled field studies to assess therapy (20) and reliable methods to prevent it are far from established.

"No effective method of E coli mastitis control has been proven under extensive controlled field conditions" (20).

"Adequate preventive measures for control of coliform mastitis are not yet available and there is a great need for effective therapeutic regimes, especially for severe cases" (21).

"The controversy that surrounds therapy for acute coliform mastitis is reflected by the variety of treatment protocols that exist" (3).

This paper represents my personal views of treatment and prevention of coliform mastitis in the field, at the current time. It is based on available literature and experiences in a three year field trial studying treatment and prognosis of the condition (13).

TREATMENT OF COLIFORM MASTITIS

The immediate problem is that in the field the organism causing mastitis is initially unknown. In a study in the USA, an accuracy of only 64% was achieved by veterinary surgeons in predicting when coliforms were the cause of mastitis, based on clinical findings (35). Treatment, therefore, has to account for the possibility that a Gram-positive pathogen may be involved, even when coliforms are suspected.

I am not going to consider treatment of mastitis which remains local (Type I), as it is generally treated successfully on farm using intramammary antibiotics.

COMMON TREATMENTS FOR SYSTEMIC AND TOXIC CASES

Oxytocin. Regular stripping of affected quarter(s) and the use of oxytocin injection to facilitate milk removal, is likely to be beneficial in all types of mastitis. It is probably of great importance in toxic coliform mastitis since the disease is thought to be perpetuated by the diffusion of inflammatory mediators from the udder to the circulation (21). Removal of these mediators is a way of limiting the disease. A study at the University of California showed that an injection of oxytocin achieved the same bacteriological and clinical cure rates as found with those obtained using intramammary antibiotics in mild cases of coliform and streptococcal mastitis (16).

Nonsteroidal anti-inflammatory drugs (NSAIDs). NSAIDs inhibit the formation of inflammatory mediators (eg. prostaglandins and thromboxane) and are therefore considered to reduce the numbers reaching the circulation. They also provide some symptomatic relief. The NSAID Flunixin meglumine (Finadyne, Schering-Plough) has been shown to improve some clinical signs (temperature, quarter inflammation and depression) and reduce the concentration of some inflammatory mediators in milk and plasma, in experimental endotoxin-induced mastitis (1,2). An uncontrolled observational study also suggested that Flunixin may have beneficial effects in cases of severe mastitis in the field (9).

Fluid therapy. Fluid therapy has been widely recommended for the treatment of toxic cases but evidence is lacking to verify its efficacy in field conditions. A recent review of fluid and electrolyte therapy for severe mastitis (12) suggested

1. Oral fluids are of very little benefit as gastrointestinal motility and function will be compromised and absorption is poor.

2. Serum chemistry changes are variable and not well understood, although hypocalcaemia has frequently been documented.

3. Near-isotonic sodium chloride-based solutions are likely to be most suitable unless specific serum chemistry results are known.

4. Hypertonic fluids (7.5% sodium chloride solutions) have shown no clear clinical benefits over equimolar amounts of sodium salts administered as isotonic solutions although they may be less expensive.

In a study comparing treatments for toxic mastitis, there was no significant difference in survival rate between isotonic fluid therapy (45 litres in a 24 hour period), Flunixin meglumine (2000 mg in a 24 hour period) or both therapies together (13).

Although infusion of hypertonic saline is currently in vogue as a therapy, caution should be used as it is unproven for toxic mastitis. It should be remembered that the mode of action of hypertonic saline is such that its effect is very transient; the main effect is thought to be an increase in ventricular preload and therefore an increase in plasma volume and blood pressure (8). This peaks at the end of infusion and is dissipated by 30 minutes after administration. Furthermore, it has been seen experimentally that plasma volume may expand in cases of endotoxin-induced mastitis (33,34). This questions dehydration as a primary mechanism of shock in toxic mastitis. Cardiogenic, peripheral vascular capacitance, neurogenic and other mechanisms may contribute to the syndrome (11) and further plasma expansion may not be sufficient to reverse these processes.

It may be that fluid therapy does not have as great an effect in cases of toxic mastitis in the field as is generally hoped.

Antimicrobials. Many authors doubt the usefulness of antimicrobials to treat coliform mastitis (16,19,21). Studies have shown that not only is self cure possible, but that some cows do not recover after antibacterial treatment, even though the coliforms involved are sensitive, in vitro, to the drugs used. The dynamics of toxic coliform mastitis also suggest that at the time of treatment, bacterial numbers in the milk will often be reducing or may have reached zero (12), and that such cows do not become bacteraemic (25). Antibacterials will probably be needed, however, because coliform infections cannot be predicted with certainty from clinical examination and gram positive organisms may be involved. Bacterial overspill from the gastrointestinal tract and impaired immune function (poor liver function) can occur in severe toxic cases in which case antibacterials are also indicated.

Steroids. Glucocorticosteroids may reduce the quantity of inflammatory mediators produced in cases of toxic mastitis, and hence be of use in treating the disease. Experimentally it has been shown that they may be of benefit if given at the time of infection in *E. coli* mastitis (21). In practice their efficacy is doubted because they act at an early stage in the sequence of inflammatory events and may not be effective once signs of disease appear (12).

Nursing. Caring for an animal which is severely sick undoubtedly plays a role in its survival. Warm, comfortable conditions with easy access to food and water are essential and the importance of regular stripping of the affected quarter(s) cannot be over emphasised.

Other therapies. Many other treatments have been suggested for toxic mastitis; from infusing yoghurt into the udder, to filling it with glucose solutions. At present they are generally lacking scientific evidence. Recently, drugs which are "anti-endotoxin" have been licensed for treating toxic conditions in other species (Stegantox, Scherring-Plough). These are, as yet unlicensed for use in cows in UK and are of unknown efficacy in toxic mastitis.

MY APPROACH - A SUMMARY

During our study we found it was possible to predict the chances of survival of cows suffering from toxic mastitis with around 80% accuracy using 2 clinical parameters; upper eyelid skintent time (STT) and rectal temperature (TEMP) (13, see appendix). I use this information to help make on-farm decisions.

A. Systemic cases (no toxic signs)

Oxytocin	Injected intramuscularly, left on farm, 30 IU approx 2 hourly for 1st day with complete quarter stripping ~10 mins after each dose.
Flunixin	(Finadyne) - 1000 mg (20 ml), i/v, initially; 500 mg (10 ml), i/m, 12-24 hours later, (repeated if necessary).
Antibiotics	Intramammary, to cover Gram +ves (even if colis suspected) Systemic, to cover Gram +ves or in case condition worsens to become toxic.
Nursing	Advise provision for warmth (?blankets, deep straw, no draughts), clean water, choice of food, regular stripping of quarter: TLC.

B. Toxic cases

1. Eyelid Skintent time < 3 seconds and TEMP > 37°C

The treatment is the same as for systemic cases excepting

Flunixin -	500 mg given i/m 12 and 24 hours after initial 1000 mg dose (repeated 12-24 hourly as nec for up to 3 days)
Calcium -	40% bottle (400 ml) given <u>slowly</u> i/v at initial visit.

2. Eyelid Skintent time > 3 seconds or TEMP < 37°C

The same treatment as advised for 1. may be used however these cases have a poor prognosis (see appendix). Possible therapies, and their expense, should be considered. It is easy to go to enormous expense in these cases and still end up with a dead patient. If it is agreed to use fluid therapy (and it is often unsuccessful in these cases), I consider it is worth trying an aggressive regime. I now use hypertonic fluids (3 litres 7.5% NaCl solution for an adult Friesian/Holstein) and recommend repeating the treatment 2 hours later and then again as I judge the case requires. This is obviously expensive which is why it is crucial to emphasise

the cost and prognosis at the outset. I use hypertonic solutions because they are quicker to administer and cheaper for the client than larger quantities of the isotonic equivalent, and not because I consider that they will improve survival rates over isotonics. A cow must have access to water immediately after infusion of hypertonic saline as it will often drink large quantities (20-30 l).

Although it seems that some cows become toxic more quickly than others after mastitis is first noticed (13), it may be true that the faster treatment is initiated, preferably before toxic signs, the better the prognosis should be. For farms which have known coliform problems (i.e. history from culture results), I am happy to leave oxytocin and flunixin (Finadyne) on farm, to be used as early as possible in the condition.

PREVENTION OF COLIFORM MASTITIS

Theoretically prevention of coliform mastitis is based on

(i) reducing organisms in the environment to decrease teat end exposure

(ii) reducing the chances of penetration of the teat by the organisms which are present and

(iii) increasing the resistance of the cows to infection should penetration occur.

Control of the disease in practice, however is often unreliable and frustrating. No control methods have been shown to be effective in reducing the incidence of infections under controlled field conditions (Blood and others 1989).

Three main periods in a cows life are responsible for providing the main risk of coliform infection.

1. Dry Period

This is possibly the most important phase because many, if not most, coliform infections originate from this time (31,32). Many infections persist from the dry period to lactation, and are seen as clinical cases around calving or soon after. It is uncertain how effective dry cow therapy is in preventing new infections in the dry period.

2. Periparturient Period

The calving accommodation is often an environment prone to a build up of coliforms because of constant use and little time for thorough cleaning. Furthermore, it is a period of high risk because the cow is likely to spend more time in recumbency and the udder also becomes more susceptible to coliform mastitis (7).

3. Lactation

The risk of coliform mastitis is high at first and reduces as lactation continues (30). Over half of toxic cases have been seen to occur within a week of calving (13).

PRINCIPLES FOR CONTROL OF COLIFORM MASTITIS

There are a number of components to a good programme.

Living environment This should be clean, dry and cool to minimise growth of coliforms ie plenty of clean bedding in a well drained, ventilated building with clean passages, collecting yards and feed areas. (This is especially applicable to dry cow and calving accommodation). This may include use of drying agents, such as lime products, under bedding.

Parlour/udder hygiene The aim is to milk clean and dry teats so as to reduce transfer of coliforms to the udder.

Cows should stand in a clean yard for 30 minutes after milking to allow closure of teat duct.

A well designed and maintained milking machine must be used.

Pre-milking teat disinfection is controversial but may be used (6,24,28).

Post-milking teat dipping should start in the last 2-3 weeks of the dry period.

Diet Rations low in selenium and vitamin E have been associated with an increased incidence of coliform mastitis (26). Appropriate dry cow rations to prevent hypocalcaemia at calving will reduce recumbency and possibly coliform infections (ie generally low calcium and high magnesium).

Vaccination Vaccines are not available yet in the UK. *E. coli* vaccines as used in the US may have some beneficial effects (23).

It seems, however, that the implementation of these strategies alone is by no means the whole answer since the incidence of coliform mastitis has not been significantly reduced in recent times (4). This is in contrast to the situation with contagious pathogens. Furthermore, well-managed, low cell count herds which may be expected to be best at disease prevention have been seen to suffer more from coliforms than other types of mastitis (11,16,18,27). Another anomaly is seen in practice; some distinctly unhygienic farms do not seem to have problems with coliform mastitis.

It is likely that the aetiology is multifactorial and that "cow-associated" factors may play an important role in determining when coliform mastitis occurs. Such factors may include:

1. Milk yield

There are correlations between milk production and mastitis and milk flow rate and mastitis such that increased yields and flow rates increase the risk of a cow becoming mastitic (14,15,29). It could be that well-managed herds tend to have higher yielding stock and the effect of high yield counters the effect of good preventive measures for coliform mastitis and hence the incidence remains unchanged.

2. Somatic cell counts

Many years ago it was seen experimentally that existing somatic cells in the mammary gland can prevent coliform mastitis (26). In one study, 46 cows seen with toxic mastitis over a 3 year period came from herds with significantly lower bulk milk somatic cell counts in the month of the case, than randomly matched control herds (13). This a particular worry to me because even if it is not the low herd cell count itself causing an increased individual risk of toxic mastitis, it suggests that there may be something about low cell count herds which causes then to become more susceptible.

3. Individual response to infection.

Individual cows are likely to have different abilities to mount effective immune responses to coliform infections. It has been shown experimentally that a poor neutrophil response can lead to more prolonged and severe coliform mastitis (17).

CONCLUSION

More research is required to establish foolproof preventive measures for coliform mastitis and also to find treatments that will significantly improve the prognosis in severe cases. At present coliform mastitis remains a great threat to cattle health and welfare in dairy herds.

