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BRIEF COMMUNICATION

Increased milk lactic acid concentration is an early indicator of mastitis

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The cost of mastitis to the dairy industry in NZ is substantial, with economic losses accruing from antibiotic costs, acute and chronic milk yield losses and reduced longevity of susceptible cows.

Early detection and treatment of mastitis is a critical factor in reducing the severity of the infection and tissue damage within the udder (Douglas *et al*., 1997). However, increases in labour productivity are reducing contact time with cows, making early detection of mastitis increasingly difficult. In particular, the development of robotic milking equipment has highlighted the need for remote detection of mastitis.

Apart from visual examination of fore-milk, somatic cell count (SCC) is used as the standard marker of milk quality. An increase in bulk milk SCC or a high SCC at herd test is used to diagnose new infections. However, one of the most widely used methods for cow-side or automated in-line testing for mastitis is the detection of changes in electrical conductivity of milk. This approach has several problems, not least of which is the limited accuracy of the method (see Woolford et al., 1998) and the relatively high cost of sensors (claws).

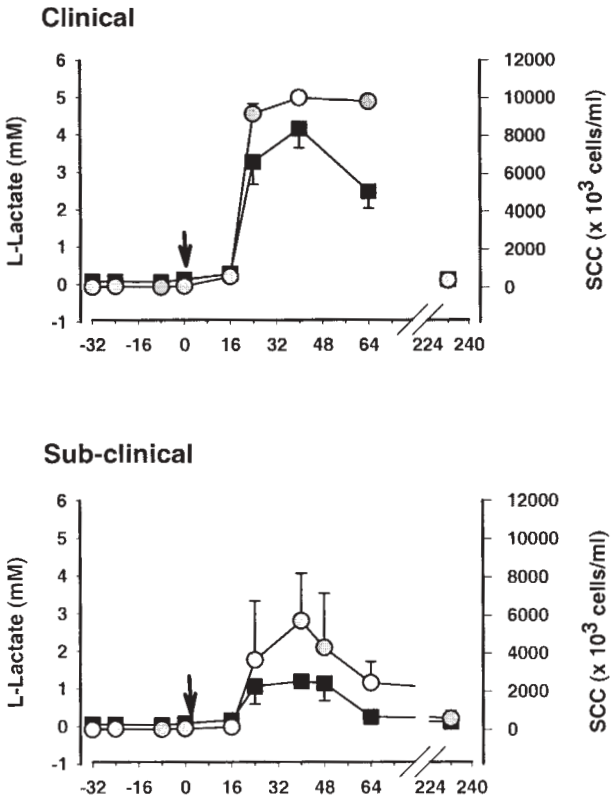
The following study was undertaken as part of a screen of a number of potential markers in milk which could be used to indicate mastitis. Further, there are relatively few data on milk lactate and its variation, a single publication suggesting a relatively slow increase in milk lactate during the first few days after drying-off (Mackie et al., 1977).

Milk was examined from Friesian and Friesian-Jersey crossbred cows in mid-lactation (November/December). Samples were taken from cows with suspected mastitis and infection was confirmed by subsequent bacteriological examination of milk. Twelve-pairs of samples were taken aseptically from control and infected quarters of cows on commercial farms. Sub-samples were taken for bacteriological examination, enzymic determination of lactic acid content (Lactic acid analytical kit; Scientific Supplies Ltd., Panmure, Auckland) and SCC. These individual quarter samples showed that milk lactate concentration was less than 0.2 mM in samples for which the SCC was less than 1 x 10^6 cells/ml. However, lactic acid concentrations increased rapidly with further increase in SCC, rising above 5 mM in some samples where SCC exceeded 1 x 10^7 cells/ml.

In the second experiment, a healthy, rear quarter of each of 10 cows in mid-lactation (average days in milk was 121 ± 5), was infused with 1000 colony-forming units of *Streptococcus uberis* in 1 ml Ringers solution (quarter strength) following an afternoon milking. Fore-milk samples were taken at each milking from control (untreated) and treated quarters and antibiotic treatment applied following the onset of clinical mastitis or after 72 h (all remaining cows).

Six treated quarters showed clinical symptoms of mastitis within 24-48h and this was associated with a thirty-fold increase, on average, in milk lactic acid (0.1 to 4.2 mM at 40h; SED 0.64, P<0.001, treated vs control quarters) and an increase in SCC from 4.5 x 10^4 to 1 x 10^7

FIGURE 1. Changes in milk lactic acid concentrations (■ ; mM) and somatic cells (●; SCC x 10^3 cells/ml) with time following intraductal injection of 1000 cfu of *Streptococcus uberis* into a rear quarter of ten lactating cows following an afternoon milking (arrowed). Graphs are shown for clinical (n=6) and sub-clinical quarters (n=3). Values for control quarters were unchanged throughout the sampling period (data not shown).



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cells/ml (see Figure 1.). Three treated quarters were sub-clinical with cell counts increasing up to 5.7 x 10^6 cells/ml at 48 h. In these animals, milk lactate was maximal at 1.2 mM at 40 h (SED 0.22; $P<0.05$) in the infected quarters relative to concentrations of 0.1mM in control quarters. Infection was not established in the infused quarter of the remaining animal.

Significant change in milk lactic acid ($P<0.001$) had occurred in all nine of the animals showing an infection by 24 h post-injection. A threshold of 0.5 mM (approximately five times control lactic acid concentrations) would have successfully diagnosed all infections at 40h and 8/9 at 24 h, whereas electrical conductivity (manual) and visual assessment only indicated the infections in the six clinical cases.

In conclusion, milk lactic acid is an excellent indicator of clinical and sub-clinical mastitis in dairy cows in mid-lactation. We also have data (not shown) which indicates that milk lactic acid will be of diagnostic value for mastitis in cattle after 3-4 days of lactation. Thick-film sensors for detection of lactic acid in blood have been produced commercially. The use of similar technology for an in-line biosensor for milk lactic acid could automate the early detection of mastitis and facilitate early therapy, increasing the frequency and extent of recovery from the infection.

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