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Facial eczema in Jersey cattle: heritability and correlation with production

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ABSTRACT

During the serious outbreak of facial eczema (FE) in 1989, blood samples were obtained from first-lactation heifers born in 1986, in order to study any genetic influence on the incidence of FE. Heifers were located in 60 herds in Northland (14 herds), Auckland (25) and Taranaki (21). The herds were members of the New Zealand Dairy Board’s 1985 Sire Proving Scheme for young Jersey bulls. Samples were collected from 1523 heifers, the daughters of 57 young Jersey sires and 19 other Jerseys including reference sires from previous years. Blood samples were analysed for gamma-glutamyltransferase (GGT), an indicator of liver damage such as due to FE. Overall, 29% of animals had elevated GGT values (≥30 IU/l), and 72% (43) of herds had 2 or more animals with elevated GGT. After setting aside data from the 17 herds where no elevations of GGT concentration were recorded, the heritability of loge GGT was 0.31±0.10 (sires with >10 daughters each, and with ancestry data on sires included). The correlations of first-lactation milkfat and protein breeding indexes with loge GGT were -0.32 and -0.16. Corresponding regressions were -2.1 (fat) and -0.9 (protein) breeding index units per genetic standard deviation change in loge GGT. Results from three serum minerals are also reported.

Keywords Dairy cows; facial eczema; heritability; correlation; milkfat; gamma-glutamyltransferase

INTRODUCTION

Facial eczema (FE) is a disease of ruminants caused by ingestion of spores of the saprophytic fungus Pithomyces chartarum. The fungus grows and sporulates rapidly in autumn conditions common in the northern North Island and its spores contain the mycotoxin sporidesmin. In susceptible dairy cattle, sporidesmin causes liver damage, photosensitivity and reduced milk yield (Steffert, 1970; Towers et al., 1975). The extent of damage caused by FE in dairy cattle was reported by the New Zealand Dairy Board (NZDB 1981), following the last serious outbreak in the autumn of 1981 when 63% of North Island herds reported cases of clinical FE, with 44% of herds affected in Northland, 73% in the Auckland province and 67% in Taranaki. Within affected herds 5.6% of animals were said to be slightly affected and 3.6% severely affected, and there was little difference between breeds. A summary of sire differences was not published.

When it became clear that another serious outbreak of FE was in progress in the autumn of 1989, a survey of genetic variation for susceptibility to FE was planned in collaboration with the NZDB. The Jersey breed was chosen for study in preference to the Friesian because fewer herds would need to be contacted and visited in order to obtain samples on 20 daughters per sire.

The objective of the study was to estimate the heritability of susceptibility to FE in Jersey cattle, and to investigate any genetic correlations of FE with milkfat or protein production. The opportunity was also taken to analyse the samples for serum Mg, Na and K.

MATERIAL AND METHODS

Sampling of Herds and Cows

The Jersey cattle studied were in the NZDB’s Sire Proving Scheme (SPS) herds. These are herds which (i) use the NZDB’s young sires (SPS sires) and proven reference sires by artificial insemination (AI), (ii) record the parentage of AI calves, and (iii) rear and subsequently record the 2-year-old milk production of these heifers.

We selected progeny of the 1985 matings in the

¹ Livestock Improvement Corporation, New Zealand Dairy Board, Private Bag, Hamilton
TABLE 1 Number of animals, sire groups and herds sampled

<table>
<thead>
<tr>
<th>Analysis number</th>
<th>Type of herd</th>
<th>Minimal number of daughters/ sire</th>
<th>Number of animals</th>
<th>Number of sires</th>
<th>Mean no. of daughters/sire</th>
<th>Number of herds</th>
<th>Mean no. of animals sampled/ herd</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>All</td>
<td>4</td>
<td>1523</td>
<td>76</td>
<td>20.0</td>
<td>60</td>
<td>25.4</td>
</tr>
<tr>
<td>2</td>
<td>All</td>
<td>10</td>
<td>1454</td>
<td>65</td>
<td>22.4</td>
<td>60</td>
<td>24.2</td>
</tr>
<tr>
<td>3</td>
<td>FE*</td>
<td>10</td>
<td>1088</td>
<td>65</td>
<td>16.7</td>
<td>43</td>
<td>25.3</td>
</tr>
</tbody>
</table>

*Only herds affected by facial eczema (i.e. at least 2 animals with a GGT enzyme concentration >30 IU/l) were included

SPS, i.e. calves born in 1986, with 2-year-old lactations in 1988/89. The sires used for the 1985 matings were young bulls (born in 1984) purchased by the NZDB in early 1985 (“1985 intake”), some young bulls born in 1983 (“1984 intake”), and reference sires born in 1978-80. About 130 herds throughout New Zealand took part in the 1985 Jersey SPS, with half being in 3 of the main regions affected by FE, Northland, Auckland and Taranaki. After eliminating 6 herds (2 per region) whose owners chose not to participate, there remained 60 herds in the study, respectively 14, 25 and 21 herds by region. All herds used seasonal (i.e. late winter/early spring) calving.

