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MASTITIS IN HEIFERS: PREVALENCE, STRATEGY FOR CONTROL DURING THE PERIPARTURIENT PERIOD, AND ECONOMIC IMPLICATIONS

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SUMMARY

Intramammary infections in breeding age and pregnant heifers are much higher than previously thought. Many of these infections can persist for long periods of time, are associated with elevated somatic cell counts, and likely impair mammary development during gestation and affect milk production after calving. Prepartum intramammary antibiotic infusion of heifer mammary glands a few weeks before calving is an effective procedure for eliminating many infections in heifers during late gestation and for reducing the prevalence of mastitis in heifers both during early lactation and throughout lactation. Prepartum antibiotic-treated heifers produced significantly more milk and had significantly lower somatic cell count scores than untreated control heifers. These observations are likely associated with or due to the lower prevalence of mastitis pathogen isolation in prepartum antibiotic-treated heifers throughout lactation. Prepartum antibiotic treatment to reduce the rate of mastitis in heifers during early lactation was economically beneficial. Prepartum antibiotic-treated heifers produced 531 kg more milk than the untreated control group. Multiplying this increase by a milk price of $0.348/kg yielded a $184.54 (£122) per-heifer increase in gross revenue. Subtracting the cost of treatment from gross revenue (including the cost of testing for antibiotic residues), the net revenue from the actual production increase amounted to $174.92 (£115) per heifer.

INTRODUCTION

Intramammary infections (IMI) in breeding age and pregnant heifers are much higher than previously thought. Many of these infections can persist for long periods of time, are associated with elevated somatic cell counts (4, 21), and may impair mammary development (21) and affect milk production after calving. Until recently, little attention has focused on methods of controlling mastitis in heifers. Consequently, there are few management practices for controlling mastitis in heifers supported by research data. The purpose of this communication is to review literature published on the prevalence of mastitis in heifers and to share our findings on heifer mastitis, strategies for control, and economic implications based on research conducted at The University of Tennessee.

PREVALENCE OF INTRAMAMMARY INFECTIONS IN HEIFERS

Intramammary infections in unbred and pregnant heifers were once thought to be very low. However, it has been shown (9) that a high percentage of pregnant heifer mammary glands were infected during late gestation, at calving and during early lactation. During the last decade, several
additional studies on the prevalence of mastitis in heifers have been published. All of these studies suggest that IMI in heifers during the prepartum period occur frequently. Marked herd variations in the rate of IMI and types of pathogens causing IMI have been reported (2, 7, 8, 10, 11, 12, 19, 20, 21).

The prevalence of IMI in unbred heifers and heifers during different stages of pregnancy may be high (21). Unbred heifers had a higher percentage (86.7%) of infected quarters compared with the overall mean for pregnant heifers (70%). *Staphylococcus* species were observed most frequently and 8 different species were isolated. The three most common species isolated from unbred and pregnant heifer mammary glands were *Staphylococcus chromogenes*, *Staphylococcus hyicus* and *Staphylococcus aureus*. Coagulase-negative *Staphylococcus* species (CNS) accounted for 67.4% of bacteria isolated.

Mammary secretions from infected mammary glands had significantly higher somatic cell counts than secretions from uninfected quarters. In addition, tissue from mammary glands of unbred heifers infected with CNS exhibited greater leukocyte infiltration and increased connective tissue compared with tissue from uninfected mammary glands (22). Thus, infection of heifer mammary glands by mastitis pathogens can occur at a very early age and some of these infections may impair mammary growth and development and influence future milk production.

A study to determine the prevalence of mastitis and types of pathogens causing IMI in pregnant heifers prior to calving and during early lactation was conducted in a herd that was *Streptococcus agalactiae*-negative and had a low prevalence of *S. aureus* (14). This pattern of infection would be typical of many dairy herds that practice postmilking teat disinfection and antibiotic dry cow therapy. Heifers (n=115) were sampled 7 days before expected calving, and 3 (C+3) and 10 (C+10) days after calving. About 90% of heifers and 61% of quarters were infected during the prepartum period. The majority of IMI (243 of 279) were due to CNS. This is higher than what we observed previously in a study conducted in another herd (9, 11), but types of mastitis pathogens isolated were similar. Trinidad et al. (1990a) also observed considerable herd-to-herd variation both in prevalence of IMI and mastitis pathogens causing IMI in unbred and pregnant heifers. For example, in one herd 44.3% of quarters were uninfected, 12.3% were infected with *S. aureus*, 41.5% were infected with CNS and 1.9% were infected with streptococci other than *St. agalactiae*. In another herd, 17.6% of quarters were uninfected, 23.1% were infected with *S. aureus*, 49.5% were infected with CNS and 9.9% were infected with *Streptococcus* species. In our studies (9, 10, 14, 15), CNS were isolated most frequently followed by environmental mastitis pathogens primarily *Streptococcus* species.

In one study approximately 46% of heifers and 19% of quarters were infected during early lactation based on duplicate samples obtained from 382 heifers within 3 days after calving (19). Coagulase-negative *Staphylococcus* species were the most prevalent bacteria isolated and were found in 22.8% of heifers and 11.4% of quarters. Another study (7) indicated that 35.5% of colostrum samples were positive for 7 different *Staphylococcus* species. Species isolated most frequently were *S. chromogenes*, *S. aureus* and *Staphylococcus simulans*. *Staphylococcus* species were isolated from
about 18% of heifer mammary glands weekly for the first 5 wk of lactation. Some 19.7% of heifer mammary glands (59 of 300) were infected at calving. CNS caused 71.2% of these IMI in another study (11). During early lactation, 15.7% of heifer mammary glands (47 of 300) were infected and 48.9% were due to CNS. Thus, the number of quarters infected with CNS decreased significantly from calving to early lactation suggesting that some CNS isolated from heifer mammary glands were either colonizing the teat duct and subsequently eliminated as a result of the milking procedure or that a high rate of spontaneous elimination occurred. Similar findings were reported in multiparous cows (5, 12).

A large survey of 28 dairies in four states was conducted to determine the prevalence of IMI in unbred and pregnant dairy heifers and to determine potential factors that influenced herd variation (2). Most IMI were due to CNS and S. aureus. Location, herd, season, and trimester of pregnancy significantly influenced prevalence of IMI in heifers. Heifers in the third trimester of pregnancy had the highest prevalence of IMI. Thus, based on all studies reported thus far, CNS will likely cause the majority of IMI in unbred and pregnant heifers and variation in the prevalence of CNS IMI in heifers should be expected among herds.

In our studies, 8 to 10% of heifer mammary glands (10, 14 15) were infected with major mastitis pathogens near calving. Most major pathogen IMI were caused by environmental mastitis pathogens, primarily Streptococcus species, which was consistent with the pattern of IMI in lactating cows in these herds (13). Conversely, other studies (2, 21) indicated that S. aureus was the most prevalent major mastitis pathogen isolated from unbred and pregnant heifer mammary glands. Differences in the incidence of IMI and types of bacteria causing IMI in pregnant heifers is likely due to the prevalence of mastitis pathogens in the herds evaluated. Thus, it is reasonable to assume that heifers from herds with a high prevalence of contagious mastitis will likely be infected predominantly by contagious mastitis pathogens such as Streptococcus agalactiae and S. aureus. Similarly, environmental mastitis pathogens such as Streptococcus uberis and Gram-negative bacteria such as Escherichia coli will likely be the predominant major pathogens isolated from heifer mammary glands from herds with an environmental mastitis problem.

CONTROL OF MASTITIS IN PREGNANT HEIFERS

Little research has focused on management strategies for controlling mastitis in heifers. Evidence suggests that IMI in pregnant heifers occurs frequently and that some infections may be detrimental to mammary gland development and influence subsequent lactational performance. Methods of controlling mastitis in heifers may eliminate or markedly reduce the deleterious effects of prepartum infections. One common denominator of all studies on heifer mastitis is the high prevalence of CNS IMI. It has been demonstrated (24) that 90% of 311 staphylococcal isolates (primarily CNS) from heifer mammary glands were susceptible to antibiotics in vitro. Some variability in antimicrobial susceptibility of bacteria obtained within and among herds was noted; however, in general, bacteria were highly susceptible to all antibiotics evaluated. The minimum inhibitory concentrations of penicillin, cloxacillin, cephalin, ceftiofur, novobiocin, enrofloxacin, erythromycin and pirlimycin against 1494 microorganisms isolated from heifer mammary glands
have been determined (25). The majority of *Staphylococcus* species were susceptible to the antimicrobial agents evaluated. However, antimicrobial susceptibility was variable for *Streptococcus* species and poor against Gram-negative enteric organisms. These data suggest that antibiotic therapy may be an effective means of eliminating *Staphylococcus* species IMI that have been shown to cause the majority of IMI of heifer mammary glands.

**A SIMPLE AND EFFECTIVE METHOD FOR CONTROLLING MASTITIS IN HEIFERS**

Our initial study to determine if prepartum infusion of lactating cow antibiotic preparations into heifer mammary glands influenced rates of IMI during early lactation was published almost ten years ago (14). Pregnant Jersey heifers (n=115) from The University of Tennessee Dairy Experiment Station research herd at Lewisburg were assigned alternately to three treatment groups as follows: group 1 (n=41) - no intramammary antibiotic infusion (negative control), group 2 (n=38) - intramammary infusion of all quarters with 200 mg sodium cloxacinil (Beecham Laboratories, Bristol, TN) 7 days before expected parturition, and group 3 (n=36) - intramammary infusion of all quarters with 200 mg cephalixin sodium (Bristol Myers, Evansville, IN) 7 days before expected parturition.

During the winter months, heifers were housed in loose housing and bedded on sawdust. In the spring, summer and fall, heifers were maintained on pasture. After calving, heifers were milked in a 12-stall trigon parlor. Milking machines were backflushed after removal and all teats of heifers were dipped with an effective postmilking teat disinfectant after milking machine removal. Lactating heifers were housed in free stalls bedded with dairy waste solids separated from a manure slurry.

Samples of mammary secretion for microbiologic evaluation were collected from all quarters of heifers in duplicate at 7 days before expected calving (C-7), and single quarter samples were obtained at 3 (C+3), 10 (C+10), C+11-30, C+31-90, C+91-150, C+ 151-240, C+241-475 days after calving and at the last milking of lactation immediately before drying off. A quarter was considered infected during the prepartum period if the same mastitis pathogen was isolated from duplicate samples obtained 7 days before expected calving. A quarter was considered infected during early lactation if the same mastitis pathogen isolated before treatment was present in samples obtained at 3 or 10 days after parturition. Differences in the percentage of heifers and quarters infected in control and antibiotic treated groups during early lactation were determined by Student's *t*-test. Microbiological data were also expressed as percent of samples containing major pathogens, minor pathogens, and percent of samples bacteriologically negative at each of the above time periods.

Almost 90% of heifers were infected 7 days prior to expected calving (Fig. 1). During early lactation, 78% of control heifers and 44.5% of quarters were infected. In contrast, 17.6% of antibiotic-treated heifers and 5.4% of antibiotic-treated quarters were infected during early lactation (Fig. 1). Fewer (*P < 0.001*) antibiotic treated heifers and quarters were infected during early lactation than in controls. Intramammary antibiotic therapy before calving was highly
effective \((P < 0.001)\) against CNS (Figs. 2 and 3). It should be noted, however, that 24 of 88 (27.4\%) CNS IMI in control heifers were not detected during early lactation suggesting a high rate of spontaneous elimination. Nine of 14 major pathogen IMI in control heifers and 3 of 22 major pathogen IMI in antibiotic treated mammary glands of heifers persisted into early lactation. Differences in major pathogen IMI between antibiotic treated and controls during early lactation were significant \((P < 0.025)\).

Mastitis pathogens were isolated from 76\% of samples obtained from untreated control quarters 7 days before expected calving, 47\% of samples obtained 3 days after calving, and 29\% of samples obtained 10 days postpartum. Throughout the remainder of lactation, mastitis pathogens were
isolated in about 30% of control quarters. A similar percentage of samples (70%) was positive for mastitis pathogens at C-7 prior to antibiotic treatment. However, only 8% of samples obtained at 3 days after calving and 4% of samples obtained 10 days postpartum from quarters of antibiotic-treated heifers contained mastitis pathogens. Throughout the remainder of lactation, mastitis pathogens were isolated from an average of about 11% of quarters. Percent of samples with mastitis pathogens was higher in untreated controls than in antibiotic-treated quarters at most sampling intervals during lactation. *Strep. uberis, Streptococcus dysgalactiae* and coagulase-negative *Staphylococcus* species were isolated most frequently in both untreated controls and antibiotic-treated heifer mammary glands.

**Figure 3. Percent quarters infected and pathogens causing infections in heifers before and after antibiotic therapy.**

**ANTIBIOTIC RESIDUES IN MILK FOLLOWING PREPARTUM TREATMENT**

One disadvantage of prepartum antibiotic administration for controlling mastitis in heifers is the potential for antibiotic residues in milk. This is especially important if heifers calve sooner than expected. To address this concern, samples of mammary secretion from all quarters of 98 heifers were collected at the first and sixth milking after calving and at 10 days after calving for antibiotic residue analysis. Samples were analyzed qualitatively for antibiotic residues by the *Bacillus stearothermophilus* disc assay (18). Zones of inhibition > 16 mm in diameter were interpreted as positive for antibiotic residues. Sensitivity of the *B. stearothermophilus* disc assay for detection of cephaparin and cloxacinil has been reported to be 0.025 μg/ml and 0.031 μg/ml, respectively (1, 3).
About 17% of colostrum samples from heifer mammary glands infused with cloxacillin were positive for antibiotic residues by the *B. stearothermophilus* disc assay (Fig. 4). The majority of positive samples were from heifers that calved within 5 days of treatment. Only 4 of 88 samples obtained at the first milking after parturition were positive for antibiotic residues if intramammary infusion of cloxacillin occurred ≥ 7 days before parturition. All samples obtained 3 days after parturition, the time when milk would likely be marketed for human consumption, were negative for antibiotic residues. Thus, the cloxacillin formulation used in the present study should not result in antibiotic residue problems in marketable milk even if heifers calve earlier than expected.

In contrast, antibiotic residues were detected frequently during early lactation in samples from heifer mammary glands infused with cephamiprin. Almost 85% of colostrum samples and 28.2% of samples obtained 3 days after parturition were positive for antibiotic residues (Fig. 4). Marked variability between time of antibiotic treatment and parturition with persistence of antibiotic residues was observed. For example, two heifers calved 8 days after treatment and all samples obtained 3 days after parturition were negative for residues. Conversely, 4 heifers calved 10 days after cephamiprin treatment and 6 of 16 samples were positive for antibiotic residues. All samples (n=24) from 6 heifers obtained 3 days after calving were negative for antibiotic residues if intramammary infusion of cephamiprin occurred ≥ 11 days before calving. Thus, it would appear that antibiotic treatment of heifer mammary glands earlier in gestation may be advantageous from an antibiotic residue standpoint. However, the timing of antibiotic treatment and subsequent persistence of antibiotics in mammary secretions following treatment could impact efficacy.

We conducted another study to determine if antibiotic treatment of heifer mammary glands earlier in the prepartum period reduced the occurrence of residues in milk (15). In this study, 82 Jersey heifers were assigned randomly to two groups: 1) negative control (n=42) and 2) intramammary infusion of 200 mg cephamiprin sodium (n=40) 14 days prior to expected calving. Mammary secretions were collected 14 days before calving, and at the first and sixth milking after calving and were analysed for residues by the *B. stearothermophilus* disc assay. Sixty of 150 samples (40%) from cephamiprin treated quarters were positive at the first milking after calving (Fig. 5). However,
only 4 of 127 samples (3.1%) obtained from antibiotic treated quarters at the sixth milking after calving were positive and 3 of the 4 positive samples were from a heifer that calved within 3 days of treatment. Thus, as observed in our earlier experiment (14), the interval between prepartum antibiotic treatment and calving was related to persistence of residues during early lactation. Intramammary infusion of antibiotics earlier in the prepartum period reduced the occurrence of residues in milk during early lactation.

Mastitis pathogens were isolated from 67% of samples obtained from control mammary glands 14 days prior to expected calving, 56% of samples obtained 3 days after calving and 36% of samples obtained 30 days postpartum (Fig. 6). A similar percentage of samples (64%) were positive for mastitis pathogens prior to antibiotic treatment. However, only 16% of samples obtained at 3 days after calving and 8% of samples obtained 30 days postpartum from quarters of antibiotic-treated heifers contained mastitis pathogens (Fig. 6). Mammary secretions were also collected from antibiotic-treated and untreated control heifers throughout lactation and at the last milking of lactation immediately before drying off for microbiological evaluation (16). Throughout the
remainder of lactation, mastitis pathogens were isolated from about 45% of quarter samples from untreated control heifers (Fig. 7). Conversely, mastitis pathogens were isolated from an average of 12% of antibiotic-treated quarters throughout lactation (Fig. 7). Percent of samples with mastitis pathogens was higher in untreated control quarters than in antibiotic-treated quarters at every sampling interval during lactation. Coagulase-negative staphylococci were isolated most frequently followed by environmental mastitis pathogens. Bacteriological results during early lactation were similar to what we observed in our earlier work (14).


datasources:
- 14: Yellow Fever Institute
More recently, we conducted a study to determine if prepartum therapy of heifer mammary glands with penicillin-novobiocin (Pharmacia Upjohn, Kalamazoo, MI) or pirlimycin hydrochloride (Pharmacia Upjohn, Kalamazoo, MI) was effective for reducing the percentage of heifers and quarters infected with mastitis pathogens during early lactation (17). Almost 73% of Holstein heifers (40 of 55) and 34.3% of quarters (73 of 213) were infected 14 days before expected calving. Of the quarters infected at 14 days before expected parturition, 76% (19 of 25) became uninfected following treatment with penicillin-novobiocin; 59% (17 of 29) were uninfected following treatment with pirlimycin, and 26% (5 of 19) were uninfected in the untreated negative control group. The majority of IMI in Holstein heifers were due to coagulase-negative staphylococci (44%) and S. aureus (30%). Almost 96% of Jersey heifers (67 of 70) and 71.3% of quarters (199 of 279) were infected 14 days before expected calving. Of the quarters infected at 14 days before expected parturition, 75% (54 of 72) became uninfected following treatment with penicillin-novobiocin; 87% (61 of 70) were uninfected following treatment with pirlimycin, and 56% (32 of 57) were uninfected in the untreated negative control group. The majority of IMI in Jersey heifers were due to coagulase-negative staphylococci (61%), Streptococcus species, primarily Str. uberis (19%) and S. aureus (8%). Prepartum therapy of heifer mammary glands with penicillin-novobiocin or pirlimycin hydrochloride was an effective procedure for significantly reducing the percentage of heifers and quarters infected with mastitis pathogens during early lactation. Studies are underway to determine the influence of penicillin-novobiocin or pirlimycin hydrochloride prepartum therapy on lactational performance and milk quality.

