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## Prevalence of clinical mastitis in 38 Waikato dairy herds

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### ABSTRACT

The apparent prevalence of clinical mastitis was determined in 38 spring calving dairy herds as part of a larger field trial conducted on behalf of Boehringer Ingelheim NZ Ltd between the 8th of July and the 21st of August, 1997. A total of 798 quarters from 595 cows were diagnosed by herdowners as having clinical mastitis. Bacterial culture was performed on all clinical quarters and antibiotic sensitivity testing was performed on selected bacterial isolates.

Clinical mastitis was diagnosed in an average of 10.0% ( $\pm 0.8\%$ , range 0.9% to 21.4%) of calved cows within the herds. No bacteria were cultured from 28.2% of clinical quarters. Of the quarters from which bacteria were cultured, 74.7% were *Streptococcus uberis*, 10.2% were coagulase negative staphylococci, 4.7% were Coliforms, 4.1% were *Streptococcus dysgalactiae*, 2.9% were *Staphylococcus aureus* and 3.3% were other species of bacteria. Penicillin resistance was detected in 25% of 44 *Staphylococcus aureus* isolates, 0% of 10 *Streptococcus uberis* isolates and 100% of 12 *E. coli* isolates.

Keywords: Dairy Cattle; Mastitis; Early lactation; Prevalence.

### INTRODUCTION

The prevalence of clinical mastitis in New Zealand dairy cattle has not been evaluated for over 30 years (Brookbanks, 1966). In that time major changes in mastitis control strategies have occurred (Woolford *et al.*, 1995) which are likely to have resulted in changes in prevalence of mastitis pathogens. Clinical mastitis is generally diagnosed and treated by herdowners without knowledge of the causative bacteria. This is because bacterial culture takes 24 to 48 hours to produce a result and because of the cost of the culture. Delaying treatment until culture results are available may result in worsening of the mastitis leading to greater production loss and greater animal suffering. However, only 25% to 85% of cases of clinical mastitis are cured following treatment during lactation (Craven, 1987). Cure rate is influenced by the bacterial species involved, the duration of infection, the type and duration of antibiotic treatment and efficacy of other treatment strategies used (Sandholm *et al.*, 1990). Knowledge of the prevalence of mastitis pathogens and the sensitivity patterns of those pathogens would allow more informed decisions to be made about the likely outcome of treatment, the type and duration of antibiotics to be used and the appropriate ancillary management strategies to be instituted to reduce incidence.

### MATERIALS AND METHODS

Herds (N=38) were enrolled in the study on the basis of willingness of the owners/sharemilkers to co-operate. Herdowners were asked to present for examination all cows that they would normally have treated with antibiotics following a diagnosis of clinical mastitis between the 8th of July and the 21st of August, 1997. Herdowners are

likely to have used swelling, heat or oedema of the udder or presence of clots or blood in the milk as indicators of clinical mastitis. No attempt was made to influence the basis on which herdowners were making the diagnosis of clinical mastitis. Twenty ml of milk from quarters diagnosed as having clinical mastitis were expressed into a 35 ml vial following cleaning of the teat end with cotton wool soaked in 70% methanol. Milk samples were frozen (-20°C) for up to 7 days before bacterial culture (Murdough *et al.*, 1996). Ten µl of milk was removed from the vial with a sterile loop and applied to a quarter plate of 5% blood agar containing 0.1% aesculin (Fort Richard, Auckland). Plates were assessed after 24 and 48 hours incubation at 37°C. Isolates were categorised on the basis of colony morphology, gram stain, haemolysis pattern, aesculin reaction, tube coagulase and CAMP tests. Coliforms were further cultured on MacConkeys agar (Fort Richard, Auckland) and had an oxidase test performed. Major pathogens were defined as *Streptococcus uberis*, *Staphylococcus aureus* and *Streptococcus dysgalactiae* and the minor pathogens included coagulase negative staphylococcus, *Corynebacterium* spp., coliforms (including *E. coli*, *Pseudomonas* spp, *Hafnia* spp.) and *Bacillus* spp. Antibiotic sensitivity testing was performed on selected isolates (44 *Staphylococcus aureus* isolates, 10 *Streptococcus uberis* and 12 *E. coli* isolates) using zone inhibition (Kirby-Bauer) methodology. The antibiotics tested included amoxicillin and clavulanic acid, cephalothin, erythromycin, oxacillin, penicillin, streptomycin, and tetracycline. Alpha Scientific (Hamilton, New Zealand) performed all the cultures and sensitivities.

