Control of Mastitis by Hygiene and Therapy

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ABSTRACT

Hygiene and therapy are two important components of a program to control mastitis. Conscientious application of these practices significantly reduces intramammary infection, especially when they are applied in concert with superior management. The two components operate independently, and response is maximum when both are applied. Hygiene acts by reducing the frequency of infection. The primary effect of therapy is to increase the rate of eliminating established infections, although dry cow treatment also provides prophylactic benefits. The most effective hygiene and therapy practices are dipping of teats after milking and treating each quarter at the end of lactation.

INTRODUCTION

Bovine mastitis is a multifactor disease that continues to be an enigma for the dairy industry. The disease is in every dairy herd, but the amount varies widely among herds. More than 80 species of microorganisms have been identified as causal agents (42). Most infections persist and are not detectable by palpation or examination of foremilk.

If a mastitis control program is to make a significant impact on a population of herds, it must be designed for all herds, not only those with special problems. Programs likely to find acceptance among dairy farmers must be economical, practical, effective under most management conditions, and must reduce the incidence of clinical mastitis. The latter is the first prerequisite of dairy farmers because they recognize clinical symptoms. The subclinical form is accepted with complacency because there is no visible manifestation of the disease or loss from it. Fortunately, the view that clinical mastitis must be reduced is not in conflict with other objectives of a control program because most clinical cases are preceded by subclinical cases.

The dairy industry is indebted to Dodd and co-workers for having developed the conceptual basis for modern mastitis control methods as determined by epidemiologic investigations in commercial dairy herds (12). The amount of infection is a function of the rate of new infection and the rate of elimination of established infections. It is not within the scope of this paper to discuss all ancillary factors that may be employed for prevention and elimination of the disease. Emphasis will be given to the role of hygiene and therapy in control programs.

HYGIENE

Approximately 95% of mastitis is caused by Staphylococcus aureus, Streptococcus agalactiae, Streptococcus dysgalactiae, and Streptococcus uberis. Maximum effort should be focused on controlling these four pathogens.

Transmission of Pathogens

If hygiene methods are to be of value in reducing incidence of new infection, they must prevent, or at least reduce, transmission of pathogens from one teat to another of the same cow as well as from cow to cow. Anything that comes in contact with an infected udder and subsequently touches another udder is a potential fomite. If transmission from udder to udder could be prevented or reduced substantially, there should be a concomitant decrease in incidence of new intramammary infection (IMI). Most transmission of S. aureus and S. agalactiae occurs during the milking process via milkers’ hands, udder cloths, and teat cups.

Some transmission occurs during the interval between milkings. Possible sources include contaminated bedding, licking of teats and udder, and contact of teats with rear legs, tail switch, and flies. Pathogens most frequently trans-
mitted during the intermilking period are *S. uberis* and coliforms. These organisms also may be transmitted during milking, particularly if udders are excessively wet when milking machines are attached.

**Sources of Pathogens**

A knowledge of the epidemiologic patterns of a disease is fundamental to development of adequate control measures. The primary reservoirs of pathogens in herds not using a mastitis control scheme are infected udders, colonized teat canals, and infected teat lesions. The relative importance of primary reservoirs, as well as secondary reservoirs, is peculiar to specific mastitis pathogens.

*Staphylococcus aureus.* Numerous reports have suggested that staphylococci were ubiquitous in the cow’s environment and that infection with them was inevitable. These conclusions were refuted by three studies (8, 34, 57) which emphasized that control of staphylococcal mastitis was dependent upon control of intramammary and udder skin infections. Staphylococci do not persist on healthy teat skin but readily colonize teat canals if a lesion is near the teat apex. Organisms multiplying in infected lesions or colonized teat canals are situated ideally for transfer into the udder.

*Streptococcus agalactiae.* The only significant reservoirs for *S. agalactiae* are infected udders. Although the organism may be isolated from bedding, milking equipment, milkers’ hands, and other objects, its presence is a consequence of recent contamination with infected milk. In absence of IMI the organism will disappear from secondary sites.

*Streptococcus dysgalactiae.* In addition to infected udders, the main sources of this pathogen are tonsils and skin lesions (31). In herds free of IMI with *S. dysgalactiae*, organisms causing new cases of mastitis probably originate from mouths of cows. The organism rarely is recovered from healthy teat skin.

