Udder shape and teat-end lesions as potential risk factors for high somatic cell counts and intra-mammary infections in dairy cows

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The association of common bacterial pathogens in milk samples during calving with udder shape or the presence of ‘teat-end’ lesions was investigated in 240 dairy cows from two herds. Sixty-three of 120 cows (53%) in one herd (herd A) and 54/120 animals (45%) in a second herd (herd B) had normal-shaped udders. The remaining animals had udder shapes defined as follows: large pendulous (18% herd A, 26% herd B); large between hindquarter (10% herd A, 17% herd B); overall small (8% herd A, 5% herd B); or small but pendulous (11% herd A, 7% herd B). At calving teat-end lesions were present in 63% and 76% of the quarters of herd A and B animals, respectively. There was no herd effect on udder shape or teat-end lesions.

Analysis of variance revealed that udder shape and teat-end lesions did not have a significant association with quarter somatic cell count. However there was some association between mammary infection and udder shape and teat-end lesions. Compared to other udder shapes, cows with large between hindquarter shape had significantly less Staphylococcus aureus and Streptococcus uberis infection (*P < 0.001). There was a similar albeit less significant negative association with Escherichia coli infection (*P < 0.01). Infection with Streptococcus agalactiae and Streptococcus dysgalactiae was more frequent in cows with large pendulous and overall small udder conformations. The results also suggest an association between intra-mammary infection at calving and the presence of hyperkeratotic teat-end lesions, given that S. aureus, coagulase-negative staphylococci, S. uberis, S. agalactiae and E. coli were cultured from significantly more quarters with such lesions than from quarters without lesions or with other types of lesion (*P < 0.001).

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Introduction

Both clinical and subclinical mastitis cause significant economic losses to the dairy industry in the United Kingdom (Kossaibati and Esslemont, 1997). Several environmental and host risk factors are associated with mastitis and most control measures are directed at reducing their impact. These include good husbandry and milking practices and the judicious use of antibiotics to treat infections and during the dry period. Despite a reduction in the incidence of mastitis through the use of such measures, the condition in the UK has an annual incidence of 40–50 cases per 100 cows (Kossaibati et al., 1998; Bradley and Green, 2001).

Improving environmental hygiene to reduce mastitis is one of the cornerstones of control. Other potential measures to reduce losses due to mastitis include the selection of cows less likely to develop the disease, and the culling of animals more susceptible to it. Previous studies have explored the potential effects of udder and teat-end size and morphology on the risks of developing mastitis (Binde and Bakke, 1984; Seykora and McDaniel, 1985a; Slettbakk et al., 1995). For example, cows with increased milk leakage, increased milk flow, asymmetric udders and flat/wide teat-ends were considered to be more susceptible to clinical mastitis (Slettbakk et al., 1995).

Given that most mastitis-causing pathogens are thought to gain entry to the mammary gland through the streak canal, the presence and shape of teat-end lesions play an important role in the pathogenesis of the disease (Chrysal et al., 2001). A few studies have suggested that some teat-end shapes act as risk factors for mastitis, and, as they have high daughter-dam heritability, can be eliminated by selective breeding (Lojda et al., 1982; Seykora and McDaniel, 1985a; Chrysal et al., 1999). The presence of teat-end lesions are also regarded as important risk factors for mastitis (Sieber and Farnsworth, 1981; Chrysal et al., 2001) and studies have investigated their relationship to somatic cell counts (Chrysal and McDaniel, 1985a; Chrysal et al., 2001). Less pointed and more inverted teat-ends have been associated with increased susceptibility to mastitis as they retain milk which can act as a substrate...
for bacterial growth (Chrystal et al., 2001) and have more dilated sphincters that facilitate bacterial invasion (Appleman, 1973).

A smooth muscle sphincter surrounds the teat canal holding it closed, thus preventing the leakage of milk and serving as the body’s first line of defence against intramammary infection (IMI). After milking, this sphincter remains dilated for 1–2 h, allowing bacteria to enter the teat canal (Sieber and Farnsworth, 1981; Neijenhuis et al., 2000). Therefore teat-ends with a shape that allows the sphincter to remain dilated for longer periods are likely to predispose cows to IMI.

