

## MASTITIS INCIDENCE IN QUARTERS WITH DIFFERENT INFECTION STATUS AT DRYING OFF AND CALVING IN TWO TREATMENT GROUPS

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### SUMMARY

A within-herd comparison of teat dipping and dry cow therapy (full treatment) with only selective dry cow therapy (partial treatment) was carried out in six commercial dairy herds for a 2-year period. In the dry and periparturient period the quarters in the full treatment group that were least susceptible to mastitis were those uninfected at drying off: in the partial treatment group quarters infected with *C. bovis* at drying off were the least susceptible and micrococcal infected quarters the most susceptible to mastitis. The infection status of quarters at calving had little effect on their susceptibility to clinical mastitis in the full treatment group, while in the partial treatment group quarters infected with *C. bovis* and micrococci were less susceptible. Withholding dry cow therapy in uninfected and micrococcal infected quarters resulted in an unacceptably high rate of new major pathogen infection. Withholding teat dipping had little effect on levels of subclinical major pathogen infection but was associated with a higher level of minor pathogen infection and a lower incidence of coliform mastitis.

### INTRODUCTION

Many factors predispose to bovine mastitis (Stovlbaek-Pederson, 1975) and an understanding of these is essential for systems of effective mastitis control to be formulated. The presence of non-pathogenic bacteria on body surfaces can protect against invasion by more pathogenic organisms (Savage & McAllister, 1970). Bacteria of low pathogenicity (minor pathogens) are frequently isolated from the healthy bovine udder and may play an important role in protecting the udder from infection with pathogenic bacteria.

Quarters with *C. bovis* and micrococcal infections are reported to be less susceptible to superinfection with major pathogens (Black, Bourland & Marshall, 1972a; Black, Marshall & Bourland, 1972b; Bramley, 1975). It might be advantageous to adopt a mastitis control system that encouraged a high prevalence of these infections.

Routine teat dipping and dry cow therapy, as well as successfully reducing levels of major pathogen infection (Kingwill *et al.*, 1970), have an even greater effect on reducing levels of minor pathogen infection (Bramley *et al.*, 1976). It has been suggested that this has increased the number of quarters susceptible to infection by opportunist pathogens and has been responsible for the increased importance of coliform mastitis in recent years (Eberhart, 1975).

In this paper, mastitis rates in quarters with different infection status are compared both between and within two treatment groups. Conclusions are drawn about the effects of the cessation of routine teat dipping and dry cow therapy on levels of minor pathogen infection and the mastitis susceptibility of individual quarters.

### MATERIALS AND METHODS

Five of the six herds which participated in the trial were established herds; the sixth was a new herd built up entirely from down calving heifers. The five established herds had used teat dipping and routine dry cow therapy for over 4 years and had had a problem with coliform mastitis. All these herds were free from *Streptococcus agalactiae* infection, had rolling mean herd milk cell counts below 500 000/ml and at the start of the trial had less than 15% of quarters infected with major pathogens. In the 2 years preceding the trial, the herds had used 3.9 milking cow intramammary tubes/cow/year (range 1.7–6.3).

The cows in each herd were divided into two groups of similar parity. Cows in the full treatment group received dry cow therapy in all quarters and were teat dipped with an iodophor disinfectant after each milking. In the partial treatment group cows were not teat dipped and only received selective dry cow therapy based on the bacteriological examination of drying off quarter milk samples; only quarters found to be infected with a major pathogen received dry cow therapy. Heifers joining the herds were allocated in turn to each group as they calved.

At the start of the trial, teat dipping and routine dry cow therapy were stopped in the partial treatment group. To allow time for the cessation of teat dipping to have an effect before drying off and to ensure that cows in the partial treatment group had had a dry period without routine therapy, cows in both groups came on trial from the beginning of the dry period which occurred more than 2 months after the start of the trial.

Milk samples at drying off, calving and from clinical mastitis cases were usually taken by herdsmen and were refrigerated as soon as possible after collection. All samples were taken after discarding foremilk and thorough cleansing of the teat end with cotton wool soaked in methanol.