Prevalence was calculated as the number of calved cows within a herd presented by herdowners following a diagnosis of clinical mastitis expressed as a percentage of all cows in the herd calved by the 21st of August.

## RESULTS

A total of 798 quarters from 595 cows were diagnosed with clinical mastitis. This represents an average of 10.0% ( $\pm 0.8\%$ , range 0.9% to 21.4%) of the cows calved within each herd (Table 1). One, two, three and four quarters from the same cow were diagnosed as having clinical mastitis in 457, 94, 22 and 22 cows, respectively.

**TABLE 1:** Prevalence of clinical mastitis in 38 Waikato dairy herds.

Variable	mean	sem <sup>††</sup>	min	max
Average number of cows in the herd	282.4	15.9	128	491
% of all cows calved by 21/8/1997*	69.4	1.7	50.0	93.1
% cows with clinical mastitis to 21/8/97*	10.0	0.8	0.9	21.4
% culture positive cows <sup>†</sup>	7.0	0.7	0.8	17.6

\* Last day for enrolment for cows;

<sup>†</sup> number of clinical cows which cultured bacteria divided by the total number of cows calved to 21/8 expressed as a %;

<sup>††</sup> sem = standard error of the mean.

More rear than fore-quarters were diagnosed with clinical mastitis (i.e. 531 vs 268 for rear and fore quarters; respectively,  $\chi^2 = 136.6$ ;  $P < 0.001$  where the actual number of diagnosed quarters was evaluated as a proportion of the total available (ie 595 x 2 rear and fore-quarters)) but there was no difference in prevalence between left and right side of the udder (401 vs 389 for left and right halves, respectively;  $P > 0.2$ ).

Bacteria were isolated from 72.4% of clinically affected quarters and major pathogens were isolated from 59.2% of all clinically affected quarters. The most common isolates were *Streptococcus uberis* (74.7% of bacteria positive quarters) and coagulase negative staphylococcus (10.2%; Table 2).

**TABLE 2:** Prevalence of bacterial species from 578 quarters presented with clinical mastitis from which bacteria were cultured.

Culture result	% *
<i>Streptococcus uberis</i>	74.7
<i>Staphylococcus aureus</i>	2.9
<i>Streptococcus dysgalactiae</i>	4.2
Coliforms	4.7
Coagulase negative staphylococcus	10.2
Others	3.3

\* as a % of all bacteria positive quarters

Resistance to penicillin and streptomycin occurred in 25% and 13.6% of 44 *Staphylococcus aureus* isolates tested, respectively. A small number ( $n=3$ ; 6.8%) of *Staphylococcus aureus* isolates were resistant to both penicillin and streptomycin. All 10 *Streptococcus uberis* isolates tested were resistant to streptomycin but were sensitive to all the other antibiotics tested. Of the 12 *E. coli* isolates tested, 100% were resistant to penicillin 75% resistant to

amoxicillin/clavulanic acid and 8.3% were resistant to tetracycline or streptomycin.

## DISCUSSION

There was a high apparent prevalence of clinical mastitis among the herds in this trial with an average of 10.0% of cows within herds being diagnosed with clinical mastitis within a 6 week period, post calving. A previous New Zealand study (Pankey *et al.*, 1996) found that 8.1% of heifers were diagnosed as having had clinical mastitis within 5 days of calving. Internationally, lower rates have been reported with monthly incidence figures of between 1.5% and 4.2% (Schukken and Kremer, 1996). Differences in calving patterns, milking management and feeding practices between New Zealand and other dairy industries may explain this variation. The seasonal calving pattern of the New Zealand dairy industry results in a large number of cows calving within a short period of time. This trial specifically examined the early lactation period when susceptibility to infection is higher than at other stages of lactation (Hogan *et al.*, 1989). Thus, annual average prevalence figures would be expected to be lower than those of the present trial as they include cows from all stages of lactation. New Zealand management practices such as restraining cows in small areas of pasture around calving and moving cattle along muddy races to and from the milking parlour may expose cows to high numbers of bacterial pathogens and increase the infection rate.