Blood samples were obtained from two-year-old females in the above herds at the end of their first lactation. Sampling dates were May 18-31 1989 in Northland, April 24 to May 10 in Auckland and May 1-12 in Taranaki.

Biochemical Analysis

Blood samples were assayed at 37°C for gamma-glutamyltransferase or GGT (E.C. 2.3.2.2), a non-specific indicator of liver damage (Towers and Stratton, 1978). Elevated GGT concentrations (values greater than 30 IU/l) were taken as indicative of the presence of liver damage associated with FE. It is possible that other toxins or an abnormal physiological status in the cow could also cause elevated GGT concentrations. However, circumstantial evidence, such as the presence of clinical cases of FE on some farms, suggested that FE was the most likely cause of liver damage and elevated GGTs in the autumn of 1989. Concentrations of magnesium were estimated from the same serum samples using standard atomic absorption procedures. Sodium and potassium concentrations were obtained by flame photometry.

Data Analysis

Table 1 shows the number of sire groups and heifers with GGT records available in Analysis 1 (sires with at least 4 daughters each). The average progeny group size and average numbers of animals sampled per herd are also shown. There were 57 1985-intake Jersey bulls, 11 1984-intake Jerseys and 8 other (older) Jerseys included. In Analysis 2 (sires with a minimum of 10 daughters), the mean progeny group size was 10% greater but 11 sire groups were lost. In Analysis 3, data from 17 herds were discarded as having fewer than 2 animals with elevated GGT values (and consequently there was probably no FE challenge and no estimable sire variance). The remaining 43 herds had an average of 25 2-year-olds per herd.

Restricted maximum likelihood (REML) procedures (Patterson and Thompson, 1971) were employed to analyse the data, using a sire model (random effect), with herd as a fixed effect. The variates were log$_e$ (GGT concentration) and concentrations of Mg, Na and K. In addition, a modified REML program was used (REMLPK), in order to include sires of the 76 sires in the study; there were only 13 of these, with 3 accounting for 84% of all the 1985-intake bulls.

Genetic correlations between log$_e$ GGT and Mg concentrations were computed, and also correlations between REML sire means for log$_e$ GGT concentration...
TABLE 2 Mean and standard deviation (s.d.) of serum mineral concentrations for each district (mmol/l)

<table>
<thead>
<tr>
<th>District</th>
<th>Number of animals</th>
<th>Mg mean</th>
<th>s.d.</th>
<th>Na mean</th>
<th>s.d.</th>
<th>K mean</th>
<th>s.d.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northland</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whangarei/Waipu</td>
<td>181</td>
<td>0.857</td>
<td>0.103</td>
<td>148</td>
<td>7.1</td>
<td>5.31</td>
<td>0.86</td>
</tr>
<tr>
<td>Helensville/Wellsford</td>
<td>84</td>
<td>0.887</td>
<td>0.117</td>
<td>149</td>
<td>4.6</td>
<td>5.29</td>
<td>0.60</td>
</tr>
<tr>
<td>Dargaville</td>
<td>76</td>
<td>0.977</td>
<td>0.089</td>
<td>146</td>
<td>3.5</td>
<td>5.13</td>
<td>0.62</td>
</tr>
<tr>
<td>South Auckland</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hamilton</td>
<td>133</td>
<td>0.945</td>
<td>0.119</td>
<td>143</td>
<td>6.6</td>
<td>4.83</td>
<td>0.58</td>
</tr>
<tr>
<td>Piako</td>
<td>426</td>
<td>0.850</td>
<td>0.200</td>
<td>145</td>
<td>6.7</td>
<td>4.93</td>
<td>0.70</td>
</tr>
<tr>
<td>South Waikato</td>
<td>148</td>
<td>0.640</td>
<td>0.146</td>
<td>144</td>
<td>8.0</td>
<td>4.35</td>
<td>0.56</td>
</tr>
<tr>
<td>Auckland</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>New Plymouth/Okato</td>
<td>235</td>
<td>0.790</td>
<td>0.175</td>
<td>147</td>
<td>5.0</td>
<td>5.19</td>
<td>0.63</td>
</tr>
<tr>
<td>Stratford</td>
<td>198</td>
<td>0.746</td>
<td>0.161</td>
<td>143</td>
<td>6.0</td>
<td>4.81</td>
<td>0.59</td>
</tr>
<tr>
<td>Patea/Hawera</td>
<td>179</td>
<td>0.839</td>
<td>0.116</td>
<td>143</td>
<td>5.8</td>
<td>5.34</td>
<td>0.56</td>
</tr>
<tr>
<td>Taranaki</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
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</tr>
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<td>0.839</td>
<td>0.116</td>
<td>143</td>
<td>5.8</td>
<td>5.34</td>
<td>0.56</td>
</tr>
</tbody>
</table>