*Streptococcus uberis.* The ecology of *S. uberis* has been studied extensively. Frequency of isolation from udder and teat skin was greater than from within the mammary gland, and skin infections were sometimes independent of IMI (51, 53). Another report indicated that the belly or lips were populated most heavily, that teat skin was a relatively unfavorable site, and that lips were the most favorable site (7). *S. uberis* is a cause of infection prior to first calving and during the dry period.

*Other pathogens.* Coliforms and pseudomonads are the most important secondary pathogens in most herds. Incidence of IMI is generally low, although outbreaks may occur when conditions develop that greatly increase exposure to the organisms. Coliforms are in manure and bedding; pseudomonas mastitis may originate from contaminated water supplies, soil, or inadequately cleaned milking machines.

**Method of Infection**

Mastitis pathogens enter the mammary gland via the teat’s streak canal, but the exact method is not known. The epidemiologic pattern most frequently observed is one of a relatively low infection rate with a high average duration of infection. Infection rates among herds ranged from less than .2 infection per cow annually to more than 2 (14, 31). Reasons for the wide variation are complex. Even under conditions of experimental exposure to large populations of pathogens, it was rare for infection to exceed 1 in 200 cow milkings (31, 37, 41).

The infrequency of new infection suggests that the streak canal is an effective barrier to penetration by bacteria. Microorganisms may overcome the streak canal barrier by multiplication, mechanical movement, or populsion during machine milking. The invasion process may begin during the brief period of milking or during the longer interval between milkings. The effect of various components of a hygiene system on new IMI depends upon both the mechanism of infection and the relative frequencies of infection during the milking and intermilking periods.

Teat lesions, particularly at the teat apex, contribute to increased incidence of IMI. In the absence of a teat dip, teat lesions become infected and act as reservoirs for mastitis pathogens. Clinical mastitis was ten times higher in quarters with a lesion at the teat apex than in quarters with unblemished teats (20).

The sequence of events leading to infection is usually one of progression from pathogen contamination of teat skin to invasion of the teat canal and proliferation within the mam-
mary gland. Theoretically, any factor that increases the number of pathogens on apical teat skin will increase the incidence while factors that decrease pathogen presence will reduce the incidence of infection.

Effectiveness of Hygienic Practices

Disinfection of milkers' hands. Milkers' hands are an important fomite. Contamination may occur when drawing foremilk, handling teat cups, stripping, or touching contaminated objects in the milking barn. Studies in England revealed that 50% of milkers' hands were contaminated before milking compared to 100% during milking (13). Effectiveness of different disinfectants in eliminating pathogens from contaminated hands was studied. An average of 30% of hands yielded positive swabs after being washed with disinfectant compared to over 95% when no disinfectant was used. The wearing of smooth rubber gloves and dipping of gloved hands in a suitable disinfectant before handling each cow reduced manual transfer of pathogens.

Udder washing. Washing udders with a sanitizing solution prior to milking is advocated for purposes of removing dirt and organic matter, destroying mastitis organisms, promoting milk let-down, and improving milk quality. The sanitizing aspect of contemporary udder washing procedures is open to serious question, and it is doubtful that washing can be justified for preventing IMI. Unless properly done, udder washing procedures may serve more to spread pathogens than to destroy them. Staphylococci survived on udder cloths after being soaked in disinfectants for 3 min (13). S. agalactiae survived on cloths for 7 days and were recovered from contaminated cloths after soaking for up to 5 h in 2% hypochlorite (4, 5). Use of single-service paper towels or separate heat-sterilized cloths reduced this source of contamination. Udders should be dried before teat cups are attached.

Disinfection of milking machine teat cups. Teat cup liners are a potent disseminator of pathogens both within and between cows. The number of pathogens recoverable from liners after milking infected cows was related to the number of bacteria in milk from the infected cow and whether the liner came in contact with infected lesions on teat skin (13). Such findings have prompted the almost universal recommendation that teat cups be disinfected between cows. The most common practice has been to dip teat cups in a disinfectant solution for a few seconds. This practice generally reduced the number of pathogens transferred from cow to cow but did not prevent all transfer. Only two cups should be dipped at a time to prevent an air block that prevents the disinfectant from reaching the full length of the liners. The most effective method for preventing transfer on liners involved circulating water through the cluster at 85°C at least 5 s (13). An alternative procedure that is gaining acceptance is back-flushing the teat cups and claw assembly between cows.

