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Moving towards low-chemical farming with sheep and cattle: the potential of a breeding approach

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ABSTRACT

On livestock farms in New Zealand, some farmers are changing their sheep, beef cattle or dairy cattle enterprises towards specialisation in low-chemical or organic systems. A vital part of the animal's ability to perform under these systems is having the disease resistance genes available (or some other means to cope), as well as having the production and quality traits. In practice, animals are likely to be under a number of disease challenges at any one time, so breeders must concentrate their genetic selection practices on the most important health problems. Other (non genetic) means may also be critical in helping to protect the animal. This paper reviews the New Zealand evidence for genetic differences in resistance to various important diseases or metabolic stresses in sheep and cattle, and shows that genetic selection could be applied successfully if required. The paper also discusses some of the challenges in realising the goal of low chemical farming, such as finding a source of rams, bulls or semen. The diseases/stresses that are covered are: *in sheep*, nematode parasites, dags, fly strike, facial eczema (FE) and ryegrass staggers (RGS); *in dairy cattle*, mastitis, bloat, FE, RGS, nematode parasites and hypomagnesaemia; *in beef cattle*, RGS, nematode parasites and hypomagnesaemia. Some breed differences are mentioned, but at present the main New Zealand evidence of genetic variation is derived from heritability estimates. In sheep, average heritability estimates are: nematode parasites in lambs, 0.23 (repeatability 0.30); dags, 0.20 (repeatability 0.4); fly strike, 0.26 (Australian data); FE, 0.45; and RGS, up to 0.17, depending on the challenge level (repeatability 0.28). Corresponding heritability estimates in cattle are: clinical mastitis 0.06 (or somatic cell count 0.09); bloat 0.19 (repeatability 0.45); FE 0.56; RGS no published estimate; nematode parasites in calves 0.32; hypomagnesaemia 0.15. The simple mean of heritability estimates for 10 traits in sheep and/or cattle was 0.26 (0.28 excluding the outlier, mastitis, at 0.06), suggesting that responses should be achieved if directional selection is applied after performance or progeny testing. Knowing the genetic correlations among these disease traits is also important, because of the multiple disease challenges likely, but few of these correlations have yet been documented. Opportunities for ranking sires using the principles of Group Breeding Schemes or Sire Reference Schemes are discussed.

Keywords: genetics; heritability; sheep; cattle; sires; low chemical farming.

INTRODUCTION

There is an increasing market expectation that meat products will come from farm systems that use lower levels of chemicals and that are caring towards both animals and the environment. This is reflected in the rapidly growing worldwide organic food market. The opportunity that this value added market creates has not gone unnoticed by the livestock sector. Growth of the sector, however, is seriously constrained because of the challenges associated with the management of animal health, and weed and plant pests, where the recourse to a chemical control is not an option if the status of the animals and their products is to be maintained (Mackay, 2001). The real risk of system collapse because of a disease outbreak discourages farmers from entering into these value added supply chains. This is highlighted in a Ministry of Agriculture and Forestry policy report (Mackay *et al.*, 2001) commissioned to identify and quantify the risks and costs associated with conversion, specifically to organic livestock production. The study concludes that without a significant investment to address the risks associated with the withdrawal of agri-chemicals, the organic sheep and cattle livestock sectors will not expand.

In farming systems where chemicals are not used as the primary means of controlling animal disease, farmers need to place greater emphasis on the breeding of replacement stock that can produce under more

challenging conditions, as part of an overall strategy to reduce disease outbreaks. This review is concerned specifically with animal health factors and some possible animal genetic solutions. Realistically, replacement female stock need to be bred at home, but there are homebred and purchasing options for male breeding stock. This review summarises genetic variation for resistance to disease traits, indicating which diseases could be tackled by a genetics approach, and it discusses some of the future challenges.

Sheep. The disease traits to be considered for sheep are nematode parasites, dags, fly strike, facial eczema (FE) and ryegrass staggers (RGS).

Dairy and beef cattle. In dairy cattle, the traits to be considered are mastitis, bloat, FE, RGS, nematode parasites and hypomagnesaemia, whilst in beef cattle they are RGS, nematode parasites and hypomagnesaemia.

MATERIALS AND METHODS

Data sources

It is our intention to limit the data sources on disease traits to New Zealand experiments, if available. The variation to be considered comes mainly from published within breed heritability (h^2) estimates, but also includes known breed differences.

RESULTS

General. In order to breed animals able to cope with

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a low chemical or organic environment, breeders must concentrate on the most important health problems. This would ensure that most or all of the selection pressure is applied to the trait(s) of greatest value to the enterprise. In practice, in a low chemical production system, animals are likely to be under a number of disease challenges at any one time. Thus other (non genetic) means will also be required to protect animals from less important disease challenges, unless there is an opportunity such as a breed substitution that would protect against a disease challenge, for example, the Finnish Landrace sheep breed carrying resistance to FE, as discussed later.

