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MILK PRODUCTION LOSSES ASSOCIATED WITH CLINICAL MASTITIS WITHIN IDENTICAL-TWIN SETS

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SUMMARY

Daily milk yield data over a period of three years have been analysed for differences in production occurring within identical-twin sets where one member was recorded as having had a clinical intramammary infection. For 22 such instances, the infected group showed 6.9% lower yield ($P < 0.01$) over the 7-day period following diagnosis and treatment. Within 14 days, yields had recovered to pre-infection levels in all but two sets. The data suggest that if clinical symptoms of mastitis are detected at an early stage, as through foremilk examination, production losses can be minimal.

INTRODUCTION

It is generally accepted that mastitis in dairy cows results in decreased production although the numerous estimates of the magnitude of loss vary widely (Janzen, 1970) because of the many possible levels of infection and associated management factors.

Experimental designs have often involved either
(a) within udder yield comparisons between infected and uninfected quarters; or
(b) correlation of yield levels of cows or quarters, with the presence or absence of pathogens, standard mastitis tests or cell counts.

Studies involving identical twins have been rare although McLeod (1958) used five such twin sets to demonstrate that within udder yield compensation by uninfected quarters offset any loss of yield in infected quarters, provided the infection was eliminated and did not become chronic.

This paper reports retrospective comparisons of daily milk yields within identical-twin sets over periods where one member was diagnosed as clinically infected while the twin-mate was observed to be clear of clinical symptoms over the entire lactation.

METHODS

The No. 1 Dairy identical-twin herd at the Ruakura Agricultural Research Centre comprises 50 sets of monozygotic-twin
cows. The herd conforms to the traditional New Zealand seasonal calving pattern, has a typical annual milkfat production of 136 kg/cow and is used for a variety of milking management and milking machine studies.

Over the three seasons 1976-79 there were 22 occasions where clinical mastitis was observed in one member of a twin set while the other was observed to be free of clinical symptoms over the entire lactation.

The criteria for the diagnosis of clinical mastitis were standardized over the three-year period. Foremilk from all quarters was inspected during pre-milking preparation by experienced dairy technicians. The presence of significant clots or, occasionally, abnormal discoloration of the foremilk was taken to indicate the presence of infection. More gross symptoms were uncommon.

On diagnosis of clinical infection a foremilk sample was taken for bacteriological assessment and an intramammary antibiotic was administered.

Daily milk yields have been retrospectively extracted for such sets, covering a period of two weeks prior to (periods 0, I) and three weeks following (periods II, III, IV) the diagnosis of the clinical infection.

The sets were at the time deployed on a variety of experimental studies and the clinical mastitis arose from exposure to normal environmental pathogens. While the primary experimental treatments may have predisposed one or other of the set members to infection, it is unlikely that such treatment regimens were responsible for the short-term within-set variations in yield for which the data were analysed.

DATA ANALYSIS

For each twin set, weekly milk yield ratios were calculated for each of the five weekly periods:

\[
\text{Yield ratio percent over period I (R_i)} = \frac{7\text{-day production infected cow}}{7\text{-day production uninfected cow}} \times 100
\]

The yield ratio for period 0 (R_0) was subtracted from the ratios for periods I - IV and a sets \times periods analysis of variance was then carried out on the differences (R_i - R_0).

All infections, including reinfections within the same set in the same year, were considered as being independent.
RESULTS

A classification of the 22 clinical infections by organism and cell count is given in Table 1 and it seems that in only one instance was a false positive diagnosis likely.

**TABLE 1: CLASSIFICATION OF DIAGNOSED CLINICAL INFECTIONS IN TERMS OF ORGANISM AND CELL COUNT**

<table>
<thead>
<tr>
<th>Cell Count (10³ cells/ml)</th>
<th>&lt; 800</th>
<th>&gt; 800</th>
<th>No Cell Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staphylococcal</td>
<td>0</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>Streptococcal</td>
<td>1</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Other organisms</td>
<td>0</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>No growth</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

Reinfections within the same lactation occurred in three twin sets and consequently the 22 clinical infections were distributed among 18 twin sets. The clinical and non-clinical groups differed by only one day in the group means for day in milk at the time of infection and over the three years infections were distributed fairly evenly between September and January.

Trends in the mean daily yield ratio (taken over the 22 infections) over the five-week pre- and post-infection period are shown in Fig. 1. Changes in the mean weekly yield ratios from the base level of period 0 are given in Table 2.

The between periods LSD for the change in weekly yield ratio was 3.5 ($P < 0.05$) and 4.6 ($P < 0.01$).

The mean yield ratio for period 0 (98%) does not suggest any initial bias towards lower yields for the clinically infected cows as may have arisen from undetected subclinical infection. The abrupt change in yield ratio between periods I and II indicated a total yield loss of 6.9% ($P < 0.01$) in the seven-day period following diagnosis and treatment. The actual volumetric losss

**TABLE 2: CHANGES IN THE MEAN WEEKLY YIELD RATIO PRIOR TO AND FOLLOWING CLINICAL INFECTION WHICH OCCURRED AT THE COMMENCEMENT OF PERIOD II**

<table>
<thead>
<tr>
<th>Yield Ratio Changes (%)</th>
<th>$R_1 - R_o$</th>
<th>$R_{ii} - R_o$</th>
<th>$R_{iii} - R_o$</th>
<th>$R_{iv} - R_o$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>-0.57</td>
<td>6.90</td>
<td>4.07</td>
<td>2.05</td>
</tr>
<tr>
<td>SE ($R - R_o$)</td>
<td>1.98</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SE (diff.)</td>
<td>1.73</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
depended on the stage of lactation and production level within individual sets. A yield recovery occurred \((P < 0.01)\) over the periods II-IV to a yield ratio which was not significantly different from that of period 0.

Trends of daily yield ratios showed that only 2 of the 22 clinical infections did not show a yield recovery, 11 showed a clear-cut transitory depression and the remaining 9 showed small effects.

**DISCUSSION**

These results are based on yield comparisons within identical-twin sets with specific criteria for the diagnosis of clinical infection. Many factors including micro-organism type and pathogenicity may be specific to this particular herd environment and may limit the generality of the results. It is also possible that undetected subclinical infections existed among the non-clinical cows and therefore that the yield losses obtained could be underestimated.

With these qualifications, the analysis shows that short-term yield losses associated with clinical mastitis are small and that a high level of yield recovery occurs. However, the data refer to a specific milking management regimen which allows early detection of clinically infected cows through foremilk examination and prompt antibiotic therapy. While this would appear to support the value of antibiotic therapy as a means of minimizing produc-
tion losses, it may also be argued that on a whole-herd basis production losses are largely the result of undetected and therefore untreated subclinicals which may persist for a much longer period.

It seems likely that early detection of infection as by foremilk examination is important in minimizing losses for clinically infected cows.

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REFERENCES