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APPENDIX

Table 1 Probability of death in cases of toxic mastitis at various temperatures and skintent times at the time of first examination by a veterinarian (13).

		UPPER EYELID SKINTENT TIMES (S)										
TEMPERATURE		0	1	2	3	4	5	6	7	8	9	10
°F	°C											
96	35.5	0.88	0.91	0.93	0.95	0.97	0.96	0.98	0.99	0.99	0.99	1
96.5		0.83	0.87	0.91	0.93	0.95	0.96	0.97	0.96	0.99	0.99	0.99
97	36.1	0.77	0.83	0.87	0.9	0.93	0.95	0.96	0.97	0.98	0.99	0.99
97.5		0.7	0.77	0.82	0.87	0.9	0.93	0.95	0.96	0.97	0.98	0.99
98	36.7	0.62	0.69	0.76	0.82	0.86	0.9	0.93	0.95	0.96	0.97	0.98
98.5		0.53	0.61	0.69	0.76	0.81	0.86	0.9	0.92	0.95	0.96	0.97
99	37.2	0.44	0.52	0.6	0.68	0.75	0.81	0.86	0.89	0.92	0.94	0.96
99.5		0.35	0.43	0.51	0.6	0.68	0.75	0.81	0.85	0.89	0.92	0.94
100	37.8	0.27	0.34	0.42	0.51	0.59	0.67	0.74	0.8	0.85	0.89	0.92
100.5		0.2	0.26	0.34	0.41	0.5	0.58	0.66	0.74	0.8	0.85	0.89
101	38.3	0.15	0.2	0.26	0.33	0.41	0.49	0.58	0.66	0.73	0.79	0.84
101.5		0.11	0.15	0.19	0.25	0.32	0.4	0.49	0.57	0.65	0.73	0.79
102	38.9	0.08	0.11	0.14	0.19	0.25	0.32	0.4	0.48	0.56	0.65	0.72
102.5		0.05	0.08	0.1	0.14	0.19	0.24	0.31	0.39	0.47	0.56	0.64
103	39.4	0.04	0.05	0.07	0.1	0.14	0.18	0.24	0.31	0.38	0.47	0.55
103.5		0.03	0.04	0.05	0.07	0.1	0.13	0.18	0.23	0.3	0.38	0.46
104	40.0	0.02	0.03	0.04	0.05	0.07	0.1	0.13	0.17	0.23	0.29	0.37
104.5		0.01	0.02	0.03	0.04	0.05	0.07	0.09	0.13	0.17	0.22	0.29
105	40.6	0.01	0.01	0.02	0.02	0.03	0.05	0.07	0.09	0.12	0.17	0.22
105.5		0.01	0.01	0.01	0.02	0.02	0.03	0.05	0.06	0.09	0.12	0.16
106	41.1	0	0.01	0.01	0.01	0.02	0.02	0.03	0.05	0.06	0.09	0.12

REFERENCES

1. ANDERSON K L, SMITH A, SHANKS R D, DAVIS L E AND GUSTAFSSON B K (1986) American Journal of Veterinary Research 47 1366-1372.

2. ANDERSON K L, KINDAHL H, SMITH A, DAVIS L E & GUSTAFSSON B K (1986) American Journal of Veterinary Research 47 1373-1377.

3. ANDERSON K L (1989) Compendium for Continuing Education for the Practising Veterinarian 11 1125-1133.

4. BOOTH J M (1993) Cattle Practice 1 125-131.

5. BLOOD & RADOSTITITS (1989) Veterinary Medicine, 7th Ed.,

6. BLOWEY R G (1993) Cattle Practice 1 197-206.

7. BREAU W & OLIVER S (1986) American Journal of Veterinary Research 47 218-222.

8. CONSTABLE P D (1994) Proceedings 12th ACVIM Forum p634.

9. CHRISTIE H (1987-1988) Proceedings of the British Cattle Veterinary Association, pp62-63.

10. EBERHART R J & NATZKE R P (1979) Journal of Dairy Science 62 1-22.

11. ERSKINE R J, EBERHART R J & SPENCER S (1988) Journal of the American Veterinary Medicine Association 192 761-765.

12. ERSKINE R J, KIRK J H, TYLER J W & DE GRAVES F J (1993) Veterinary Clinics of North America 9 499-517.

13. GREEN M J (1994) Dissertation for Diploma in Cattle Health and Production, Royal College of Veterinary Surgeons.

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

15. GRINDAL R J, WALTON A W & HILLERTON J E (1991) Journal of Dairy Research 58 383-388.

16. GUTERBOCK W M (1994) Proceedings of the 26th Convention of the American Association of Bovine Practitioners p 6.

17. HILL A W (1981) Research in Veterinary Science 31 107-112.

18. HOGAN J S, SMITH K L & HOBLET K (1989) Journal of Dairy Science 72 1547-1556.

19. HOGAN J S, SMITH K L & TODHUNTER D A (1987) Proceedings of the International Mastitis Symposium, Quebec Canada p302.

20. JONES T O (1990) Veterinary Bulletin 60 198-220.

21. LOHUIS J A C M (1990) Cornell Veterinarian 80 223-227.

22. LOHUIS J A C M, VAN LEEUWEN J, VERHEIJDEN J, VAN MIERT A & BRAND A (1988) Journal of Dairy Science 71 2782-2789.

23. LUCAS M J & HEWETT G R (1994) Proceedings of British Mastitis Conference (1994) p3-8.

24. PANKEY J W & DRECHSLER P A (1993) Veterinary Clinics of North America 9 519-530.

25. POWERS M J, WHITE M E, DINSMORE P & GUARD C (1986) Journal of the American Veterinary Medicine Association 189 440-441.

26. SCHALM O W, LASMANIS J, CARROLL E J, (1964) American Journal of Veterinary Research 25 83

27. SCHUKKEN Y H, (1989) Veterinary Record 25 60-63.

28. SHEARN M F H, LANGRIDGE S, TEVERSON R M, BOOTH J M & HILLERTON J E (1992) Veterinary Record 131 488-489.

29. SHOOK G E (1993) Veterinary Clinics of North America 9 563-582.

30. SMITH K L AND HOGAN J S (1993) Veterinary Clinics of North America 9 489-498.

31. SMITH K L, TODHUNTER D A & SCHOENBERGER P S (1985) Journal of Dairy Science 68 53-533.

32. TODHUNTER D A, SMITH K L, HOGAN J S & SCHOENBERGER P S (1991) American Journal of Veterinary Research 52 84-88.

33. TYLER J W, DE GRAVES F J & ERSKINE R J (1993) Journal of the American Veterinary Medicine Association. [Sourced from Erskine 1993]

34. TYLER J W, WELLES E G & SORGONEN D C (1992) Proceedings of the 10th Annual Veterinary Medicine Forum p505.

35. WHITE M E & 9 others (1987) Cornell Veterinarian 77 13-20.

COLIFORM MASTITIS : A VIEW FROM THE FARM

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SUMMARY

Farming on the Somerset levels gives a challenge to keep cows dry in the winter and there is also copper deficiency problem to deal with. My approach to mastitis prevention seems to have reduced any winter problem but an increased incidence of clinical cases at turnout requires a different strategy. This has led to development of a personal approach to mastitis prevention and treatment explained here.

INTRODUCTION TO FARM

General

The farm is described factually in Table 1. The enterprise is staffed by one full-time employee whilst I milk along with the numerous other jobs necessary.

Table 1 Land and stock

Land	Stock
185 acres	80 cows, Holstein/Friesian
45 acres rough steep pasture.	80 followers
35 acres maize, followed by Italian rye grass leys	200 ewes, lambing Feb/Mar
30 acres clover leys	
20 acres leys for cutting/grazing	
20 acres permanent pasture	

The farm is just on the edge of Somerset levels which means it is very wet in the winter but becomes burnt up by mid July.

Recent production has averaged 6300 l/cow with 3700 l from forage. With a butter fat level of 4.15% and protein at 3.24%, the margin over purchased feed has been 20.0 p/l . I use DAISY and have calculated that in 1994 some 94% cows conceived requiring 1.7 serves/conception. The calving interval was 359 days.

Mastitis

The bulk milk cell count varies through the year from 80,000 cells/ml in the winter to 180,000 cells/ml in the winter. There were 14 clinical cases of mastitis in the last year, equivalent to 18 cases/100 cows/year. Most of these occurred at turnout and hence the higher cell count in the summer months. I have a number of ideas on why this increase occurs.

These include

- cows lying on cold ground?
- cows slipping in yard in rush to get to grass
- higher yield at turnout leading to more milking machine 'damage'
- by lambing just prior to turnout the milking routine may be skimped, clinical case identification poorer. I suspect this to be true for at least 2 cases this spring.
- the end of the winter may create extra stress and lower resistance
- I try to introduce grass gradually but the cows never seem entirely happy
- a severe problem with copper deficiency.

MY MASTITIS PREVENTION STRATEGY

Housing

My principle intentions are to provide the cows with dry, clean, airy beds which are comfortable. This includes having plenty of room. The milking cows were moved from loose housing to cubicles 5 years ago to help to combat mastitis, they always seemed to become mastitic and never lame, and also to use less straw. We have trouble getting straw.

We have an Old Shed containing 50 Dutch comfort cubicles each 4 ft wide and 9.6 ft long. There is a lunging space of 2 ft in front of the brisket board. The step into the cubicles is 9 inches high. This may be too high but alteration would mean 4 loads of concrete and a higher roof. The apex of the roof has been removed to help ventilation.

Our New Shed has 40 cubicles just 2 years old. The building has the same design but a higher roof and so is more airy and lighter. The beds are always slightly drier and there is a wider passageway. However, the cows prefer the Old Shed!

Straw is blown in twice weekly (Tuesday and Fridays) using 6-8 bales per shed. The straw is blown well to the front giving a big build up and allowing the cows to drag it back.

The passageways and the feed area are scraped twice daily by tractor. Any dung on the beds is scraped off and a little fresh straw covering applied.

I believe that it is important to have the water troughs outside, especially in loose housing, as the cows always waste water.

In winter the dry cows are in cubicles until 2-3 weeks before calving and then loose housed at a low stocking rate. We bed up when dirty with plenty of straw added especially into the corners where the cows prefer to calve. The cows leave the loose housing when they are steady on their feet.

In the spring and autumn all cows are out during the day and in at night. Access to the cubicles is always available, even in summer.

Diet

In winter the milkers are fed a diet of 50-75% maize silage and 25-50% grass silage to give M+12-15 kg . I particularly like the maize as it makes the dung stiffer and may contribute to less mastitis. Concentrates are mix/blend/straights formulation as necessary fed with the silage. Some concentrates are fed as a top-up in the parlour. I use a mineral supplement.

At grass in the spring some silage or straw is provided to slow down scouring effect of young grass, as the cows seem more prone to general disorders when the dung is very loose. Again maize silage seems good for this. When short of silage this spring we were unable to feed enough and this may have contributed to an increased rate of mastitis. [As the cows appear dirtier at grass their tails are cut to prevent 'painting' of slurry!]

The dry cows are fed hay if possible to give a better mineral balance and drier dung. They get straw and silage 2 weeks before calving, then 1-2 kg dairy concentrate with a gradual change to a milking diet. I hope that this stops milk fever and mastitis after calving.

To cope with the copper deficiency cows should be injected at drying off and again at calving and copper included in the cake. I often forget to inject each cow as she calves, therefore they are injected three times each year and have 200 ppm copper in the cake.

Parlour routine

The herd is milked through a Fullwood 6/12 herringbone parlour. It has a 3" vacuum line, jars, clear-flow claws. The liners are replaced approximately every 6 months.

The cows are dry wiped at every milking unless the teats are really dirty, when they are washed and dried.

ACRs are not fitted, but I do try to remove the cluster as soon as possible to prevent teat end damage.

All teats are sprayed with a chlorhexidine disinfectant using a garden sprayer. Cows are then let out into the yard to stand about for up to 1/2 hour after milking, whilst the parlour is washed out. Fresh silage is provided twice daily during milking so it is another 1/2 hour before most cows go to lie down. [I also believe that there is less build up of acid in the rumen from concentrate when cows have access to silage after milking, this may also help to prevent other various disorders.] I try to move the cows quietly so they do not slip. If they fall, I often wash the udder and re-spray the teats, especially fresh calvers.

