SUMMARY

Fleece rot and footrot are two bacterial skin diseases of potential significance in sheep. Both diseases are associated with sheep production in high rainfall environments. Fleece rot is currently the most efficient indicator trait known for genetic improvement in resistance to body strike. Exploitation of genetic variation in resistance to fleece rot is feasible through selection of sheep within flocks, between flocks within strains, and between strains. Identification of resistant genotypes is at present only realistic after expression of the disease, either in the flock, sire evaluation schemes, or suitable flock comparisons. Potential scope for identification of resistant genotypes without the expression of fleece rot, is discussed for both between-flock and within-flock selection.

In the case of footrot, breeding strategies should aim to reduce the impact of virulent or intermediate footrot to that of benign footrot so that no specialised control strategies are warranted and the disease has minimum impact on production. An alternative strategy could be to improve the responsiveness of sheep to vaccination so that footrot could be managed similarly to the clostridial diseases, with annual booster vaccinations offering long-term effective protection.

Host factors, including sources of genetic variation, contributing to the expression of footrot have not been adequately investigated. Because of insufficient information it is not possible to include resistance to footrot in well-designed breeding programmes for Merino sheep. Preliminary results suggest a low to moderate heritability for the expression of footrot following challenge and vaccination.

FLEECE ROT

Fleece rot is a mild superficial dermatitis induced by moisture and bacterial proliferation at skin level. Exudation, resulting in a matted band of fibres, combined with extensive staining of the fleece are the major signs. A number of bacterial species occur in fleece rot, but Pseudomonas aeruginosa and P. maltophilia are considered the causal agents. The major significance of fleece rot is the predisposition of affected sheep to body strike. Fleece rot is at present the most efficient indicator trait for genetic improvement of resistance to body strike.

Incentive to improve resistance to fleece rot

Although resistance to fleece rot could be taken as a breeding objective in its own right, the more serious sequel of body strike has a relative economic value which is far greater (Ponzoni 1985). In the case of resistance to body strike, both a reduction in the prevalence and an increase in the number of years for which no preventive control is needed, are the main reasons to consider fleece rot in a breeding programme (Raadsma 1991a).
Genetic variation in fleece rot

Raadsma (1991b) recently reviewed studies describing sources of genetic variation in resistance to fleece rot. It was concluded that on an underlying scale of liability, the heritability was in the order of 30-40%. No conclusive evidence of differences between breeds in their susceptibility to fleece rot are available. Lack of reliable comparisons was seen as the major reason. Within the Merino breed, considerable differences exist between strains, and flocks within strains, in their susceptibility to fleece rot. The implications of this large source of variation may be of considerable importance to the Australian Merino industry, since the majority of commercial breeders purchase their replacement rams from outside sources.

Exploitation of genetic variation in fleece rot

Complete replacement of a susceptible bloodline with a more resistant bloodline, or introduction of a more resistant bloodline through continued purchase of rams, are two options to make rapid increases in resistance. The major limitations are that resistant bloodlines cannot be readily identified unless they participate in formal and well designed/organised flock comparisons (Hygate and Atkins 1988). Furthermore, the economic performance of a bloodline does not depend solely on resistance to fleece rot. Combined information on resistance and performance in all other major production traits is thus needed before sensible decisions can be made.

The role of exploiting within-flock genetic variation in resistance to fleece rot becomes clear when viewed as a potential indicator trait for resistance to body strike. In the latter case, genetic progress in resistance is limited under direct selection. The often low and variable prevalence of body strike limits effective selection differentials. Utilising fleece rot as an indirect selection trait is an attractive alternative, since fleece rot is normally expressed at a higher prevalence than body strike. It is also easy, simple and cheap to measure, and it has a high co-heritability (defined as $h_1 h_2 r_{12}$) with liability to body strike. Raadsma (1991a) predicted rates of progress in resistance to body strike for mass selection, utilising various selection strategies, and considered fleece rot to be a suitable indicator for selection against body strike. The response in selection flocks, selected solely for increased and decreased expression of fleece rot, showed a decrease in the prevalence of body strike and an increase in the number of years free of body strike, in the flock selected for less fleece rot (Raadsma 1991b).