It has been demonstrated that intramammary infusion of an antibiotic formulation for nonlactating cows into breeding age and pregnant heifers during different trimesters of pregnancy was effective in reducing the prevalence of mastitis and somatic cell counts at parturition (23). However, efficacy of prepartum antibiotic therapy at 7 or 14 days prior to expected calving in our studies (14, 15) was considerably higher than that reported from Louisiana (23). This could be due, in part, to the time when heifers were treated with antibiotics, differences in the pathogens causing IMI, and the time when IMI occur. In support of this hypothesis, it has been indicated that the prevalence of heifer IMI was highest during the last trimester of pregnancy (2). Thus, methods of controlling mastitis in heifers would likely be more effective if administered during the last trimester of pregnancy as opposed to early gestation.

INFLUENCE OF PREPARTUM INTRAMAMMARY ANTIBIOTIC TREATMENT OF HEIFERS ON LACTATIONAL PERFORMANCE: ECONOMIC IMPLICATIONS

We were also interested in determining the influence of prepartum antibiotic treatment on subsequent lactational performance of heifers. Milk production and somatic cell count score data from 82 control heifers and 111 heifers treated with antibiotics before calving were evaluated and data are presented in Table 1. Mean 305-day milk production was significantly higher in heifers treated with antibiotics. Heifers treated with antibiotics before calving had a significantly lower somatic cell count score than control heifers (2.63 vs. 2.04).
Table 1. Lactational performance of antibiotic-treated and control heifers.

<table>
<thead>
<tr>
<th>Treatment group</th>
<th>Milk production (kg)</th>
<th>Somatic cell count score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (n=82)</td>
<td>Actual: 5195</td>
<td>305-day: 5005</td>
</tr>
<tr>
<td>Treated (n=111)</td>
<td>5726*</td>
<td>5464*</td>
</tr>
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</table>

* Significantly different (P<0.05).

Prepartum antibiotic treatment to reduce the rate of mastitis in heifers during early lactation was economically beneficial (6). Prepartum antibiotic-treated heifers produced 531 kg more milk than the untreated control group. Multiplying this increase by a milk price of $0.3476/kg yielded a $184.54 (£122) per heifer increase in gross revenue. Subtracting the cost of treatment from gross revenue, the net revenue from the actual production increase amounted to $174.92 (£115) per heifer. These net revenue figures included the cost of testing for antibiotic residues. The relationship between net revenue increases and the increase in milk produced due to treatment, given a wage rate of $6.25 (£4.13)/h and a milk price of $0.3476/kg was determined. Treatment would be profitable as long as the increase in milk production is greater than 27.7 kg (6).

CONCLUSIONS

Intramammary infections in breeding age and pregnant heifers are much higher than previously thought. Many of these infections can persist for long periods of time, are associated with elevated somatic cell counts, and likely impair mammary development during gestation and affect milk production after calving. Prepartum intramammary antibiotic infusion of heifer mammary glands a few weeks before calving is an effective procedure for eliminating many infections in heifers during late gestation and for reducing the prevalence of mastitis in heifers both during early lactation and throughout lactation. Prepartum antibiotic-treated heifers produced significantly more milk and had significantly lower somatic cell count scores than untreated control heifers. These observations are likely associated with or due to the lower prevalence of mastitis pathogen isolation in prepartum antibiotic-treated heifers throughout lactation. Prepartum antibiotic treatment to reduce the rate of mastitis in heifers during early lactation was economically beneficial. Prepartum antibiotic-treated heifers produced 531 kg more milk than the untreated control group. Multiplying this increase by a milk price of $0.348/kg yielded a $184.54 (£122) per-heifer increase in gross revenue. Subtracting the cost of treatment from gross revenue (including the cost of testing for antibiotic residues), the net revenue from the actual production increase amounted to $174.92 (£115) per heifer.
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REFERENCES


A PRACTISING VETS APPROACH TO THE HIGH CELL COUNT HERD

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SUMMARY

Dairy farmers currently use a variety of approaches to dealing with a high bulk tank cell count. Simply they may only consider each case of clinical mastitis individually, have some form of compliance with the five-point mastitis control plan or increasingly adopt a plan of strategic management. The cattle vet has a major role to play in the more sophisticated approaches need to plan mastitis control and to manage milk quality. The main component of the approach must remain the five-point mastitis control plan but with stricter attention to limiting the duration of infections and targeting of treatment for new infections. The vet now has many tools to deploy from obtaining good data to help understand the farm and its problems, easier identification of causative bacteria and introduction of a preventive medicine approach as part of a cattle health plan tailored to the individual farm.

INTRODUCTION

Udder health is central to profitable milk production. Not only are there significant losses from poor udder health but there are now significant penalties for failing to achieve particular levels of cell count. In recent years, the somatic cell count target levels have reduced and many producers are now penalised at levels as low as 150,000 cells/ml. Ten years ago many of many clients were comfortable with cell counts of 400-600,000 cells/ml. In 1990 33% of the national herd had average herd cell counts greater than 400,000 cells per ml. In October 1991 a penalty of 0.2 p/l was imposed on producers exceeding 700,000 cells/ml and a bonus was payable of 0.2p/l for milk quality better than 400,000 cells/ml (1). This improved milk quality and 2 years later only 26% of producers were producing milk with a bulk supply cell count above 400,000 cells/ml. (2). The penalties increased to 0.5 and 2.0 p/l for exceeding the thresholds of 400,000 and 1,000,000 cells/ml respectively. By 1996 the Milk Marque penalty for milk exceeding 400,000 cells/ml had increased to 2.0 p/l (3). Since introduction of the European Milk Directive, 92/46/EEC, milk with a cell count greater than 400,000 cells/ml is not acceptable for human consumption. The current penalties have now increased further with market place split between first purchasers pursuing a low somatic cell count milk with penalties ranging between 0.8 p/l to 1.8 p/l for slipping from 150,000 cells/ml to greater than 200,000 cells/ml. Other purchasers have a more lenient view on somatic cell count penalties and typically the penalty loss from below 250,000 to 350,000 cells/ml would be 0.2 p/l. Nearly all first purchasers levied a penalty of 1-10 p/l for somatic cell counts greater than 400,000 cells/ml. With this economic background the control of udder health has never been more important and the margin for error is minimal. The aim of this paper is to give a practising vet view of udder health control within the current standards and marketplace.

THE PROBLEM OF CONTROLLING UDDER HEALTH - A VETS VIEWPOINT

Not all clients will telephone the practice to seek advice when they develop a high cell count problem in the herd. Many farmers are not willing to accept defeat and often persevere with
managing a disease problem alone before eventually calling for help. All my clients have access to bulk cell counts and the majority will use individual cell counts on a monthly basis.

Mastitis has, typically, been poorly recorded in the past and more than half of the more keen dairy farmers have not had a system in place to record mastitis incidence and treatments. This is now changing and with the introduction of the BCVA Herd Health plan (3) and National Dairy Farm Assurance schemes this will be largely rectified.

The consequence of poor mastitis control and recording can be the development of a “shoot or treat” mentality to udder health control. The cowman, when presented with a problem cow, makes an “urgent” decision to treat the immediate problem on a case by case basis. Rarely is there a more measured approach to prevent future cases in the herd. This is not unique to farmers and the vet may also fall into the trap of treating the urgent clinical case and not initiating a proper preventive investigation due to pressures of clinical work.

The incidence of mastitis varies enormously within my client base with the annual incidence varying between herds from 5 to 100 cases/100 cows/year. In all these herds the bulk cell count will be less than 400,000 cells/ml. In some of the problem herds the overall bulk milk cell count is managed by increasing the cull rate by “brinkmanship culling” i.e. culling out high cell count cows with a high individual contribution to the bulk tank or not putting the cows’ milk into the bulk tank. In other herds the cell count may not be an issue as the problem is one of high incidence and low cell count. In my experience culling criteria change with the level of the bulk cell count and clients, when asked “what criteria you use to cull cows”, often answer that “it depends on the herd cell count”.

Good udder health depends on performing 20 or so small tasks correctly every day 365 days a year. Some farm systems achieve these standards easily. On others as litres sold per man and cow susceptibility to new infections increase maintaining udder health to current standards can be problematic.

A TYPICAL AD HOC APPROACH.

In the majority of herds the main method of mastitis control remains the 5-point plan. This is applied with varying degrees of efficiency and success. Even 30 years after the plan was devised it is not uniformly understood. Multiple use udder cloths are still used in some herds and these clients can be difficult to convince of the benefits of the 5-point plan. In herds with diligent stockmen and well-maintained housing and plant udder health is not a major problem. However, achieving a consistently low bulk milk cell count, below 150,000 cells/ml, is a challenge as a single missed case of clinical mastitis may put the herd bulk milk SCC above the bonus band. High levels of culling and treatment can become the norm rather than be seen as broadly unacceptable and requiring of investigation. This is largely due to a conspicuous failure of the dairy farmer to understand the true costs of mastitis and a false perception of high costs of investigation (typically around 0.02 p/l compared to the financial losses of more than 0.5 p/l).
Treatment of clinical and sub clinical cases is usually based on previous responses to a particular antibiotic preparation or historic bacteriological investigations and seldom based on individual culture on a planned basis.

Currently most intramammary tubes are licensed to be used as a short duration treatment with the aim to reduce milk losses rather than produce a high cure rate during lactation, especially to pathogens such as *Streptococcus uberis* and *Staphylococcus aureus*.

Increasingly sub-clinical cases may be treated based on individual quarter or cow cell counts and/or California Milk Tests without reference to bacteriology on grounds of expense. Again the focus can be over reliance on therapy rather than prevention.

**THE STRATEGIC APPROACH**

In order to tackle a problem cell count herd the 5-point plan is still the main basis of the investigation. The broad aims are:

1. Prevent new infections. Rigorous implementation of the 5-point plan and attention to environment.

2. Remove existing infections from the herd/bulk tank. This is achieved by culling, early drying off, or removing the cow from the milking herd, for treatment or to suckle calves.

3. Treat existing infections more effectively. Improved lactation and dry cow therapy. Both 2 and 3 will reduce the exposure to infection of susceptible cows and in turn reduce the new infection rate.

**THE TACTICS**

The method of investigation has been covered in more detail in previous papers (5,6,7,8,9) The approach I use is via a questionnaire and the current one in use was developed by five of us in conjunction with Pfizer Animal Health (10).

1. Establish the extent of the problem. This is based on a study of mastitis records, intramammary tube usage and cell count data. The records, combined with knowledge of the farm, will often give strong indications of the areas of weakness.

2. Identify the pathogens. This is central to success. Unless the main pathogens are identified, the strategy cannot be implemented.

3. Perform an in-depth investigation on farm, based on discussions with the farmer, milker and observations at milking.

4. Discuss the results with the farmer and herdsman and agree a list of recommendations that will be applied.
5. Review and monitor progress. Encourage compliance with recommendations on an ongoing basis.

**DIAGNOSING THE BACTERIA INVOLVED IN ELEVATING CELL COUNTS: PRACTICAL CONSIDERATIONS**

- Bacteriology of individual *quarter* samples of all cows having a persistent monthly cell count more than 200,000 cells/ml is the best way to determine the causative bacteria. In many instances this is prohibitively expensive in large herds or herds with high numbers of cows with a high cell count. Complications come if many infections more than one type of bacteria. Intermittent excretion of bacteria can complicate accurate diagnosis when using single samples.

- The numbers can be reduced by selecting the highest 10 cows, or the 10 cows with the most recent new sub clinical infection (i.e. an increased in cell count less than 200,000 to more than 200,000 cells/ml) or by using the CMT test to identify individual quarters.

- Bacteriology of bulk milk samples may give an indication of the organisms present in the herd. Although this may seem to be a “broad brush” approach it has the advantage of screening the whole herd. The results will not allow an action plan for each cow more an overview of the key organisms involved. One advantage of the bulk milk examination is that it provides useful information plant cleaning and general levels of organisms in the milk (11).

- Bacteriology of samples from the ten most recent clinical cases, frozen if necessary and submitted to the lab in bulk, is often most useful. The clinical cases may not always be representative of the bacteria causing the elevated cell counts as low grade infections by some bacteria may not feature as causes of clinical mastitis. Bacteriology on 3 samples or any problem clinical case may not be representative. Determination of the bacteria involved will help to identify appropriate treatment protocols (12,13).

**COMMON PITFALLS OF AN INVESTIGATION**

These are some of the key difficulties that I have experienced when investigating problem herds. This is not comprehensive list.

- **Do not jump to conclusions.** The clinical presentation of mastitis may be different to the cause of high cell counts in the herd. The severe *Escherichia coli* mastitis cases are obvious and the assumption may be made that the key organisms involved in the sub-clinical mastitis are environmental. This may not be the case. A fuller investigation is necessary.

- **What is mastitis?** Carefully discuss with the milker what are his criteria for diagnosing mastitis and what treatment protocol he applies. This may be different to what you and or the farmer think it is. In some instances the change of incidence of mastitis in the herd is more a reflection of a change in criteria of case selection rather than a change in incidence. A milker can become sensitised to the occurrence of clinical mastitis and embark on a campaign of
treatment which will effect results. There may be no system in place to diagnose mastitis save for visual examination of the udder, which has its limitations.

- **Check the tube usage against the clinical records.** This will give an indication of tubes used per case and is relatively easy to do.

- **Clarify the culling policy.** This policy can be an absolute muddle on some farms, ranging from culling on the basis of one or two elevated cell counts to an absence of any culling policy. The policy is often fluid and varies with the number of replacement heifers available rather than rigid scientific criteria. More use of bacteriological cultures prior to deciding to cull may aid the decision process.

- **Liner changes.** Commonly on problem herds the milking machine is taken for granted and there is often not a system in place to ensure that liners are changed at the appropriate interval. Even if the liner life is calculated and agreed putting a system in place to carry out the changes at the required frequency is often difficult. Milking the liner “till it splits” is not the ideal option!

- **There is no substitute for observing milking.** This gives a general overview of what the milking routine and approach is like. Partial teat dipping and spraying is the norm rather than the exception and in many herds the criteria appears to be if there is a drip of teat dip on the end of the teat this is enough. Covering the whole teat per se may not be seen as part of the procedure. Checking the volume of teat dip or spray used is useful.

- **Dry cow therapy.** Establishing how this is done is essential. This can be very rough and ready and the concept of bacteria being introduced at drying off is difficult to explain. Just checking that DCT is in use is not enough.

- **Producing a report.** Written reports and then a verbal follow up is essential. If you are asking someone to change the habits of a lifetime, then you must carefully explain the benefits of the change and why they are necessary. Writing down the recommendations and methods will emphasise the points but is no substitute to discussion.

- **Defining expectations.** Do not promise success quickly. The speed of response will depend on the cause of the problem, the solutions available and the diligence with which they are applied. The aim is not to go for a quick fix but a lasting solution.

- **Encouragement and follow up.** Giving ongoing guidance is part of the solution.

**BACTERIOLOGY OF SUB CLINICAL AND CLINICAL CASES - AN OBSERVATION IN A HERD**

The herd had 380 cows calving all year round, housed on straw yards, an averaging 7500 litres. The straw yards were introduced 2 years earlier to a building designed for cubicles. The straw yards were overcrowded. The rolling bulk tank cell count was 315,000 cells/ml. There was a rapid increase in the incidence of clinical mastitis, to more than 100 cases in 100 cows.
Table 1. Results of clinical cases sampled for culture March–June 2000

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>% Quarters</th>
<th>No. Quarters</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Streptococcus uberis</em></td>
<td>67</td>
<td>21</td>
</tr>
<tr>
<td>CNS</td>
<td>12</td>
<td>4</td>
</tr>
<tr>
<td>Bacillus sp.</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td><em>Corynebacterium bovis</em></td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Escherichia coli</td>
<td>16</td>
<td>5</td>
</tr>
<tr>
<td><em>Streptococcus dysgalactiae</em></td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Totals</strong></td>
<td><strong>31</strong></td>
<td></td>
</tr>
</tbody>
</table>

Initial investigation of 12 clinical cases of mastitis revealed *Str. uberis* in 10 cows, CNS in 4 cases and *Staphylococcus aureus* in one case. The herd was chosen to be involved in a trial run by Mo Milne and Andy Biggs to evaluate the use of pulse therapy on failed initial treatment of clinical mastitis caused by *Str. uberis*. Sampling of clinical cases continued and the results suggested a problem caused by *Str. uberis* (Table 1). A further screen of 113 cows with individual cell counts more than 200,000 cells/ml was performed to establish the pathogens isolated from 452 individual quarters (Table 2).

Table 2. Results of a screen of 113 cows, quarter samples taken from cows with cell counts above 200,000 cells/ml.