Summary of basic requirements for prevention

1. Dry comfortable bed to lie on
2. Teat dip or spray
3. Quick milking routine
4. Correct diet, especially in dry period
5. Dry cow therapy
6. Cull persistent offenders or make sure there is no contact with contaminated teat cups.

A PERSONAL APPROACH TO TREATMENT OF CLINICAL CASES

Mild cases

I define these as observing a few clots in the milk, some hardness and tenderness in the udder but not usually any loss of appetite.

I strip the quarter and use intramammary tubes, Synulox or Tetra Delta. Synulox has a 48 hour milk out so I always try it first. I use Tetra Delta milking cow for the more severe cases or if there is a poor response to Synulox. If the cow has a big udder I may use 2 tubes at the first signs then one at the next milking, followed by one every day for at least 3 days. Sometimes a cow with a big udder also gets a Compropen (Ampicillin) injection.

Severe cases

My estimation of severity level changes when there is obvious parlour shyness and loss of appetite. I still find that these cases respond to the same treatment with tubes etc.

Very severe cases

These cows have glazed eyes, they may be scouring, there may or may not be a change in the milk at this stage and the cow could be stiff when walking.

A first thought is for veterinary help as dehydrated cows need fluids to flush out the toxins produced by the mastitis. Along with intravenous fluids we may give 4-5 gallons of tepid water via a funnel and long tube down the throat. This can have amazing results.

I try to strip out the quarter, administer Aureomycin (Chlortetracycline) tubes and give an Engemycin (Oxytetracycline) injection into the vein. Twenty ml Finadyne (Flunixin), an anti-inflammatory might also be given. Milk fever minerals No 6 are given sub-cutaneously.

If the cow is very ill you can't sell her anyway, you may just as well make every effort possible at the start. There is no point in doing half a job, the cow may well make a full recovery.

With a very severe coliform infection 50% of the cows may die, so early detection and treatment is necessary. There is likely to be a very sick 600 kg animal lying on the quarter to be stripped and the animal is dehydrated. It might be thought best to shoot there and then but I ALWAYS try to cure the cow. My methods have been developed because the vet stays for half an hour, charges a fortune and advises stripping the quarter!!

There is no doubt that prevention must be best and my methods seem to suit my farm.

PROBLEMS RELATED TO MILKING MACHINES

COMMON PROBLEMS ENCOUNTERED IN MILKING MACHINES

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SUMMARY

The need for good user servicing of the milking machine is explained with a service programme outlined. Failure to service and poor understanding of the required machine performance leads to many possible faults. These have been identified in a large examination. The major faults and the problems they create are described.

INTRODUCTION

Items of fixed equipment on the farm probably receive less maintenance than they deserve. This is especially true of the milking machine. Given a sound electricity supply, the routine is to switch on the electric motor to drive the vacuum pump, switch on ancillary services and start the 'chore' of milking cows two or three times daily. Minimal time is spent on planned servicing of the milking machine and yet elements of this machine are coming into contact with quite delicate parts of the cow's anatomy on a regular basis. So many times it has been proved that a faulty milking machine has aggravated udder health and yet basic maintenance, to manufacturers instructions, is often neglected on the dairy farm.

This paper will establish the scope of service required and identify machine faults from research of milking machine tests from various sources.

SERVICE INTERVAL

For each item of milking equipment used on the dairy farm, the manufacturer will have provided, via the dealer, instructions for the use and care of the equipment. The service intervals quoted must only be used as a guide, because rates of wear and deterioration will depend upon

- the type of installation and layout
- herd size in relation to number of milking units
- milk yields
- washing system
- whether two or three times daily milking is practised (1)

Effective servicing should restore the machine to an *'as new condition'* with respect to vacuum level, speed, ratio, air flow etc.

EQUIPMENT REQUIRING SERVICE

The installer should provide instructions for routine servicing, including the replacement of individual parts, in particular, with regard to the lubrication and filter servicing requirements. Table 1 shows usual service items and the frequency of checking based on the recommendation of several manufacturers (1).

Table 1 Service items and frequency for various milking machine components and operations.

TIME INTERVAL						SERVICE TASK
D	W	M	3M	6M	A	
*	*	*		*	*	Vacuum pump (various tasks)
*						Vacuum level
*						Pulsation rate
*						Abnormal noises
*----------					----------*	Cleaning filters
*	*	*				Pipe work faults
	*					Hydraulic oil levels (fast exit parlour)
*	*	*		*		Automatic cluster remover
		*				Grease gate linkages
		*				Wash out main and air pipeline pulsators
*----------					----------*	Inspect/renew rubber tubes
				*	*	Dealer servicing
	*					Cleaning air bleeds
					*	Gasket replacement (cluster)
*----------					----------*	Good contacts on safe extra
						Low voltage systems (SELV)
		*				Cleaning the interceptor
*						Water heater time clock setting
*						Water heater temperature
*						Washing system performance
		*				Drain valves
		*				Feeder calibration
	*					Milk pump seal
	*					Claw automatic shut off valve
				*		Renew teat cup liners
*						Bulk tank cleaning

D - daily, W - weekly, M - monthly, 3M - 3 monthly, 6M - 6 monthly, A - annually

In a little more detail these include

Vacuum pump

Daily	- oil level (note abnormal oil usage)
Weekly	- belt tension - pulley alignment and security - belt system guard
Monthly	- oil consumption and adjustment - exhaust system
Six monthly	- vacuum pump performance
Annually	- inspection of vanes (through inlet port)

Cleaning Filters

These are mostly sponge filters requiring removal and cleaning in warm soapy water, a rinse in clean water, drying and then re-fitting followed immediately by a performance test.

However some components utilise small paper filters that are non-serviceable items, giving a cost saving on valuable labour time.

Typical components that require filter cleaning/replacement are

Regulator
Pulsator (clean air supply)
Automatic cluster remover (clean air supply)
Interceptor
Pulsators operating feeders

Maintaining a good low voltage supply

Safe extra low voltage (SELV) electricity is widely used for many items of dairy machinery. SELV is defined as an extra low voltage system which is separated electrically from earth so that a single fault cannot give rise to the risk of electric shock (3).

The most common causes of failure include changes in the environmental conditions, damage by livestock and vermin, vibration in stallwork caused by restless livestock, poor electrical connections - use of simple terminal blocks and over enthusiasm with the power washing hose.

MACHINE FAULTS FOUND DURING PLANT TESTS

A survey of 250 milking machine tests was conducted as a spot check to identify the type and frequency of faults that occur on milking machines (4). The commonest problems are shown in Table 2.

Table 2 Results of a survey of 250 milking machine tests for correct operation and common faults.

ITEM	NUMBER	%
Complete compliance to BS5545	15	6
Zero faults to BS5545 but does not include exhaust test point	94	38
Incorrect vacuum level	57	23
Insufficient vacuum reserve	36	14
Regulator fault	113	45
Pulsation fault	177	71
Other items requiring service	134	54

The results of the tests clearly show that faults on certain elements of the milking machine especially vacuum level and reserve, control, and pulsation that may cause discomfort and injury to teat tissue. Other faults have not been identified, but one may assume that as the machine deteriorated it would adversely affect the milking process.

That such an array of problems is so common seems to suggest that many farmers consider the test to be a service for their milking machine.

The results of the survey prompted an investigation of the results of tests over the previous two years. Results from nearly 25,000 tests covering herds of all sizes and many different milking machine layouts were available. Obviously, it would be impossible to look at all the tests individually. A random selection of one hundred tests within a 24 month period have been examined. Care has been taken that no farms were duplicated. The summary data are shown in Table 3.

Table 3 Frequency of faults in the milking machine found in 100 tests examined in detail.

ITEM	% FAULTY
Vacuum pump	56
Regulator	60
Interceptor	26
Sanitary trap	14
Air pipeline (pulsators)	18
Air pipeline (milking vacuum)	14
Transfer pipeline	8
Milk meter	4
Recorder jar	6
Cluster	28
Pulsator	44
Air losses	14
Vacuum reserve	6
Releaser milk pump	30
Vacuum gauge	6
Pipe size	4
Cleaning problems	28
Mastitis filter	4
Flexible tubes	22
Valves	6

The specific faults found comprise a very long list (see Appendix), the more frequent are shown in Table 4.

Table 4 Commonest faults for the main machine components.

FAULT	%
Regulator renewal	8
New regulator filter	4
Interceptor drain flaps	10
Interceptor lid gasket	5
Main air pipeline dirt	6
Main air pipeline leaks	6
Releaser milk pump leaks	27
Incorrect pulse tubes	12
Faulty wash spreaders	12
Claw gasket leaks	20
Pulsator	40

There are agreed standards for the construction, installation and testing of milking machines, which should provide satisfactory performance of the milking equipment. As with other farm machines, this is a minimum performance level which although perfectly adequate can be improved on to suit the individual herd and farm requirements.

Continual optimal performance can only be obtained if the system operates properly and the basic requirement is to check this by a daily maintenance routine of the milking equipment by dairy staff. It starts with as little as checking the vacuum level and listening to the pulsation.

The results from the examination of machine test reports show that there is a widespread lack of understanding of the working principles of the machine. This would appear to result in reduced maintenance and hence poorer average performance than required.

Instructions for the use and maintenance of the milking machine are often not available, they may assume too much prior knowledge or are **not user friendly.**

Time for maintenance appears to be limited in the working day but is this only because the relative importance of the need for maintenance is not understood? This could also be helped if there were more co-operation between operators and dealer/manufacturers to establish a sensible level of maintenance which is within the technical scope of the operator using tools and equipment normally available on the farm.

Several manufacturers have recognised these problems and have implemented, via their dealers, flexible service contracts to identify with service intervals etc., mentioned earlier (1). There is evidently scope for training in basic servicing of milking equipment to the standard expected by the manufacturer.

The static machine test should not be used as a routine service for the milking machine. The need to perform extensive cleaning and adjustment of mechanisms can only confuse effective testing.

Further in depth analysis, using commercially available computer software, would help to identify more precisely the scope of milking machine faults.

A milking machine which is wrongly designed, worn or badly maintained can present a series of risks to udder health (Table 5).

Table 5 The risks from milking machine faults for udder health

FAULT	RISK
Unstable vacuum	Impacts on teat end
Faulty pulsation	Damage to teat end Damage to streak canal
Excessive vacuum	Damage to teat end
Liner slip	Impacts on teat end

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REFERENCES

1. Routine and Corrective Maintenance of Milking Machines. MPB.3.A.8 Agricultural Training Board.

2. Milking Machine Installations. Part 2 Specifications for construction and performance Section 24: Instruction for use and maintenance. British Standard BS5545: Part 2 (1988).

3. Requirements for Electrical Installation. British Standard BS 7671 (1992) IEE Wiring Regulations 16th Edition.

APPENDIX Description of faults found on machine tests.

