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BREEDING FOR FACIAL ECZEMA RESISTANCE
— A PROGRESS REPORT

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SUMMARY

Since 1975 one hundred and sixty Romney sires have been progeny tested by sporidesmin challenge for facial eczema resistance status, as estimated by degree of liver damage. The most resistant and susceptible sires have been used to generate resistant and susceptible flocks for biochemical and field study. The heritability estimate \( h^2 \) for resistance, based on a total of 1,455 progeny, was 0.42 ± 0.09. Exposure to severe field challenge during a natural disease outbreak did not swamp the resistance mechanism, and long-term survival and liveweight was considerably higher in resistant than susceptible progeny following such challenge. There is some evidence that Booroola merinos may be more resistant than the Romney and Perendale breeds.

INTRODUCTION

In 1975 Campbell et al. reported a seemingly high heritability for resistance to facial eczema (FE) or, more accurately, a high heritability for liver damage score (LDS) in animals either exposed to a field outbreak \( (h^2 = 0.6) \) or directly challenged by intra-ruminal intubation of sporidesmin on a liveweight basis \( (h^2 = 0.4) \).

MATERIALS AND METHODS

Since 1975, surplus progeny from numerous single-sire mating groups from the animal breeding research areas at Tokanui and Rotomahana, from Massey University and from selection lines at Whatawhata Hill Country Research Station, have been challenged with sporidesmin and the breeding values (BV) of their sires estimated. In total 160 Romney rams have been progeny tested in this way. The two or three most resistant (R) and most susceptible (S) in each year have been mated to random ewes at Ruakura and their progeny, born and raised in a common environment, have been challenged to confirm their sires’ ranking. The most resistant and susceptible rams have been used to generate R and S flocks by initially mating to random ewes and subsequently to F, R and S ewes to produce first backcross \( (B_1) \) and at the 1980 lambing, the first crop of \( B_2 \) lambs.

In each season the selected R and S flock sires (usually two of each category) have also been mated, along with prospective R and
Sires, to a number of ewes at random for subsequent progeny challenge so that sire status is continually confirmed and its BV updated throughout this useful life.

RESULTS

BREEDING VALUE AND HERITABILITY

These parameters are assessed using only progeny LDS, a subjective assessment of liver damage on a 0 to 5 scale (Campbell and Sinclair, 1968). This is a discrete and probably non-linear variable, so some caution is attached to its use in conventional BV and heritability calculations.

BV$s$ (Table 1) are calculated for each sire by contemporary comparison with other sires represented in the same trials, the weighting for sires being the harmonic mean of number of progeny relative to number of contemporaries per trial. Re-use of rams was limited to trials in which all sires represented had been selected in equal numbers from the LDS extremes of previous trials. Thus the mean LDS for such trials were similar to those for trials with initial-use only rams and sire BV$s$ were updated relative to the respective trial means.

Mixed-model least squares procedures were used to estimate the heritability of LDS for progeny groups of lambs dosed with sporidesmin in 12 separate trials over six years. For sires represented in more than one trial, only their first progeny group was included in the analysis. Trials were regarded as fixed effects with sires within trial being random. In total, the 160 Romney rams were represented by 1455 progeny. The calculated overall heritability estimate was $0.42 \pm 0.09$. On occasions when several rams from each of a number of sources were included in the same trial, no evidence of between-source variation was found.

‘R’ AND ‘S’ FE FLOCK Sires

The reaction to sporidesmin challenge of $F_1$ progeny of the major sires so far used to generate the resistant and susceptible flocks is shown in Table 1. The older sires, on the left of each category list, have taken part in more progeny trials and so have had more progeny challenged. Cumulative breeding values (CBV) include all data available to March 1980. The negative CBV for the resistant sires indicate LDS below the trial means. Clearly all sires have not taken part in all progeny trials run so far and to the extent that between trial differences may affect LDS, percent clinicals, percent
deaths and liveweight change it is not accurate to compare sires directly. However HO 152 and R 161 were represented in the same 5 progeny trials and LF 107 and LM 100 in the same 2. Other R and S sires had progeny in several common trials. Nevertheless the overall disparity and the magnitude of the difference between R and S sires' progeny for LDS, percent clinicals and deaths, and liveweight change leaves little doubt that considerable productive gains should accrue from using resistant rather than susceptible sires.

**TABLE 1: F1 PROGENY RESPONSE — MAJOR FLOCK SIRES**

<table>
<thead>
<tr>
<th>Sire</th>
<th>HO 152</th>
<th>HF 42</th>
<th>LF 107</th>
<th>FE 275</th>
<th>R 161</th>
<th>KF 11</th>
<th>R 139</th>
<th>LM 100</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of Trials*</td>
<td>5</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>5</td>
<td>3</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>No. of Progeny</td>
<td>55</td>
<td>51</td>
<td>23</td>
<td>10</td>
<td>57</td>
<td>31</td>
<td>24</td>
<td>20</td>
</tr>
<tr>
<td>Mean LDS</td>
<td>1.2</td>
<td>1.1</td>
<td>0.8</td>
<td>0.5</td>
<td>2.7</td>
<td>2.5</td>
<td>1.9</td>
<td>2.9</td>
</tr>
<tr>
<td>Clinicals %</td>
<td>18</td>
<td>18</td>
<td>0</td>
<td>0</td>
<td>54</td>
<td>39</td>
<td>37</td>
<td>50</td>
</tr>
<tr>
<td>Deaths %</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>25</td>
<td>26</td>
<td>13</td>
<td>20</td>
</tr>
<tr>
<td>Lwt. change (kg)**</td>
<td>0.8</td>
<td>1.3</td>
<td>1.1</td>
<td>2.1</td>
<td>-2.6</td>
<td>-0.7</td>
<td>-0.4</td>
<td>-1.2</td>
</tr>
<tr>
<td>CBV</td>
<td>-0.9</td>
<td>-0.9</td>
<td>-1.3</td>
<td>-1.3</td>
<td>2.0</td>
<td>1.4</td>
<td>1.0</td>
<td>1.9</td>
</tr>
</tbody>
</table>

* Excluding 1st trial on which initial selection was made.
** In 6 weeks from challenge or earlier death.