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Cows</th>
<th>Quarters</th>
<th>Quarters per cow</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>%</td>
<td>Number</td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td>2</td>
<td>1.8</td>
<td>2</td>
</tr>
<tr>
<td>CNS</td>
<td>69</td>
<td>61.1</td>
<td>197</td>
</tr>
<tr>
<td><em>Streptococcus uberis</em></td>
<td>60</td>
<td>53.1</td>
<td>94</td>
</tr>
<tr>
<td><em>Streptococcus dysgalactiae</em></td>
<td>29</td>
<td>25.7</td>
<td>40</td>
</tr>
<tr>
<td><em>Corynebacterium bovis</em></td>
<td>41</td>
<td>36.3</td>
<td>70</td>
</tr>
<tr>
<td>Coliforms</td>
<td>6</td>
<td>5.3</td>
<td>6</td>
</tr>
<tr>
<td><em>Streptococcus faecalis</em></td>
<td>40</td>
<td>35.4</td>
<td>67</td>
</tr>
<tr>
<td><em>Bacillus spp.</em></td>
<td>59</td>
<td>52.2</td>
<td>114</td>
</tr>
<tr>
<td>No growth</td>
<td>15</td>
<td>13.3</td>
<td>20</td>
</tr>
<tr>
<td><em>Streptococcus agalactiae</em></td>
<td>0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

It is interesting to note the relatively low prevalence of *Str. uberis* from the quarter samples compared to the clinical incidence (20% cases versus 67% quarter isolation rate) and the absence from the 31 cows sampled for clinical mastitis of *Corynebacterium bovis*. *Streptococcus dysgalactiae* occurred in 26% of the quarter samples from sub clinical cases and only 6% of the clinical cases. Coagulase negative staphylococci were mainly associated with sub clinical cases.

Care must be taken in assuming that the pathogen isolated from the clinical sample is representative of all the bacteria present in the udders. Many types of bacteria appear to be
Present at a subclinical level and depending on the bacteria may contribute to the overall bulk tank SCC. This neatly illustrates the difficulty of identifying the causative organism and there is a potential in some herds for the bacteria such as *C. bovis* and CNS to be cause of cell count problems at the bulk tank level of 150,000 cells/ml.

**CONCLUSIONS**

The control of udder infections using the 5-point mastitis control plan is as relevant today as it ever has been. The targets have changed and lower cell counts are now expected. The increasingly high standards of milk quality and medicine usage that farmers have to achieve require complete adherence to the mastitis control strategies.

The key issues vets and farmers need to address, are:

- Increasingly sub clinical udder infections will cause significant financial problems for the farmer as even low-grade may jeopardise the milk price bonus payable. Treating clinical mastitis alone may not always be sufficient and treatment of sub clinical infections may be required in some circumstances. There are new questions being raised on how low cell counts should be, the possibility of an increase in susceptibility to more severe mastitis in low cell count cows, and if there is an optimal balance between any target SCC and the likelihood of increased antibiotic usage in the milking cow.

- To achieve the higher standards being demanded, a higher level of management and attention to detail is required and given these difficult financial times this can be difficult to achieve as more output per man is expected, for no increase in income to deliver any capital investment necessary. Although the 5-point plan has been in existence for 30 years it can not be assumed that all farmers know the importance of the 5-point plan in detail.

- As sub clinical infections become more relevant financially to the farmer the use of the California Milk Test, individual cow cell counts and quarter cell counts become more relevant in the approach to udder health. Early detection of udder infection may be more important than visual changes in the milk. This is currently difficult to achieve. The farmer needs a better guide as to the SCC of the milk before it enters the tank. This is not easily achieved either.

- More effort should be spent in the investigation of problem herds aiming to prevent new infections rather than simply coping with the consequences.

- If treatment and culling are required they should be based on scientific approaches and involve bacteriology and detailed veterinary advice rather than as responses to an empirical cell count.

- Treatment and culling should be seen as failure to prevent rather than accepted as the main part of mastitis control strategy in some herds. These control measures are only part of the 5-point plan.
More sampling of cows to identify pathogens will aid not only in diagnosis but also in decision making for the individual cow. For instance improved cure rates with augmented dry cow therapy with Tylosin in cows infected with \textit{Str. uberis} and \textit{S. aureus} (14) have been described. A proportion of cows identified for culling on the basis of SCC alone might be saved if more effort on correct diagnosis was applied. It is a maxim that 3 cases in one quarter is a criterion for culling. A more sophisticated approach may reduce wasted culls.

The whole area of disease control should move from treating the urgent case to developing a more lasting preventive approach, which is much more profitable and essential in the increasingly competitive market place. This will only occur with vets and farmers working closer together to achieve these aims.

Acknowledgements

To Pfizer Animal Health for the sponsoring of the quarter sample screening and assistance in developing the mastitis guide and to Andrew Biggs and the Vale Veterinary Laboratory for assistance with access to trial work and cultures (sponsored by Schering Plough and Intervet).

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4. British Cattle Herd Health Plan for Farm Assurance. \textit{British Cattle Veterinary Association}.
MASTITIS – DEALING WITH THE HIGH INCIDENCE HERD

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SUMMARY

A practitioner’s approach to investigating outbreaks of clinical mastitis is outlined through techniques such as specific case sampling, sampling high cell count cows and the use of bulk milk samples. This information is combined with analysis of mastitis records and a farm visit. The results are used in conjunction with a working knowledge of how the various forms of mastitis behave to decide on an action plan.

INTRODUCTION

There are basically only 2 types of high clinical incidence herd – those that know about it and those that don’t. Many herds accept a high level of clinical mastitis by either not recognising the problem for what it is or becoming accustomed into accepting certain patterns of infection. These are difficult herds and without good records they will only be recognised by their high use of intra mammary treatment. By far the most common situation in practice is the sudden outburst of cases in an otherwise settled pattern of mastitis incidence. Most herds will have fluctuations in the frequency clinical cases but there is often a need to look closely at some herds when the cluster of cases becomes such that some action is needed – not simply buying 2 boxes of tubes instead of the normal single box.

The main principle to keep in mind is that clinical mastitis is the end result of a process that begins with invasion of the teat, that allows infection to establish in the udder, and then the cow fails to remove the infection before it produces disease. Removing infection through therapy offers poor prospects and so the key areas are and must be to:

- Prevent new infections getting in (invasion)
- Assist the animal in preventing the pathogens establishing and producing damage

Where does this leave us with the herd with high levels of mastitis?

An overall plan can be summarised under the following headings:

1. Shift the emphasis from therapy to prevention
2. Investigate – specific and general techniques
3. End up with some idea of the epidemiology
4. Implement an action plan
1. THERAPY

There is still a great misconception that you can get yourself out of a clinical situation through therapy. The first and most important message to clients is that there is little or no prospect in therapy for the control of mastitis. There is unfortunately nothing of more interest to clients and veterinarians than to discuss the merits of various techniques for therapy but the principle must be that you cannot treat your way out of trouble. Having established this point however the immediate concern is that all clinical cases are dealt with correctly with the aim of:

- Relieve pain and discomfort for the animal
- Prevent a chronic ongoing situation
- Protect the food quality of the milk produced
- Reduce economic losses from high Somatic Cell Counts (SCC)
- Preventing excretion of infective organisms with the risk of spread

Although antibiotics are the single most important factor in the eyes of the client and it is important to make sure this choice is effective – it is the supportive role of other therapies that often makes such a profound difference. The use of anti-inflammatory drugs along with oxytocin may be more important in proper effective therapy.

2. INVESTIGATION

There are 4 key areas we need information about

- Infection – what types of infection are we dealing with
- Environment – what are the surroundings and housing the cows are kept in
- Background – what is the management of the cow from feeding to milking
- Disease levels – how much mastitis and what is the pattern?

Practical approach
There are various options, each with its own problems and shortfalls:

- Individual case sampling
- Sampling high cell count cows
- Bulk milk sampling
- Looking at the records
- Farm visit

**Individual case samples**

A milk sample taken from a clinical case prior to treatment is likely to give the best direct information about the cause of the mastitis. However, by the time a run of mastitis cases has occurred, much material will have been lost for diagnosis, as the cows will have been treated. There are also practical problems with this approach:

- Sampling technique to obtain a milk sample
- Early diagnosis of a case of mastitis
- Storage and transport of the sample

Farm sampling may mean that it is very difficult to get quality samples when asking herdspersons to carry out the procedure. There is the risk of a sample being heavily contaminated with organisms, as it is difficult for the herdsperson to keep clean when actively involved in actually carrying out milking and it is a very demanding procedure to get a sample from a quarter without allowing outside contamination. The sample then has to be cooled and received by the laboratory within the working day if there is to be any chance of a realistic diagnosis.

Other workers have shown that some good material can be obtained with farm personnel being trained to sample the quarter and the sample then frozen. This allows more flexibility with transport and storage although there is the possibility that some organisms will not survive freezing and thawing before culture at the laboratory. However, the system does give some scope as a routine in all mastitis cases to examine samples pending the results of treatment or as an insurance against a run of mastitis.

**High Cell Count Cows**

Looking at high SCC cows may be an option if there appears to be a SCC problem in the herd or a danger of there becoming one. Sampling high SCC cows is much easier as it can be planned, carried out by trained staff and samples transported for laboratory diagnosis from a prearranged visit to get the best results. High SCC cows however:

- May not represent the clinical picture – they may be a totally different problem
- They may be difficult to recognise in the early stages as individual SCC vary enormously

Most investigations show a variety of organisms isolated from clinical cases or high SCC cows and this should be no surprise although it may not be as “clear” to explain as when there is a consistent theme. Even if there is a range of organisms isolated it often gives some information about the pattern of mastitis and more general information about the herd and the way it is managed. All this information will be needed in the end.
**Bulk Milk**

A sample is taken from the bulk milk tank for bacteriology according to a set protocol. The aim is to get the most representative sample of the bacteria present in the bulk milk. These bacteria come from various sources:

- Mastitis
- The milking plant
- Environmental contamination of teats
- Contamination during milking

A bulk milk sample is therefore far from specific. It may point towards a possible cause for the mastitis outbreak but its general approach makes it more useful to give background information about the herd:

- The level of environmental contamination
- Overall plant hygiene
- Milking technique
- Possible causes of mastitis

**Records**

Look for both farm and practice records to see if the level of mastitis can be assessed and to see if there is any trend in the disease pattern over a period of time. Information should ideally be based on the farm record of cases and treatments but a practice record of tube usage is a very useful qualification of either the accuracy of recording (the usual problem) or the apparent success of treatment.

In any investigation a working knowledge of the farm is vital if a simple diagnosis of the mastitis cause is to be turned into knowledge of the dynamics of the disease (its epidemiology) and an action plan for prevention. Once you have enough background information from handling samples and records a site visit is needed to interpret this against what is actually happening on the farm and in the parlour.

3. **EPIDEMIOLOGY**

Before deciding on an action plan it is necessary to bring together findings from the investigation and compare these against some basic knowledge about how the various forms of mastitis behave.

**Environmental mastitis – look at……**

- The environment of the milking cow
- The environment of the dry cow
- Milking technique
  - opportunities for invasion by teat end damage
  - the load on the teat end due to preparation etc.
  - the mechanics of milking allowing entry
- Immune responses especially in the fresh calver.
Infectious mastitis – look at……

- What is the bulk level of this organism – the amount of contamination?
- Are there high SCC cows in the herd and have they been sampled?
- The milking routine – is there increased opportunity for contamination to spread?
- Examine the teats and udder for signs of skin damage or sores – the reservoir

Incidental bacteriology
Other results from bacteriology of any sort may not fit into a disease picture but can give a very good idea of many factors about the farm, the cows and the way they are milked. *Corynebacterium bovis* may indicate poor teat hygiene – especially post dipping. *Enterococcus faecalis*, yeast moulds and sometimes bacilli could indicate poor teat preparation and a high environmental burden – dirty teats and dirty housing.

4. ACTION PLAN

Source - can we identify it?

Invasion – can we prevent it?

Establishing infection – can we help with “immunity”?

(Elimination)

The key to tackling the problem lies in preventing new infection. This means looking at what we have found out about the source of the organism what factors could be introducing infection into the udder and possibly information about why the infections are becoming established and clinical.

Sources
- Dry cow infection – environmental
- Environmental conditions of the milking cows
- Sources of infectious organisms – other cows
- Teat damage and skin injuries

Invasion
What factors are involved with invasion – the whole milking process?

Establish
What may help infection to establish – cow condition and feeding and the management of the fresh calved cow?
CONCLUSIONS

It is common to find that there is no one single cause for the high incidence of mastitis. It is usually an accumulation of problems and faults over a period of time, which then tips the balance to produce an outbreak of cases. The aim is to try and address these multiple problems and not simply look for the single “Holy Grail”. The whole exercise of investigating a herd and producing reports on the findings will increase awareness. Increased awareness makes for better techniques and this in itself will produce improvement. Talking about mastitis even though there may be no specific fault identified will produce results.
COLIFORM MASTITIS - THE IMPORTANCE OF THE DRY PERIOD

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SUMMARY

Despite significant progress in the control of contagious pathogens, environmental mastitis continues to be a major cause of financial loss to the UK dairy industry. This paper presents some findings from a field study designed to investigate the incidence and significance of coliform infections acquired during the non-lactating period. The bacteriological status of two quarters from each of 629 cows was assessed through the non-lactating period (two quarters were left as unsampled controls); samples were collected from all mastitic quarters of these cows during the subsequent lactation. A significant rise in the incidence of intra mammary infection was detected between drying off and prior to calving. When compared to unsampled controls, quarters sampled during the dry period did not show a significantly different incidence of infection at calving, or subsequent clinical mastitis. Quarters infected with a coliform during the dry period were significantly more likely than uninfected quarters to develop mastitis caused by that pathogen. Of all coliform mastitis occurring in the first 100 days of lactation, 52% arose in quarters previously infected, during the dry period, with the same species/strain of bacteria (as identified by DNA fingerprinting). These findings suggest that management during the dry period may influence the incidence of coliform mastitis in the subsequent lactation.

INTRODUCTION

In recent years implementation of the five-point mastitis control plan has resulted in a marked reduction in the incidence of contagious mastitis (1). This decline has not been accompanied by a comparable fall in the incidence of environmental mastitis; which has therefore become of relatively greater importance (2).

Classically, the non-lactating mammary gland has been considered refractory to ‘coliform’ infection (3). However research in the US, from as early as 1943, has implicated the dry period as being the time of greatest risk for the acquisition of new Gram-negative intra-mammary infections (IMI) (4,5,6,7,8), with some 61% of new IMI occurring at this time. Further studies have illustrated the ability of such infections to remain quiescent within the udder until calving, subsequently causing clinical mastitis in early lactation (9).

To date, despite some anecdotal evidence, similar studies, to validate these findings, have not been carried out in the UK. As a consequence the importance of the dry period in the control of Gram-negative mastitis remains equivocal.

The aim of the research outlined here was to investigate the incidence of intra-mammary infections acquired during the non-lactating period of dairy cattle, under UK field conditions and their subsequent importance in the ensuing lactation.
MATERIALS AND METHODS

Salient aspects of the study methodology are outlined below. Full details have been described elsewhere (10).

Herd Selection: Herds were selected on the basis of location (Somerset), low bulk milk somatic cell count (generally <200,000 cells/ml), calving pattern and likelihood of owner compliance with the study protocol. All herds were milk recorded (NMR/DAISY). Herds were not selected on the basis of a previous history of coliform mastitis.

Sampling Strategy: Duplicate quarter samples were collected by the authors at ‘drying off’ and during the week following calving. During the dry period, duplicate, samples were taken from two ipsilateral quarters (LF and LH (odd numbered cows) or RF and RH (even numbered cows)) once in each of the two weeks prior to the anticipated calving date. The other two quarters remained as unsampled controls to eliminate the sampling procedure as a cause of new intra mammary infections. Any cow not calving by her ‘due date’ was sampled weekly until parturition. During the subsequent lactation milk samples were collected from all mastitic quarters identified by the herdsmen and the disease graded for severity.

Sampling Procedure: Prior to sampling, teats were cleansed of gross contamination and dipped in a solution containing 2800 ppm available chlorine (Agrisept, Pharmacia-UpJohn). Following a minimum 30-second contact time the teats were wiped dry. Each teat was subsequently scrubbed with surgical spirit and allowed to dry. Immediately before sampling the teat ends were scrubbed for a second time using surgical spirit and foremilk was discarded (strict foremilk was collected from udders assessed as having little secretion present). Duplicate samples were then collected, following a third scrub of the teat ends. Following sampling, teats were again dipped with the dame disinfectant and the cows were confined to a loafing yard for at least 30 minutes. Samples were immediately stored in a cool box and maintained at or below 4°C. Bacteriology was performed within 24 hours. The herdsmen, using the sampling procedure outlined above collected a single quarter sample from all mastitic cows. These samples were frozen and batched each week for submission to the laboratory. Gloves were worn throughout the sampling procedure, and were changed both between cows and between duplicate sample sets.

Dry Cow Therapy: Dry cow therapy was administered by the authors following collection of the ‘drying off’ samples. The teat ends were scrubbed with surgical spirit for a fourth time prior to partial insertion of the tube canula. Following treatment teats were dipped with disinfectant and the cows confined to a loafing yard for at least 30 minutes. Dry cow antibiotics used were cloxacinil (Orbenin Extra, Pfizer), cephalonium (Cepravin, Schering-Plough) or procaine penicillin G (Mylipen, Schering-Plough).

Bacteriology: Samples were submitted to Langford Veterinary Investigation Centre for bacteriology. Approximately 10 µl of secretion was inoculated onto sheep blood agar and Edward’s agar; 100 µl of secretion was inoculated onto MacConkey agar to enhance coliform detection (7). Plates were incubated at 37°C and bacteria colonies observed after 24 and 48 hours. Organisms were identified and quantified using standard laboratory techniques. Escherichia coli was identified by colony morphology, oxidase and indole tests, other coliforms were identified using a microtube identification system (RapiD 20 E, bioMérieux).
Definition of an Intra mammary Infection: Isolation of a recognised pathogen, in pure growth was considered an intra mammary infection. If a screening sample was obviously contaminated or contained >1 enterobacterial isolate the duplicate sample was submitted for bacteriological examination and intra mammary infections diagnosed on the basis of re-isolation of the organism.