Vacuum pump

Poor output
Worn drive belts
Incorrect drive belt tension
Worn pulley
Exhaust inclined upwards from vacuum pump - no oil collector
Loose pulley
Small bore exhaust pipe
Exhaust discharge into pump room
Low/no oil in lubricator
No belt guard

Regulator

Dirty filter
Split diaphragms
Abnormally high air leakage
High vacuum level - incorrect adjustment
Sticking valves
High vacuum level - no adjustment on regulator
Regulator requires total overhaul/renewal (8%)
New filter required (4%)

Interceptor

Poor drainage
Automatic drain flaps (10%)
Automatic shut off valve missing
Lid gasket perished (5%)
Float missing from automatic shut off valve
Hole in interceptor vessel

Sanitary Trap

Automatic drain valve leaking
Lid split
Air leaks in pipework entering sanitary trap lid
Perished gasket
Automatic shut off valve float missing

Main air pipeline/air pipeline (pulsators)

No drainage
Milk/dirt contamination inside pipework (6%)
Excessive number of elbows in pipework
Pipework reduces in diameter back to the vacuum pump
High air leaks (6%)
Poor pipe alignment

Milk system pipework

High air losses
Air leaks from control valves
Air leaks from 3-way valves
Perished/split pipe couplings
Sagging/slipped pipework

Transfer pipeline

Air leaks on milk transfer valves
Diameter of transfer pipe too small for number of recorder jars

Milk Meter

High air losses from milk meter air bleeds
Air bleeds blocked

Recorder jar

Hole in side of recorder jar
Poor vacuum feed to the top of the jar
Air leaks from milk sampling tap
Slow recovery of recorder jar to vacuum because of poor routing of vacuum pipe
Debris in spreader valve at top of jar
Jar not vertical

Releaser milk pump

Drive shaft worn - seals leaking
Air leaks past non-return valve (27%)

Farm vacuum gauge

Poor position
Unable to adjust to true zero
Sticking
Under-reading

Rubber tubes

Incorrect diameter of long and short pulse tubes (12%)
Perished/weak tube prone to collapse under vacuum
Splits in bends and connectors
Reduced pulsation phases due to incorrect long pulse tube

Wash system

Excessive use of dairy chemicals
Faulty jar spreaders (12%)
Poor fit - jetter to teat cup
Poor flow rate due to blocked jar spreader and restriction on transfer valve
Milkstone
Incorrect balance of flow between recorder jar and cluster

3 way valve

Air leaks

Mastitis filter

Air leaks
Incorrectly fitted

Receiver

Air leaks from lid

Cluster

Blocked air bleeds
Worn liners causing a modification to the phases of the pulsation system
Small claw piece bowl - below 80 ml capacity
Air leaks on claw gasket (20%)

Pulsator

Pinched pulse tubes
Dirty clean air filters (slow liner collapse)
Dirty contamination in pulsator clean air pipeline
Poor wiring to pulsator relays
Excessive pulsation rate - up to 76 pulses per minute (p.p.m.)
Slow pulsation rate - down to 35 p.p.m.
Incorrect pipe size for air pipeline (pulsator)
Excessive milk to rest ratio 75:25
Split diaphragms in pulsators
Poor rest phases ('d' phase)
Generally poor condition due to dirt contamination

MASTITIS AND MILK MARKETING

WHAT THE PURCHASER REQUIRES IN QUALITY FROM THE FARMER

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SUMMARY

The UK milk industry, as a direct result of deregulation, is going through fundamental changes as it moves from a producer led industry to a consumer led industry.

The consumers' perception of quality is changing. Consumers not only want products that are fresh and of high quality they are also increasingly seeking reassurance on the welfare of the animals (and their calves!) that produce the milk.

Quality Assurance at farm level will take on increasing importance as buyers ensure compliance with the milk hygiene requirements of Council Directive 92/46 EEC as implemented in the UK by The Dairy Products (Hygiene) Regulations 1995 (1) and move to approved suppliers whose premises and milking practice meet the buyers' standards.

The opening up of the market place will introduce innovation and continuing changes in milk marketing with closer relationships between direct buyers and their producers.

INTRODUCTION

This paper describes the views that have been developed as Nestlé has prepared for the changes in the milk market and especially the quality standards being set. Quality is definable as "the degree of excellence".

This can be considered in two parts. First, by looking at the attitude of Nestlé, its products and view of the quality required to achieve the company goal of being the number ONE branded food company in the UK and a major exporter of dairy products, and to show what has been achieved in the first year of purchasing milk directly. Secondly, to comment on the Nestlé view of the future changes in the market place and to implement the milk hygiene directive.

NESTLÉ

Nestlé is one of the worlds leading branded food manufacturers employing 200,000 people in 440 factories world wide. Annual purchases include 7.6 million tonnes of fresh milk to supply 98 factories in 38 different countries. In the UK purchases total 700 million litres for manufacture into branded milk products for home and world wide trade. Four factories, Omagh in Northern Ireland, Girvan in Scotland, Dalston in Cumbria and Ashbourne in Derbyshire, receive this milk.

Raw milk is manufactured into Whole Milk Powder, Evaporated Milk, Rice Pudding, UHT Drinks, Cream, Sweetened Condensed Milk, Cappuccino and Chocolate Crumb for the confectionery division.

Under the old Milk Marketing Scheme there was a barrier between the producers and the users with many of the decisions on the way the industry was run decided by the Milk Boards who represented the producers. The market was not consumer led and the individual producers had less awareness, and even less interest (!), in what was happening in the market place. As long as the milk was collected each day and paid for regularly they were happy.

The changes which occurred on 1 November 1994 were quite fundamental. The statutory requirement for the Boards to buy all of the milk produced was removed and direct purchasers took on the responsibility for not only collecting the milk but also the enforcement of the milk hygiene standards and compliance with quota regulations.

Direct contracts with farmers allow purchasers to influence quality by being proactive with the producers and by allowing full traceability of the supply. Milk bought from single voluntary co-operatives can only be tested by the purchaser on arrival at the factory and the cooperative has to be relied upon to impose and improve quality standards.

Previously 'wet white stuff' was bought down a phone line! Producers were paid for constituent value yet the manufacturer paid by the litre. In an area of the country with poor compositional quality this could be tough for the purchaser.

In England and Northern Ireland prior to 1 November 1994 core producers were 'allocated' to the Nestlé factories. Statistics on the milk quality achieved by these producers were compiled and to a limited extent there developed an impassive relationship between them and the company. In Scotland there was no traceability. After nine months of direct buying it is possible to compare the quality achieved from core producers allocated by the MMB (England & Wales) to our Ashbourne factory for the period January - April 1994 with that by direct producers in the same period of 1995 (Table 1) .

Table 1 Comparison of milk quality between MMB supply in 1994 and direct suppliers in 1995 to Nestlé Ashbourne.

Month	No. of producers	TBC	SCC	Antibiotic failures	Added water failures
MMB supply 1994					
January	347	12	288	4	16
February	346	11	277	8	14
March	340	12	268	4	34
Ashbourne direct farmers 1995					
January	260	8	214	2	10
February	260	6	187	8	7
March	260	7	181	5	1

These statistics indicate a better quality of supply with the new direct contracts, though it is uncertain if this results from an improved quality of recruited suppliers or Nestlé's expertise.

To achieve our marketing objectives it is necessary to keep abreast of public taste and to back this with good packaging and advertising - this requires innovation and expenditure. However QUALITY is the bottom line and the consumer, whether from the Middle East or Middle England, must be totally satisfied with his last purchase and have confidence in the brand name and manufacturer.

To achieve quality throughout the supply chain "from Farm to Factory to Consumer" the Nestlé policy is to manage the risk, identify the critical control points and reduce the hazard to acceptable levels. The management of the risk at supplier level is done increasingly by working with suppliers across all products so that they put into practice the quality procedures. This "quality assurance" plus full traceability is the preferred route rather than trying to achieve the objectives by ever more vigorous testing at the factory.

Under the Milk Board regimes hygienic quality of milk received a lot of attention and to their credit the consumer had confidence in milk quality. There was encouragement for high quality milk producers and discouragement for poor quality production. With direct contracts, the influence on quality can be proactive with the farmers whereas with alternative suppliers it is only possible to test milk on arrival at the factory and to rely on the suppliers to impose and improve quality standards.

Quality in milk is determined at the farm level. The composition depends on feeding and breeding; cell count and bacterial count depend on mastitis management, milking practice, animal husbandry and plant hygiene. Antibiotic use and misuse happens at farm level.

MILK - PRODUCT SPECIFICATION FOR MANUFACTURING

Composition

Currently milk is standardised to achieve the correct ratio of fat to other solids (SNF - solids not fat) depending on the compositional quality of the milk supplied and the specific product to be manufactured. This equates to a required fat level of approximately 3.5%. At current fat levels the small surplus of fat goes to other Nestlé factories or is sold. Higher levels of SNF contribute to improved yields of finished product.

The concept of designer milk is getting increasing publicity with ADAS researchers confidently predicting that they can reduce fat in milk by up to 25% by change of diet. Whilst this may be very useful as a marketing initiative for the liquid market and a help to those producers over quota, Nestlé would not want to become a buyer of cream because the milk received at the factories was too low in fat!

The most efficient way to adjust milk is to **separate**. The designer milk idealists and researchers have a different perception of the industry. Such milks will only, in our view, supply niche markets with limited volumes.

More important are the process stability problems from poor composition in the spring prior to turnout and the even worse consequences when producers start cutting back on concentrates to manage quota.

Total bacterial count (TBC)

Low TBC levels are vitally important for all users of milk and for Nestlé the factory throughput and end product quality depend on input milk of the highest hygienic quality.

The industry currently determines the hygienic quality of milk by means of a total bacterial count using an automated loop plating technique, incubating the culture for 72 hours and counting the colonies. This technology is labour intensive, slow and in fact only determines the count of viable bacteria. More modern methods using a fluorescent staining technique and counting all cells have been introduced successfully in several countries eg Holland introduced this system in March 1995.

This new equipment is about to be introduced in the UK at ADAS and Milk Marque and will produce a total count, including psychrophilic bacteria. This figure is then converted to give a viable count that will equate to current results for most producers. The significance of this change is that some producers with low TBC plate counts may have higher total counts using the new system because all bacteria are stained. For the producers with outlying results investigations will be made during the introduction period to identify the bacteria and the source so that the total counts may be controlled.

The main advantage of the new system is the speed of results. They will be available the day after sampling rather than after the incubation period.

Frequency of collection

The industry is pulling in two directions on this subject. The customers for liquid milk want fresh low TBC milk collected daily and delivered daily to their supermarkets. Several supermarket buyers have recently announced that they are launching special quality milk schemes from approved producers with milk that is collected daily using dedicated tankers, collection silos and production lines. The milk co-operatives that have replaced the Boards are trying to move to alternate day collection to reduce their very high transport costs.

Nestlé are promoting alternate day collection and considering third day collection from producers with low TBC for milk for some products. The Nestlé experience in Europe is that third day milk will process well provided it is of good initial quality and has been kept at or below 4°C. There is an obvious concern from producers about the perception of milk collected every three days.

Somatic cell counts (SCC)

Nestlé set a Gold standard at 250,000 cells/ml in March 1993 when Nestlé was the first purchaser to launch direct purchase contracts and this has been adopted as a standard for the industry.

High cell count causes a decrease in SNF and fat which reflects in the payment to the producer. For the manufacturer any change in the physical characteristics of the milk will lead to potential processing problems eg throughput, stability as well as any effects on the quality of the end product and its shelf life.

The new hygiene regulations (1) mean that milk with a monthly geometric mean somatic cell count of 400,000 cells/ml on collection from the farm will, after 1st January 1998, be totally unmarketable.

Nestlé are introducing new and tighter bands and penalties from April 1996 and will be helping producers who currently do not meet these higher standards.

Antibiotics

Nestlé will not accept milk testing positive for any antibiotics.

Prior to 1 November 1994, routine tests were carried out at the factories both on milk from core producers and on the other additional milk supplies. Since 1 November 1994 milk has been sampled from every collection from all producers and a factory based antibiotic testing service provided to contracted producers. This service has provided antibiotic testing for farm vats and for individual cows and has been taken up by producers supplying to all of the factories particularly in England and Wales where the MMB (England & Wales) had, unlike the SMMB, not been sampling every collection in the past.

Nestlé direct suppliers are responding positively to their responsibilities in this requirement.

Any relationship of Nestlé with the veterinary profession is strictly through the producer/vet arrangement and is viewed as proactive and a positive contribution to milk quality rather than being another cost centre.

Nestlé set out in 1992 to secure independent milk testing for deliveries to the factories and now use the very modern facilities at ADAS Wolverhampton, the Anser laboratory in Northern Ireland and SAC Auchincruive. These laboratories along with the rest of the industry use the Delvo test and samples from failures are held (up to one month) for further tests if required. The Charm test is currently under trial to help identify the antibiotic found.

Maximum residue limits (MRL)

The Dairy Products (Hygiene) Regulations 1995 (1) specify the requirement of the buying establishment to test for the residues of substances having pharmacological or hormonal action and of antibiotics, pesticides, detergents and other substances including added water which are harmful to human health or which might alter the organoleptic characteristics of dairy products or make their consumption harmful to human health if those residues exceed permitted tolerance limits.

Under Council Regulation (EEC) 2377/90 MRLs are set for antibiotics, antiparasitic agents and pesticides. Nestlé will be carrying out these tests and expect producers to observe label recommendations, abide by the regulations and not to knowingly sell milk that has any adverse effect on products in terms of taste, performance and residue levels.

It is of concern that with the changes in the regulations that the approval of sanitation products now comes under the Environmental Health Officers and that these products will now be marketed without label approval.

The future

The number of producers will fall dramatically if the pundits are to be believed. ADAS has predicted that the number of producers in England and Wales could fall from 29000 to 20000 by the turn of the century and Jim Brown from Scottish Milk suggested at the Peebles Conference this year that the present 2000 Scottish producers be reduced to only 1000 in 10 years time.