Teat dipping. Transfer of some pathogens is inevitable at milking time, even under the best of hygienic conditions. To destroy pathogens remaining on teats at the end of milking, some form of postmilking teat antisepsis is necessary. The most widely used procedure involves dipping teats in a suitable disinfectant soon after milking machines are removed. This technique was recommended first in 1916 (26). A considerable body of evidence has accumulated in recent years to support the concept (15, 29, 30, 33, 36, 37, 49, 50, 56).

Teat dipping with an effective solution will reduce new IMI at least 50% for the majority of dairy herds. Correct use of an appropriate product may be the most effective single prophylactic procedure that dairy farmers can employ for preventing IMI. British workers have demonstrated that dipping of teats at drying off reduced infections in the dry period (35).

The concept of applying postmilking teat sanitizers by spraying is becoming more popular, particularly in larger dairy herds where the milking operation is mechanized. The value of teat spraying is not well documented and limited data suggest that it may not be as effective as dipping (24, 38). One possible reason is insufficient teat coverage. A thorough evaluation of the practice is needed.

Teat dips containing oil as the principal vehicle should be avoided because none of the oil-based dips tested has been effective (41, 48). Oil-based products should not be confused with water-based dips containing small amounts of lanolin or glycerine as emollient.

At least three studies involving single herds
failed to demonstrate efficacy of teat dips reported by other workers to be highly efficacious (17, 27, 47). Reasons for lack of prophylaxis were unclear. It seems likely that the test herds were unsuitable for evaluating efficacy of dips because of a low infection rate with "dip-controllable" infections, or, perhaps, a majority of the infections were machine mediated.

The ability of a teat dip preparation to reduce microbial populations on teat skin is a useful measure of effectiveness. This ability should not be equated with efficacy to prevent IMI. This can be determined only by controlled infection trials.

Though postmilking teat antisepsis usually will reduce the rate of new IMI 50%, the effect on IMI is modest in the short term. This is because dips do not affect established infections. In fact, if a program of milking management could be devised to prevent all new IMI, IMI would fall by less than one-third in a year (9). This explains why dairy farmers do not see prompt evidence of the effect of prophylactic measures on infection. In the long term, mastitis is best held to a low frequency by effective preventive methods such as correct use of functionally adequate milking machines and teat dipping.

THERAPY

It is necessary to increase the rate at which established infections are eliminated to achieve a rapid decline in IMI. This is best accomplished by effective therapeutic procedures. Some of the information reported was taken from materials prepared by the author for the National Mastitis Council.

The aim of antimicrobial therapy is to destroy pathogens without damaging the host. Elimination of pathogens depends upon getting the necessary concentration of the appropriate drug to all the foci of infection for an adequate time. Unfortunately, a wide gap exists between what is known about medicaments and the manner of use.

Types of products. The cure rate achieved by given products is dependent upon dosage, solubility of the drug salt, type of base, frequency of administration, and synergistic or antagonistic action of drug combinations. These subject areas have been reviewed (2, 25, 28, 45, 46, 52, 55, 59).

Extensive use of antibiotics for treating clinical mastitis has not had a major effect on the prevalence of IMI in dairy herds. The prime limitation of relying upon treatment of clinical cases alone was that only 40% of infections identified by laboratory culture were recognized by clinical abnormalities in milk (21). The problem was compounded because a majority of clinical cases appeared to respond to treatment as evidenced by disappearance of clinical symptoms. Unfortunately, many of the infections were not eliminated and persisted at subclinical levels. This is exemplified by data from a study in Louisiana in which 270 cases of clinical mastitis were treated (40). In each case the clinical symptoms disappeared and the affected quarter, seemingly, returned to normal. The cure rates are in Table 1. These rates were similar to reports by workers in England (12, 21). If therapy is to make the desired contribution to mastitis control, it is necessary to treat subclinical as well as clinical cases.

Subclinical mastitis. Treatment of subclinical mastitis during lactation is indicated primarily when the dairy farmer is in danger of losing his market due to a high percentage of cows being infected. The prognosis of treating streptococci is good while staphylococcal infections often are refractory. Intramammary treatment with the full series of recommended infusions yields best results. Intramuscular treatment is often ineffective and is not recommended. To achieve cure rates commensurate with those from intramammary therapy requires exceptionally high doses of antibiotics repeated (59).