Statistical Analysis: Results were collated and analysed using Access (Microsoft Corporation), Excel (Microsoft Corporation) and Epi-Info (CDC, Atlanta, GA). Statistical analysis was performed using the $\chi^2$ test; results were considered significant when $p \leq 0.05$.

RESULTS

Clinical mastitis incidence and aetiology

The incidence and aetiology of clinical mastitis during the study period has been described elsewhere (2). In summary 337 cases of clinical mastitis occurred during the study period. The mean annual incidence was 41.6 cases/100 cows/year (Range 13-75). *Escherichia coli* was the commonest cause of clinical mastitis accounting for 34.7% of all isolates.

Dry period infections

These results are based on 629 cows calving between 22 March 1997 and 4 April 1998. Infection data were collated for the first 100 days of the subsequent lactation. A more detailed presentation of these results has been presented elsewhere (10).

Major pathogens

The percentage of quarters infected at each sampling point with coliform organisms is outlined in Table 1 and illustrated in Figure 1.

<table>
<thead>
<tr>
<th></th>
<th>Drying Off</th>
<th>3 Weeks Pre-calving</th>
<th>2 Weeks Pre-calving</th>
<th>1 Week Pre-calving</th>
<th>Post Calving</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>2565</td>
<td>423</td>
<td>1003</td>
<td>1197</td>
<td>2503</td>
</tr>
<tr>
<td><em>E. coli</em></td>
<td>2.14</td>
<td>4.73</td>
<td>3.69</td>
<td>5.18</td>
<td>5.27</td>
</tr>
<tr>
<td><em>Enterobacter spp.</em></td>
<td>0.27</td>
<td>0.71</td>
<td>0.80</td>
<td>1.00</td>
<td>0.32</td>
</tr>
<tr>
<td><em>Klebsiella spp.</em></td>
<td>0.04</td>
<td>0.00</td>
<td>0.30</td>
<td>0.08</td>
<td>0.28</td>
</tr>
<tr>
<td><em>Serratia spp.</em></td>
<td>0.08</td>
<td>0.24</td>
<td>0.60</td>
<td>0.25</td>
<td>0.12</td>
</tr>
<tr>
<td><em>Citrobacter spp.</em></td>
<td>0.08</td>
<td>0.24</td>
<td>0.40</td>
<td>0.50</td>
<td>0.20</td>
</tr>
<tr>
<td><em>Proteus spp.</em></td>
<td>0.08</td>
<td>0.95</td>
<td>1.30</td>
<td>1.25</td>
<td>0.52</td>
</tr>
<tr>
<td><em>Morganella spp.</em></td>
<td>0.08</td>
<td>0.24</td>
<td>0.10</td>
<td>0.17</td>
<td>0.08</td>
</tr>
<tr>
<td><em>Salmonella spp.</em></td>
<td>0.04</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>All coliforms</td>
<td>2.69</td>
<td>7.09</td>
<td>6.58</td>
<td>7.77</td>
<td>6.39</td>
</tr>
</tbody>
</table>
Figure 1: Percentage of quarters infected with *E. coli* and all coliforms at each sampling time point

![Bar chart showing percentage of quarters infected with E. coli and all coliforms at different time points.](chart.png)

There was a significant increase in the proportion of quarters infected with coliforms between drying off (69/2565 quarters) and one week prior to calving (93/1197 quarters, p<0.001). There was a similar significant increase in *E. coli* infections (55/2565 quarters at drying off and 62/1197 quarters at one week prior to calving, p<0.01). There was no significant difference in the proportion of quarters infected with coliforms between three and two weeks pre-calving (p>0.2). There was a trend towards a higher prevalence of infection with the coliforms, in the late dry period, in higher parity cows. No seasonal effects were observed other than a significantly higher prevalence of infection at drying off in the summer than in the winter and spring.

Of 41 quarters infected with *E. coli* at drying off only 3 apparently persisted until calving, though subsequent fingerprinting demonstrated that in all cases this was due to re-infection with a different strain. New intra mammary infections with *E. coli* were detected in 8.6% of quarters during the dry period. New quarter infections accounted for 97% of all *E. coli* infections detected during the dry period. New coliform intra mammary infections were detected in 12.8% of quarters during the dry period. New quarter infections accounted for 96.6% of all coliform infections detected during the dry period.

**Mastitis in cows on the study**

153 cases of clinical mastitis occurred in cows on the study, within the first 100 days of lactation. In 40.5% cases a coliform was identified as the causal pathogen. *E. coli* was isolated on 50 occasions, *Klebsiella spp.* on 4, *Serratia spp.* on 6 and *Citrobacter spp.* on 2.

Of 1197 quarters sampled during the dry period, 154 (12.87%) quarters were found to be infected with a coliform at one or more of the sampling points. Some 13 (8.44%) of these infected quarters later developed mastitis due to the same species/stain of bacteria; these quarters had 20 cases in total. Conversely of 1043 quarters not infected with a coliform...
during the dry period, 15 (1.44%) which then developed coliform mastitis, some 18 cases in total, there was a significantly greater risk of a previously infected quarter later developing mastitis than an uninfected quarter (p<0.001).

In quarters sampled during the dry period, a total of 38 cases of coliform mastitis occurred in 28 quarters. Of these 38 cases, 20 (52.6%) occurred in quarters previously infected, with the same species/strain of bacteria, during the dry period. In 71% of coliform mastitis cases, occurring in sampled quarters, the same species of pathogen had been detected previously (i.e. at drying off, pre- or post calving).

**Sampled and control quarters**

There was not a significantly increased incidence of coliform infections in the routine post-calving milk samples of quarters sampled during the dry period (82 of 1194 quarters) compared to those which were not sampled (71 of 1194 quarters) (p=0.40). There was also no significant increase in incidence of coliform mastitis in quarters sampled during the dry period (28 of 1194 quarters) than those not (21 of 1194) (p=0.39).

**DISCUSSION**

The data from this study on a small, though arguably typical, cohort of dairy herds would tend to support the anecdotal evidence, which has suggested an increase in the incidence of mastitis due to coliform organisms over the past few decades. The coliforms have classically been classified as opportunistic environmental pathogens capable of colonising the udder, causing a transient infection and mastitis, not uncommonly accompanied by severe systemic disease. Sub-clinical infections, although recognised (11,12) have not been implicated as a significant cause of subsequent disease.

The dry period has been implicated as a crucial period for acquisition of new Gram -ve intra mammary infections (IMIs), with >60% of all new IMIs occurring at this time (8). The data from this study would also support this finding as a significant rise in the level of infection was detected during the dry period.

Previous studies have been unable to implicate conclusively these new infections, acquired during the dry period, in subsequent mastitis (8). Also, the absence of unsampled control quarters means that the role of iatrogenic introduction of infection was unknown (8). The design of this investigation controlled for the effect of sampling quarters during the dry period and has more conclusively implicated dry period infections in subsequent mastitic episodes.

Perhaps the most compelling results generated from this study are that more than 50% of all clinical mastitis due to coliform organisms arose from quarters previously infected during the dry period; and that 71% of coliform mastitis occurred in quarters that had previously been found to be infected at any one time point. These phenomena can be ascribed to two possible scenarios; either bacteria reside within the udder awaiting conditions conducive to multiplication and subsequent disease, or certain quarters show an increased susceptibility to re-infection with the same species of pathogen. DNA fingerprinting studies of the coliform isolates from cases of mastitis apparently arising from dry period infections confirmed that this phenomenon was as a result of persistent infection and not re-infection as may previously have been assumed (10).
Clinical implications and dry period management options

An in-depth review of strategies for minimizing the risk of intra mammary coliform infections and subsequent clinical coliform mastitis is beyond the scope of this paper, however a few management options and issues, affecting the dry cow, are briefly outlined and discussed below

Method of drying off: The act of administering antibiotics to a potentially sterile quarter is the first potential area of hazard and the need for cleanliness in the procedure cannot be over-emphasised. Cleaning, disinfecting and drying the teat, followed by stripping the quarter and at least two teat-end scrubs with spirit is the minimum recommended prior to administration of an antibiotic tube and post dipping. The impact of infections introduced at this stage is difficult to quantify; they are unlikely to persist until the next lactation but can result in acute coliform mastitis prior to involution of the gland.

Dry cow therapy: The selection of antibiotic dry cow therapy may be an important element of a coliform mastitis control scheme. Studies currently being undertaken by the authors are investigating the potential impact of different dry cow formulations on subsequent coliform mastitis incidence.

Teat sealants: ‘External’ teat sealants are already available in the UK and though their efficacy under UK field conditions has yet to be established, they may aid in the control of new intra-mammary infections in the late dry period, by protecting the teat end from environmental contamination. An internal sealant ‘Teatseal’ (Bimeda) has shown promise in New Zealand as an alternative to antibiotic dry cow in preventing new Streptococcus uberis intra mammary infections during the dry period (13).

Environmental management: ‘Clean, dry, cool and comfortable’. It is common for dry cows to be housed in the oldest, poorest maintained buildings or kept on pasture with little attention, a foolhardy strategy, when the dry period is probably critical not only in terms of udder health, but also in other aspects of health and production. As a general rule, it is useful to ask the question ‘is the dry cow environment at least as good as for the milking cows’. It is rare that any new accommodation is built for dry cows but it is usually possible to make the best of existing facilities. Cubicles can be adapted and if used inorganic bedding materials such as sand will inevitably support less bacterial growth than straw.

Straw yards provide a better type of system than cubicles for foot health but are almost always worse for environmental mastitis pathogens. Recent work (14) has indicated that below the surface of even a recently bedded straw yard, warmth and moisture provide the ideal environment for sustaining environmental bacteria. Even using recommendations such as bedding up daily, cleaning out every 3-4 weeks and maintaining low stocking densities (10m² per cow bedded area and a cubic capacity of 15m³), infections will still occur. Even in well managed straw yards, intra mammary infections in the dry period are inevitable.

Mixing of groups: It has been reported that housing pregnant heifers and dry cows together increases the risk of E. coli mastitis (15). This is something that can often be avoided through the use of simple partitions, without huge building costs.
Leaking milk: Leaking of milk may be a risk factor for mastitis (Peeler, 1999). Problems with leaking milk may be associated with dry cows which are over-fed before calving (rare), are in sight/sound of the parlour which makes milk let down more likely, or are in the presence of calves (i.e. with recently calved cows). These should be avoided if milk leakage is a problem on the unit.

Nutrition: Negative energy balance (16) and number of feed spaces per cow (15) have been related to the severity or incidence rate of $E_{coli}$ mastitis respectively. Dry matter intakes, energy balance and mineral supplementation (particularly vitamin E and selenium (17)) in the late dry (transition) period, around calving and in early lactation are probably important in reducing clinical episodes of coliform mastitis.

Vaccination: Recently a vaccine for coliform mastitis, based on a rough (R) mutant $E_{coli}$ (strain J5) has become available in the UK. Use of the vaccine is not associated with a reduction in the number of new dry period coliform intra mammary infections (18), but has been shown to decrease the incidence and severity of clinical coliform cases. This vaccine will have a role, alongside other preventive measures, in the control of Gram-negative mastitis. It should be stressed that the vaccine is not a substitute for correct management but should be used as an aid to further reduce the incidence and severity of coliform mastitis.

FUTURE/ONGOING WORK

Many outstanding questions raised by this research which warrant further investigation. Why do only some quarters develop intra mammary infections and why do only some of these subsequently develop mastitis? Are quarters denuded of ‘commensal’ minor pathogens, by dry cow therapy more susceptible to infection? Also, why is there a delay between acquisition of infection and subsequent mastitis, and are there strains of $E_{coli}$ adapted to colonising the bovine udder, which can persist and subsequently cause disease? It is hoped that some of these questions can be answered in further studies, drawing upon the vast stock of pathogens and samples acquired during this study. Additional work, currently underway, include intervention studies to attempt to decrease the incidence of new enterobacterial IMI in the dry period and mastitis in the subsequent lactation. Another interesting ongoing area of study is the feasibility of seasonal, selective use of dry cow therapy in low somatic cell count cows.

CONCLUSIONS

Preliminary findings from this study support those of previous studies and have far reaching implications for our understanding of ‘environmental’ mastitis and our approach to control of outbreaks of disease. In the past, control has centred on the management of the lactating and peri-parturient cow; these findings would suggest that significant effort also needs to be directed towards management of the dry cow. The apparently high ‘new infection’ rate from 3 weeks pre-calving would imply that the whole of the dry period is critical in preventing new infections and as such needs to be addressed by minimising bacterial challenge and enhancing pre-existing host defences.
ACKNOWLEDGEMENTS

The authors gratefully acknowledge the financial support of Leo Laboratories without which this study could not have been undertaken, the Langford Veterinary Investigation Centre for undertaking bacteriology, the patience of farmers for allowing weekly access to their herds. A.J. Bradley is supported by a fellowship funded by the Wellcome Trust, London.

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determine efficacy of an *Escherichia coli* J5 mastitis vaccine. *J. Dairy Sci.* 75, 78-84.
TO DRY COW TREAT OR NOT?

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SUMMARY
Total dry cow therapy is the current recommendation for non organic dairy herds in the United Kingdom but is not allowed on organically managed cows. The consequences of ceasing to use dry cow therapy on conversion need to be demonstrated to advise on likely problems and alternative management. Results from a selective dry cow trial, comparing total dry cow treatment with no treatment, on four herds in England are presented. These confirm that dry cow treatment for cows remains highly effective in preventing new infections. It reduces the rate of new infection by approximately 80% and prevents clinical mastitis in the dry period, especially when these are likely to be caused by Streptococcus uberis.

INTRODUCTION
The use of intramammary antibiotic to prevent intra mammary infections in the dry period was originally developed as a control measure for summer mastitis (1). Its use, applied immediately after the last milking, was later found to result in an enhanced cure rate for existing infections and to reduce the chance of new infections in the dry period (2) when these were most likely to be caused by Staphylococcus aureus or Streptococcus agalactiae. It was then combined with several other recommendations to give what is known as the “Five Point Mastitis Control Plan.”

Organic production is an increasing niche market that specifies no prophylactic antibiotic use. With current milk prices this form of milk production presents an attractive proposition. The present economic climate has also necessitated a review of current practices including total dry cow therapy.

Currently in the UK, dry cow therapy for all cows all year round is the recommended practice in conventional dairying. Increasing concerns over the widespread use of antibiotics along with the observation that now many cows at drying off are uninfected has resulted in the re-evaluation of the use of total dry cow therapy. Alternatives include selective dry cow therapy, selective quarter therapy, administration of lactation intra mammary antibiotic often for several days prior to drying off, systemic therapy, no therapy and the use of teat sealants (3-6).

Selective quarter or cow treatment requires initial screening to identify an infection and general preventive benefits of total dry cow therapy are lost. Identification of infected quarters or cows can be carried out using cell counts and or the California Mastitis Test as an initial screening method, with bacteriology for further confirmation. These are not always accurate and can add expense. Success rates for systemic treatments either alone or in combination with intra-mammary treatment have been no better than intra-mammary treatment alone. The use of systemic antibiotics at drying off may pose an extra antibiotic residue risk, with antibiotics excreted in the urine and faeces in possibly sub therapeutic concentrations.
In Norway dry cow intra-mammary formulations are not available and lactation treatments are used for infected cows (4). This generally means that more antibiotic is used and there is increased handling stress. Some organic farms are using this as an alternative to dry cow therapy.

External teat sealants have been used to try and prevent new infections during the dry period with limited success (7). Inert bismuth subnitrate as an intra-mammary sealant is available in Ireland and New Zealand, with a reported 90% reduction in new infections (6, 8). This has been used with an antibiotic or the bacteriocin lactocin or alone. This product looks very promising as an alternative to total dry cow therapy.

To assess the benefits and problems of the different strategies several factors need to be taken into account. These include –

- the current infection status of the cow and herd,
- parity
- environmental factors
- season
- potential market to be supplied
- economic circumstances.

Only the first is relevant in the initial study reported here.

**MATERIALS AND METHODS**

**Experimental design**
Initially four herds were recruited for a selective dry cow trial, two herds at the Institute for Animal Health and two herds undergoing conversion to organic status. Cows were assigned randomly within each herd to two groups: treatment and non treated. Treatment was use of Cepravin DC (Schering Plough plc) for three herds and Orbenin Extra DC (Pfizer plc) for one herd. All cows were dried off abruptly at the end of the designated milking.

**Sampling procedure**
Cows were sampled aseptically using 70% ethanol solution to clean the teats. A single 10 ml foremilk sample from each teat was collected. Samples were taken one week prior to drying off, at drying off, within 24 hours of calving where possible and 7 to 14 days after calving. Extra samples were taken if any of the previous samples were not suitable or for confirmation of infection. Sampling at pre drying off, drying off and post calving was normally on a fixed day each week and samples were examined within 24 hours of sampling. Samples at calving were stored at 3-8 °C for no more than 3 days until assay. Those from external farms were frozen if there was going to be a delay of more than 3 days between sampling and assaying.

**Laboratory methods**
Cell counting was carried out on all suitable samples using a Fossomatic machine. Microbiological examination was carried out according to IDF recommendations (9) For routine samples 0.05 ml of milk was inoculated onto aesculin blood agar. Plates were incubated for 48 hours at 35-37 °C and examined after 24 and 48 hours incubation. Colonies were provisionally identified on gross morphology and number and type of colonies were recorded. Appropriate tests were carried out on colonies
isolated, where necessary to identify the pathogens. Samples from cows with infections identified as clinical mastitis were assayed as above, in addition 0.05ml from the suspected clinically infected quarter was inoculated onto sheep blood agar, an additional aesculin blood agar and MacConkey agar.

All strains isolated from bacteriologically positive samples were frozen in 50% v/v glycerol/water solution at −20°C.

Definitions
Where the same pathogen was isolated in two consecutive samples or two out of three samples or a pathogen in one sample with a cell count elevated in comparison to the other quarter cell counts this was defined as an infection. An elevated cell count was defined as two times greater than those of the other quarters and greater than 200,000 cells per millilitre.