**RESPONSE TO FIELD OUTBREAKS OF FE**

The method of sporidesmin challenge employed is clearly artificial and probably does not accurately simulate what happens in a natural field outbreak. There is also the concern that a sufficiently severe field challenge may swamp the resistance mechanism and obliterate differences between R and S progeny.

Two earlier trials using F, lambs indicated that the response of progeny to toxic grazing was similar to their response to direct sporidesmin challenge. Table 2 compares the response of first backcross (B,) ram lambs to a severe toxic grazing challenge in the 1980 season. These lambs grazed pastures whose spore trap counts ranged from 90,000 to 130,000 spores/28 l or air passing through the Brook spore trap — three to four times the accepted danger level. They grazed these toxic pastures for 4 weeks, were then removed to safe pasture and slaughtered for liver examination 4 weeks later. Despite the severity of the challenge the resistance mechanism was not swamped. The difference in death rate is particularly marked and the data for percentage clinicals mask the
much milder clinical symptoms in the resisters than in the susceptibles. A subjective 0-3 scoring of clinical symptoms (none, mild, moderate and severe) showed a three-fold difference in mean severity of symptom scores between R and S groups.

**TABLE 2: B; LAMBS GRAZED 4 WEEKS ON TOXIC PASTURE**

<table>
<thead>
<tr>
<th>Sire</th>
<th>No. of Progeny</th>
<th>Mean LDS</th>
<th>Clinicals (%)</th>
<th>Deaths (%)</th>
<th>Lwt. Change (kg)</th>
<th>R v S</th>
</tr>
</thead>
<tbody>
<tr>
<td>LF 107</td>
<td>12</td>
<td>3.1</td>
<td>58</td>
<td>17</td>
<td>-3.0</td>
<td>**</td>
</tr>
<tr>
<td>FE 275</td>
<td>11</td>
<td>2.8</td>
<td>64</td>
<td>18</td>
<td>-3.4</td>
<td>**</td>
</tr>
<tr>
<td>R 139</td>
<td>13</td>
<td>3.8</td>
<td>92</td>
<td>38</td>
<td>-4.5</td>
<td>***</td>
</tr>
<tr>
<td>LM 100</td>
<td>11</td>
<td>4.7</td>
<td>100</td>
<td>82</td>
<td>-5.4</td>
<td>*</td>
</tr>
</tbody>
</table>

1 Significance level applies equally to an analysis of LDS as an ordinal variable.

The longer term effects of exposure to toxic pasture conditions of R and S progeny can be seen in Fig. 1. These were 1975-born F, dry/dry ewes run on similar toxic pasture in 1980 but not slaughtered. The carry-over effect on liveweight and survival of the susceptible ewes persisted and the differences between the groups increased despite removal to ample, safe, fungicide-sprayed pasture at the end of the period on toxic grazing. Current survival rates are 75% and 22% for these R and S groups respectively.

**FIG 1: Liveweight (kg), deaths of 5-year-old F, resistant and susceptible ewes following 24 days grazing on pastures carrying a high spore load.**
Breeds Effects

Flocks on the Rotomahana Research Station have generated purebred Romney, purebred Perendale and their crosses with the Booroola. Ram lamb progeny of these breeds and crosses were included in the 1979/80 Romney sire-proving progeny trial. Table 3 summarises the data. It indicates an approximate halving of LDS in Booroola cross animals, which, although not statistically significant, nevertheless tends to confirm findings of B. L. Smith and S. D. Aust (pers. comm.) that Merinos are more resistant to FE than are English breeds.

<table>
<thead>
<tr>
<th>TABLE 3: RESPONSE OF ENGLISH STRAIGHTBREDS AND THEIR BOOROOLA MERINO CROSSES TO SPORIDESMIN CHALLENGE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No. Progeny</strong></td>
</tr>
<tr>
<td>Romney</td>
</tr>
<tr>
<td>Perendale</td>
</tr>
<tr>
<td>Booroola x Rom.</td>
</tr>
<tr>
<td>Booroola x Per.</td>
</tr>
</tbody>
</table>

For LDS: \( \frac{R + P}{2} - B = 0.58 \pm 0.35 \) (N.S.)

Conclusion

It is clear that it is possible to breed for resistance to the worst manifestations of the facial eczema exotoxin: severe liver damage, clinical symptoms, liveweight loss and death. The resistance mechanism is highly heritable and is not over-ridden by severe challenge. The resistance mechanism itself is now being studied by comparing the metabolism of sporidesmin in the genetically characterised R and S flocks generated at Ruakura in the hope of uncovering a biochemical marker of value in the field selection of resistant sires.

Acknowledgements

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