Nestlé see the future as producing high quality milk efficiently and that this will increasingly become the job of specialist dairy farmers.

The rapid changes in the industry in the UK recently leaves few opportunities for new entrants and with other EU countries protecting their smaller producer there may emerge a two-speed milk industry in the EU.

With deregulation the UK dairy industry will, after the current rash of closures and restructuring, be more competitive in what will be in postGATT terms a very competitive EC market place.

Nestlé have recruited the more progressive producers with above average hygienic quality. This is a very good starting point and the way forward is to develop further a relationship with these producers so that the businesses can be developed jointly. The future in this relationship is in moving towards "approved" farms that meet Nestlé standards in quality of the milk, milking facilities and practice, herd health and increasingly the welfare of the animals producing the milk.

The Dairy Products (Hygiene) Regulations 1995 (1) come into force this year. The implementation of these regulations will be partly by MAFF, the Scottish Office and DANI through regular inspection and approval of premises. The direct buyer is however in the front line on the implementation of these regulations and has the primary responsibility to ensure that the milk meets the standards in the directive.

Welfare issues have a high profile and must be tackled by the industry as there is increasing pressure to assure customers that the milk comes from happy cows always receiving tender loving care.

Since deregulation there has been an upward price spiral in a sellers market. This will not always be the case and more stable pricing that is fair and sustainable to both parties will provide the greater long term security that we all seek.

REFERENCES

1. The Dairy Products (Hygiene) Regulations 1995, London, HMSO.

APPENDIX Nestlé UK Ltd Somatic Cell Count Penalties

The band that your milk falls into will be determined by geometric mean of at least two samples taken each month over a period of 3 months

1 November 1994 to 31 March 1996

Somatic Cell Count	Band	Price Deduction
Less than 250,000	Gold	Full Price
250,001-400,000	Silver	-0.1 ppl
400,001-500,000	Bronze	-0.4 ppl
500,001-750,000	Sub Standard	-1.0 ppl
750,001-1,000,000	Sub Standard	-1.5 ppl
More than 1,000,000	Sub Standard	-2.0 ppl

1 April 1996 to 31 March 1997

Somatic Cell Count	Band	Price Deduction
Less than 250,000	Gold	Full Price
250,001-400,000	Silver	-0.1 ppl
400,001-500,000	Sub Standard	-1.0 ppl
Over 500,000	Sub Standard	-6.0 ppl

From 1 April 1997

If your milk falls into the sub standard band for more than four consecutive months it will be deemed that your milk does not meet the requirements set out in the EU directive 92/46 and it will be the responsibility of the producers to dispose of such milk.

Somatic Cell Count	Band	Price Deduction
Less than 250,000	Gold	Full Price
250,001-400,000	Silver	-0.2 ppl
Over 400,000	Sub Standard	-6.0 ppl

IMPACT OF EU DIRECTIVES ON THE UK DAIRY FARMER

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SUMMARY

The legal aspects of the Milk Hygiene Directive 92/46/EEC and the relative UK legislation Dairy Products (Hygiene) Regulations 1995 (DPHR) are explained simply. The requirements of the new standard, especially new improvements in milk quality are discussed. Finally the need for high quality milk following good mastitis control is considered.

INTRODUCTION

Today's consumer quite rightly demands high quality food. To ensure consistent high quality requires that every stage of the food chain operates at the correct standard. Farmers recognise the crucial role they play in this process. European and national rules help ensure that the product leaving the farm is of the quality required to enter the food chain.

The EU directives referred to in this paper form the backcloth for UK domestic legislation including the DPHR 1995 in force today. These regulations provide for higher standards in some instances than were previously required. However the main purpose of Directive 92/46/EEC was to provide for a uniform high standard across the EU to facilitate trade and to introduce the EU health mark.

This paper discusses the effect of the directives and the national regulations on the UK dairy farmer.

EU LEGISLATION

(a) Background (Directive 85/397/EEC)

The first common quality standards for milk in the EU were set out in Directive 85/397/EEC. This covered only milk intended for trade between EU member states; milk produced for sale on the national market was left to national legislation. Directive 85/397/EEC covered all aspects of milk production from the farm, through the processing establishment, to the consumer.

For raw milk, two sets of quality standards were established and became known as "Step 1" and "Step 2". The standards covered plate count at 30°C, somatic cell count, freezing point (as a check for added water) and checks for the presence of penicillin and other antibiotics.

Table 1 "Step 1" and "Step 2" Standards

	Step 1	Step 2
Plate Count at 30°C (per ml)	≤ 300,000	≤ 100,000
Cell Count (per ml)	≤ 500,000	≤ 400,000
Freezing Point (°C)	≤ - 0.520	≤- 0.520
Antibiotics (per ml) • **penicillin** • **other**	< 0.007 IU undetectable	< 0.007 IU undetectable

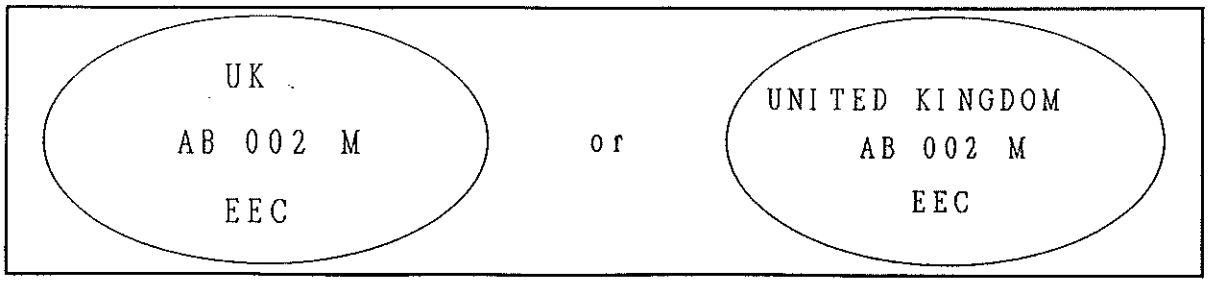
The lower Step 1 standard applied to all intra-EU trade in heat treated milk from 1 January 1989. The UK chose to apply Step 2 standards to the domestic market from this date, enabling us to require the same Step 2 standards of imported liquid milk.

When Step 2 standards were introduced, the MMB undertook a programme of visits by field advisers to farms where a problem had been identified. The MMB ensured that milk from those farms which did not meet the Step 2 requirements was excluded from the liquid market and used only for manufacture.

(b) Current Situation (Directive 92/46/EEC)

Directive 85/397/EEC, which covered only intra-EU trade in liquid milk, was replaced by a much more comprehensive Directive on milk hygiene, Directive 92/46/EEC. It was intended that this Directive be in force throughout the EU by 1 January 1994. However, prolonged discussions in Brussels over amendments to the Directive contributed to delays in implementation in all member states. At the time of writing, several member states had still not implemented the Directive fully. The Directive was implemented in the UK on 9 May 1995.

Under the provisions of Directive 92/46/EEC, all approved processing establishments in the EU will be given an approval number. Any dairy product produced in accordance with the requirements of Directive 92/46/EEC may carry the EU "Health Mark" - a small oval showing the country of origin and the processor's approval number. All products traded between EU member states must carry the health mark.



Directive 92/46/EEC sets hygiene requirements for the production and marketing of all milk and milk products in the EU, whether for national markets or export. As in Directive 85/397/EEC, all aspects of milk production, processing and marketing are covered - from the farm to the final consumer.

Small processors (defined throughout the EU as those establishments processing less than 2 million litres of milk per year) can obtain derogations from some of the building and equipment requirements of the Directive but not from the hygiene standards themselves.

In the UK, all of the requirements of Directive 92/46/EEC (and some additional national requirements) have been brought together in the DPHR 1995. The DPHR came into force on 9 May 1995, and largely replaced the existing milk hygiene legislation, the Milk and Dairies (General) Regulations 1959.

(c) Requirements on UK Producers

Schedule 1 of the DPHR sets conditions for production holdings including general conditions of hygiene for the holding, staff and animal housing. Conditions for handling, cooling and storing raw milk on the farm are also covered here. Schedule 2 deals with the approval of dairy processing establishments. Later Schedules cover product requirements as well as the storage, transport and packaging of dairy products.

Schedule 3 of the DPHR deals with requirements for raw milk in three parts: animal health standards; raw milk standards; and checks for added water.

- *Animal Health Standards*

Animals must not show any sign of infectious disease, give the milk no abnormal organoleptic characteristics *("organoleptic" relates to the effects on the human senses, particularly taste or smell)* and be in a general state of good health with no visible udder wounds. Cows must be yielding at least 2 litres of milk per day. Where animals have been treated with substances potentially hazardous to human health, the correct withdrawal period must be observed.

- *Raw Milk Standards*

Milk must have no added water or excessive residues of antibiotics or other harmful substances, and must satisfy the criteria shown in Table 2. A standard for *Staphylococcus aureus* is set for milk to be used for raw milk products. At least three samples out of five must have fewer than 500 bacteria per ml; none may have more than 2000 bacteria per ml.

Table 2 Raw Milk Standards

	For Liquid	For Manufacturing
Plate Count 30°C (per ml)	≤ 100,000	≤ 400,000 now ≤ 100,000 1.1.98
Somatic Cell Count (per ml)	≤ 400,000	≤ 500,000 now ≤ 400,000 1.1.98

- *Checks for Added Water*

Raw milk is to be sampled randomly and the freezing point checked to ensure that no added water is present. No specific freezing point standard has been set, but any suspicion that water has been added will be followed up with further sampling and testing. The main responsibility for checking for the presence of added water rests with the purchaser of the milk.

(d) **Enforcement of the New Standards**

The standards apply to raw milk as it leaves the production holding, and are to be checked by random sampling. The responsibility for ensuring that the sampling is properly undertaken rests with the occupier of the processing establishment.

- The ***plate count*** (at 30°C) is to be calculated on the basis of a geometric average over a period of two months, with at least two samples per month.

- The ***somatic cell count*** is to be calculated on the basis of a geometric average over a period of three months, with at least one sample per month.

The pronounced seasonality of milk production in Ireland means that it will be difficult to obtain useable somatic cell count results during certain months of the year. An alternative method of calculation has been proposed, but has yet to be approved formally by the Commission. This would allow the results from two consecutive months to be combined into one at the times of the year when production is at its lowest.

The UK has in the past checked somatic cell count at the point when bulk milk is delivered to a dairy. As a result, the higher cell counts of some individual farms have been somewhat disguised. The move to testing at the individual holding may present problems for some farms in the UK, despite the general improvements in cell counts made over recent years. The problem can be most pronounced in herds with very seasonal production, as animals tend to have a higher cell count before drying off.

The UK secured a derogation from Directive 92/46/EEC, enabling cell counts to be tested at the dairy until 1 July 1997. This will give those UK producers with high herd cell counts valuable extra time to meet the new standards. This derogation applies to all member states not just the UK. Hence the right of UK producers to carry the EU health mark will not be affected.

EFFECT ON UK DAIRYING

(a) Somatic Cell Count Situation (England & Wales)

Cell counts improved markedly under the system of payment penalties and incentives introduced with the MMB Cell Count Scheme in October 1991. From April 1993, milk was classified into four bands, with Band 1 milk meeting the cell count requirements of the two EU hygiene directives.

- Band 1 < 401,000 cells/ml
- Band 2 401,000 - 500,000
- Band 3 501,000 - 1,000,000
- Band 4 > 1,000,000

In the 1993/4 year - the latest full year for which MMB figures are available - 75% of producers (supplying 85% of milk) were in Band 1. A further 9% of producers (or 7% of milk) were in Band 2. This indicates that approximately 16% of producers in England and Wales (accounting for 8% of all milk supplies) could have difficulty meeting the new cell count standards. Fewer than 3% of producers (supplying under 1% of milk) had cell counts in excess of 1,000,000 cells/ml.

Since vesting day on 1 November, each milk purchaser can only present a comprehensive picture of cell counts in milk from its own supplying farms. A *Milk Quality Forum* (MQF) has been established by the NFU, the Dairy Industry Federation (DIF) and the UK Federation of Milk Producer Organisations (UKFMPO), with MAFF as observers.

The inaugural meeting was held on 27 June 1995. It is intended to provide an opportunity to discuss issues relating to raw milk quality and to give an overview of the milk quality situation in England and Wales. A Technical Working Group has also been set up, comprising representatives from the MQF, members of the DIF, members of the UKFMPO and the milk testing laboratories.