and the breeding index (BI) for milkfat or protein. Data from each cow at each monthly test were also checked for a sire relationship between production and loge GGT concentration.

Farmers were asked to complete a one-page questionnaire presented to them during the visit to blood sample their cows. This was to collect data on numbers of animals (of any age) showing signs of clinical FE.

RESULTS

Facial Eczema Incidence

Overall 29% of the 1523 animals showed elevated GGT levels, evidence of the presence of at least subclinical liver damage. Classifying by GGT value over all 60 herds, there were 12% of animals with 31-100 IU/l, 8% 101-330 IU/l, 5% with moderate liver damage (331-1100 IU/l) and 4% severe cases (>1100 IU/l). On a regional basis, 3, 8 and 15% of 2-year-olds respectively in Northland, Auckland and Taranaki herds had moderate or severe liver damage (9% overall). Forty-three herds (72% of all herds) had 2 or more animals with elevated GGT levels, i.e. 7/14 herds in Northland, 16/25 in Auckland and 20/21 in Taranaki.

Twenty-four (40%) of the farms reported clinical cases of FE amongst milking cows and some farms also reported clinical FE amongst non-milking stock. In total, farmers reported 139 clinical cases over their whole herds, about 6 per herd in clinically affected herds; 22 animals over all 60 herds either died or were culled immediately. Forty-one (68%) of farms took active precautions against FE.

Serum Mineral Concentrations

Marked variations among individual farms in mean serum concentrations of Mg (0.55-1.04 mmol/l), Na (137-156 mmol/l) and K (4.35-6.45 mmol/l) were recorded. Table 2 shows the means for each district. The between-farm and between-region differences probably relate to soil type and exposure to coastal salt deposition. The lowest serum Mg concentrations were in South Waikato and Taranaki, areas of known poor Mg status. The highest Na levels were from coastal Northland and Taranaki.
TABLE 3  Heritability estimates and standard errors for facial eczema (as indicated by elevations of GGT*) and for Mg concentration

<table>
<thead>
<tr>
<th>Analysis number</th>
<th>Type of herd</th>
<th>Minimal progeny no.(^{c})</th>
<th>Log(_{e}) GGT concentration</th>
<th>Mg(^{d})</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>No ancestry</td>
<td>With ancestry(^{d})</td>
</tr>
<tr>
<td>1</td>
<td>All</td>
<td>4</td>
<td>0.21±0.07</td>
<td>0.23±0.07</td>
</tr>
<tr>
<td>2</td>
<td>All</td>
<td>10</td>
<td>0.23±0.07</td>
<td>0.25±0.08</td>
</tr>
<tr>
<td>3</td>
<td>FEb</td>
<td>10</td>
<td>0.28±0.09</td>
<td>0.31±0.10</td>
</tr>
</tbody>
</table>

* See text for description
\(^{b}\) Only herds with elevated GGT, indicating facial eczema (see Table 1)
\(^{c}\) Minimal number of daughters per sire
\(^{d}\) Sire pedigrees of sires included.

Heritabilities

Table 3 shows the heritability estimates for GGT concentration from Analyses 1-3, with and without the inclusion of sire ancestry data. The preferred estimate is 0.31±0.10, based on animals in affected herds only, at least 10 daughters per sire, and with ancestry included. The heritability estimate for Mg concentration (Analysis 2) was 0.15±0.06. The estimate for Mg was higher from Analysis 3, but this excluded herds with no elevated GGT (i.e. no liver damage), a fact probably not relevant to the Mg data.

Heritabilities for Na and K were not significantly different from zero.

Correlations of log\(_{e}\) GGT with magnesium and fat and protein yields

The phenotypic and genetic correlations (between log\(_{e}\) GGT and magnesium) from the REMLPK analyses were respectively 0.03±0.03 and -0.04±0.27 (Analysis 2, which included all 60 herds) and 0.05±0.03 and -0.05±0.26 (Analysis 3).