Another source of IMI is the presence of other teat lesions resulting from trauma, chemical injury and external environmental conditions. Trauma occurs during milking following liner slips due to excessive temporary vacuum losses, low vacuum reserve, abrupt removal of the milking unit without shutting off the vacuum, vacuum fluctuations caused by inefficient vacuum regulation, blocked air vents, restrictions in the short milk tube and poor cluster alignment. Such lesions can be readily colonised by a variety of bacteria that serve as important reservoirs of infection. Pathogens may be spread from animal to animal through the use of udder cloths, on milker’s hands or via milking machine components (Bruun, 2003).

Initially, teat-end lesions appear as small white circular or horseshoe-shaped rings around the teat opening. As the lesion develops, the teat opening may turn inside out or protrude and vertically oriented fissures appear in the skin covering the teat (Chrystal et al., 2001). When bacteria enter the mammary gland they multiply and produce toxins, enzymes, and cell-wall components, which stimulate an inflammatory response (Nickerson et al., 1995), the magnitude of which is influenced by the causative pathogen and by cow factors such as stage of lactation, age, immune status, genetics and nutritional status (Hamann and Mein, 1996). In many cases large numbers of neutrophils and other leucocytes migrate to the infected quarters, following chemotactic signals and pass between the epithelial cells into lumen of the lactiferous tubules (Zeconci et al., 1992).

The objective of the present study was to investigate the possible relationship between udder shape and the presence of teat-end lesions and somatic cell counts (SCCs) and bacterial IMI.

Materials and methods

Herd selection

Between September 2003 and May 2005, two commercial dairy farms in North-West Cheshire were recruited on the basis of location, on having non-seasonal calving patterns and on the owners’ compliance with the study protocol. One herd consisted of 120 Friesian–Holstein cows managed under intensive husbandry conditions and the other had the same number of cows of the same breed but was managed under organic conditions. Monthly bulk milk and individual cow SCCs were available as both herds participated in a national milk record scheme.

During the summer months all cows were at pasture and during the winter they were housed in either free stalls or in straw yards. Periparturient cows were kept on pasture and during the winter they were housed in either free stalls or in straw yards. Periparturient cows were kept on pasture in summer and in loose boxes in winter. Cows on both herds were treated for tuberculosis and other diseases.

Milk sampling

Milk samples collected at calving were cultured for bacteria. Duplicate quarter samples were collected aseptically from all cows, labelled with the cow and quarter identification as well as with the date of collection and 15 mL were sent to a laboratory for SCC measurement (On Merit Ltd., Berkshire, UK). A portion of each sample was cultured for bacteria and colonies were provisionally identified on the basis of their gross morphology and number of colony forming units (Cowen, 1974; Quinn et al., 1994). The tube coagulase test (Boerlin et al., 2003) was used to identify Staphylococcus aureus and other coagulase-positive staphylococci from coagulase-negative staphylococci (CNS) and streptococci were further identified on the basis of their ability to split aesculin and on the presence of Lancefield Group polysaccharide antigens. Isolated corynebacteria were not differentiated into species.

All Gram-negative bacteria were tested for the production of oxidase. Escherichia coli and the other oxacide-negative enterobacteria were identified further by assessing their production of indole and urease using a microtube identification system (RapidID 20 E, bioMérieux).

Isolated bacteria were classified either as major (S. aureus, Streptococcus agalactiae, S. dysgalactiae, S. uberis, E. coli, Arcanobacterium pyogenes and Klebsiella spp.) or minor pathogens (Corynebacterium spp. and coagulase-negative staphylococci CNS). Only samples yielding a pure growth of 1–3 species of bacteria were considered to have caused IMI. Samples yielding >3 species were re-cultured and if similar results were obtained, the sample was considered contaminated and was not used in the analysis.

Analysis of data

Data were initially recorded and stored in Excel spreadsheets. Log-transformed data were analysed using the Minitab statistical computer package (release 12.21, Minitab Inc.) and Genstat (Version 8, SAS Institute). The chi-square (p2) test and analysis of variance (ANOVA) were used to determine possible associations between udder/teat confirmation or teat-end lesions and quarter SCC and bacterial IMI.