(b) Consequences of High Cell Counts

Producers who fail to meet the new cell count standards face serious financial consequences. Milk with a cell count in excess of 500,000 cells/ml (or 400,000 cells/ml from 1 January 1998) - the acceptable limit for manufacturing milk - cannot carry the health mark and hence can be sold only on the domestic market. This would entail separate collection and processing arrangements which most purchasers are unlikely to consider worthwhile. High cell count milk is likely to become unmarketable once the new standards are in effect.

(c) Pricing Incentives

Since 1 November 1994, all major purchasers have applied financial incentives and penalties to the producer milk price in order to encourage producers to reduce somatic cell counts. Most have followed a system similar in principle to that started by the MMB. An explanation of Milk Marque's pricing system follows, as an illustration.

Milk Marque's Incentive Scheme

Milk Marque currently pay their "standard" price for milk with a total bacterial count (or plate count) of less than 20,000 cells/ml and a somatic cell count of between 251,000 and 400,000 cells/ml. Producers who supply milk with a TBC in excess of 20,000 cells/ml face financial penalties starting at 0.25 pence per litre. Financial penalties for cell counts in excess of 401,000 cells/ml start at 0.5 ppl. At the moment, producers receive a bonus of 0.2 ppl for cell counts below 250,000 cell/ml. This will change from April 1996 when the Cell Count Quality Payment Scheme is tightened.

Table 3 Milk Marque Cell Count Scheme

Cell Count (cells/ml)	April - Sept 1995	Oct 95 - March 96	From April 1996
0-250,000	+0.2 ppl	+0.2 ppl	-
251,000 - 400,000	-	-	-0.2 ppl
401,000 - 500,000	-0.5 ppl	-1.0 ppl	-2.0 ppl
≥501,000	-3.0 ppl	-4.0 ppl	-6.0 ppl

REDUCING CELL COUNTS

(a) The Five Point Plan

The basic "5 Point Plan" is still the best method of controlling mastitis in the herd and hence reducing cell counts.

(i) *Disinfect all teats at every milking*

Cover teats and teat ends with an approved disinfectant.

(ii) *Identify and treat cows showing clinical mastitis*

With the help of your vet.

(iii) *Treat every cow with a dry cow tube*

Do it cleanly and effectively when the cow dries off, in accordance with the advice of your vet.

(iv) *Cull cows with persistent infection*

Cows which do not respond to treatment can put unaffected animals at risk.

(v) *Fix the milking machine*

All machines should be tested once or twice a year, preferably including a dynamic test.

The provision of ***good cow housing*** - designed for maximum comfort and cleanliness - is also important, and should form the sixth point in the plan.

In some cases, very tight single season calving patterns pay produce temporarily high herd cell counts which may have to be addressed.

Schemes such as NMR's individual cell count service can be invaluable in identifying problem animals. The proportion of NMR milk recording members using this service has risen dramatically in recent years - from 42% in the autumn of 1990 to 82% in June 1995.

CONCLUSIONS

The demand for high quality food will continue, as will the need for high standards of health and hygiene on the farm. No doubt first hand buyers of ex-farm milk will continue to use bonus and penalty schemes to encourage the delivery of high quality ex-farm supplies.

The "five point plan" has provided a very useful set of management guidelines to help reduce mastitis levels and somatic cell counts. However more attention will have to be given to all aspects of the dairy cow's environment and particularly to winter housing. Many cow cubicle sheds and other buildings were designed for smaller cows than today's animals, and much has been learned over the years about the management of cow housing. Investment will be needed in this area on many farms in the coming years.

In summary, UK dairy farmers can and will continue to supply high quality raw material to the dairy industry and the consumer.

RESEARCH UPDATE

EARLY DETECTION AND EARLY TREATMENT OF MASTITIS - CAN IT WORK?

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SUMMARY

The electrical conductivity of milk is a simple physical property which can be very easily altered by a host of physiological events including infection of the mammary gland. Accurate means of measuring and computing changes in milk electrical conductivity are becoming available and appear to be useful in detecting mastitis. Subclinical infections may be diagnosed less accurately than clinical disease. Experimental studies suggest that developing clinical mastitis caused by *Streptococcus uberis* or *Staphylococcus aureus* may be identified reliably 1-2 milking before the first clots in milk. An advantage of early diagnosis could come from early antibiotic treatment which has been shown to improve the cure rate and reduce the length of time poor quality milk is produced.

INTRODUCTION

Mastitis detection systems used in the milking parlour, often included as part of the milking machine, are usually based on measurement of changes in the electrical conductivity of milk. Such systems have been under development for over 50 years (1) and more are becoming available commercially so it is timely to consider

- what the systems are supposed to do
- how they work
- what could be beneficial
- how information might be applied.

This will be description of recent work from Compton, from Holland and of collaborative work. The Dutch aim is to develop an accurate and sensitive system whilst at Compton the aim is to evaluate how such a system might be best used. So far, all automatic detection systems of promise use changes in milk electrical conductivity but increasingly rely on incorporation of other information.

MILK ELECTRICAL CONDUCTIVITY (EC) AND THE EFFECT OF MASTITIS

Resistance to an electrical current is a commonly understood concept, it results in the generation of heat and light in the light bulb. Conductivity is the reciprocal of this, how well a current passes through a medium. In bacteriological solutions, such as milk or blood, current is conducted by charged particles, mostly sodium (Na^+), potassium (K^+) and chloride (Cl^-) ions. Simply, the EC of milk might be considered as how many of these ions are in the milk.

In the secretory cell of the mammary gland there is an active system to keep K^+ in the cells and Na^+ in the fluid around the cells and, because there is a concentration gradient between the two, ion flow (current) tries to restore the balance. Similarly there is a difference in the ionic content between blood and milk which is maintained by an active secretion system and a relatively impermeable cellular barrier to ion passage between blood and milk.

As an infection develops to become disease the active maintenance system breaks down and milk becomes more like blood especially in terms of ion composition and concentration. This occurs because various products of the bacteria causing the infection and invasion of the mammary gland by defensive white blood cells change the permeability of the blood vessels and the barrier. The tight junctions between the cells which maintain the barrier are disrupted and the active transport systems become partly disabled.

NORMAL EC

EC varies markedly under normal conditions especially

- between cow and herds
- with time; stage of lactation, parity and diurnally
- with temperature
- with milk composition (fat), oestrus, nutrition etc.

Changes induced by infection have to be distinguished against this background.

EC DETECTION SYSTEMS

Systems to measure EC in experimental studies have been available for decades and under continuous development for farm use. They only need two electrodes, an applied voltage and a meter. However, functional systems need quality, non-fouling electrodes, some electronics and a way of storing and/or processing the data, hence the slowness in commercial availability. A lot of data is generated when measurements are made at various times during a milking, for each quarter, each cow, each milking and each day in milk. This is all data and in itself is of little use. Practical use requires information derived by processing the data. A number of calculation methods have been used to try to show differences between quarters or changes for any given quarter with time. These include

- measurement relative to an absolute threshold
- differences between quarters
- calculation of an inter-quarter ratio
- differences between milk fractions
- changes with time eg between milkings

Usable data processing systems tend to be sophisticated requiring computers. It is sometimes useful to investigate the individual cow using a hand-held meter, but the main drive is to create a monitoring system for the whole herd and in practice this must be automatic.

EC is usually related to other health indicators often for simple understanding eg cell count, bacteriology, clinical signs or milk yield depression.

To use changes in EC it is necessary to decide what information is needed. Is it information on subclinical infection or clinical disease ?

SUBCLINICAL INFECTIONS

High EC could relate to a high cell count but this does not mean disease. The high cell count could be historical, and usually is, as very many high cell count quarters do not have an existing infection. Information on subclinical infections can be useful to

- identify sources of bacteria and high cell count for milk quality purposes [an uncommon/infrequent need]
- identify cows which could later have a clinical case of mastitis [useful to know]
- identify cows posing a risk to others [not urgent]
- identify cows for dry cow treatment or culling [not immediate]

In general this information is not required immediately and every milking. There are probably more cost effective ways of getting the information.

Evidence from use of a prototype on-line system shows that identification of subclinically infected quarters based on EC is fairly poor (2). The problem of failing to detect an infection might be recoverable, but indication of infection when there is none, a false positive, does not engender confidence.

Table 1 Indication of subclinical infection by EC (2).

	E.coli	*S.aureus*	*S.uberis*	*S.dysgalactiae*	No pathogen
EC positive	2	4	0	0	48*
EC negative	0	4#	1#	8#	517

* false positive results # false negative results

The accuracy varies with the pathogen amongst other factors. The results in Table 1 support earlier information, from using a hand-held system, that detection of subclinical streptococcal infections is poor to impossible (3).

CLINICAL MASTITIS

It may seem overkill to use a complicated and expensive machine to detect something that any competent stockman should spot easily - the clinical quarter. Doubtless many will still consider value in such a system, but most value may be in unattended eg robotic, systems when there will be no other way of signalling disease easily.

It is likely that EC will be much more useful to detect <u>developing</u> clinical quarters.

INCIPIENT MASTITIS

There is a time delay, the length varying according to bacterial pathogen, between bacteria establishing in the mammary gland, changes in cell count, change in EC and observation of clots (if the infection becomes so serious) (Table 2). This shows that a change in EC can be used to indicate the developing infection one or two milkings before the first visual observation can be made.

Table 2 Time sequence of development of disease following experimental infection of the mammary gland with *Streptococcus uberis* or *Staphylococcus aureus* (4)

	Average no. of milkings post infusion until			
	Bacteria recovered	1 Log increase in cell count	Change in EC	Clots in foremilk
S. uberis	2.3	3.1	3.5	5.2
S. aureus	1.1	2.3	2.7	4.2

The Dutch system has been used in a parlour on natural infections and different methods of analysing data examined (5). This was not real-time analysis. Detection at the milking when clots appeared was good, but slightly poorer for previous milkings. The sensitivity of the system varied with the method of data analysis. Work is continuing on these problems and further improvements appear likely, especially when use is made of milk temperature and milk yield data in correct association with historical patterns of change. This is a very complicated computation of changes in a number of indirect responses to infection. There are three main complications.

1. Detecting clinical mastitis is rather like looking for a needle in a haystack. At 40 cases/100 cows/year a case is likely only every 6100 quarter milkings. A lot of data has to be gathered and processed before one case is likely to occur. This may mean

2. A false negative diagnosis is made. An infection may exist but is not indicated. This is not too disastrous, there is always another chance at the next milking and other ways of spotting the infection later. Frequent false negatives will however undermine confidence in the system. More important are likely to be

3. False positive signals which will be more dangerous if irrecoverable action is taken eg administration of antibiotic therapy. Much of the damage can be avoided if the EC detection system is simply a primary screening tool, but delay make preclude any advantage from the early detection.

ADVANTAGES OF EARLY DETECTION

The simple advantages are confined to safeguarding milk quality, limiting spread of infection and improving the health of the infected animal. The first is easily achieved by milk diversion and the second by separation of the infected animal. The opportunities to improve the health of the animal lie with implementing early treatment and achieving better cure.

Initial studies have been undertaken on developing clinical cases, created experimentally using *S. uberis*, when accurate early diagnosis was possible and therefore early administration of intramammary antibiotics could be used (6). On average, EC first indicated the infection in conventionally treated cases some 4 milkings before clots and full clinical mastitis occurred. However, when intramammary antibiotic therapy was given, solely in response to EC changes, no clots occurred in the milk from any of the five animals used. The early treatment reduced the number of milkings when milk contained bacteria, fewer tubes of antibiotic were required to achieve clinical cure and all quarters were cured bacteriologically. A 50% bacteriological cure only was achieved with conventional diagnosis and treatment. Early treatment also gave a significant reduction in the period when high cell count milk, undetectable without sampling and laboratory analysis, was produced. The early treated group produced milk with a cell count >500,000 cells/ml for 14 fewer milkings (Table 3). Benefits from less udder damage (a yield effect may also be found), better cure and an earlier return of milk quality seem very achievable. Alternative treatments and disease by other pathogens have still to be investigated.