Dry cow treatment. The best time to treat most subclinical infections is at drying off. If not treated, most will persist until calving. Advantages of dry cow treatment are that the

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>No. quarters treated</th>
<th>% cured</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staphylococcus aureus</td>
<td>121</td>
<td>24.8</td>
</tr>
<tr>
<td>Streptococcus agalactiae</td>
<td>31</td>
<td>51.6</td>
</tr>
<tr>
<td>Other streptococci</td>
<td>111</td>
<td>36.0</td>
</tr>
<tr>
<td>Coliforms</td>
<td>7</td>
<td>71.4</td>
</tr>
</tbody>
</table>
cure rate is higher than for cows treated during lactation, incidence of new IMI during the dry period is reduced, damaged tissue may be regenerated before freshening, clinical mastitis at freshening is reduced, and salable milk is not contaminated with drug residues (11, 23, 28).

Effectiveness of dry cow treatment is improved by the use of slow-release products that maintain therapeutic amounts of antibiotics for long periods in the dry udder. Products designed for lactating animals should not be used to treat cows at drying off. The preferred time to treat is following the final milking of lactation.

When dry cow treatment is routinely part of a mastitis control program, it is necessary to decide which quarters should be treated. Treatment of all quarters has the advantages of reaching each infected quarter, being more effective in preventing new dry period IMI, and requiring no screening or laboratory procedures. Selective treatment based on clinical history or the results of screening tests may fail to reach 30 to 60% of infected quarters. A theoretical rating of six systems for dry cow mastitis management has been reported (28).

A 4-year study in Louisiana with 267 cows compared four dry cow mastitis management systems on commercial dairy farms (43). Treatments were Group 1, none; Group 2, at drying off; Group 3, at calving; and Group 4, at drying off and calving. Treatments at drying off were single infusions in all four quarters with a dry cow product while treatments on day of calving were single infusions in all quarters with a product formulated for lactating cows. Data are in Table 2. Spontaneous cure rates in untreated quarters were staphylococci, 27%, and streptococci, 70%. Cure rates following treatment at drying off were staphylococci, 53%, and streptococci, 88%. Reductions in the dry period infections were staphylococci, 52%, and streptococci, 61%. Reductions in number of quarters infected at freshening following dry treatment were staphylococci, 48%, and streptococci, 60%. Cure rates following a single treatment at freshening were staphylococci, 20%, and streptococci, 60%.

The IMI 4 wk after calving was highest in Group 1. The lowest IMI was in Groups 2 and 4, which did not differ significantly. Group 3 had a slightly lower infection at 4 wk following calving than Group 1, but the difference was

<table>
<thead>
<tr>
<th>Group</th>
<th>Staphylococci</th>
<th>Streptococci</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>18</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>14</td>
<td>10</td>
</tr>
<tr>
<td>3</td>
<td>27</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>22</td>
<td>6</td>
</tr>
</tbody>
</table>

Table 2. Summary of efficacy data from four dry cow treatment systems.

not sufficient to justify the added effort and expense of treating at calving.

Treatment at drying off reduced the incidence of clinical mastitis from 25 to 11 cases during the critical 1st wk following calving. Treatment only at calving reduced the number of cases from 25 to 17, and treatment both at drying off and calving reduced the incidence from 25 to 8. Differences in incidence between 8 and 60 days after calving were small and nonsignificant among the four groups.

Variation in herd response. Wide variation in response to therapy exists both within and among herds. In one study, involving 30 herds, the cure rate against staphylococci ranged from 5 to 60% in lactation and 30 to 80% at drying off (12). All of the isolates were highly sensitive to the drug used in vitro. In another study, cure rates ranged from 43 to 93% against all pathogens (18).

In a third study, the cure rate was reported at least 50% higher in cows in the first three lactations compared to the sixth or later lactation (16). Treatments administered on the first occasion of clinical mastitis eliminated approximately 50% of infections, but, if that treatment failed, the figure fell to approximately 10% in subsequent treatments (10).

The principal cause of therapeutic failures was tissue barriers rather than drug resistance (44). This was confirmed in a study in Louisiana in which practically all isolates of staphylococci were sensitive to the antibiotics used. The cure rate ranged from 68% when the affected quarter yielded a score of CMT-0 to 13% for CMT-3 quarters (39).

Some of the differences in cure rates among herds may be related to overall herd management. In one study, the cure rate of all infections was 67 and 88% for herds employing good and excellent management skills, respectively (18). A 13% difference in response was reported in excellently and poorly managed herds in another study (6).