Results

Udder conformation and teat-end lesions

The distribution of udder shapes and teat-end lesions within the herds is detailed in Tables 1 and 2. In herds A and B, 53% and 45% of cows had udder shapes classified as normal, ‘Large pendulous’ (category 1) and ‘overall small’ (category 3) udder shapes were least frequently described (Table 1). There was no significant difference between the herds with regard to udder shape. In herd A, 45% and 37% of teats had no lesions at drying off and at calving, respectively (Table 2). The corresponding figures for herd B were 44% and 24%, respectively.
Table 3 details the mean and standard deviation of the SCCs of the quarters with different lesions for both herds. An analysis of variance revealed that teat-end lesions did not have a significant effect on quarter SCC (Table 4). On bacterial culture no association was identified between udder shape and IMI (Table 5). However, compared to other udder shapes, IMI with *S. aureus* and *S. uberis* were significantly less common in cows with 'large between hind-quarter'-shaped udders (category 2) (Pearson's $\chi^2 = 21.59$ and 26.07, respectively; df = 3; $P < 0.001$). There was also the same, albeit less significant, trend with IMI with *E. coli* (Pearson’s $\chi^2 = 10.99$; df = 3, $P = 0.012$). *Streptococcus agalactiae* and *S. dysgalactiae* were more frequently isolated from quarters of 'large pendulous' and 'overall small' udders. Therefore overall, the results suggest that at calving, 'large between hindquarter'-shaped udders are associated with lower rates of bacterial IMI.

Table 3
Relationship between presence of teat-end lesions and mean (standard deviation) quarter somatic cell count (SCC) at calving

<table>
<thead>
<tr>
<th>Teat-end lesion (category)</th>
<th>Somatic cell counts (Log$_{10}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RF</td>
</tr>
<tr>
<td>No lesions (0)</td>
<td>5.84 (0.54)</td>
</tr>
<tr>
<td>Smooth/slightly rough ring (1)</td>
<td>5.76 (0.65)</td>
</tr>
<tr>
<td>Rough chronic ring (2)</td>
<td>5.760 (0.71)</td>
</tr>
<tr>
<td>Very rough/severely</td>
<td>5.76 (0.61)</td>
</tr>
<tr>
<td>hyperkeratotic (3)</td>
<td>5.81 (0.63)</td>
</tr>
<tr>
<td>Unclassified (4)</td>
<td>5.65 (0.72)</td>
</tr>
</tbody>
</table>

RF, right front quarter; LF, left front quarter; RH, right hind quarter; LH, left hind quarter.

Table 4
Relationship between teat-end lesions and quarter somatic cell count (SCC)

<table>
<thead>
<tr>
<th>Quarter</th>
<th>df</th>
<th>s.s.</th>
<th>m.s.</th>
<th>v.r.</th>
<th>F pr</th>
</tr>
</thead>
<tbody>
<tr>
<td>RF</td>
<td>4</td>
<td>0.7574</td>
<td>0.1893</td>
<td>0.49</td>
<td>0.741</td>
</tr>
<tr>
<td>LF</td>
<td>4</td>
<td>0.9662</td>
<td>0.2416</td>
<td>0.42</td>
<td>0.795</td>
</tr>
<tr>
<td>RH</td>
<td>4</td>
<td>0.2656</td>
<td>0.0664</td>
<td>0.17</td>
<td>0.955</td>
</tr>
<tr>
<td>LH</td>
<td>4</td>
<td>2.1795</td>
<td>0.7265</td>
<td>2.20</td>
<td>0.089</td>
</tr>
</tbody>
</table>

RF, right front quarter; LF, left front quarter; RH, right hind quarter; LH, left hind quarter; df, degrees of freedom; s.s., sum of squares (residual); m.s., mean square; v.r., variance ratio; F pr, F probability.