Table 3 Effect on milk cell from early treatment following detection of developing mastitis (6)

Cow	Infected quarter cell count ('000 cells/ml)		
	Pre-infection	1st treatment	28 milkings post treatment
Early treatment			
0165	39	3050	162
1179	31	2320	206
7519	39	1080	201
1070	53	4733	94
2227	39	837	450
Conventional treatment			
0142	39	>20000	294
1151	29	8180	185
7986	30	>20000	678
1104	71	>20000	734

All studies so far suggest that early detection of developing clinical mastitis can be made. Acceptable accuracy has really only been achieved with systems under experimental conditions. Recent results from the joint UK and Dutch studies promise more accurate diagnosis especially in a large reduction in the number of false positive diagnoses. Further development of computer software and elaboration of benefits when infection is caused by different pathogens is essential. The systems then have to be tested in farm trials.

CONCLUSIONS

There may be benefits from early diagnosis of a developing clinical mastitis by changes in EC. This has to be possible by automatic detection with the information available in real time. The information has to be more accurate than currently possible. Early indications suggest marked benefits in treatment efficacy and milk quality. The economics are far from testable but the potential remains real.

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REFERENCES

1. DAVIES J G, JONES V E & WARD S J (1943) Proceedings of the Society of Agricultural Bacteriology 14 43.

2. LANSBERGEN L M T E, NIELEN M, LAM T J G M, PENGOV A, SCHUKKEN Y H & MAATJE K (1994) Journal of Dairy Science 77 1132-1140.

3. HILLERTON J E & WALTON A W (1991) Veterinary Record 128 513-515.

4. MILNER P, PAGE K L, WALTON A W & HILLERTON J E (1996) Journal of Dairy Science (In press).

5. NIELEN M, SCHUKKEN Y H, BRAND A, HARING S & FERWEDA-VAN ZONNEVELD R T (1995) Journal of Dairy Science (In press).

6. MILNER P, PAGE K L & HILLERTON J E (1995) Proceedings of the 3rd International Mastitis Symposium vol. 2, s5, 44-48.

POTENTIAL VACCINE STRATEGIES TO CONTROL STREPTOCCOCCUS UBERIS MASTITIS

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SUMMARY

Immunisation of dairy cows with a live *Streptococcus uberis* vaccine has been shown to reduce the incidence of clinical mastitis, reduce the somatic cell count and reduce the numbers of bacteria found in the milk following experimental infection. The mechanism by which the vaccine exerts its effect is not yet known but protection against infection was not achieved simply by the action of antibody or the influx of neutrophils. The potential mechanisms and the future directions for effective vaccination of cattle against *S. uberis* are discussed.

INTRODUCTION

The introduction of hygienic milking practices has significantly reduced the incidence of mastitis caused by 'contagious pathogens' including *Staphylococcus aureus, Streptococcus agalactiae* and *Streptococcus dysgalactiae*. Consequently, the commonest causes of clinical mastitis are now *S. uberis, Escherichia coli, S. aureus and S. dysgalactiae*. Control of these diseases is still dependent on good husbandry and the large-scale prophylactic and therapeutic use of antibiotics. Alternative strategies based on vaccination have been sought by many groups around the world although their success has been limited. This paper describes work which attempts to immunise dairy cows against mastitis caused by *S. uberis*.

VACCINATING RUMINANTS AGAINST MASTITIS

The development of any mastitis vaccine has been retarded previously by the belief that immune protection can only be achieved by inducing a rapid inflammatory response to infection which is characterised by the rapid influx of circulating neutrophils into the mammary gland and by enhancing the ability of those neutrophils to phagocytose and kill the bacteria once they reach the gland. This approach has been proposed for *S. aureus* (1,2) and effectively induces a "mastitis to prevent a mastitis". Hence vaccination does not prevent infection and still results in a reduction in milk quality.

Other vaccines have been directed towards the neutralisation of toxic bacterial components. For example, the J-5 *E. coli* bacterin was designed to produce neutralising activity to the toxic portion of the bacterial lipopolysaccharide (3) and vaccination with inactivated haemolysins from *S. aureus* is designed to neutralise toxin activity (4). Both these preparations are intended to reduce the incidence of clinical disease and/or lessen the systemic effects of infection with these two pathogens.

In the case of *S. uberis*, virulent strains appear to be highly resistant to phagocytosis by bovine neutrophils during the initial stages of infection (5) and this has been reproduced in the laboratory (6) where isolates of *S. uberis* can be induced to become resistant to phagocytosis even in the presence of serum from immunised animals (7). Potential vaccines

against *S. uberis* must therefore overcome the ability of the bacterium to resist phagocytosis once within the mammary gland or be directed to other essential properties of virulent strains.

VACCINATION WITH LIVE *S.uberis* VERSUS A SURFACE EXTRACT DERIVED FROM *S. uberis*

The effects of vaccinating dairy cows subcutaneously with live *S. uberis* or with a surface extract derived from *S. uberis* has recently been described (8). The vaccination protocol used was similar to that reported to induce high levels of rotavirus-specific antibody in colostrum (9) and comprised subcutaneous immunisations administered 14 days before drying off and again within 48 hours of calving. Intramammary infusions of the surface extract were given to all vaccinated cows 7 days after drying off and a third group remained non-vaccinated controls. All animals were challenged with *S. uberis* strain 0140J 2-6 weeks into lactation.

All challenged quarters of the animals in the control and extract vaccine group developed clinical mastitis and required antibiotic therapy 2-4 days post-challenge, although the numbers of bacteria present in the milk and the average cell count were lower in the animals vaccinated with extract compared to the controls. The average loss in milk yield was also less in the extract vaccine group, although the variation between animals was considerable.

In contrast, the live vaccine regime reduced the incidence of clinical disease when compared with the control group. Of the 8 quarters challenged, only two quarters developed clinical mastitis and required antibiotic therapy. Of the remaining quarters, 3 showed intermittent recovery of low numbers of bacteria and 3 further quarters shed low levels of bacteria until day 4 after which numbers gradually increased. The cell count was also reduced considerably in the animals given the live vaccine and their milk yield was only slightly reduced following challenge.

This live vaccine regime appeared to modify the speed at which the mammary gland became colonised by bacteria, particularly during the early stages of infection. This was not associated with the infiltration of neutrophils into the mammary gland, since an increase in cell count only occurred in response to high bacterial numbers. This is supported by microscopic examination of milk or tissues from animals following infection which revealed an extensive infiltration of neutrophils into the gland and yet very few neutrophils appeared able to phagocytose the bacteria (10). The level of *S. uberis* antibody (particularly the isotype IgG2) was elevated in the serum of cows given the live vaccine, yet the ability of the serum and milk samples to opsonise the bacteria to aid phagocytosis was not altered. Hence prevention of clinical mastitis was achievable without changed opsonic activity suggesting that, unlike other forms of mastitis, protection against infection by *S. uberis* is not achieved simply by the action of antibody and neutrophils.

In contrast to the live vaccine, immunisation with the surface extract material which contained cell wall and capsular material, evoked a poor response. The bacterial capsule is believed to be an important factor in the ability of the bacterium to resist host defences; it may prevent antibody or other opsonic factors from binding (11). Alternatively, it may allow opsonic factors to bind beneath the capsular surface so that the capsule then prevents contact between the opsonins and their receptors on the phagocytic cell (12). The generation of an immune response to capsular material has become a major goal for many bacterial vaccines.

It has been shown that both encapsulated, phagocytosis-resistant strains of *S. uberis* and non-capsular, phagocytosis-sensitive strains can bind equal amounts of potentially opsonic antibody (7) and that encapsulated and non-encapsulated strains are phagocytosed equally well by bovine macrophages (13). These findings indicate that the capsule is no deterrent to the interaction of the antibody and phagocytic cell with *S. uberis*. The failure of the capsular material to protect against infection contrary to vaccination with the live organism suggests that *S. uberis* may elaborate factors during growth *in vivo* which may be important virulence determinants.

CONCLUSIONS AND FUTURE DIRECTIONS

Vaccination with live *S. uberis* has been shown to reduce the occurrence of clinical disease following experimental infection. The mechanism by which the vaccine exerts its effect is not yet known although, unlike other mastitis-causing pathogens, protection against infection by *S.uberis* is not achieved simply by the action of antibody and neutrophils. It is likely therefore that *S. uberis* produces/secretes factors during growth *in vivo* which may be important virulence determinants and targets for potential vaccine antigens.

Two such 'factors' are the subject of current research at IAH. Studies indicate that *S. uberis* is capable of producing a component from the capsular matrix which may exert a toxic effect on bovine neutrophils (14). The characterisation of this component and its mode of action are the subject of current investigations and it is hoped that a vaccination regime which induces neutralising antibody to this molecule could prove to be a valuable vaccine antigen. A second 'factor' may be involved in the degradation of milk proteins to produce the amino-acids and peptides which are essential for growth within the mammary gland. *S. uberis* has been found to produce an extracellular protein which is capable of activating bovine plasminogen to the caseinolytic enzyme plasmin (15). The concentration of plasminogen in normal bovine milk is around 1.3 μg/ml, therefore activation of plasminogen to plasmin during the early stages of infection, prior to an inflammatory response, could increase the availability of peptides and amino-acids for bacterial growth. Antibody directed at this extracellular protein could significantly reduce the availability of these nutrients and restrict bacterial growth. The plasminogen activator from *S. uberis* may prove to be an essential determinant of infectivity and therefore become a very useful vaccine antigen.

REFERENCES

1. WATSON D L (1988) Research in Veterinary Science 45 16-21.

2. WATSON D L & SCHWARTZKOFF C L (1990) International Symposium on bovine Mastitis, Indianapolis p73.

3. GONZALEZ R N, CULLOR J S, JASPER D E, FARVER T B, BUSHNELL R B, OLIVER M N (1989) Canadian Journal of Veterinary Research 53 301-305.

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

5. LEIGH J A, FIELD T R & WILLIAMS M R (1990) Research in Veterinary Science 49 85-87.

6. LEIGH J A & FIELD T R (1991) Veterinary Research Communications 15 1-6.

7. LEIGH J A & FIELD T R (1994) Infection & Immunity 62, 1854-1859.

8. HILL A W, FINCH J M, FIELD T R & LEIGH J A (1994) FEMS Immunology & Medical Microbiology 8 109-118.

9. SAIF L J, REDMAN D R, SMITH K L & THEIL K W (1983) Infection & Immunity 41 1118-1131.

10. THOMAS L H, HAIDER W, HILL A W & COOK R S (1994) American Journal of Veterinary Research 55 1723-1728.

11. HOROWITZ M A & SILVERSTEIN S C (1980) Journal of Clinical Investigation 65 82-94.

12. KING B F & WILKINSON B J (1981) Infection & Immunity 33 666-672.

13. GRANT R G (1995) PhD thesis, University of Bristol.

14. LEIGH J A & FIELD T R (1993) XII Lancefield Symposium on Streptococci and Streptococcal Diseases. St. Petersburg.

15. LEIGH J A (1993) FEMS Microbiology Letters 114 67-72.

ABSTRACTS OF POSTERS

DEVELOPING A HERD HEALTH PROGRAMME

PETER MAY, MRCVS Swindon, **RAY WILLIAMS, MRCVS** Malmesbury and **ROSEMARY BARFOOT**, ATB Landbase Ltd., NAC, Kenilworth, Warwickshire

Mastitis is just one of the diseases that can affect the dairy herd, but because of its obvious disruption to the milking routine and sometimes dramatic signs, it tends to be ranked as highly important by the dairy farmer and herdsman.

The price of mastitis can be high. There are direct costs and hidden production losses, which vary from farm to farm. As with any inefficiency, disease has to be managed. It is absolutely imperative to develop a successful strategy, which involves everyone working with the herd. The successful strategy must also be cost effective.

It is important however not to deal with mastitis in isolation, but to develop a herd health programme which looks at all diseases and production disorders. To do this successfully, the knowledge and advice of the farm's veterinarian needs to be fully utilised. There needs to be a team approach in tackling disease and a good working relationship and understanding between the farmer and his/her veterinarian.