Bacteriological examination of milk samples was carried out in the practice laboratory by inoculating 0.01 ml of milk on to aesculin nutrient blood agar plates. After incubation for 48 h at 37°C, provisional identification of bacteria was made on the basis of colony morphology and where necessary confirmatory tests. Strains of *Staph. aureus* which did not produce  $\alpha$ - and  $\beta$ -haemolysins were identified by performing slide or tube coagulase tests using bovine plasma (Julian & Sissons, 1970). Inulin and raffinose sugar reactions were used to identify *Str. uberis*. Coliform isolates were subcultured on to MacConkey agar plates to determine growth characteristics and lactose utilization. The identification of unusual or atypical isolates was carried out by staff at the Central Veterinary Laboratory.

Macroscopically normal milk samples from clinical mastitis cases were subjected to a Whiteside test (Schalm, Carroll & Jain, 1971). Samples which gave a negative reaction were excluded from the results. A quarter was considered to be infected if five or more colony forming units of a recognized mastitis pathogen were present on a plate; quarters were resampled if fewer colony forming units were obtained or if the samples were contaminated.

A monthly visit was made, usually during afternoon milking, to check teat dipping procedures, to assess the environment of the cows and to deal with any problems arising during the course of the trial. In two herds, milk sampling and treatments at drying off were carried out by one of the authors. In the others, the herdsmen were given written instructions on which cows and quarters were to receive dry cow therapy.

## RESULTS

There were large differences between the treatment groups in the infection status of quarters at calving and drying off (Table I), when there were significantly more uninfected quarters in the full treatment group and more *C. bovis* infected quarters in the partial treatment group. Significantly more quarters were infected with major pathogens at calving in the partial treatment group but the difference was insignificant at drying off. In the partial treatment group there were more quarters infected with micrococci at drying off but at calving the prevalence of these infections was similar in both groups.

**Table I**  
**The infection status of quarters at drying off and calving**

Infection status	Calving		Drying off	
	Full treatment	Partial treatment	Full treatment	Partial treatment
Uninfected	1424*	986*	1008†	747†
Infected with major pathogens	142*	268*	175	197
Infected with <i>C. bovis</i>	100*	319*	585†	715†
Infected with micrococci	115	125	104†	188†
Total	1781	1698	1872	1847

\*†Significant ( $P < 0.05$ ) difference between treatment groups.

During the dry period (Tables II, III), nearly twice as many quarters became infected in the partial treatment group due to large and statistically significant differences ( $P < 0.05$ ) in the susceptibility of uninfected quarters and those infected with micrococci. Quarters in the partial treatment group, infected with major pathogens or *C. bovis*, had a similar susceptibility to corresponding quarters in the full treatment group.

In the full treatment group quarters already infected with minor pathogens at drying off were significantly ( $P < 0.05$ ) more likely to develop new subclinical or clinical major pathogen infections in the dry period than quarters which were uninfected at drying off.

In the full treatment group quarters already infected with *C. bovis* at drying off were significantly ( $P < 0.05$ ) more likely to acquire new subclinical or clinical major pathogen infections than uninfected quarters treated at drying off. In contrast, in the partial treatment group, *C. bovis* infected quarters were significantly ( $P < 0.05$ ) less likely to develop mastitis in the dry period than uninfected quarters.

**Table II**  
**The rates° of clinical mastitis associated with new infections in the dry and periparturient periods in quarters of different infection status at drying off**

<i>Infection status</i>	<i>Rates°</i>	
	<i>Full treatment group</i>	<i>Partial treatment group</i>
Uninfected	1.7*	4.8*
Infected with major pathogens	6.9†	6.1
Infected with <i>C. bovis</i>	3.6†	2.5†
Infected with micrococci	1.9*	9*†
Total	2.8*	4.5*

°Cases of mastitis/100 quarters.

\*Significant ( $P < 0.05$ ) difference between treatment groups.

†Significant ( $P < 0.05$ ) difference between infected and uninfected quarters in same treatment group.

**Table III**  
**The rates° of new major pathogen infection during the dry and periparturient periods in quarters of different infection status at drying off**

<i>Infection status</i>	<i>Rates°</i>	
	<i>Full treatment group</i>	<i>Partial treatment group</i>
Uninfected	6.6*	14.9*
Infected with major pathogens	14.3†	18.3
Infected with <i>C. bovis</i>	10.2†	13.4
Infected with micrococci	7.7*	22.9†*
Total	8.5*	15.5*

°New infections/100 quarters.

\*Significant ( $P < 0.05$ ) difference between treatment groups.