Bacterial culture was also used to assess the relationship between teat-end lesions and IMI (Table 6). The results suggest some association between infection and type of lesion. For example, significantly more samples from quarters with very rough/severely hyperkeratotic teat-ends (category 3) yielded growth of *S. aureus* compared to those without or with other types of lesions (Pearson’s $\chi^2 = 26.78$; df = 4, $P < 0.001$). Infection with *E. coli* was also significantly more frequent (Pearson’s $\chi^2 = 38.36$; df = 4, $P < 0.001$) in samples obtained from quarters with very rough/severely hyperkeratotic teat-ends and with unclassified lesions compared to those obtained from normal teats and those with category 1 or 2 lesions. The same trend was observed with *S. uberis*, *CNS* and *S. agalactiae* but not with *S. dysgalactiae* (Table 6). There was no significant difference between the two herds with regard to teat-end lesions.

**Discussion**

The control of clinical and subclinical mastitis in dairy herds depends on the identification and elimination of risk factors associated with the environment, with animal management and with individual animals. Whilst most risk factors associated with management and the environment are addressed by introducing good management and hygiene measures, selecting dairy cows which are less susceptible to mastitis is also a control measure worthy of consideration (Nash et al., 2003). Previous studies have attempted to identify host factors that predispose cows to IMI (Schukken et al., 1990; Burvenich et al., 2003; Bannerman et al., 2008a, b). Whilst an animal's general resistance to infection is important, the obvious target of study is the mammary gland itself. In this context the current study investigated what, if any, effect udder shape and the presence of teat-end lesions have on the incidence of IMIs. The results indicate that less than half of the cows studied had udder shapes that could be described as normal although there was insufficient evidence to associate particular udder shapes with IMI or increased SCC.

The study results did not indicate a clear association between IMI with a particular pathogen and udder shape, but compared to other udder shapes, infections with *S. aureus* and *S. uberis* were significantly less common in cows with large between hindquarter. However, infections with *S. agalactiae* and *S. dysgalactiae* were more frequently detected in animals with 'large pendulous' – and 'overall small' – shaped udders.

A reduction in teat-end-to-floor space is associated with increased SCC (Sletbak et al., 1990) and some udder and teat-end shapes are associated with milk leakage, a significant risk factor for clinical mastitis (Sletbak et al., 1995; Klaas et al., 2005). Increased risk of clinical mastitis has been linked to asymmetric udder balance, in which the caudal half of the organ is larger than the cranial portion (Grohn et al., 1990). High milking rates and large teat-canal diameter have also significant associations with increased SCC (Seykora and McDaniel, 1985a; Jørstad et al., 1989) and increased risk of IMI (Grindal and Hillerton, 1991).

As the teat-end or orifice is the first line of defence against invading bacteria, changes or damage to this part of the udder may reduce its effectiveness in preventing IMI (Sieber and Farnsworth, 1981; Gleeson et al., 2004). It has been reported that changes to the orifice, brought about by milking methods and changes in weather conditions, may favour bacterial invasion (O'Shea, 1987; Gleeson et al., 2004). Sieber and Farnsworth (1981) demonstrated that quarters with severe teat-end lesions and those which were traumatised or leaked milk had increased rates of IMI. In the present study almost every animal had some kind of teat-end lesion on one or more teats, although only a small proportion of the lesions were severe in nature. Sieber and Farnsworth (1981) also reported that teat-end lesions were common...
and that most farmers considered chronic ring lesions to be a ‘normal’ feature of the teat-end.

We found little association between udder shape and SCC score or IMI but there was evidence of some association between infection with nearly all of the major pathogens and CNS and type of teat-end lesion. Significantly higher proportions of quarters with ‘very rough/severely hyperkeratotic’ teat-ends and with unclassified teat-end lesions, were infected with the major pathogens (except S. dysgalactiae) and with CNS and other minor pathogens. Previous studies have shown that the presence of teat-end lesions are associated with increased risk of clinical mastitis but most of the lesions observed in the present study were chronic in nature. Although there are clear associations between acute, severe teat lesions and clinical mastitis (Sieber and Farnsworth, 1981), the role of chronic lesions in this disease is more controversial. While Seykora and McDaniel (1985a) identified a positive association between teat-end lesion score and SCC, Farnsworth (1995) found no relationship with infection prevalence and the presence of hyperkeratotic teat lesions.