The aims in planning a herd health programme are:

- protecting the herd from disease introduction
- exercising constant disease surveillance
- disease recording and monitoring
- setting targets for production and disease levels
- knowing when intervention is necessary
- preventing disease and maintaining productivity
- reducing disruption caused by disease
- ensuring contracts with milk buyers
- maintaining profitability of the business

Training may be required to help develop a programme of this kind, and this is now available. The benefits of training in a herd health programme are:

- ensured compliance with legislation
- improved knowledge and greater understanding
- greater motivation and commitment
- exchange of ideas and opportunity for better communication

SURVEY OF MASTITIS CONSULTANCY SERVICE

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A survey was carried out on 836 members of the Genus Mastitis Consultancy Service in England and Wales. Bulk tank geometric mean cell counts at May 1995 were compared with those on joining the service and grouped into the current payment bands - below 250,000 cells/ml, between 251-400,000 cells/ml, over 400,000 cells/ml. These were further divided between clients who had been on the service over two years and those who had joined within the last two years.

There were significant improvements in the percentage of members achieving geometric mean cell counts below 250,000 cells/ml, most noticeable for those members having been on the service over two years.

There are still a number of clients, who, despite having been receiving mastitis control advice for over two years, have still not been able to reduce their cell count to below 400,000 cells/ml. The reasons given by the Mastitis Consultants in order of frequency are:- not culling; poor record keeping; not teat dipping; poor milking routine; milking machine; housing management; not using dry cow therapy. The clinical incidence was calculated only from those keeping accurate clinical records (85%) and was 33 cases/100 cows/year for those on the service over two years and 41 cases/100 cows/year for those joining within the last two years.

MILK QUALITY MASTITIS AND SOMATIC CELL COUNTS
CONTROLLING YOUR BULK TANK SOMATIC CELL COUNT

D N LOGUE, J GUNN and S CHAPLIN, Dairy Health Unit, S.A.C., Veterinary Services, Auchincruive, Ayr KA5 5EA

EC directive 92/46 on milk hygiene states that milk for human consumption must have a Bulk Tank Somatic Cell Count (BTSCC) of less than 400,000 cells/ml. The EC derogation for collection of milk in excess of 400,000 cells/ml ends in 1997 so after that date producers must ensure that this threshold is ALWAYS met. Thus it is essential that producers protect their businesses by reducing their BTSCC.

Research at SAC since 1990 (Fenlon *et al* 1994, Logue *et al* 1994, Gunn 1995) has shown that there are a number of elements of control which will allow a more rapid reduction of BTSCC than simply applying the "Five Point Plan". The major elements are based around the use of Individual Cow Somatic Cell Counts (ICSCC) which are now widely available. However, these elements must be applied alongside the full implementation of the "Five Point Plan".

Firstly, producers should aim for annual BTSCC means of less than 250,000 since sometimes herds in excess of this, but under 400,000 can exceed 400,000 for a few months in the year. Even herds with low annual figures can have a slightly raised BTSCC when a lot of cows are calving or nearly dry, despite having very few infected cows.

Secondly, use ICSCC figures to target the essential elements of mastitis control by:

i) Identifying the problem cows, ie those with a consistently high ICSCC.
ii) Determining the major bacteria causing subclinical mastitis.
iii) Treating infected milking cows if appropriate, eg *Streptococcus. agalactiae* infections.
iv) Drying off early/cull high SCC cows - SEEK VETERINARY ADVICE FIRST.
v) Milking low SCC cows first, especially heifers. High SCC cows carry infection so milk them last to avoid transferring infection to low SCC cows at milking.
vi) Targetting milk. In emergencies avoid putting milk from the highest ICSCC cows into the tank, perhaps by feeding to calves.

Finally, Do **NOT** ignore the basics - these elements must be seen as adjuncts to the proper application of the "Five Point Plan".

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IS THERE A ROLE FOR THE CALIFORNIA MASTITIS TEST?

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The California Mastitis Test (CMT) has been in use for many years to help to determine if any individual quarter of a cow may be infected. It allows an indirect relative assessment of cell count. However, the CMT is not very useful within a few days of calving or near to drying off, as the cell count increases naturally in these periods.

The CMT can be a useful tool where mastitis clots have been seen on an in-line mastitis detector but no clots are found in the quarter milk and no other signs are found.

The display presented aims to update the use of this simple piece of apparatus in investigating a mastitis problem.

MACHINE MILKING AND TEAT CONDITION

ALISTAIR McIVOR Milking machine specialist, 24 Meadow Close, Kempsey, Worcester

Modern milking is a sensitive relationship between the cow and the milking machine. Field and experimental evidence clearly demonstrates the problems that can arise when milking equipment is not operating correctly. Static milking machine tests will highlight any operational deficiences according to defined standards. It can also be important to observe the changes in teat condition before and after milking and also over subsequent lactations.

This poster details on observations that may be carried out when inspecting milking. Observations may be made on preparation of the teats for milking, machine attachment, unit removal and the post milking routine of the milker. During the actual milking, tests can be made on the machine to measure pulsation and vacuum changes when the units are removed and attached, to determine liner slip and other extraneous happenings in a particular milking system.

THE VALUE OF EMOLLIENTS IN TEAT DISINFECTANT

MARTIN F H SHEARN, Institute of Animal Health, Compton, Newbury, RG20 7NN

Teat disinfectants first appeared on the market in the mid 1960's, following trials at the National Institute for Research in Dairying (1). These disinfectants were based primarily on udder wash formulations and did not contain emollients. If teats were affected by lesions glycerin was often added. The commonest emollients now in use are glycerin, lanolin and sorbitol. They act differently but all help to maintain teat skin condition.

Later trials showed that the optimum level of emollient is around the 10% (v/v) (2). The level of emollient necessary to keep teat skin in excellent condition varies widely between herds. Whereas 5% emollient would be sufficient during the summer it may not be not high enough in the winter. When management/weather conditions change so may the need for a different emollient level. The condition of the teats, soft and supple, needs to be regularly appraised and the formulation of the dip adjusted as necessary. This does not necessarily mean a change of teat disinfectant!

References

1. SHEARN M F H, KINGWILL R G, GRIFFIN T K, WESTGARTH D R, NEAVE, F K & KING J S NIRD Annual Report (1973-74) p57.

2. GODINHO K S & Bramley A J (1980) Journal of Applied Bacteriology 48 449-455.

HYPERKERATOSIS OF THE TEAT ORIFICE - UPDATE FARM B 1994

MARTIN F H SHEARN, Institute for Animal Health, Compton, Newbury, Berks. RG20 7NN

In early 1994 hyperkeratosis levels were observed to have increased dramatically in a herd. A further visit to the herd in October 1994 showed a return to more normal levels of hyperkeratosis.

Orifice hyperkeratosis (%) on Farm B

Month	No.of teats	% orifice hyperkeratosis score					
		0	1	2	3	4	5
Apr 93	480	47.8	18.1	25.6	7.5	0.2	0.6
Aug 93	456	56.6	20.2	20.2	2.6	0.4	0
Oct 93	497	57.7	16.9	22.1	2.2	1.0	0
Apr 94	**432**	**34.9**	**19.3**	**36.8**	**7.8**	**1.2**	**0**
Oct 94	428	53.3	21.7	20.1	4.2	0.7	0

* 0 = normal 1 = slight 2 = moderate 3 = moderate to severe
4 = severe 5 = very severe

The marked deterioration of the teat orifice was probably related to a failure of the vacuum controller at the end of December 1993 for **over two weeks** resulting in excessive vacuum. Some of the apparent improvement in hyperkeratosis was probably due to the influence of heifers joining the herd and a number of cows going through a dry period.

Footnote: No full milking machine test has been carried out on this herd for many years!

HOUSING MANAGEMENT

J R BAINES, ADAS National Milking Technology Specialist, Lawnswood, Leeds

Mastitis pathogens such as *E. coli* and *S. uberis* survive in the cows' environment. The management of housing and loafing areas are important in reducing the teat-end challenge from these organisms.

Poor design causes stress to cows. Bad management produces wet bedding and this in turn increases the teat-end challenge from mastitis bacteria. Both of these factors lead to increased levels of mastitis incidence.

Cows with dirty teats require increased preparation time before milking. This upsets the milking routine, which in turn may lead to overmilking, if automatic cluster removers are not in use. An increase in TBC and milk sediment may well follow where udder preparation is inadequate.

Buildings for dairy cows should be well ventilated, draught free and provide comfortable and dry lying areas. Lying areas should be well covered with clean dry bedding. Excreta should be removed frequently.

RISK FACTORS FOR SUMMER MASTITIS

J ERIC HILLERTON, MARTIN F H SHEARN & JONATHAN WEST, Institute for Animal Health, Compton Laboratory, Newbury, Berks. RG20 7NN

A series of data on the management, disease patterns, structure and environment of paired, adjacent farms which either 'regularly' or 'rarely' experienced summer mastitis was collected. The data were analysed to determine if any particular factors were related to the occurrence of isolated or initial cases of summer mastitis. These cases appear to occur very much at random. The study emphasised the positive control achieved by use of dry cow therapy to prevent summer mastitis. The major factor(s) related to epidemics of summer mastitis (2 or more cases linked in time) appear to be farm efficiency and the quality of management. Farms selling milk of low cell count and low TBC, and close to quota suffer less summer mastitis.

MAJOR HISTOCOMPATIBILITY COMPLEX CLASS II EXPRESSION ON MILK MONONUCLEAR CELLS DIFFERS IN COWS WITH LOW OR HIGH SOMATIC CELL COUNTS

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[1]Department of Veterinary Medicine, [3]Department of Veterinary Pathology, University of Glasgow Veterinary School, Bearsden, Glasgow and [2]University of Paisley

Milk somatic cells comprise of neutrophils, macrophages, lymphocytes and epithelial cells. The single most important factor associated with high somatic cell count is the presence of intramammary infection and neutrophils account for the greatest proportion of cells present in infected mammary secretions. However, macrophages have been shown to be capable of presenting antigen to T lymphocytes, and these T lymphocytes, in turn, play a central role in orchestrating the immune response. It is possible that high cell count cows which do not completely clear intramammary infections, have some deficiency in these mononuclear cell (macrophage and lymphocyte) populations. The aim of this work was to compare the numbers of milk mononuclear cells isolated from high and low cell count cows and to determine if the expression of the Major Histocompatibility Complex (MHC) class II molecule, which is involved in antigen recognition by T-helper lymphocytes, differs on the mononuclear cell population in the two groups of cows.

Mononuclear cells were isolated from milk by density centrifugation and high cell count cows (>400,000/ml milk for at least three consecutive monthly samples) were found to have a similar number of mononuclear cells per ml of milk to low cell count cows (<150,000/ml milk). Milk mononuclear cells expressing MHC class II were identified by immunofluorescence and analysed by flow cytometry. Milk from the high cell count group had a significantly greater number of MHC class II expressing cells and a significantly higher intensity of fluorescence on cells from the high cell count group than on cells from the low cell count group. These results suggest that the inability of immune defences to eliminate intramammary infection in high cell count cows is not a result of lack of cells expressing MHC class II in the local mammary gland environment.

COWS WITH CHRONIC INFECTIONS OF THE MAMMARY GLAND WITH *STREPTOCOCCUS UBERIS* MOUNT LITTLE INFLAMMATORY RESPONSE AND HAVE AN INTACT HUMORAL IMMUNE RESPONSE.

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Eight cows with a history of recurrent mastitis associated with infection with *Streptococcus uberis* were examined histologically (6 cows) and serologically (4 cows).

The mild histological reaction comprised: infiltration of the interstitial mammary tissue with lymphocytes, macrophages and plasma cells, a few focal areas of neutrophil exudate in acinae, prominent and numerous corpora amylaceae, vacuolated ductular and secretory epithelium and widespread involution. *S. uberis* was identified by immunoperoxidase labelling in several locations including macrophages in the interstitial spaces, free or phagocytosed in acinae, penetrating the secretory or ductular epithelium and occasionally in the cytoplasm of single ductular "epithelial" cells. In two cows *S. uberis* was located in the interfollicular areas of the supra mammary lymph nodes. A lack of inflammatory response associated with immunolabelled cocci was noted.

Mean specific serum IgG2 levels by ELISA in the four cows examined were enhanced (16635.5 $\pm$ 8855.2) when compared with control cows (3020.2 $\pm$ 1263.5).

From these limited observations it was concluded that the mild histological reaction was in sharp contrast to earlier experimental studies in naive animals (1) and that a state of tolerance appeared to exist between these animals and the resident bacteria. The failure to eliminate the bacteria was not due to an impaired humoral response.

References

1. THOMAS L H, HAIDER W, HILL A W & COOK R S (1994). American Journal of Veterinary Research 55, 1723-1728.

Applied research at the Institute for Animal Health

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