Sheep. The most serious production limiting animal health problem on low chemical or organic sheep farms is the control of nematode parasites (Mackay *et al.*, 1998). Numerous publications in New Zealand have shown that faecal worm egg count (FEC) in lambs after weaning is a heritable trait (Morris *et al.*, 1995a). Combined estimates are shown for this and other disease traits in Table 1. Selection progress for reduced FEC in lambs can be achieved in practice, leading to lower levels of nematode parasite infection in lambs, with associated reductions in FEC in peri-parturient ewes. However, reducing FEC in lambs and ewes will only reduce the effects of parasitism on sheep performance indirectly, via lower pasture larval contamination and therefore lower challenge (Bisset *et al.*, 2001). Under grazing conditions commonly experienced by New Zealand lambs, where the main nematode genera are *Trichostrongylus* and *Ostertagia* rather than *Haemonchus*, low FEC is genetically correlated with high dag score, slightly reduced post weaning growth, and reduced fleece weight in hoggets and ewes (Morris *et al.*, 2000). Index selection combining low FEC and high production traits, such as offered by the Sheep Improvement Limited service “WormFEC™”, potentially provides an answer to this problem. Another answer, which is still in the research/development stage, may be selection for increased resilience traits, with or without selection for low FEC at the same time (Morris *et al.*, 2001a). Betteridge *et al.* (1997) found that sheep selected for resilience out performed control lines in their first year of life, in a high nematode challenge environment, but their growth still fell well short of the performance of either the resilient or control animals in

the low nematode challenge environment.

Dag score, which shows, in part, the level of an animal’s immunological responses, is also heritable ($h^2 = 0.20$; Table 1), with a repeatability of approx. 0.4 (depending on whether animals are drenched and/or shorn between dag scores). The realised genetic correlation between dags and FEC is negative (unfavourable) in AgResearch FEC selection lines (Morris *et al.*, 2000), although paternal half sib estimates of the correlation from other data sets are highly variable in sign and magnitude (Morris *et al.*, 1995a). Because the correlation is not close to -1 , probably only some of the genes are in common, coding for low FEC and high dags.

Another important factor often associated with high dag score is a pre disposition to fly strike. Selection for low dag score would presumably reduce the potential for fly strike, since a high proportion of fly strike in New Zealand is breech strike (Heath & Bishop, 1995). In Australia, the h^2 of fly strike is 0.26 (Raadsma, 1991), and there is a positive genetic correlation (> 0.9) with fleece rot, although the correlations may not necessarily be the same in New Zealand breeds as in the Merino. Recently it has been shown that a major gene is segregating in lines of experimental Merinos genetically selected for or against fly strike (Mortimer *et al.*, 2001). Much less research has been carried out on the genetics of host susceptibility to fly strike in this country than with fly borne pests in Australia, but with the advent of the Australian blow fly (*Lucilia cuprina*) into New Zealand (Heath and Bishop, 1995), there is a need for intensive study. This is of particular significance in low-chemical systems because of the limited control options and the immediate animal welfare issues associated with an outbreak of fly-strike. In those environments where fly-strike is a threat, the lack of an effective control is a major barrier to a low-chemical enterprise.

According to Raadsma (2000), the sheep host (in Australia) does not produce an antibody response to flies, but it generates wool and skin substances in response to moisture and bacterial agents in the fleece rot; these may deter flies from landing or from laying eggs. The mechanism may, thus, be different in principle from the major gene for resistance of *Bos taurus* cattle (Frisch, 1994) to ticks (*Boophilus microplus*), in which at least one of the resistance pathways is for host eosinophils to secrete histamine in response to ticks (Schleger *et al.*, 1981).

Resistance to FE in sheep is well known to be heritable ($h^2 = 0.45$; estimated by Morris *et al.* (1995b) from the resistant (R), control (C) and susceptible FE selection lines at Ruakura). Genetic progress for FE resistance has been achieved by single trait selection at Ruakura, where the R line is now over 3 times as resistant as the C line after 25 years of selection. Progress has also been achieved by the Auckland Romney Development Group in selecting for greater FE resistance using an index that includes increased production (Morris *et al.*, 1999). An AgResearch service known as “Ramguard” provides ram breeders with the materials and the sample analyses to test and rank their rams for FE resistance (Morris *et al.*, 1994a).

TABLE 1: Heritability (h^2) and repeatability (r) estimates^a for disease traits in New Zealand sheep and cattle.