†Significant ( $P < 0.05$ ) difference between infected and uninfected quarters in the same treatment group.

In the partial treatment group, quarters infected with micrococci were significantly ( $P < 0.05$ ) more likely to develop clinical mastitis and to acquire new major pathogen infections than quarters uninfected at drying off in the same group (Tables II and III).

In both treatment groups quarters infected with a major pathogen at calving (Table IV) were nearly three times more likely to develop clinical mastitis in the 3 months after calving than were uninfected quarters. Of these cases, 70% were associated with the same major pathogen as was found at calving and many were likely to be persisting infections. The probability of quarters infected with major pathogens at calving developing clinical mastitis caused by a different pathogen was similar to that of uninfected quarters.

**Table IV**  
**The rates<sup>o</sup> of clinical mastitis during the first 3 months of lactation in quarters of different infection status at calving**

	Rates <sup>o</sup>	
	Full treatment group	Partial treatment group
Uninfected	5.9	6.6
Infected with major pathogens	19.7 <sup>†</sup>	17.2 <sup>†</sup>
Infected with <i>C. bovis</i>	8.0*	2.2 <sup>†</sup> *
Infected with micrococci	7.8	4.0
Total	7.2	7.2

<sup>o</sup>Cases of mastitis/100 quarters.

\*Significant ( $P < 0.05$ ) difference between treatment groups.

<sup>†</sup>Significant ( $P < 0.01$ ) difference between infected and uninfected quarters in the same treatment group.

In the partial treatment group quarters infected with *C. bovis* or micrococci at calving were less likely to develop clinical mastitis in the first 3 months of lactation than quarters uninfected at calving, but the difference was only statistically significant for *C. bovis*. In the full treatment group the differences in the susceptibility to mastitis of *C. bovis* or micrococcal infected quarters on the one hand and uninfected quarters on the other were statistically insignificant.

In the total there was no difference in the susceptibility to post-calving mastitis in the two treatment groups: this was despite the fact that *C. bovis* infected quarters were nearly four times as likely to develop clinical mastitis in the full treatment group than in the partial treatment group.

In both treatment groups quarters with different infection status at calving showed little difference in their susceptibility to acquiring new major pathogen infections before the next dry period (Table V).

**Table V**  
**The rates<sup>o</sup> of new major pathogen infection during lactation in quarters of different infection status at calving**

Infection status	Rates <sup>o</sup>	
	Full treatment group	Partial treatment group
Uninfected	11.2	13.6
Infected with major pathogens	12.0	13.1
Infected with <i>C. bovis</i>	14.0	12.5
Infected with micrococci	14.8	15.2
Total	11.7	12.4

<sup>o</sup>New infections/100 quarters.

In the full treatment group, 23 of the 24 coliform infections first found clinical in the first 3 months of lactation, occurred in quarters which were uninfected at calving: this is significantly ( $P < 0.05$ ) higher than the rate of new infections first found clinical due to other major pathogens which occurred in previously uninfected quarters (Table VI). In the partial treatment group, the incidence of coliform mastitis and mastitis due to other major pathogens was similar in quarters that were uninfected at calving. Major pathogens other than coliforms showed no tendency to cause significantly different rates of clinical mastitis in infected or uninfected quarters at calving.

**Table VI**  
New coliform and other major pathogen infections, first found clinical in the first 3 months of lactation, in quarters of different infection status at calving

Infection status at calving	Full treatment group		Partial treatment group	
	Coliform	Other major pathogens	Coliform	Other major pathogens
Uninfected	23 (96%)	34 (69%)	11 (61%)	25 (68%)
Infected with major pathogens	1 (4%)	3 (6%)	6 (33%)	5 (13%)
Infected with <i>C. bovis</i>	0 —	5 (11%)	1 (6%)	5 (13%)
Infected with micrococci	0 —	7 (14%)	0 —	2 (6%)
Total	24 (100%)	49 (100%)	18 (100%)	37 (100%)

## DISCUSSION

In the partial treatment group quarters infected with *C. bovis* at calving were the quarters least likely to develop clinical mastitis during the first 3 months of lactation. This supports the findings of Black *et al.* (1972a, b) and Bramley (1975) and is at variance with those of Honkanen-Buzalski, Griffin & Dodd (1984). Bramley (1975), however, reported no difference between teat dipped and non-teat dipped groups while in this trial *C. bovis* infected quarters were more susceptible to mastitis in the full treatment group than in the partial treatment group.