Clinical mastitis was defined as occurring when visible changes in the milk were seen such as watery milk, clots or flakes and changes in the udder such as swelling or heat. These were either detected by the herdsman or at one of the routine sampling points.

Analysis
Data were analysed using Minitab version 7.

RESULTS
The results for infection status are limited to streptococci, coliforms, Staphylococcus aureus, Arcanobacterium pyogenes and Proteus spp. Infections likely to be caused by coagulase negative staphylococci or Corynebacterium bovis are not included.

Logistical regression analysis was carried out on herd and dry period length. The only significant factor was a dry period length of more than 16 weeks and these cows were excluded from the analyses of results.

Table 1 Rate of clinical mastitis occurring during the dry period for untreated and dry cow treatment animals in IAH herds.

<table>
<thead>
<tr>
<th></th>
<th>No infection</th>
<th>Clinical mastitis</th>
<th>Total no. cows</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treated at drying off</td>
<td>111</td>
<td>0</td>
<td>111</td>
</tr>
<tr>
<td>Not treated</td>
<td>118</td>
<td>7</td>
<td>125</td>
</tr>
<tr>
<td>Total no. cows</td>
<td>229</td>
<td>7</td>
<td>236</td>
</tr>
</tbody>
</table>

\( \chi^2 = 7.383 \)  p\#0.01

The incidence of clinical mastitis in the dry period at IAH was 0% in dry cow treated animals and 6.5% in untreated animals, a statistically significant difference (Table 1). There was also a 100% reduction in clinical incidence in the two organic herds although this was barely statistically significant due to the small group size (Table 2).
Table 2 Rate of clinical mastitis occurring during the dry period for untreated and dry cow treatment animals in the two organic herds.

<table>
<thead>
<tr>
<th></th>
<th>No infection</th>
<th>Clinical mastitis</th>
<th>Total no. cows</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treated at drying off</td>
<td>24</td>
<td>0</td>
<td>24</td>
</tr>
<tr>
<td>Not treated</td>
<td>26</td>
<td>4</td>
<td>30</td>
</tr>
<tr>
<td>Total no. cows</td>
<td>50</td>
<td>4</td>
<td>54</td>
</tr>
</tbody>
</table>

$\chi^2=3.173$  $p=0.075$

During the dry period all four herds had cows that suffered dry period clinical mastitis; for 10 of 12 cows this was caused by *S. uberis*. Two cows also acquired an intra mammary infection caused by *A. pyogenes* in one quarter in addition to *S. uberis* in one or more other quarters. Two cows from IAH that had clinical mastitis in the dry period were culled before calving.

At calving all herds had a significantly greater number of new infections in the untreated cows compared to the treated cows. The percentage of cows infected at calving varied between herds and ranged from 30% to 50% at calving for untreated cows and 0% to 15% for treated cows. The difference was statistically significant at both IAH (Table 3) and on the organic herds (Table 4).

Table 3 Rate of new intra mammary infections detected at calving for IAH cows.

<table>
<thead>
<tr>
<th></th>
<th>Not infected</th>
<th>Infected</th>
<th>Total no. cows</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treated at drying off</td>
<td>98</td>
<td>13</td>
<td>111</td>
</tr>
<tr>
<td>Not treated</td>
<td>89</td>
<td>34</td>
<td>123</td>
</tr>
<tr>
<td>Total no. cows</td>
<td>187</td>
<td>47</td>
<td>234</td>
</tr>
</tbody>
</table>

$\chi^2=12.547$  $p<0.001$

Table 4. Rate of new intra mammary infections detected at calving in the two organic herds.

<table>
<thead>
<tr>
<th></th>
<th>Not infected</th>
<th>Infected</th>
<th>Total mo. cows</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treated at drying off</td>
<td>24</td>
<td>0</td>
<td>24</td>
</tr>
<tr>
<td>Not treated</td>
<td>15</td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>Total no. cows</td>
<td>39</td>
<td>15</td>
<td>54</td>
</tr>
</tbody>
</table>

$\chi^2=16.615$  $p=0.000$

Some 15 cows (15%) at IAH that had received dry cow treatment developed an infection in the dry period whilst no new intra mammary infections occurred in the two organic herds. However, all IAH cows were uninfected at drying off whilst of the 24 cows in the treated group on the organic farms 6 were already infected in one or more quarters at drying off. There was a different risk attached to the two groups.

The two organic herds both untreated groups had a higher percentage of cows (50%) infected at calving than the IAH herds (28%). In the untreated group 60% of new infections were caused by *S. uberis* compared with 50% in the untreated group. In those cows infected at calving that subsequently showed clinical signs in the same quarter with the same pathogen, 50% were caused by *S. uberis*. 
At drying off no cows enrolled at the Institute for Animal Health (IAH) were infected with *S. uberis, S. aureus, S. agalactiae, Streptococcus dysgalactiae* or coliforms. On the two organic herds the cows were allocated to treatment or non-treatment groups irrespective of their infection status at drying off. In these herds 14 cows were infected with *S. aureus* at drying off and only one cow of these cows, in the treated group, appeared uninfected at calving indicating a very low cure rate. The spontaneous cure rate for infections from the untreated group was zero (Table 5).

**Table 5. Number of infections persisting through the dry period in cows from the two organic herds.**

<table>
<thead>
<tr>
<th></th>
<th>Infected at drying off</th>
<th>Infections persisting at calving</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treated</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Not treated</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
<td>13</td>
</tr>
</tbody>
</table>

**DISCUSSION**

There were significant differences in the new infection rates and the incidence of clinical mastitis during the dry period, at calving and in the subsequent lactation between the untreated and treated cows from all herds. There were many more cases of sub clinical and clinical mastitis in the untreated cows. More than 50% of the infections were caused by *S. uberis*. This indicates the importance of dry cow therapy in both low cell count, uninfected cows and infected cows under current management situations. In this trial provisional analysis has indicated that there is no reduced risk of infection when cows are outdoors during the summer months.

The non lactating udder is highly susceptible to certain infections and 50% of intramammary infections acquired during the dry period will persist into the next lactation if not eradicated by appropriate treatment (10). During the dry period new infection rates are highest in the early dry period, lowest when involution is complete and increase as parturition approaches. New infection rates at calving in this trial varied between herds and ranged from 30% to 50% of cows at calving in untreated groups and 0% to 15 % in treated groups. Over 50% of these infections at calving were due to *S. uberis*. The significantly lower rate of infection caused by *S. uberis* in dry cow treated cows shown in this study highlights the importance of dry cow therapy for preventing infections during the early dry period.

All the cows that showed clinical signs during the dry period had a least one quarter infected with *S. uberis*. The clinical infection rate during the dry period was not as high as that reported from Australia workers where cows were sampled periodically during the dry period (11). This may be due to the fact that their cows were more closely observed during the dry period. The dry period clinical mastitis incidence in the two IAH herds declined over the trial period. One possible reason for this may be an improvement in dry cow management. Alternatively, if dry cow management had not changed, more attention may have been paid to the cows in the initial stages of the trial due to reservations by the farm staff on the impact of not treating cows. This change in incidence was also noted in one of the organic herds.
There was a low cure rate in both treated and non-treated groups of infections present at drying off. As all these infections were caused by *S. aureus* this probably influenced the low rates obtained.

**CONCLUSIONS**

Dry cow therapy is still an essential part of controlling intra mammary infections during the dry period and the subsequent lactation, even in low cell count herds, in England throughout the year. The consequences of failing to use dry cow treatment are likely to include an increased prevalence of intra mammary infection, more dry period clinical mastitis and poorer milk quality. The later is shown by an increase in the bulk milk tank cell count for all herds. This might be assumed to increase faster if no cows received dry cow treatment. These factors will contribute to a decline in cow well-being.

**ACKNOWLEDGEMENT**

To everyone who has helped.

**REFERENCES**


WHAT TO DO WITH STAPH COWS? GUIDELINES FOR THE CONTROL OF S. AUREUS SUBCLINICAL MASTITIS BY CULLING USING RECOMMENDATIONS BASED ON A BIO-ECONOMIC COMPUTER MODEL

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INTRODUCTION

Reasons for Project

Since 1992 there has been an increasing demand by Milk Buyers for milk with quality standards considerably better than those set by the EC in their Directive 92/46. As a result of this, and concerted advisory drives over the last decade, virtually all UK herds have moved to being consistently below the EC threshold figure for bulk tank somatic cell counts (BTSCC) of 400,000 cells/ml. However recent figures from Scotland (see Figure 1) and the UK as a whole (1), indicate that this improvement has reached a plateau. One reason for the lack of further progress may be the difficulty of managing strategic culling. It is well known that culling plays a major role in the control of the common mastitis pathogen S. aureus. This is because it causes a very intractable condition and as a result it is the main organism targeted by the advice within the FIVE POINT PLAN to cull animals with repetitive cases of clinical mastitis. Unfortunately this pathogen also accounts for a considerable proportion of subclinical infections in our dairy herds (3). Hence understanding how farms can best manage culling related to high SCC figures could allow us to give better advice on the use of strategic culling in the control of high BTSCC figures caused by this bacteria.

Figure 1. Progress in control of Bulk Tank SCC figures since 1993
Farmers will be only too well aware of the competing claims for culling. Stott and Kennedy (5) first described the use of Dynamic Programming to tackle the economics of culling dairy cows with mastitis. The present study further develops this theme and in doing so allows linkage with other studies in particular weighing the importance of criteria such as longevity and SCC in the selection of superior breeding stock. Thus information from this study is already being applied to other areas of vital interest to dairy farmers.

**Aims of the project**

The overall objective of this work was to identify cost effective strategies for the control of subclinical mastitis due to *S. aureus*. This was to be done by:

1. Development of suitable databases from existing data.
2. Using these to develop further aspects of the Dynamic Program (DP) Model and to refine it.
3. Using this DP Model to examine the economic impact of control by culling under different price, production and intramammary infection scenarios.

**The DP model**

The foundation of all these studies is a computer based "model" dairy herd. The DP uses field data to specify the replacement decision for a “typical herd”. Then, by using the likely economic benefit to the farm of culling an animal, it computes likely culling rates under pre-determined scenarios (such as overall yield output of the herd). The original DP model (4) used data which is now outdated. In the present project our aim was to revise and develop the Model to include subclinical mastitis caused by *S. aureus*. We considered that this approach was particularly relevant for *S. aureus* as we had data showing very poor cure rates after quarters were infected with a strain of *S.aureus* taken from a field case of subclinical mastitis (2). In this DP model the effects of clinical mastitis were considered as a constant characteristic and the effects of subclinical mastitis caused by organisms other than *S. aureus* were not considered at all. Thus, our figures only partly represent the full extent of mastitis problems in the dairy herd and particularly concentrate upon the use of voluntary culling to control BTSCC figures due to intractable subclinical *S. aureus* mastitis. The Model also assumes that other parts of mastitis control, such as limiting transfer at milking, and other predisposing causes, such as milking machine performance, are equally applied across all herds irrespective of the level of infection. This is clearly a simplification and thus decisions based on the output produced from the model should take this into account.

The program uses a mean projection of penalties and premiums for BTSCC figures based on those recently used by a number of the major buyers (Table 1).

The DP Model then relates these penalties to the individual cow within the herd and how her yield and individual SCC (ICSCC) (which is related to infection) contributes to the overall BTSCC figure. This is quite a complex relationship; for example, at its simplest a cow with a high ICSCC has a greater likelihood of being culled in a herd if the BTSCC is close to one of the penalty thresholds shown in Table 1 than if a herd is in the middle of a band. This is because the risk of losing a milk price premium or gaining extra penalty are greater at these points if the replacement decision is postponed.
Table 1. Projected penalties and premiums used in the DP Model showing present projected figures and for comparison those estimated on a similar basis for 1999

<table>
<thead>
<tr>
<th>SCC Band (kcounts/ml)</th>
<th>Penalty/Premium (ppl) 1999</th>
<th>Penalty/Premium (ppl) projected</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 &lt;150</td>
<td>0.2</td>
<td>0.3</td>
</tr>
<tr>
<td>2 151-250</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3 251-400</td>
<td>-0.5</td>
<td>-2.0</td>
</tr>
<tr>
<td>4 401-500</td>
<td>-10</td>
<td>-10</td>
</tr>
<tr>
<td>5 501+</td>
<td>-10</td>
<td>-14</td>
</tr>
</tbody>
</table>

Costs of culling to obtain such a “return” we have called Penalty Loss (PLOSS). The extent of PLOSS will depend not only on the yield and ICSCC of the individual cow, but also on herd size and volume of milk produced, as these will affect the relative impact of a potential cull on BTSCC. To account for this, herd size was assumed to be 100 milking cows producing milk evenly throughout the year. The variability of the rolling monthly geometric mean of BTSCC (based on the average of a sample of 350 farms in 1999) was also taken into consideration, as this too will affect the impact of culling on the risk of a change in milk price penalty/premium. We found it very difficult to account accurately for the influence of different levels of penalty and premium. Fig. 2 illustrates how increasing/decreasing the BTSCC by 1,000 cells/ml changes the expected future milk price given the current BTSCC. This is expressed as the change in the annual weighted average of all possible prices (18ppl plus the values in Table 1). The weighting factor is the probability of occurrence of each possible price. This figure therefore mirrors the relative “incentive” to cull high ICSCC cows (i.e. those over the herd BTSCC mean) and to retain those with low ICSCC. As can be seen this incentive depends on the BTSCC and the penalty and premium scheme. Firstly, in both the old and the new schemes, if the graph is continued to the 400,000 cells/ml threshold the peak is exceedingly high and outwith the range of the graph in Fig. 2 i.e. the incentive to cull high ICSCC cows in high BTSCC herds (>300,000 cells/ml) is huge. This graph also predicts that with both the 1999 figures and the “new” projected penalty and premium scheme herds will have a BTSCC “comfort zone” at about 170,000. Thus, it suggests that a herd with a BTSCC of 151,000 cells/ml is more likely to cull a cow with a high ICSCC than a herd with a BTSCC of 171,000 cells/ml. This is because there is a greater chance that this act will have an immediate effect in reducing the BTSCC to below the premium threshold (150,000 cells/ml). However if the herd has a BTSCC of 230,000 cells/ml then it shows that then there is an even greater incentive to cull to reduce the chance of having a BTSCC in the next penalty range (>250,000 cells/ml) (see Fig 2).

Finally the program contains an estimate of the loss of efficiency of milk production (or Yield Loss-YLOSS) associated with an increased ICSCC. In other words a cow with subclinical mastitis will give less milk than she would if she was not infected. Thus there will be an incentive to cull according to this YLOSS effect in addition to the PLOSS effect. A conservative estimate of YLOSS, based on an earlier study in Scotland, has been used. A figure of approximately a 5% milk yield depression for every unit increase in the natural log of SCC (a rise from 200,000 to 544,000 cells/ml is a unit increase) was obtained (6). This figure is in good agreement with other larger and more sophisticated studies in the USA and Canada.
Figure 2. Effect of BTSCC on change in expected milk price caused by increasing BTSCC by 1,000 cells/ml

The study has looked at three different output herds (Low, around 5,700 l/cow/year; Average around 6,800 l/cow/year and High around 8,900 l/cow/year). These are then considered at three levels of infection (low or “Control”, average or “Intermediate” and high or “Infected”). The cell counts for these putative infection rates were derived from NMR and Scottish data. The first two were derived directly from NMR 1998 data and reflect low and average BTSCC herds. These herds had no known bacteriology but we have reasonably assumed that they correspond to a “Control” and “Intermediate” status. The high or “Infected” herd was derived from figures from herds with a known infection with *S. aureus*. While these were not the actual figures, as we had to “correct” them to a lower figure to reflect the present status of the UK National herd, the increase in ICSCC by lactation in the Infected herd was in proportion to these earlier figures (from 1993-95). This pattern of ICSCC by lactation number with increasing “infection” is shown in Table 2.

Table 2. Effect of “level of infection” upon mean ICSCC for cows in that lactation.

<table>
<thead>
<tr>
<th>Lactation no.</th>
<th>Mean ICSCC by lactation number in the three types of herd</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
</tr>
<tr>
<td>1</td>
<td>79</td>
</tr>
<tr>
<td>3</td>
<td>101</td>
</tr>
<tr>
<td>6</td>
<td>156</td>
</tr>
</tbody>
</table>

In order to understand the way in which infection and BTSCC affect culling the model was run a considerable number of times. These take place over a 20-year scenario and show how the herd will balance culling to give an optimal economic output under a variety of herd descriptions. The output also gives us an estimate of the relative importance of the criteria.
that we use in the model. Additionally it has allowed us to develop a simple decision support table to give guidance to farmers on how best they might cull potentially infected cows to help reduce the level of *S. aureus* subclinical mastitis and so their BTSCC.

Results from the DP model

While there were subtle differences between the way the DP dealt with culling in herds with a similar “infection” but different yield outputs the latter did not materially affect the decision support. In other words, the effect of yield on culling strategy was relatively small compared to the impact of the level of infection. Thus the results here are only those for the average yield output herd (i.e. average overall herd sales of around 6,800 litres per cow per annum).

The DP economic outputs showed that the annuity difference per cow, due to the loss of efficiency of milk production alone, or YLOSS, (i.e. before there was any consideration of BTSCC penalties and premiums) between an average yielding herd with a low infection (Control) and an “Infected” herd was £38/cow. The introduction of the new projected penalties and premiums (from Table 1), and the resultant PLOSS, created an even larger demand for culling against high individual SCC cows.

| Table 3. Reductions in Annuity for Control, Intermediate and Infected herds with Average output (6800 l/cow/year) after 20-year runs by DP Model when YLOSS and YLOSS & PLOSS are used. |
|---|---|---|---|---|---|
| **Infection Type of herd** | **Reductions in Annuity (£/cow/year) computed by DP Model** | **After culling for Yield Loss (YLOSS)** | **Additional BTSCC Penalties (PLOSS)** | **Overall from Control herd at base** | **Difference** |
| **Control** | Base* | 1 | 1 |
| **Intermediate** | 22 | 11 | 33 |
| **Infected** | 38 | 22 | 60 |

*Baseline annuity (£148/cow/yr)

As a result, a further annuity loss of £22/cow/year was calculated, again taking the Control herd as the baseline (see Table 3). Thus in total there was a “loss” of £60/cow/yr for the Infected herd compared to the Control. The Intermediate herd (average infection) was less affected but still had a total loss of £33/cow/year compared to the Control (Table 3).