Prevention of drug residues. Label directions should be followed carefully and milk from all quarters discarded regardless of the number infused. Milk also must be discarded following parenteral drug treatment. Treated animals should not be sold for slaughter until the drug withdrawal time for meat has elapsed.

Cellular response. Time required for somatic cells to decrease after elimination of pathogens depends upon the organism involved and the amount of tissue damage from the infection. This may range from a few days for some streptococci to months for some staphylococci.

RESULTS FROM FIELD TRIALS

Field trials have been conducted in several countries to measure the effect of practicing good milking management, disinfecting teats after every milking, and treating quarters at drying off with a specially-formulated antibiotic preparation on level of infection. The first three investigations (MFE-1, MFE-2, and MFE-3) were conducted in England.

MFE-1. The study involved 700 to 800 cows in 14 herds for 12 mo (13,33). Seven herds were assigned to a control group and milked without any hygiene except udder washing with a common cloth and warm water. Seven herds were placed on a “full hygiene” system that included a .01% solution of chlorhexidine udder wash, a .5% chlorhexidine teat dip solution, single service paper towels for udder washing, and pasteurization of teat cups between cows. In addition, half of the cows in each group were treated at drying off. Decrease in incidence of new IMI in full hygiene herds as compared to control herds was S. aureus, 40%; S. agalactiae, 29%; S. dysgalactiae, 76%; and S. uberis, 62%.

MFE-2. Three hygiene systems were compared on 1,000 cows in 15 herds for 18 mo (13, 32). Two systems were the same as in MFE-1 except that with the full hygiene system milkers wore smooth rubber gloves, udders were washed with .01% iodophor solution, and teats were dipped after milking in a .5% iodophor preparation. A third “partial hygiene” system was included and was the same as full hygiene except that teat cups were not disinfected. Half of the cows in each group were treated at drying off. Full hygiene reduced new IMI during lactation by 58%, compared to 44% for partial hygiene. Differences were not significant. The investigators concluded that “providing the teats and particularly the ends of the teats were disinfected immediately after the end of milking the spread of pathogens on the milking machine clusters did not greatly affect new infection rates.”

MFE-3. This trial involved 32 herds during 3
yr (22). The partial hygiene routine employed in MFE-2 was followed in 16 herds except that udders were washed in .06% hypochlorite solution. In the other 16 herds, teats were dipped after milking in 4% hypochlorite solution, but no other hygiene measures were applied. Teat cup disinfection was not practiced in any of the 32 herds but infected cows in each group were treated at onset of the trial, and all cows were treated with an improved antibiotic formulation at drying off. Results are in Table 3.

Infection with any pathogen decreased from 27.0 to 7.6% in the partial hygiene group, and from 30.8 to 7.5% in the teat dip only group. Infections caused by *S. aureus*, *S. agalactiae*, and *S. dysgalactiae* declined by at least 75% in both groups. Reductions in amount and rate of infection caused by *S. uberis* were less. Infections with gram-negative organisms remained about the same throughout the 3-yr study. None of the differences between the partial hygiene and teat dip only groups was significant. The new infection rate varied 15-fold among the 32 herds. Wide variation was possibly due to differences in management, environment, strain of organism, milking machines, milking procedures, and presence of teat lesions.

*Cornell study.* The study involved 2,000 cows in 24 herds and was similar to MFE-3 in design and execution. Teats were dipped after milking in 4% hypochlorite solution, and all quarters were treated at the end of lactation. After 3 yr the infection with mastitis pathogens had decreased from 28.1 to 7.1% of quarters (30).

*New South Wales study.* This study was in 35 herds (19). The hygiene routine involved washing the udder with running water and soap containing hexachlorophene and drying with a paper towel prior to milking. Teats were dipped immediately after milking in an iodophor teat dip containing .5% iodine plus glycerine. Treatment was administered during lactation only to quarters with clinical mastitis. All quarters of every cow received treatment at the first dry period with a specially-formulated product in a long acting base. In subsequent dry periods, treatment was administered only to cows whose milk yielded positive CMT reactions during the month prior to drying off. An effort was made to see that the milking machines were adjusted properly on all test farms.