Bramley (1978) and Linde, Holmberg & Astrom (1975) showed that pre-existing micrococcal infections protected quarters from super-infection with major pathogens during lactation and in this trial, in the partial treatment group, such quarters were the second most resistant group, although they were not significantly less susceptible than uninfected quarters.

During the dry and periparturient periods in the partial treatment group, quarters with untreated micrococcal infections were significantly more at risk. The fact that infection with *C. bovis* reduced the susceptibility of quarters in the dry period while micrococcal infection increased it, may be explained by possible differences in the pathology of these two infections: Black *et al.* (1972a, b) showed that 75% of *C. bovis*

infections are limited to the streak canal and therefore do not induce a significant leucocytosis. The experiments of Bramley (1978) and Linde *et al.* (1975) were carried out in quarters where micrococcal infections had been artificially produced within the mammary gland and had caused a significant leucocytosis. Reiter, Elisabeth Sharpe & Higgs (1970) concluded that the higher rate of infection in the dry as opposed to the lactating udder was mainly due to the cessation of milking which allowed the penetration of pathogens through the streak canal, and not that the dry udder was inherently more susceptible to infection. Bramley (1975) concluded that the reduced susceptibility of micrococcal infected quarters during lactation was due to the increased leucocytosis that the infections caused. However, the ability of neutrophils to phagocytose may be greatly reduced in the dry udder due to the nature of the secretion (Jensen & Eberhart, 1981). Thus in the dry period, streak canal *C. bovis* infections preventing migration of pathogens may have a greater protective effect than intramammary micrococcal infections producing leucocytosis.

The presence of untreated micrococcal infection not only failed to protect quarters in the dry period but actually increased the susceptibility of these quarters to mastitis. It is possible that the presence of certain intramammary infections could interfere with inhibitory mechanisms which help to prevent the dry udder becoming infected with other pathogens. Thus Reiter & Oram (1967) showed that one such mechanism (the lactoperoxidase/thiocyanate/peroxidase system) successfully inhibited the growth of *Str. uberis*, the major pathogen most frequently found causing new infections during the dry period in this trial.

Oliver, Dodd & Neave (1956) reported that cows with a previous history of major pathogen infection, although free at drying off, contracted more than twice as much new major pathogen infection in the dry period as cows with no history of infection. In this trial the increased susceptibility to mastitis of quarters infected with major pathogens, over uninfected quarters in the full treatment group, despite seemingly successful therapy, appears to confirm these findings. At calving, however, quarters with major pathogen infections appeared to be no more susceptible to mastitis caused by new infections than uninfected quarters, perhaps because none of the infections at calving was eliminated by antibiotic treatment.

Armstrong (1977) suggested that routine dry cow therapy should be stopped if coliform mastitis could not be controlled successfully. The significant increase in the level of other major pathogen infections at calving in the partial treatment group suggests that such a procedure would be inherently risky and should only be considered if coliform mastitis is a particular problem at calving, with an associated high incidence of acute infection. If a selective dry cow therapy regimen is to be considered for the control of this disease, the results of this trial would suggest that only *C. bovis* infected quarters should remain untreated.

In contrast, withholding teat dipping in the partial treatment group did not result in a significant rise in the level of major pathogens at drying off, although it did cause significantly lower levels of uninfected quarters due to an increase in minor pathogen infections. The incidence of coliform mastitis in this trial in the full treatment group remained high both in the first and second thirds of lactation (Robinson, Jackson & Marr, 1985), suggesting that teat dipping maintained a high level of uninfected quarters susceptible to coliform infection, and emphasizing the need for strict attention to hygiene if this control system is employed.

*C. bovis* infections protected quarters from super-infection with major pathogens and reduced the incidence of clinical mastitis. Cessation of teat dipping rather than routine dry cow therapy would appear to be a more acceptable way of promoting higher levels of *C. bovis* infection although if very high levels of these infections could be achieved at drying off, perhaps by artificial means, then cessation of dry cow therapy might not result in an increased incidence of major pathogen infections and may be a tenable proposition. The different effects of pre-existing *C. bovis* and micrococcal infections at drying off emphasize the need for the elucidation of the role played by these minor pathogens and the natural defence mechanisms present in the dry udder.

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