Trait	Sheep		Cattle	
	h^2	r	h^2	r
Nematode parasites ^b	0.23	0.30	0.32	
Dags	0.20	0.40		
Fly strike	0.26 ^c			
Facial eczema	0.45		0.56	0.85
Ryegrass staggers	0.17	0.28 ^d		
Mastitis			0.06 ^c	
Somatic cell count			0.09 ^c	
Bloat			0.19	0.45
Hypomagnesaemia			0.15	

^a Generally from animal model restricted maximum likelihood analyses.

^b Faecal egg count.

^c Not New Zealand data.

^d Between years.

An unexpected finding from the Ruakura FE selection lines is that there is a genetic correlation of 0.3 between resistance to FE and resistance to RGS (Morris *et al.*, 1995c). This has been confirmed in two additional single trait selection lines of sheep at Ruakura, selected for resistance or susceptibility to RGS ($h^2=0.17$; repeatability across seasons, 0.28; Amyes *et al.*, 2002). Using the Ruakura RGS selection lines, it has been possible to create genetic divergence for RGS, although most of the progress so far has been in the susceptible direction.

It would also be of interest to know the genetic correlations between resistance to FE and to other diseases. A genetic correlation estimate of resistance to FE and to nematode infection has been published, combining results from two experiments, and the value was statistically significant at 0.19 ± 0.09 (Morris *et al.*, 1996). There is a need for greater understanding of the interactions among disease traits.

Finally, in addition to within breed genetic variation in disease resistance, some between breed differences have also been shown. For nematode parasites, McEwan *et al.* (1994a, b) published breed differences showing that Perendales were slightly more resistant to nematode infection than Romneys or Coopworths, and also that Texel x Romneys were more resistant than Romneys. For dags, Meyer *et al.* (1983) showed that Perendales were less prone to dags than Romneys and Border Leicester x Romney crosses. In relation to FE, Finnish Landrace are more resistant than Romneys (Morris *et al.*, 1994b). East Friesians are similar to, or more FE resistant than Romneys (Morris *et al.* 2001b), depending on whether assessments are made using, respectively, clinical incidence or enzyme assays (for gamma glutamyl transferase, GGT). It is generally believed that Texels are more susceptible to FE than Romneys (J. L. Dobbie, personal communication, January 2002).

Cattle. There are fewer h^2 estimates available for disease traits in New Zealand cattle than in sheep. In dairy cattle, the primary disease that will need to be controlled in a low chemical system is mastitis. The next ranked disease traits probably depend on the animal management practices, pasture species, pasture quality and time of year, but increased resilience to nematodes in dairy heifers, resistance to FE and to bloat, are candidates. Literature review estimates of the genetics of clinical mastitis and somatic cell count (SCC) in dairy cattle (Colleau & Le Bihan Duval, 1995) gave mean h^2 estimates of 0.06 ($n=9$ international publications) and 0.09 ($n=15$), respectively (Table 1). Another review of similar (but not identical) references by Mrode & Swanson (1996) gave values of 0.04 and 0.11, respectively. The genetic correlations between the two traits averaged 0.65 and 0.7 in the two reviews, respectively. Looking specifically at New Zealand herds, Smit & Wickham (1986) found an h^2 of 0.04 for SCC in Holstein Friesians and Jerseys. If the New Zealand parameters continue to be as low as these reported in 1986, then large progeny groups will be needed in order to make genetic progress to reduce SCC and, hence, mastitis, as demonstrated effectively in Norway (Heringstad *et al.*, 2001). There is, however, the advantage of having multiple records per cow, with a repeatability

for SCC of 0.21 across herds and lactations (Smit & Wickham, 1986).

For bloat, Table 1 shows parameter estimates from the AgResearch experimental herd of 0.19 ± 0.03 for h^2 and 0.45 ± 0.02 for repeatability (updated from values published originally by Morris *et al.*, 1991). For FE, the most recent estimate is an h^2 of 0.56 with a very high repeatability (0.85) among consecutive GGT monitor samples (Morris *et al.*, 1998). These authors also found a breed difference in resistance to FE (Jerseys more susceptible than Holstein Friesians), and the suggestion of a breed difference in h^2 (Jerseys having a higher h^2 than Holstein Friesians). For RGS, there are no published h^2 estimates. However, some unpublished Holstein Friesian data collected by AgResearch in collaboration with Dexel showed significant correlations between the RGS scores of daughters and their dams in the same herd, suggesting that RGS susceptibility is probably inherited, as it is in sheep. These three diseases (bloat, FE and RGS) probably have the same mode of inheritance in dairy and beef cattle.

Table 1 also shows an h^2 of 0.32 for nematode parasites in New Zealand beef calves (C. A. Morris, 2002, unpublished data). Nematodes can be the single biggest challenge to both rearing and finishing prime cattle. Similar estimates have been derived in dairy cattle (C. A. Morris, 2002, unpublished data). Compared with progress made in understanding resilience to nematodes in sheep, little is known in cattle.