There was a large among-herd variation in the prevalence of clinical mastitis with between 0.9% and 21.4% of cows within herds being diagnosed. A similar wide range in prevalence of clinical mastitis was reported in the study on heifers (Pankey *et al.*, 1996). The reasons for this large between herd variation are not clear but may include inherent differences in prevalence of different bacterial species due to differences in milking and feed management or antibiotic usage. Additionally, herdowners may differ in their ability to detect changes in milk or the udder resulting in a diagnosis of mastitis. Herdowner diagnosis was used as the basis for determining prevalence as herdowners generally make diagnostic and treatment decisions about mastitis in New Zealand. Variation in herdowners ability to detect mastitis needs to be further evaluated and specific education programmes implemented or the use of diagnostic aids (rapid mastitis tests, conductivity meters) encouraged to increase the sensitivity and specificity of diagnosis of mastitis by herdowners.

*Streptococcus uberis* was the most prevalent pathogen isolated in this trial. It is an environmental pathogen as it can survive in extra-mammary sites including cows' skin, the respiratory tract and/or faeces (King 1981; Robinson *et al.*, 1985). Pankey *et al.*, (1996) isolated *Streptococcus uberis* or *Streptococcus dysgalactiae* from 67.6% of clinical cases of mastitis in heifers within 5 days of calving, which is in close agreement with the present trial. In contrast, the survey performed in the 1960's (Brookbanks, 1966) isolated only 22 *Streptococcus uberis* from 1137 bacterial positive quarters with *Staphylococcus aureus*, *Streptococcus dysgalactiae* and *Streptococcus agalactiae*

being more prevalent. Although not directly comparable, since Brookbanks used the rapid mastitis test as the basis of diagnosis for sampling and the sampling occurred throughout lactation, a comparison of the studies indicates that introduction of mastitis control strategies such as the 5 point plan and the SAMM plan (Woolford *et al.*, 1995) may have altered the prevalence of mastitis pathogens in New Zealand.

The second most common pathogen isolated was coagulase negative staphylococcus, in agreement with a previous study in heifers (Pankey *et al.*, 1996). Coagulase negative staphylococci are regarded as 'minor' pathogens (Sandholm *et al.*, 1990), implying that they result in little cellular damage or immune response following infection. However, the herdowners used the presence of clots, blood or pain and swelling of the udder as the basis of diagnosis, indicating that coagulase negative staphylococcus does cause sufficient changes to milk or the udder to result in a diagnosis of clinical mastitis. It is not known however, how many subclinical cases of coagulase positive staphylococcus were present in these herds and hence what proportion of these infections produce changes in the udder or milk that are clinically evident.

The bacterial sensitivity patterns of the small number of *Streptococcus uberis* isolates tested indicate that no resistance to penicillin has developed despite over 30 years use of penicillin based intramammary treatments (Brookbanks, 1966). Although *Streptococcus uberis* is inherently resistant to streptomycin, this is not of concern as no streptomycin-only intramammary preparations are available in New Zealand. The resistance patterns of the 44 *Staphylococcus aureus* isolates tested were variable within and between herds. Variation in cure rate between cows and between herds may be at least partly due to variation in pattern of *Staphylococcus aureus* resistance. However, *Staphylococcus aureus* represented only 2.9% of the bacterial positive cases of mastitis or 1.7% of all presented cases of clinical mastitis. Approximately 25% of the *Staphylococcus aureus* isolates were resistant to penicillin. All *E. coli* were penicillin resistant. The percentage of the clinical mastitis cases with penicillin resistance is the sum of all the coliforms (ie 4.7%) and a quarter of the *Staphylococcus aureus* isolates ( $0.25 \times 2.9\%$ ) producing a total of 5.4% of the bacteria positive or 3.9% of all cases of clinical mastitis cases. Thus, penicillin resistance is present in only a small percentage of the total population of clinical cases.

In conclusion, there was a high apparent prevalence of clinical mastitis in dairy herds from the Waikato region of New Zealand during the first 6 weeks of lactation. There was a large, unexplained variation in prevalence among herds. A majority of isolates (74.7%) were *Streptococcus uberis*.

In the absence of culture and sensitivity data, treatment of clinical mastitis in early lactation should proceed

on the basis that the infection is most likely due to *Streptococcus uberis* and is sensitive to penicillin.

This study also indicates that management of clinical mastitis in early lactation needs to focus on methods that reduce new infections with *Streptococcus uberis* and co-agulase positive staphylococcus. As *Streptococcus uberis* is an environmental pathogen, provision of 'clean' pasture for cows around calving, provision of clean, dry races, maintenance of moderate body condition, milking out of cows soon after calving and prevention of cross-suckling are management strategies that may reduce incidence of infection (Pankey *et al.*, 1996).

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