Further studies of larger numbers of herds will be necessary to further elucidate the role of teat-end lesions as risk factors for IMI, as the current study and that of Sieber and Farnsworth (1981) indicated the prevalence of these lesions varies from herd to herd.

Conclusions

A few studies have indicated that certain aspects of udder and teat conformation may be useful predictors of the incidence of mastitis in cows (Thomas et al., 1984). Pendulous and deeper udders have been associated with a higher incidence of mastitis (Young et al., 1960), while non-pendulous udders were reported to be more resistant to the disease (Bakken, 1981; Thomas et al., 1984). Although the present study did not investigate the association of teat-end shape with IMI and SCC, previous studies have found that teat-ends that were plate- or funnel-shaped were associated with higher incidences of clinical and subclinical mastitis (Seykora and McDaniel, 1985a; Chrystat et al., 2001). It should be possible to select for animals with udder and teat features that result in a being less predisposed to mastitis, as the heritability of these traits is high (White, 1974; Norman et al., 1983; Seykora and McDaniel, 1985a, b). A large US study found that genetic selection for lower SCC, longer productive life, shorter teats, or closely spaced front teats may reduce the incidence of IMI at first parturition (Nash et al., 2003).

Conflict of interest statement

None of the authors of this paper has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

References


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Table 6

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>No lesions n = 290</th>
<th>Smooth/slightly rough ring n = 350</th>
<th>Rough chronic ring n = 178</th>
<th>Very rough/severely hk n = 74</th>
<th>Unclassified n = 63</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staphylococcus aureus</td>
<td>61 (21.0)</td>
<td>44 (12.7)</td>
<td>22 (13)</td>
<td>28 (38.3)</td>
<td>7 (11.1)</td>
</tr>
<tr>
<td>Streptococcus agalactiae</td>
<td>27 (9.3)</td>
<td>11 (3.1)</td>
<td>0 (0)</td>
<td>11 (15.)</td>
<td>8 (12.6)</td>
</tr>
<tr>
<td>Streptococcus dysgalactiae</td>
<td>24 (8.2)</td>
<td>36 (10.4)</td>
<td>13 (7.3)</td>
<td>4 (5.4)</td>
<td>8 (12.6)</td>
</tr>
<tr>
<td>Streptococcus uberis</td>
<td>34 (11.6)</td>
<td>38 (10.9)</td>
<td>16 (9)</td>
<td>20 (27.3)</td>
<td>15 (23.8)</td>
</tr>
<tr>
<td>CNS</td>
<td>30 (10.2)</td>
<td>35 (10.1)</td>
<td>10 (5.6)</td>
<td>12 (16.4)</td>
<td>10 (15.8)</td>
</tr>
<tr>
<td>Escherichia coli</td>
<td>23 (7.9)</td>
<td>27 (7.8)</td>
<td>28 (15.9)</td>
<td>25 (34.2)</td>
<td>23 (36.5)</td>
</tr>
<tr>
<td>Corynebacterium spp.</td>
<td>25 (8.5)</td>
<td>20 (5.7)</td>
<td>14 (7.2)</td>
<td>19 (26)</td>
<td>10 (15.8)</td>
</tr>
<tr>
<td>Bacillus spp.</td>
<td>23 (7.8)</td>
<td>25 (7.2)</td>
<td>17 (9.6)</td>
<td>9 (12.3)</td>
<td>6 (9.5)</td>
</tr>
<tr>
<td>Other</td>
<td>6 (2.2)</td>
<td>15 (4.3)</td>
<td>11 (6.2)</td>
<td>15 (20.5)</td>
<td>3 (4.7)</td>
</tr>
</tbody>
</table>

1–3 pathogens/quarter; CNS, coagulase-negative staphylococci; hk, hyperkeratotic.

TABLE 6

Relationship between teat-end lesions and intra-mammary infection (IMI) by major pathogens at calving

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Alice L. Bhutto et al. / The Veterinary Journal xxx (2008) xxx–xxx

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4


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