The DP Model predicted that all herds will do some culling and will reduce their BTSCC in response to the effects of subclinical *S. aureus* on milk yield and on milk price penalties/premiums. The incentive to cull against the effect of ICSCC on BTSCC however declines with decreasing BTSCC (see Fig. 2). In order to establish PLOSS the equilibrium BTSCC must be found where the incentive to further culling is balanced by the cost of doing so. This was done by finding the equilibrium “threshold ICSCC” which represents the boundary between cows that tend to lower the BTSCC and so enhance their chances of being kept and those above which do the opposite and hence increase their chances for replacement. This equilibrium ICSCC can be seen as the point above which this computerised herd manager (for effectively that is what this DP Model is) would examine cows closely to see
whether the yield and other considerations merited keeping the animal. The equilibrium “thresholds” for the three different levels of infection (Control, Intermediate and Infected) are shown in Table 4. Considerations of yield and cost of culling indicate that there will be an incentive to keep some cows with ICSCC above this ICSCC threshold. This explains why the equilibrium determined by the DP Model for BTSCC is higher than the equilibrium it suggests for ICSCC. These equilibria are established at the end of the 20-year DP model run, however it usually only takes around 5 years for the model to approach close to these end states.

Table 4. Equilibrium ICSCC (.000 cells/ml) and equilibrium BTSCC for Average (6,800 l/cow/year) yield herds in face of economic penalties of subclinical mastitis for yield output and milk price. These represent long-run targets for the herd and individual cow respectively.

<table>
<thead>
<tr>
<th>Infection type of herd</th>
<th>BTSCC “Threshold” figures</th>
<th>ICSCC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before any SCC culling</td>
<td>After culling for Yield Loss</td>
</tr>
<tr>
<td>Control</td>
<td>149</td>
<td>130</td>
</tr>
<tr>
<td>Intermediate</td>
<td>213</td>
<td>191</td>
</tr>
<tr>
<td>Infected</td>
<td>318</td>
<td>260</td>
</tr>
</tbody>
</table>

DISCUSSION

These results indicate that with both the 1999 figures and the “new” projected penalty and premium scheme the need for culling infected high ICSCC cows until the BTSCC is down to at least 300,000 cells/ml and preferably to 170,000 cells/ml is imperative. This agrees well with our independent findings from field observation (3) and with the recent field figures shown in Fig. 1. However we have already pointed out that recent UK BTSCC figures are essentially static around 170,000 cells/ml. This is despite the fact that milk buyers have been steadily increasing BTSCC penalties. In some cases these can mean a reduction of almost 13% of the milk price even for figures consistently below the EC threshold of 400,000 cells/ml. We believe that the conflicting demands of the economics of culling and of BTSCC thresholds are the prime reasons for this static appearance of UK BTSCC figures.

Culling alone without limiting the subsequent rise in infection merely leaves the herd in a state similar to that seen in either the Infected or Intermediate herds shown here. As can be seen these have their most economic steady state at a BTSCC figure between 150,000 and 200,000 cells/ml. For these reasons we suggest that quite a number of those herds within this range of BTSCC may be culling hard for ICSCC and therefore have a much higher level of infection with subclinical mastitis than is often thought to be the case. J M Booth (pers. com.) has already raised this possibility and we strongly agree that there is a need for a structured
investigation into the extent of subclinical mastitis in groups of herds targeted by BTSCC and other characteristics highlighted by this model.

On the other hand, if the herd, having culled infected cattle, controls subsequent reinfection well, then the DP Model suggests that it can move steadily from Infected status through Intermediate to Control status within a decade. The figures from herds in the field suggest that this is conservative but it gives a realistic target. Control of *S. aureus* is a slow business and farmers and herdsmen must listen to the advice of their veterinarians about mastitis control, apply ALL the strategies with vigour and keep faith that it will make a difference long-term. Finally to avoid the problem of the “hump” at 170,000 cells/ml (shown in Fig. 2) creating a buffer to national progress we would suggest that a policy of a price penalty based on overall SCC contribution to the general milk supply above a threshold of 115,000 cells/ml (our Control ICSCC threshold) would be a better approach.

**Developing decision support for the farmer**

Admittedly culling in a real dairy herd has even more constraints than in our DP Model but using this information we can develop tactics for the field from how the model is approaching this problem. Once it is established that *S. aureus* is a major cause of subclinical mastitis in the herd and that many high ICSCC cows are infected by this organism then the herd is open to the application of the culling strategies developed by the DP. These are governed by the extent of infection and its output.

The DP Model makes an economic case for increasing the voluntary culling of herds with a high level of infection by selecting and culling lower yielding high ICSCC cows at approximately double the rate recommended for the Control herd. The way in which the Model develops this culling in either the Infected or Control herd, both with Average outputs, is detailed below in Table 5 and Figure 3. These show that the DP Model is consistently culling more cows with both a lower ICSCC and yield in the Infected herd than the Control herd.

**Table 5. Optimum culling rates in Control, Intermediate and Infected herds with Average outputs.**

<table>
<thead>
<tr>
<th>Category of infection</th>
<th>Culling rates computed by DP Model</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Involuntary</td>
<td>Voluntary (for low yield and high ICSCC)</td>
</tr>
</tbody>
</table>

The Model suggests that in order to achieve the most economic return the “Infected” *S. aureus* herd can “afford” to annually cull approximately 5% more of the herd than the Control. An in-depth study of the way the DP targets these cows shows that this culling is particularly concentrated in the first and second and again at the 4th and 5th lactations in the Infected herd by comparison to the Control (Fig. 3). This finding for early lactations has not been described before and emphasises the value of such a study in developing novel targeted decision support.

**Figure 3.** Optimum replacement rates for Control and Infected herds with average outputs by lactation number.

In conclusion the DP Model makes an economic case for an increase in voluntary culling against a high level of infection by nearly two times the level seen in the Control herd and for particularly targeting the 1st, 2nd, 4th and 5th lactation cows. Doing this will improve the financial performance of the herd quite considerably because it is able to move away from higher penalties and stay safely in lower bands. There are economic differences between the low and high yielding herds largely related to the milk output and this does affect culling to a limited extent. Essentially culling rates in infected herds are highest in those herds with a high milk yield. High output infected herds could justify about 2% more voluntary culling than low output infected herds.

Tables 6 and 7 show the threshold points for culling taken by the model for the Control and for the Infected herds.

**Table 6.** Maximum ICSCC (’000) and associated minimum yield (kg) that results in a 'keep' decision in a herd with Average outputs.

<table>
<thead>
<tr>
<th>Lactation</th>
<th>Control’ Herd</th>
<th>Infected’ Herd</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICSCC</td>
<td>Yield</td>
<td>ICSCC</td>
</tr>
</tbody>
</table>
Table 6 shows how high the yield of the cow at lactations between 1 and 5 has to be in order to offset a high ICSCC and ensure a 'keep' decision. On the other hand Table 7 shows how low the ICSCC has to be to offset a low yield. In essence the pressure for culling very low yielding cows is very large and ICSCC has little influence!

Table 7. Minimum yields (kg) and associated ICSCC (’000) that results in a 'keep' decision in a herd with Average outputs

<table>
<thead>
<tr>
<th>Lactation</th>
<th>'Control' Herd Yield</th>
<th>ICSCC</th>
<th>'Infected' Herd Yield</th>
<th>ICSCC</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4145</td>
<td>31</td>
<td>4145</td>
<td>57</td>
</tr>
<tr>
<td>2</td>
<td>4869</td>
<td>31</td>
<td>4869</td>
<td>66</td>
</tr>
<tr>
<td>3</td>
<td>5208</td>
<td>33</td>
<td>5208</td>
<td>66</td>
</tr>
<tr>
<td>4</td>
<td>5541</td>
<td>38</td>
<td>5288</td>
<td>41</td>
</tr>
<tr>
<td>5</td>
<td>5770</td>
<td>38</td>
<td>5770</td>
<td>57</td>
</tr>
</tbody>
</table>

These boundaries are shown graphically in Figs. 4 and 5, which illustrate the decision to keep a cow (shaded) or cull (unshaded). In lactation 1 the thresholds are very similar regardless of herd level of infection. Although culling criteria are similar in lactation 1, culling rates will be higher for infected herds as more cows will exceed the threshold ICSCC level (Fig. 3). The effect at this lactation is however small, with about 5% of lactation 1 cows falling into the replace category for infected herds. The equivalent figure for control herds was 4%.

Figure 4. Shading represents a 'keep' decision, unshaded 'replace' in this simple culling guide showing the difference between Infected and Control herd for lactation 1 (based on yield and ICSCC) in a herd with average output.
The situation in lactation 5 contrasts with lactation 1. For yields of 7,800 l or more in control herds, the optimal decision is to 'keep' regardless of ICSCC. For infected herds, consistently high ICSCC is a culling criterion regardless of milk yield. For cows with an ICSCC of 400,000 cells/ml or more, the decision is always to replace. This explains the lower plateau for infected herds in Fig. 5. These widely differing culling criteria and the wider range of ICSCC found in infected herds leads to quite different culling outcomes in lactation 5 (Fig. 3). For the infected herd, 36% of cows at this lactation fall into the 'replace' category. The equivalent figure for the control herd was 9%.
Figure 5. Shading represents a 'keep' decision, unshaded 'replace' in this simple culling guide showing the difference between Infected and Control herd for lactation 5 (based on yield and ICSCC) in a herd with average output.

In summary, ICSCC is an important culling criterion for herds heavily infected with *S. aureus* mastitis but must still be considered even in herds which, like the Control herd, have a low level of infection. The culling recommendations vary greatly between lactations and between infection status. This study illustrates how useful decision support strategies can be provided by simple modelling techniques. We feel that this DP Model fairly reflects the bio-economic situation in quite a number of dairy herds in the UK but we acknowledge the limitations of the data on which the Model is based. There is a need for further work to examine the sensitivity of culling guides to the assumptions on which the DP model is based and to test these against practical experience. This will establish whether the quite simple rules of thumb illustrated here are adequate for decision support or if more sophisticated farm-based computer models are required to customise the support to individual circumstances. A targeted intervention study of herds with various BTSCC figures and levels of *S. aureus* infection would achieve these aims.

We have also made some preliminary studies as to how a reducing level of infection can be incorporated into this DP Model. However this has not proved easy and will need further work and ideally access to a large reliable source of information on the interaction between infection rate and culling. Finally our experience with this DP approach leads us to believe that it can be expanded towards a more comprehensive system encompassing all five of the major pathogens associated with mastitis.

ACKNOWLEDGEMENTS
The Milk Development Council financed this work and in addition SAC receives financial support from the Scottish Executive Rural Affairs Department. We also gratefully acknowledge the help and advice of Victoria Edge, Julie Fitzpatrick & James Booth, all the Milk Buyers in Scotland who contributed to the data in Fig. 1 and finally the veterinarians and farmers who have helped in our investigations.

REFERENCES


A PRACTICAL EVALUATION OF MILK CONDUCTIVITY MEASUREMENTS

HELEN BIGGADIKE (1), IAN OHNSTAD (1), DR ERIC HILBERTON (2) (1)ADAS Bridgets, Martyr Worthy, Winchester, Hampshire, (2) Institute for Animal Health, Compton, Newbury, Berkshire

SUMMARY

The milk electrical conductivity of 31 cows milked with a Liberty automatic milking system was monitored for 15 weeks. An alert triggered when conductivity rose by 17.5% corresponded with a rise in cell count to more than 400,000 cells/ml. When a trigger equivalent to a cell count rise to more than 200,000 cells/ml was applied then the sensitivity for mastitis detection was 80% and the specificity was 63%. When milk samples were analysed for pathogenic bacteria then the rate of false positive triggers was 12%.

INTRODUCTION

Mastitis results in changes in the electrical conductivity of milk, primarily because of changes in the concentration of sodium, potassium and chloride ions. Measurement of conductivity can therefore assist in the early identification of mastitis. Equipment is commercially available for the in-line measurement of milk conductivity but currently there are no agreed guidelines on how best to use this information to maximise the sensitivity and specificity of these measurements. Milk conductivity values can show substantial variation in the absence of mastitis due to factors such as stage of lactation, milking interval and oestrus (1). These non mastitis factors complicate the interpretation of conductivity changes and the accurate selection of cows for early antibiotic therapy. There is currently a desire to reduce total antibiotic use, improve the targeting of antibiotic used and improve the bacteriological cure of udder infections. Early identification of mastitis using changes in electrical conductivity and early antibiotic intervention has been shown to be an efficient method of achieving a bacteriological cure in cows infected experimentally with Streptococcus uberis (2). Early identification and treatment of mastitis has the potential to improve the efficacy of treatment, improve bacteriological cure rate and hence reduce recurrence rates and potentially overall antibiotic usage per cow. A recent field study was undertaken by ADAS Bridgets and the Institute for Animal Health to evaluate individual quarter conductivity and its relation to somatic cell count (SCC) and mastitis.

OBJECTIVE

Evaluation of individual quarter conductivity and its relation to somatic cell count and mastitis
MATERIALS AND METHODS

Data were collected from 31 Holstein cows in a 70 cow herd for a period of 15 weeks between November 1999 and February 2000. Cows were milked through a two-box Liberty automatic milking unit that had the facility to monitor conductivity changes on an individual quarter basis. The system was set to record increases in conductivity of 10% or more when compared to the mean of that quarter over the previous 14 milking. Milk yield, milking interval and days in milk were collected at each milking and analysed on a whole cow basis, somatic cell count samples were collected weekly and analysed on an individual quarter basis and bacteriology samples were collected from 35 quarters following a conductivity trigger and were also analysed on an individual quarter basis. All data were collated to give weekly means resulting in a total of 465 ‘cow-weeks’ of data for whole cow values or a maximum of 1600 ‘quarter-weeks’ of data for individual quarter values.

RESULTS

Occurrence of conductivity triggers

Over the recording period 194 cow-weeks (42%) had one or more quarter conductivity triggers, 271 (58%) had no trigger. The overall occurrence of conductivity triggers by cow-week is summarised in Table 1.

Table 1. Frequency of conductivity triggers by week of study

<table>
<thead>
<tr>
<th>Week</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3 or 4</th>
<th>Total 1 or more</th>
<th>Total cow-weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weeks 1-15</td>
<td>271 (58)</td>
<td>83 (18)</td>
<td>68 (15)</td>
<td>43 (9)</td>
<td>194 (42)</td>
<td>465</td>
</tr>
</tbody>
</table>

Assessed on a quarter-week basis, 310 quarter-weeks (20%) had a conductivity trigger of 10% or more, while 1229 quarter-weeks (80%) had no trigger.

Somatic cell count and occurrence of conductivity triggers

The geometric mean SCC was significantly higher for quarter-weeks in which there was a conductivity trigger than for quarter-weeks with no trigger (P<0.001). This is summarised in Table 2.
Table 2. Mean log SCC for quarter-weeks with and without conductivity trigger

<table>
<thead>
<tr>
<th>Occurrence of conductivity trigger</th>
<th>Geometric mean SCC</th>
<th>SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>3.52 (34)</td>
<td>0.044</td>
</tr>
<tr>
<td>Yes</td>
<td>5.28 (196)</td>
<td>0.087</td>
</tr>
</tbody>
</table>

Figure 1. Trend in geometric mean SCC at increasing conductivity trigger percentages

Geometric mean SCC at increasing conductivity trigger percentage

The geometric mean SCC was significantly higher for quarter-weeks with a greater increase in conductivity (P<0.001). The geometric mean SCC was lowest for quarter-weeks with no conductivity trigger, SCC increased as the conductivity trigger percentage increased from 10% to 20%. This is illustrated in Fig. 1. The numbers shown above the histogram bars indicate the number of quarter-weeks in each conductivity band category. With the exception of the small number of quarter-weeks with a conductivity trigger in excess of 30%, quarter-weeks in conductivity trigger bands greater than 17.5% had a mean SCC in excess of 400,000 cells/ml.

Estimation of sensitivity and specificity

Each quarter-week was categorised as uninfected, with a SCC ≤ 200/000 cells/ml, or infected, with a SCC >200,000/ml, and then this was related to the percentage of quarter-weeks which had a conductivity trigger of 10% or more. This is summarised in Table 3.
Table 3. Estimation of false positive and false negative conductivity triggers (SCC at 200,000/ml)

<table>
<thead>
<tr>
<th>Conductivity trigger</th>
<th>Quarter SCC ≤ 200,000 cells/ml (%)</th>
<th>Quarter SCC &gt;200,000 cells/ml (%)</th>
<th>Total numbers</th>
</tr>
</thead>
<tbody>
<tr>
<td>None (&lt;10%)</td>
<td>1080 (70.2)</td>
<td>149 (9.7)</td>
<td>1229 (80)</td>
</tr>
<tr>
<td>10% or more</td>
<td>162 (10.5)</td>
<td>148 (9.6)</td>
<td>310 (20)</td>
</tr>
<tr>
<td>Total numbers</td>
<td>1242 (81)</td>
<td>297 (19)</td>
<td>1539 (100)</td>
</tr>
</tbody>
</table>

Just below 10 percent of quarter-weeks had no conductivity trigger but had a SCC of more than 200,000 cells/ml and were, therefore, described as false negatives. Just over 10 percent of quarter-weeks with a conductivity trigger of 10% or more had a SCC of less than 200,000 cells/ml and were described as false positives. From these data the sensitivity was calculated to be 50 percent and the specificity 87 percent. The positive predictive value was calculated to be 48 percent and the negative predictive value to be 88 percent.