At the end of the 1988/89 lactation, first proofs were published by the NZDB on 51 of the 57 1985-intake sires in the present study, with first-lactation data for milkfat and protein BI. Table 4 shows the correlations among regressed sire means of log\(_{e}\) GGT and fat or protein BI, and regressions of BI on log\(_{e}\) GGT. Correlations between log\(_{e}\) GGT and the breeding indices for average daily yield in 'spring' (September to December), or in 'autumn' (January to April) or overall (September to April) are also given for the 43 affected herds. Corresponding correlation estimates between log\(_{e}\) GGT and breeding indices for average daily yield from all 60 herds were -0.20 (spring), -0.31 (autumn) and -0.26 (overall) for fat, and -0.04, -0.15 and -0.10 respectively for protein.

TABLE 4  'Genetic' correlations\(^{a}\) between first-lactation milkfat or protein and breeding values for log\(_{e}\) GGT\(^{b}\) from 51 '1985-intake' bulls

<table>
<thead>
<tr>
<th>Statistic</th>
<th>Fat</th>
<th>Protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genetic correlation, BI x log(_{e}) GGT</td>
<td>-0.32</td>
<td>-0.16</td>
</tr>
<tr>
<td>Regression(^{c}), BI on log(_{e}) GGT</td>
<td>-2.1</td>
<td>-0.9</td>
</tr>
<tr>
<td>Genetic correlation(^{d}), average daily yield1 x log(_{e}) GGT</td>
<td>-0.13</td>
<td>0.01</td>
</tr>
<tr>
<td>average daily yield2 x log(_{e}) GGT</td>
<td>-0.27</td>
<td>-0.14</td>
</tr>
<tr>
<td>average daily yield3 x log(_{e}) GGT</td>
<td>-0.21</td>
<td>-0.07</td>
</tr>
</tbody>
</table>

*Correlations among regressed sire means; correlations were significant (at P<0.05) if <−0.29.
\(^{b}\) GGT: gamma glutamyltransferase, an indicator of liver damage due to facial eczema.
\(^{c}\) Fat or protein breeding index (BI) units per s.d. of BI for log\(_{e}\) GGT.
\(^{d}\) Yield1 = daily yield from September to December
            Yield2 = daily yield from January to April
            Yield3 = daily yield from September to April
DISCUSSION

Regional Differences

In 1989 the most serious FE encountered was in Taranaki, where 20/21 herds were affected, and 43% of all animals tested were affected. Over the three regions studied, the 43 affected herds accounted for about one-third of all Jersey SPS herds in New Zealand.

For the 3 minerals studied, regional differences were consistent with known facts about soil type and proximity to the sea (Grace, 1983).

Heritabilities

The heritability of log GGT was 0.31±0.10, compared with the estimate of 0.42±0.09 for FE susceptibility in sheep (Campbell et al., 1981). However, the sheep study involved administering controlled doses of sporidesmin, whereas neither pasture intake nor the degree of selective grazing (and hence the sporidesmin challenge) was controlled in this cattle survey. Nevertheless, it would be possible to select successfully for reduced susceptibility to FE in dairy cattle, using blood sampling of cows after a field challenge on toxic pasture. The more difficult problem, from an individual farmer’s viewpoint, would be to control the FE genes of bulls or semen used.

The heritability of Mg concentration, 0.15±0.06, was lower than for GGT or milk yield traits, but still significant. Cows were sampled at a time of year when Mg status was generally good and no Mg supplementation was practised. Greater variation in individual, herd and regional serum Mg levels could be expected during late winter and early spring when Mg status is poor in many areas. How this would affect the heritability is not known.

The non-significant heritabilities for Na and K probably reflected the degree of homeostasis applied to these minerals in the body.

Correlations

The apparent negative sire correlations between log GGT and milkfat BI (-0.32) or protein BI (-0.16) are important. Table 4 shows that the correlations with log GGT were negative for yield in ‘spring’ as well as being negative for yield in ‘autumn’, when there was likely to be an FE challenge. Each bull’s breeding index for log GGT may thus be important for bull selection decisions in the NZDB’s Sire Proving Scheme.

The survey also provides an opportunity in the future to examine the relationship between FE susceptibility and next season’s milk production.

ACKNOWLEDGEMENTS

We wish to thank Dr B.W. Wickham and staff of the Livestock Improvement Corporation (Newstead and New Plymouth) for assistance in contacting farmers; members of MAF Quality Management (particularly Mr Jim Bailey (Hamilton), Mr Winston Kaka (Whangarei) and Mr Brian Mylestone (New Plymouth) and their staff) for assistance with collecting blood samples, and also our Ruakura staff for assistance with collecting blood samples; and finally Mrs Lyn Briggs for analyses of gamma-glutamyltransferase.

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