For hypomagnesaemia, an h^2 of 0.15 was reported for dairy cows (Morris *et al.*, 1990). In contrast, a study of beef weaner calves in Florida (Odenya *et al.*, 1992) provided a larger estimate for Mg of 0.36, along with 0.39 for Ca and 0.40 for P.

In spite of similar probable modes of inheritance, the mean incidences of some diseases (and their heritabilities) may differ between dairy and beef cattle because of differences in breed, or in intensity of animal husbandry applied to each type of cattle, or in the terrain/altitude/pasture types encountered by dairy vs. beef cattle. For example, it is not automatic that the heritabilities of say, nematode parasite levels in calves, will be the same in dairy and beef cattle because of different rearing experiences and ages at weaning from the dam.

What then can be done in practice with selection for disease resistance traits?

DISCUSSION

The potential. The simple mean of h^2 estimates for 10 traits in sheep and/or cattle summarised above was 0.26, including mastitis or SCC but not both (or 0.28 for 9 traits if the low value of 0.06 for mastitis was excluded). This mean of 0.28 is very similar to that for many production traits, suggesting that effective responses can be achieved under commercial circumstances, if directional selection is applied after performance or progeny testing, as with say weight selection. An earlier review by Morris (2000) reported h^2 estimates for disease traits summarised *only* from selection studies in progress, and the mean value was the same at 0.28. The genetic correlation between each of these traits would also be of

interest given that, in practice, and particularly in a low-chemical production system, the animal is likely to be under a number of disease challenges at any one time. Apart from the genetic correlations between FEC and dag score, FE and RGS resistance, and FEC and FE, little is known of the interaction between traits and their consequences on animal health, performance and product attributes.

Ranking sires. The major genetic questions for sheep and beef farmers intending to move their enterprises towards specialising in low-chemical or organic systems are: a) Should I breed my own sires? b) If buying sires from outside, where should I buy them? c) What information is available to help in ranking outside flocks/herds, and in ranking sires within them? Finally, (d) how much progress needs to be made in resistance to several challenges before the resulting animals will be able to withstand a low chemical environment? The same questions, (a) to (c), have been discussed for over 30 years in New Zealand in relation to production traits. For some breeds of sheep and beef cattle, Sire Reference Schemes and centralised recording of pedigree and production data with organised data analysis systems have provided a solution, but only for a limited number of production traits. There are now published data on, for example, liveweight and carcass traits in some breeds, ranking both potential young sires, and also sires with existing progeny, so that buyers can choose which sires to purchase, as live animals or in some cases as semen.

The same principles could be applied to disease traits, for all breeders, or specifically for those interested in low chemical or organic systems. For resistance to nematode parasites in sheep, breeding-value summaries are already available through “WormFEC™”. For resistance to FE in sheep, a framework within the Sheep Improvement Limited recording scheme has been established recently to provide recording/ranking procedures using “Ramguard”. For other diseases no such schemes exist at present. There are opportunities for farmers with similar interests and objectives to pool resources to achieve the above examples of sire recording and ranking, as was done with Group Breeding Schemes to improve production traits in sheep and beef cattle, beginning in the 1960s and 1970s (Rae, 1974). The basic advantages of a Group Breeding Scheme over small isolated sire breeding flocks/herds are: large population size, high selection intensity, and objective recording, often with combined breeding decisions on each sire. There are also spin offs from the (human) social contact, from opportunities to observe and record performance in the central elite nucleus flock (herd), and from the sire multiplication opportunities offered by the Group Breeding Scheme, for the transfer of elite and 2nd tier sires to members’ farms or for public sale.

For the final question, on how much progress is required in resistance for several traits before a low chemical system is sustainable, no answer is available yet. However it is suspected that selection for disease resistance traits may not offer benefits in the early stages and/or in high challenge years. This is unlike the situation with production traits, for which the incremental genetic progress translates to tangible gains. van der Waaij *et al.*

(2000) have modelled constant infection pressures from nematode parasites, as they affect production traits expressed by animals with varying resistance. Possibly this model could be adapted for use under our local conditions.

In the case of dairy cattle, a major question is whether an artificial insemination organisation will provide breeding values for any disease traits of sires, or will use such breeding values to select resistant sires in the dairy industry for general or specialist use.

Unlike the dairy industry, the more fragmented nature of performance recording data in New Zealand’s sheep industry, and especially in the beef cattle industry, would lead to few opportunities for pooling disease data from seedstock flocks (herds), unless somebody chooses to set up reference sire connections among flocks (herds) and then analyse the structured data collected.

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