Bacteriology

Over a period of 14 weeks, 35 quarter samples were submitted for bacteriology. Of these samples, nine were later discarded being classed as repeat samples from quarters that had previously been sampled and had the same pathogen isolated. Of the remaining 26 samples, 24 had a SCC of more than 200,000 cells/ml, 16 revealed major mastitis causing pathogens and eight samples contained mixed growths or contaminants making positive identification of the mastitis causing pathogen impossible. Two samples contained no pathogens. Overall, three of the samples (12%) were categorised as being false positives. All three had a SCC of below 400,000 cells/ml (42,000, 116,000 and 353,000 cells/ml) and none had mastitis pathogens identified.

Milk yield and occurrence of conductivity triggers

Cow-weeks with no conductivity trigger had a significantly higher mean milk yield compared to cow-weeks with one or multiple quarter triggers (P<0.001). This is illustrated in Table 4.

Milking interval and occurrence of conductivity triggers

There was a significant difference in milking interval between cow-weeks with zero, one or multiple quarter conductivity triggers (P=0.01). Although no clear pattern occurred, there was a trend for cows with one quarter (8.7 h), two quarters (9.2 h) or three or four quarters (8.3 h) triggered to have a longer milking interval than those with no quarter triggers (8.4 h).
Table 4. Milk yield/milking for cow-weeks with no, one or multiple quarter conductivity triggers

<table>
<thead>
<tr>
<th>Number of quarters/cow triggered</th>
<th>Mean milk yield (kg/milking)</th>
<th>SEM</th>
<th>Number of cow-weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>8.4</td>
<td>0.16</td>
<td>228</td>
</tr>
<tr>
<td>1</td>
<td>7.7</td>
<td>0.27</td>
<td>83</td>
</tr>
<tr>
<td>2</td>
<td>7.4</td>
<td>0.29</td>
<td>68</td>
</tr>
<tr>
<td>3+</td>
<td>6.6</td>
<td>0.37</td>
<td>43</td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**DISCUSSION**

Identifying high risk quarters as those with a 10% or greater increase in milk electrical conductivity compared to the mean conductivity of the previous 14 milkings resulted in 20% of quarter-weeks and 42% of cow-weeks being triggered. As the conductivity trigger increased above 10% the geometric mean SCC also increased.

To make use of conductivity changes in the early detection of mastitis a high level of sensitivity and specificity is desirable to avoid ‘missing’ infected cows or treating uninfected cows unnecessarily. A quarter SCC of 200,000 cells/ml was used as the divide between infected and uninfected quarters, as recommended by the International Dairy Federation (3). Using these criteria the data indicated a specificity of 87% but a sensitivity of only 50% suggesting that under this system only a relatively small percentage (13%) of uninfected cows would be wrongly identified and treated unnecessarily but an unacceptable percentage (50%) of infected cows (SCC>200,000/ml) would be missed. It has been reported (1) that at low conductivity trigger thresholds the frequency of false positives was high (low specificity) and that even with high sensitivity and specificity, a high level of false positives can occur if the prevalence of clinical mastitis is very low (4). The positive predictive value, which expresses the probability that the quarter with a positive conductivity trigger is infected, was 48% while the negative predictive value, which expresses the probability that a quarter with no conductivity trigger is not infected, was 88%. The assessment made from the small subset of bacteriology samples, however, suggests that 88% of conductivity triggers were associated with either a raised SCC or the presence of mastitis causing pathogens or both and it can be concluded that the quarters sampled for bacteriology were not representative of the whole population of study cows.
Milk yield and milking interval were both significantly different in cow-weeks with conductivity triggers and agree with the trends reported in earlier research. Conductivity has been reported as being higher in cows with longer intervals between milkings (5) and typically cows in early lactation (higher yields) have lower milk conductivity than cows towards the end of lactation (6).

CONCLUSIONS

Over a 3-month period, 42% of cow-weeks and 20% of quarter-weeks had an increase in quarter conductivity of 10% or more compared to the mean quarter conductivity of the previous 14 milkings.

Geometric mean SCC was higher in quarter-weeks with a 10% plus increase in conductivity compared to quarters with a conductivity change of less than 10%.

When determined only on weekly SCC measurements the specificity of this system was estimated to be 87%, the sensitivity only 50%, the negative predictive value 88% and the positive predictive value only 48%.

The limited number of bacteriology samples indicated a higher specificity than that estimated with SCC data alone with the number of false positives being estimated to be only 12 percent.

ACKNOWLEDGEMENTS

The Milk Development Council funded this project. Milking facility and cows were made available and sampling undertaken by Mr C Hamilton. Technical support was provided by Liberty Milking Systems.

REFERENCES

HOW I PRODUCE HIGH QUALITY MILK

LIZ BEST, Poole Farm, Leighterton, Glouc.

INTRODUCTION

The farm is run by my husband Chris and I, with me doing all of the milking. We started in Somerset on a 15 acre small-holding in 1983. After several applications we obtained the 43 hectare tenancy of Poole Farm in 1985 part of the Gloucestershire County Council estate. Like everyone else milking cows, we have enjoyed the short good spells and endured the hard times over the past 15 years, from quotas being brought in just as we started to today’s milk price. Our problems are increased by the limitations of the farm, particularly its size with no opportunity to expand, and its shape. The buildings are across the road from the grazing which is wedge shaped with the farm at the point of the wedge. We are adopting two strategies to stay afloat. First, Chris works long hours off the farm thus I have a full day every one of the 7 days a week. Secondly, we are now 5 months into organic conversion. It appears that trying to sustain our standards will bring us new challenges when we lose many of the tools in current use.

THE FARM

We have 65 cows, mostly black and white but with a few Brown Swiss, breed our own replacements and calve all year round. We graze from April to November on grass and stubble turnips. There are 20 sheep on the farm to help to maintain the pastures. Previously we grew maize but not this year, we are trying lucerne.

Production is creeping up, averaging 7235 litres in 1999, with a target of 7500 litres in 2000. We manage 3.30% protein and 4.20% fat.

One of the first views of the farm on arrival is the dairy. This is our shop window. The aim is to keep the rest up to this standard.
The structure of the farm is such that when the cows are out then they are out. When in, there are 62 cubicles, straw on mats, plus a loose housing area for the calving cows, pens for young stock and separate boxes for isolation if needed. When housed, the cows have access to the outside yard and a feed area. These areas are scraped twice daily.

Dry cows, and any lame animals, are on loose-housed straw until they can cope with cubicle life. Heifers are integrated with the cows so that they lie in cubicles and are accustomed to passing through the parlour. Heifer calves are kept with the cow for 4 days, bull calves are kept with the cow for 3-4 weeks, until they are fit for market.

Since moving to Poole Farm we have upgraded the parlour, in 1992. It is a simple but effective 6-stall tandem, jar plant where the cows are fed all their concentrates. We have a new bulk tank (1997) and a new plate cooler to help with every other day collection, we have improved the muck and effluent storage and, when we can, we try to improve the feeding and cubicle areas.
One of our main goals is to obtain the highest possible standards in milk quality. The Bactoscan is usually below 10 and our cell count below 60,000 cells/ml. We do not have individual cows above 200,000 cells/ml, at least not for long. Health of the cows is a priority. We achieve this by

**VIGILANCE** - we look at everything that is going on, try to spot when things are going wrong and attend to them immediately. This is essentially attention to detail which starts with being tidy around the farm. Everything has its place and is kept there, maintenance is a priority but essentially we try to keep everything clean and working properly.

**CLEAN, DRY and COMFORTABLE** – we start with everything clean. This means it is easier to keep everything clean. This includes the parlour, the equipment and machinery, the yards and the cubicles. To be clean needs the cows and the beds to be dry. We have easy access to good straw that we keep undercover, except for some round bales. The beds are deep. The cubicles are cleaned off twice daily and straw added daily.

**MONITORING** – we keep a close watch on the cows, checking foremilk, using the CMT and paying close attention to individual cow cell counts. Any cow over 200,000 cells/ml is looked at carefully. We pay particular attention to mastitis control. Cows get individual attention. This system allows us to spot even the smallest behavioural changes.

All teats are washed and dried with individual paper towels at every milking. This allows us to feel the udder, changes here often indicate mastitis before clots appear in the foremilk. Disinfectant is sprayed after milking and the cows stand, being fed big bale silage in a clean yard, for at least 20 minutes.

High quality milk is our goal. Fig. 1 shows the Bactoscan results for 32 weeks, it has never exceeded 10 and averages less than 7 for this year. This year the cell count averages 74,000 cells/ml (Fig. 2), the same as for the equivalent period in 1999. The rolling annual average is 73,000 cells/ml.

![Figure 1. Monthly bulk tank Bactoscans](image-url)
All cases of mastitis are treated. We average about 25 cases/100 cows/year using approximately 2 intra mammary tubes per case. *Streptococcus uberis* is, not surprisingly given our straw bedding, the principal cause. That is why so much attention is given to bedding and calving areas. This includes the calving paddock used in the summer, it has no bare areas. Until now we have dry cow treated all cows. Culling has usually been only 20% with few animals culled for mastitis. Our health programme is developed and maintained in close association with Philip Marsh, our vet. A major part of our system depends on us keeping good records and using the information we get from NMR etc. Converting to organic dairying will require us to review much of what we do, especially managing dry cows.

**SUMMARY**

In summary we produce high quality milk consistently by attention to cleanliness and detail. We need to produce the very best to minimise our costs, to stay in dairying and because we owe it to our cows. Dairying is our chosen way of life so we want to do it properly.
MILK QUALITY - A RETAIL PERSPECTIVE

C M BROWN, Marks & Spencer, Baker St, London W1A 1DN

Marks & Spencer has a principal aim in food retailing.

It is to be:

'The most enticing, innovative, high quality and trusted chain of food stores in the world'

The customers of Marks & Spencer milk, expect several key areas to be right, each time milk is purchased. The milk has to be

- Fresh tasting without off-flavours or odours
- Hygienic, lasting in the fridge
- Produced from cows, which have good welfare conditions and are fed the right types of feed

Milk is perceived as wholesome. It is used to feed the newborn, infants and young children. This leads to greater customer expectation than for other products. In response our producer specification includes details on all these aspects.

As a product, milk is perceived as having several positive attributes

- It is natural, healthy, fresh and produced from friendly farming.
- Animal welfare is not seen as an issue with cattle outside, where they are eating grass and are publicly visible.

There are some negatives aspects

- Milk is perceived as having high fat levels. This fat is not considered to be 'good' fat as it is saturated animal fat.
- Occasional issues of pesticide residues and any health-related matters cause consumer concern.

Milk hygiene has not been directly mentioned. The public has little or no understanding of antibiotic use or mastitis problems in dairy production. This places obligations on the milk-producing sector. It is important that there should be no cause for the consuming public to become concerned over these issues. An insurance policy of maintaining and improving hygiene standards has to be in position. The areas of potential concern have to be researched and counteracting strategies developed. For example, the strategy of reducing the level of mastitis by increasing antibiotic use is not a solution. Neither is the use of filtration or dilution a suitable response to high cell count milk.
Marks & Spencer consider milk hygiene to be extremely important in their purchase specifications. The Marks & Spencer Code of Practice for Dairy Production has hygienic milk production as a high priority.

This Code requires, for example:

- An animal health plan to be developed in consultation with the responsible veterinary surgeon
- Interior walls, floors and ceilings to be in sound condition and able to be kept clean
- Milking machinery to be serviced annually with further testing according to the manufacturers recommendations.

Further medium and long-term aims in the area of milk hygiene have been developed and are used to provide guidance to milk suppliers.

To emphasise, there is a crucial need to protect milk's image with consumers. There are also direct benefits from hygienic milk production. Cheese yield is reduced in high SCC milk compared to low SCC milk. The spores from spore forming bacteria can pass through milk pasteurisation causing spoilage problems. Additionally, legislation is reflecting consumer concerns over food safety with changes in EU and UK law.

In conclusion, milk is a product with which consumers identify many benefits. This status place responsibility on the industry to maintain and improve the attributes of fresh, high nutritive value and wholesomeness. High hygienic standards are essential to support and protect the high status for milk with consumers.
THE COST OF MASTITIS - AN OPPORTUNITY TO GAIN MORE MONEY

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SUMMARY

The total cost of mastitis at herd level will consist of four main fractions. 1: The loss due to milk quality. 2: The loss due to less efficient milk production due to chronic subclinically infected cows. 3: The loss due to discharged milk, veterinary fee and antibiotics due to treatment and 4: The loss due to increased replacement rate or culling of cows at suboptimal time in lactation. Under Norwegian condition with low somatic cell counts the largest loss is due to treatment cost of clinical cases (48 %) and replacement costs (27 %). The distribution of these costs will vary from farm to farm and region to region according to the mastitis control strategy and environmental condition. The distributions of the costs will indicate the correct way of putting in place a mastitis control program. The total losses are not the important figure. The most important for the farmers and veterinarians are how much these costs can be reduced. The reduction in costs is the hidden benefits in a mastitis control program that will encourage the farmer to improve udder health and make a market for preventive veterinary medicine. These profits are not very obvious and it is a pedagogical difficulty to envisage the figures and thus be able to take out the increased gross margin.

(Note 1 NOK = approximately 13 £)

INTRODUCTION

The cost of mastitis is very apparent for most people working with dairy cows, however, the correct costs are not easy to calculate. Farmers will be focused on the most understandable costs that are veterinary fees, antibiotics, extra labour and discharged milk. However, veterinary fees in a high-quality practise could also be seen as investment in a certain situation. Data from Norway has shown that there is very little or marginal association between treatment rate of mastitis and the gross margin in the farm economics. Why is that? It is because the treatment cost is only parts of the total mastitis cost, where most of the cost are so called hidden costs, where therapy could be seen as an investment to decrease these hidden costs.

Cost of mastitis can be looked upon at different levels as quarter level, cow level, herd level, regional level, country level and world level. At each level an impression of the cost will be important for decision making like: Should the quarter be dried off or treated? Should the cow be treated or culled? What is the benefit or potential for preventive investment in a herd? What is the correct strategy for mastitis control at regional or national level? In this lecture I will concentrate on herd level, which is from preventive site the most interesting and difficult. Some of the calculation at herd level could also be used at cow level, as the herd very often will add up the effects of all cows in the herd.

The costs will be presented and discussed in four parts.
1. Quality cost
2. Cost in loss of production
3. Cost of therapy
4. Cost of replacement
Finally the possibilities of doing correct and wrong investments in mastitis control is discussed.

**QUALITY COST**

Milk is one of the most important and natural nutrients in the world. It is used for consumption, cheese and ingredients in other foodstuffs. In later years there has been overproduction of milk in the western part of the world in connection with a competition from other foodstuffs. When there is overproduction and competition the prices will go down. Under such circumstances it is even more important to keep the quality at a very high level. Mastitis is per definition an inflammation in the udder (1). Such inflammation will change the blood circulation and thus also influence the production of milk within the udder. These changes are well documented and can be summarised as:

- Decreased production of lactose
- Decreased production of casein
- Increased influx of blood proteins
- Increased influx of blood cells
- Increased influx of enzymes (especially proteolytic)
- Increased influx of salts
- Increased influx of immunoglobulines
- Changes in fat quality
- Decreased quality of fat membrane
- Etc, etc.

These changes starts already at a SCC at 100,000 per ml (2)

All these changes will have an effect on the quality of milk and milk products. Some of these changes are:

- Unstable and rancid taste of milk
- Less cheese yield from the same amount of milk
- Longer renneting time in cheese production
- Less stability of cheese texture and taste
- Longer whipping time for cream

In 1990 there was at least 30 references on the relation between milk quality and products quality (3).

All these changes will make a less valuable product on the market and the product will be unstable in quality after shorter time of storage. This all will have an impact on the consumers attitude to the product and thus also to the willingness to buy the products at a high price in the long run.

It is this fact that has imposed many dairies to put withdrawals and premium of milk delivered from farms to the dairy industry. Because the dairy industry have seen the extra value of a good quality products. These premium and withdrawals are mostly based on SCC because SCC reflects the inflammation process and thus the changes in milk composition. It is not the cell count by itself that is important but the association of SCC with the changes in composition. In Norway (TINE Dairy Association) these limits are at premium at below 230,000 per ml (+ 4.5 % of milk price of 3.05 NOK), first withdrawal at 300,000 BMSCC per ml (- 2.0 %), second withdrawal at 350,000 per ml (- 4.0 %) and warning and closing the delivery at 400,000 per ml. These quality limits should reflect the value of the changes in milk composition under high quality conditions. It is hard to argue for those specific limits, as the changes in milk starts at around 100,000 per ml in SCC and progress continuously upwards at a logarithmic scale (4). However, this is a decision making process and will at time reflect the goal for each dairy industry and the standard of products placed on the market.
The regulatory limit of 400,000 per ml is more relevant from the aspect of safety for human consumption as the higher the SCC the higher is the risk of the cow having mastitis or other pathogens that will have impact on human health in some or another way.

The quality loss is originally a loss that will affect the dairy processor. It most therefore be the decision of the dairy processor if this cost is distributed to the farmers that contribute most to bad quality or is distributed evenly to all farmers through the economic impact the quality will have on milk prices. It is of course, hard to argue for a specific limit and specific prices at each limit. In addition there are lots of technical problems in calculation of these figure in a faire way. This is outside the scope of this paper.

However, there is no date that without premium or withdrawals there is very few incentive factors from the farmers point of view to improve milk quality through mastitis control acting at subclinical infections, except for hidden costs. An effect of these limits could probably be seen in the decrease in BMSCC in Norway since 1984 and increased treatment of mild clinical cases at the same time (figure 1).

**Figure 1.** BMSCC and incidence rate of clinical mastitis treatment in Norway 1980 till 1999.

**PRODUCTION LOSS**

It is well known and well documented that inflammation and also mastitis is connected to loss in function (functio leasa) one of the cardinal symptoms of inflammation. The loss of production in association with SCC is well documented by both Raubertas and Shook (5) and Hortet et al. (6). Both groups illustrate a difference in production loss from first calvers and older cows. The production loss is also correlated with the logarithmic scale of the cell count. Figure 2 illustrate the
association in milk production loss and SCC. Some Norwegian data also illustrate the association at herd level between BMSCC and production per cow-year.