**Table 3. Summary of results from MFE-3.**

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>% quarters infected with</th>
<th>Treatment</th>
<th>Start</th>
<th>1 yr</th>
<th>2 yr</th>
<th>3 yr</th>
<th>1 yr</th>
<th>2 yr</th>
<th>3 yr</th>
<th>1 yr</th>
<th>2 yr</th>
<th>3 yr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any bacteria</td>
<td>27.0</td>
<td>Partial hygiene</td>
<td>2.2</td>
<td>1.1</td>
<td>1.2</td>
<td>1.5</td>
<td>7.6</td>
<td>9.5</td>
<td>11.2</td>
<td>7.6</td>
<td>9.5</td>
<td>11.2</td>
</tr>
<tr>
<td><em>P. aeruginosa</em></td>
<td>7.6</td>
<td>Partial hygiene</td>
<td>2.2</td>
<td>1.1</td>
<td>1.2</td>
<td>1.5</td>
<td>7.6</td>
<td>9.5</td>
<td>11.2</td>
<td>7.6</td>
<td>9.5</td>
<td>11.2</td>
</tr>
<tr>
<td><em>S. aureus</em></td>
<td>7.6</td>
<td>Partial hygiene</td>
<td>2.2</td>
<td>1.1</td>
<td>1.2</td>
<td>1.5</td>
<td>7.6</td>
<td>9.5</td>
<td>11.2</td>
<td>7.6</td>
<td>9.5</td>
<td>11.2</td>
</tr>
<tr>
<td><em>S. agalactiae</em></td>
<td>7.6</td>
<td>Partial hygiene</td>
<td>2.2</td>
<td>1.1</td>
<td>1.2</td>
<td>1.5</td>
<td>7.6</td>
<td>9.5</td>
<td>11.2</td>
<td>7.6</td>
<td>9.5</td>
<td>11.2</td>
</tr>
<tr>
<td><em>S. dysgalactiae</em></td>
<td>7.6</td>
<td>Partial hygiene</td>
<td>2.2</td>
<td>1.1</td>
<td>1.2</td>
<td>1.5</td>
<td>7.6</td>
<td>9.5</td>
<td>11.2</td>
<td>7.6</td>
<td>9.5</td>
<td>11.2</td>
</tr>
</tbody>
</table>

Quarters infected with *S. aureus* decreased from 26 to 13%, *S. agalactiae* from 4.9 to 5%, other streptococci from 3.1 to 2.3%, and CMT positive reactions from 34 to 12%.

**Other studies.** A small trial involving 120 cows for 2 yr was in Pennsylvania (15). Half the cows were subjected to teat dipping with a 1% iodophor product during lactation and dry treatment in all quarters during the 1st and 2nd wk of the dry periods. The infusion product contained $2 \times 10^6$ units of penicillin and 100 mg dihydrostreptomycin. The other half of the cows were untreated controls. Infections with *S. aureus* decreased in the treated group from 9.5 to 2.9% of quarters, and *S. agalactiae* decreased from 21.8 to 1.6%. The control group had much smaller changes. The numbers of new coiform infections were 37 and 25 for the treated and control groups. In a second trial involving 80 cows for 60 wk the overall infection rate was reduced 51.1% in the treated group.

To encourage application of MFE-3 methods, four field trials were implemented in England. Progress was measured by changes in somatic cells in herd milk. The first involved 15 herds in a veterinary practice area for 2 yr (54). The geometric cell level in herds following the control routine were intermediate in response. A total of 386 herds participated involving 500 herds in Somerset for 3 yr (3). In herds in which intensive milking time hygiene was practiced and dry treatment for 3 yr (58). In herds in which intensive milking time hygiene was practiced and dry treatment in all quarters during the 1st and 2nd wk of the dry periods. The infusion product contained $2 \times 10^6$ units of penicillin and 100 mg dihydrostreptomycin. The other half of the cows were untreated controls. Infections with *S. aureus* decreased in the treated group from 9.5 to 2.9% of quarters, and *S. agalactiae* decreased from 21.8 to 1.6%. The control group had much smaller changes. The numbers of new coiform infections were 37 and 25 for the treated and control groups. In a second trial involving 80 cows for 60 wk the overall infection rate was reduced 51.1% in the treated group.

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A trial involving 55 herds, ranging in size from 15 to 120 cows, was conducted in Israel for 3 yr (58). In herds in which intensive milking time hygiene was practiced and dry cow treatment was administered to all cows, infection with *S. aureus* fell from 30 to 10%. The level of IMI was reduced less when control measures were limited to teat disinfection and dry treatment of infected cows only.

**REFERENCES**