Milk production loss is not obvious for the producer because this is milk never produced and never seen. It is therefore a hidden cost or lost opportunity income. Another problem with this cost is to put a price on it. As the milk is not produced one should probably withdraw the feed cost from the opportunity income, as milk could not be produced from any feed. Another argument (and there is some indication) is that the inflammation process also needs some energy. This extra energy should also be counted for.

![Graph](image)

**Figure 2.** Production loss associated with SCC (according to Raubertas and Shook (5) and produced milk per cow-year associated with BMSCC (data from Norway).

In Norway we have tried to calculate this loss by estimate the theoretical loss in production according to the recorded BMSCC using the results from Raubertas and Shook (5). The estimated loss is then multiplied by the gross margin for milk production (NOK 2.50). Another argument from farmers is that due to milk quota there is no need for increased production, however, there is other tools to meet the goal of the quota at correct time and level.

**LOSS DUE TO THERAPY**

The loss due to therapy is very obvious and visible. There are very few arguments against the losses, although also the argument of filled quota can be put in place. The loss due to therapy will consist of:

- Value of discharged milk
- Value of fed milk minus saved calf feed
- Veterinary fees
- Cost of antibiotics or other therapeutics
- Extra labour due to therapy

Eventually milk losses due to decreased production, decreased quality is accounted for in the two previous chapters and eventually early culling will be accounted for under replacement costs.

The discharged milk will be the daily production at the time of onset of a clinical case or therapy and usually multiplied with 8 days. The 8 days are usually four days of therapy and additionally four days of withdrawal time due to residuals. This amount of milk will be multiplied with the milk
price given to the farmer (the opportunity income if the milk was delivered to the dairy processor). There is no withdrawal for feed costs because the milk is already produced and the effect of feed is lost. Some of this milk could in some farms be used as calf feed. If so we will withdraw the value of saved feed for calves.

The veterinary fee and antibiotic cost is fairly easy to identify and this is what the most farmers account for as the total cost of mastitis and nothing more. In one sense in fact it is an investment when the situation is put in place. The total loss due to clinical treatments according to daily milk yield is illustrated in figure 3.

![Cost vs. Loss in NOK](image)

**Figure 3.** The increased losses due to clinical cases of mastitis according to the daily milk yield.

Extra work is also a cost when treating clinical cases of mastitis. The problem is how to calculate the price per hour of work. In agriculture unfortunately opportunity income could be rather small at least for the few hours struggling with a mastitis case. Another argument could be willingness to pay for getting rid of all the frustration and stress associated with clinical cases of mastitis.

**REPLACEMENT COST**

The replacement cost due to mastitis is probably one of the largest costs. However, it is also a hidden cost and thus a hidden opportunity income. It is very difficult if possible to calculate in a correct way. Several ways of doing this have been presented. Some have tried to calculate every cost from birth to culling and compared with healthy cows not culled. Others have been building very complex models using linear programming to optimise the decision on replacement. However, every cost by raising a heifer is very difficult to find and is probably not even needed. A more pragmatic way is to state that the market value of an animal is at a certain time the sum of the cost of raising the cow plus the fee for doing that. In this way one could withdraw the income from a slaughtered cow (the culled cow) from the price of a pregnant replacement heifer. This difference will be the extra replacement cost arguing that the opportunity income of that heifer is the value by not needing her for replacement but putting
Figure 4. Example of the dynamic costs and income during a replacement, according to Østerås (7).

Figure 5. The simulated replacement cost at culling in different stages and different lactation's according to Østerås (7).

her out for sale on the market for live animals. Alternatively one could argue that if the farmer do not have a heifer to replace from his own herd he have to buy one also at the market of live animals. Under this concept the frame of the replacement cost will be the value of a pregnant heifer minus the value of the slaughtered dairy cow that are replaced. This difference could then be reduced according to the expected lifetime of a cow (example four years). However, we would like to correct this difference by the extra amount of milk an older cow will give (or first calving cow would give approximately 1.500 litre of less milk). Also we would correct for the value of part of an extra calf to be born when the replacement rate is increased.

Another concept is to simulate the production line of two cases (figure 4). One of a cow living for five years and another line with one replacement at different points in lactation and different lactation's as done by Østerås (7).

Figure 5 illustrate that the optimal replacement cost when replacing a mean Norwegian dairy cow with a mean Norwegian heifer vary from approximately 200 US$ till 400 US$. This optimal
replacement stage in lactation is at month 4 to 6 dependent on lactation number. Figure 5 also illustrate that culling in first lactation month will give a replacement cost of approximately 400 US$ till 800 US$. Culling at this stage in lactation will often be due to diseases like milk fever, mastitis or others. This indicates the lost opportunity income if the disease would not occur and is thus a hidden cost of disease. Probably replacement costs is one of the largest costs of dairy diseases.

DISTRIBUTION OF DIFFERENT COSTS IN MASTITIS (NORWEGIAN CONDITIONS)

Figure 6 and 7 illustrate the distribution of cost of mastitis under the Norwegian suppositions. Figure 6 illustrates that the total mastitis loss has decreased from 384 million NOK in 1991 till 245 million NOK in 1999 or 23.3 øre per litre milk delivered to the dairy processors in 1993 till 15.9 øre in 1999, a reduction of 32 percentage.

![Graph showing total mastitis loss in Norway from 1989 till 1999.](image)

**Figure 6.** Total mastitis loss in Norway from 1989 till 1999.
Figure 7 illustrates the distribution according to the different fragments of mastitis loss. As an example the mastitis loss in 1989 was 22.0 mill due to quality, 114.2 mill due to production loss, 170.7 mill due to clinical mastitis and 60.9 mill due to replacement cost. In 1999 the same figures were 3.2, 56.4, 119.8 and 65.4 respectively. This is all at the supposition set in 1989. If these suppositions are changed in 1999 to the real 1999 money values the losses would be 3.4, 32.5, 115.8 and 65.4 respectively.

**Figure 7.** Distribution of the total mastitis loss to quality, production loss, clinical mastitis and replacement during 1989 through 1999.

It is relevant to register that the farmers are very keen of keeping the quality premium because they see the benefit at each delivery and is thus very encouraged to improve BMSCC. However, the economic effect at farm level totally is very small. The effect this motivation has on production loss and milk quality is fare more. Setting up premium levels has therefore been very successful.

**Evaluating the Cows Place in the Farm (Cow Level)**

At cow level it is very important for a farmer to know if this cow in a certain situation is so valuable in the future that she can give a better future value than a heifer replacement. The calculation to do so would be mainly the same as at the herd level. As example the cost of therapy have to balance the gain in expected higher milk price (due to premium), expected higher production and less chance of culling and later clinical cases. If the future value despite therapy is less than the future value of the replacement heifer the cow should be replaced. It is complicated to do such estimation exact, however, it is now through spreadsheet and other tool possible to do so.

**The Benefit of Mastitis Control According to Opportunity Income (Herd Level)**

As stated earlier in this lecture the total loss is relatively unimportant if it is not possible to decrease it by reasonable tools or changing management. An example of the total mastitis loss and possible investment strategy is illustrated in Table 1.
Table 1. Estimated total mastitis loss and possible investment in a herd with 117 cow-year in 1998.

<table>
<thead>
<tr>
<th>Estimated loss</th>
<th>NOK</th>
<th>Investment</th>
<th>NOK</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quality loss (210 BMSCC, 4month lost premium)</td>
<td>19,411</td>
<td>Income in n year</td>
<td>5 years</td>
</tr>
<tr>
<td>Production loss (210 BMSCC)</td>
<td>43,558</td>
<td>Interest rate</td>
<td>5 %</td>
</tr>
<tr>
<td>Loss due clinical mastitis (88 cases)</td>
<td>88,510</td>
<td>Net present value</td>
<td>223,617</td>
</tr>
<tr>
<td>Replacement cost (20 replacements)</td>
<td>40,099</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total mastitis loss</td>
<td>191,578</td>
<td>Proposed investment</td>
<td></td>
</tr>
<tr>
<td>Goal to reduce the cost</td>
<td>27 %</td>
<td>First year</td>
<td>5,000</td>
</tr>
<tr>
<td>Potential extra income each year</td>
<td>51,650</td>
<td>Second year</td>
<td>5,000</td>
</tr>
<tr>
<td>Estimated interest rate</td>
<td></td>
<td>Thereafter yearly</td>
<td>500</td>
</tr>
</tbody>
</table>

This herd was loosing 190,000 NOK per year in 1998 with a potential gain of approximately 50,000 NOK each year if reducing with 27 %. If the investments were 5,000 first and second year the interest rate would be above 2000 %. A tremendous large interest rate. However, we also see that the limit for investment lasting for 5 years are approximately 220,000 NOK. Close to this amount of money the approximation of reducing the loss is being critical. The closer to this limit you are the more confident one should be on the outputs of that investment. With large investments and uncertainty the output of the preventive work is a risky process. The possibility to loose money will be large.

Table 2. Estimated total mastitis loss and possible investment in a herd with 117 cow-year in 1999.

<table>
<thead>
<tr>
<th>Estimated loss</th>
<th>Investment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quality loss (214 BMSCC, 4 month lost premium)</td>
<td>17,762</td>
</tr>
<tr>
<td>Production loss (214 BMSCC)</td>
<td>43,193</td>
</tr>
<tr>
<td>Loss due clinical mastitis (51 cases)</td>
<td>44,192</td>
</tr>
<tr>
<td>Replacement cost (9 replacements)</td>
<td>17,726</td>
</tr>
<tr>
<td>Total mastitis loss</td>
<td>122,873</td>
</tr>
<tr>
<td>Goal to reduce the cost</td>
<td>18 %</td>
</tr>
<tr>
<td>Potential extra income each year</td>
<td>22,117</td>
</tr>
<tr>
<td>Estimated interest rate</td>
<td></td>
</tr>
<tr>
<td>Estimated interest rate</td>
<td></td>
</tr>
</tbody>
</table>

In this herd (a real case) there was put pressure on milking routines (management changes) and selective dry cow therapy as well as teat dipping. There was a large *Staph. aureus* problem. Results the next year are illustrated in Table 2.

We see that the herd has gained approximately 68,000 NOK and there had been a large interest rate of the invested money. There is still a way to go according to the Norwegian standard. The loss per cow is still 1,040 NOK while the Norwegian mean is 850 NOK. The loss could still be reduced by 18 % to meet the Norwegian general standard. With the same investment for five years the interest rate of the money would be 342 %.

If instead of 5,000 NOK each year the investment was increased to 10,000 each year (more consultants) the interest rate will decrease to 120 %, still a incredible large interest rate. However, if the farmer and the veterinarian decided to invest approximately 90,000 NOK in the barn or milking equipment the interest rate went down to - 20 %. The farmer would loose money supposed the same gaining time and a reduction of 18 % of mastitis loss. A sensitivity analyses will show that during the same span of time the reduction must be 25 % of the total loss then the gain would be 11%
interest rate. The same result was obtained with 18% reduction in loss and a gain over 10 years. Eleven per cent is still a fairly high interest rate, however it is close to zero and there is a warning saying that you have to be careful or very convinced on the effect of the proposed investment.

In conclusion there could be stated that in preventive medicine there is not long distance from large money earned till large money lost. This, because we are working with biological processes that are influenced by lots of factors. If one or more of these factors are out of normal range the whole biological system is becoming unstable. Unstableness is typical for biological systems under diseased conditions. Therefor preventive work is a risky business where there is very close range from large money earned till large money lost. This risky process could be more safe only in one way - knowing the biological system and how it works not only from reading books but most important with experience from the field.

REFERENCES


MARKETING MASTITIS SERVICES

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Marketing is about identifying and satisfying customer needs. It is estimated that each year in the U.K. the losses due to mastitis and related milk quality problems exceed £100m, or almost £50 per dairy cow. This identifies a need to tackle the problems. The market relates to any dairy farmer who is having mastitis related problems. Mastitis is probably the costliest disease in the U.K. and these losses are easy to quantify. The farmer can see a deduction, as a somatic cell count penalty, from his milk cheque and is also aware of losses when he is discarding milk from treating clinical cases.

REASONS TO TACKLE MASTITIS PROBLEMS

1. High bulk tank somatic cell count
2. High Bactoscan and/or TBC
3. High incidence of clinical mastitis
4. Sub optimal animal welfare
5. Quality assurance.

During the current period when there has occurred a significant reduction in milk price a dairy farmer has to try to maintain profitability by reducing costs and losses. In the past many dairy farmers were prepared to accept a higher level of short-term loss as they could recover these when they were paid much more for milk. The great majority of dairy farmers now or in the future cannot sustain these losses.

For many farmers price economics focuses on achieving a low somatic cell counts and a low milk bacterial content. Some farmers are not concerned about the level of clinical mastitis occurring provided they do not have price deductions for cell count or Bactoscan. It is estimated, at present, that each case of clinical mastitis costs about £80. This varies from £30 for a very mild case that responds swiftly to treatment up to £700 for a severe Escherichia coli mastitis resulting in the death of a cow. These figures are based on the milk price, cull cow value etc. at 2000 prices.

Herd health plans are going to be introduced to all dairy farms over the next three years. Some farmers already have a herd health plan, following the BCVA (British Cattle Veterinary Association) format. This allows for the monitoring of various diseases including mastitis and will allow the veterinary surgeon to be able to quantify losses relating to mastitis and identify a need for improvement. In addition, milk buyers are more concerned about quality assurance and animal welfare, and as mastitis is responsible for reduced welfare of the cow, herds with high incidence will be expected to take appropriate measures to reduce the incidence of this disease.
Mastitis cannot be eradicated. It is estimated that clinical mastitis costs the dairy industry £100m each year and it should be possible to be reduce these costs significantly in the majority of dairy herds by adoption of appropriate control measures.

SKILLS

Some mastitis problems are reasonably easy to identify and resolve. However, there are a considerable number that are complex. For this reason a considerable degree of skill and knowledge is required in order to be able to advise and problem solve effectively. The ideal person to advise is the veterinary surgeon, however the level of knowledge of the new graduate is quite limited and so extra training is required.

These extra skills can be provided by CPD (continuing professional development) such as attending specialists meetings (including the British Mastitis Conference), reading, spending time with experienced people relating to mastitis, and membership of specialist associations such as the US NMC (National Mastitis Council). An enthusiastic approach to this subject is also needed. It is also very important that each person realises his or her limitations so that extra help can be drawn on as necessary.

SPREADING THE WORD

The fatal error is to assume that just because you are knowledgeable or enthusiastic about a particular topic that everyone knows this. It is important to market your services and constantly remind farmers that you want to work with them to help resolve mastitis problems. This can be achieved by supplying newsletters, attending farmer meetings, arranging discussion groups, and generally taking the time and effort to talk to dairy farmers about their specific problems, aims and goals. All too frequently people are rushing around from A to B without taking the time or trouble to talk to their clients or customers about their problems or specific needs. Time spent in this area will pay healthy dividends.

IDENTIFYING PROBLEM HERDS

Problem herds can be identified from a variety of sources. These include the following data.

1. Somatic cell count results
2. Bactoscan results
3. DAISY or other computer data
4. Intra mammary tube sales
5. Herd health plans and monitoring for quality assurance

Target levels for cell count and Bactoscan will predominately be set by the dairy company who establish a threshold value above which the farmer will be penalised. The aim is to remain below this threshold at all times. There is a considerable difference in threshold value between dairy companies. These values will vary according to the end use of milk and the value that the milk buyer puts on the different components.
Irrespective of these targets it is advisable that all herds have a somatic cell count in the order of 100,000 to 150,000 cells/ml. This will ensure that animals will have a low level of sub-clinical mastitis, little damage to udder tissue and therefore maximise yield.

All herds should be able to have a mastitis rate of 30 or less. The mastitis rate is the number of cases of mastitis per 100 cows per year, where one case of mastitis is one quarter infected once. This allows a comparison of clinical mastitis irrespective of herd size. There is a considerable variation in the mastitis rate between herds, and many farmers will be unaware that they have a clinical mastitis problem, as this data may never have been worked out.

Once you work out the mastitis rate, the cost of clinical disease to the dairy herd per annum and quantify the potential savings, dairy farmers suddenly become very interested in mastitis control. This is one of the benefits of computerised disease recording, herd health plans and regular monitoring of disease.

Having identified suitable dairy herds the next step is to be able to go on farm and tackle the problem.

**APPROACH TO THE PROBLEM HERD**

It is important that the person who is advising has an enthusiastic approach and a good knowledge of the subject. Once a person realises that the problem is beyond their limitations, and then the problem should be referred to a suitable expert. Many people feel that if they have to refer the subject to someone else it is a sign of failure. This is far from the case as the primary aim is to provide a solution as swiftly as possible at a reasonable cost.

A referral to an expert in mastitis control will help ensure that the issues are addressed fully, other areas that may have been overlooked are identified and the referring veterinary surgeon leaving extra skills from the person carrying out the referral as does the dairy farmer. Overall this is a win: win situation.

It is advisable that if the problem is to be referred this occurs early on in the investigation. Some of the most successful referrals that the author has been involved in have resulted from the referring vet ringing up knowing that the problem is out of his depth and this has resulted in early intervention, with a rapid resolution without unnecessary expense.

**SUMMARY**

There are large potential savings to be made in the majority of herds in mastitis and milk quality. The cost benefit of advice is very high and while this may initially be to resolve an existing problem, this will also help prevent other problems occurring in the future.

There are many dairy farmers who are unaware that they have a problem that can be addressed and result in an increase in profit, better animal welfare, improved milk quality and more satisfaction from their farming enterprise. It is important to try and identify these farms and work with dairy farmers rather than assuming farmers will come forward knowing that they have a problem. A proactive rather than a reactive approach is required and this will help to maintain the profitability of dairy farms in the future.