Unfortunately, fleece rot also suffers from the same problems as body strike, in that the prevalence is often not high enough for optimum progress. The search for indirect selection traits for fleece rot has not yielded indicators which are simple, cheap, and effective, despite comprehensive efforts (see Raadsma 1991b). Although a number of fleece traits were considered suitable for increasing resistance to fleece rot, they were less effective when considered in breeding programmes which included other important breeding objectives. The importance of the relationship between the indicator traits and other objectives negated potential improvement in resistance to fleece rot (Atkins, unpublished data). At present there is no single indicator trait which describes resistance to fleece rot more efficiently than direct expression of the disease. The use of sire reference schemes has been advocated as an alternative means to identify sires which can be used to improve resistance to fleece rot (Mortimer and Raadsma 1991). The relatively small number of sires which can be tested and potential sire by environment interactions, may limit the scope of this approach.
Developments with new indirect selection traits for fleece rot

Raadsma (1988) suggested that the susceptibility of flocks to fleece rot could be predicted on the basis of a "between-flock index". Combined information on a number of simple fleece traits (greasy fleece weight, yield, staple length, and fleece yellowness), resulted in a "between-flock" correlation of 0.88 with observed fleece rot in 40 flocks (n=600). The same index predicted fleece rot in the same flocks in the following year (r=0.79). The index could also be used to predict fleece rot in a different sample of 22 flocks (n=220) in a different environment(r=0.78 and 0.74 respectively for observations over two years). It may thus be possible to predict the susceptibility of flocks to fleece rot without the expression of the disease. Other traits which were equally effective in a "between-flock index", included fibre diameter, variability in fibre diameter, crimp frequency, fleece value, and wool handle. The "between-flock index" was highly repeatable (r=0.92) for measurement of 40 flocks over 2 years.

For within-flock indirect selection, Raadsma (1991a) suggested that it was unlikely that a single indicator would reflect genetic variation in resistance to fleece rot. The reason is that variation in resistance to fleece rot is brought about by different biological mechanisms (fleece, fleece-skin interface, and immunological barriers), all of which are likely to be partially under genetic control. Indirect selection will likely be based on; (a) an index of a range of indicators from each of the important barriers, (b) a screen directly at DNA level of genes which are linked and/or contribute to resistance, (c) some form of challenge which measures the net effect of all the factors contributing to resistance. Raadsma et al. (these proceedings) indicated a preliminary role for DNA hoof printing in fleece rot and body strike. The group with Sandeman, has indicated the possibility of using a skin challenge with blowfly larval antigens as a screening test for resistance to fleece rot and flystrike (O'Meara et al., in press).

FOOTROT

Breeding sheep for resistance to footrot needs to be considered in the context of factors controlling its expression and benefit of reducing its economic impact. This has recently been reviewed by Egerian and Raadsma (1991). The following is a brief summary.

Expression of footrot

Footrot is a contagious bacterial disease of sheep. Its occurrence results from an interaction between a specific transmitting agent, *Dichelobacter nodosus*, and other microorganisms; the innate and acquired resistance of the host; and the environment in which sheep are kept. Although most of the work has focussed on bacterial and environmental factors, only sheep factors are discussed here.

In naturally infected flocks, the prevalence is seldom 100%. Furthermore some sheep have the ability to overcome the disease through "self cure", whereas others remain chronically infected. The role of the epidermal skin barrier and subsequent inflammatory response are thought to be important in innate resistance (Parker et al., 1983, 1985). Host factors responsible for self cure are not known at present.
Acquired resistance following natural infection or colostral transfer does not appear to be important. Active immunisation against fimbral antigens results in resistance to infection which is sero-group specific and usually of limited duration. Sheep need to be vaccinated with antigens from all known sero-groups to cover field challenge. Recent evidence suggests that sheep differ strongly in their ability to produce antibody (Raadsma et al., 1990) and the phenomenon of antigenic competition (Egerton et al., unpublished data), strongly limits the effectiveness of multivalent footrot vaccines.

**Incentive for genetic improvement in resistance to footrot**

The prevalence of severe cases of footrot (lesions which underrun the sole and hard horn of the hoof) in a flock is the main criterion which determines the biological and economic impact of the disease. Reduced production and high labour costs associated with control, are a main feature of virulent footrot. Benign footrot on the other hand has minimal impact on sheep health and production if left untreated. In the absence of regulatory control, the occurrence of footrot on a farm does not always justify treatment. Egerton and Raadsma (1991) presented estimates of losses likely to arise when owners take one of three options; (a) take no action, (b) implement control, (c) proceed through control to eradication. A number of important observations were made:

- For benign footrot the cost of control or eradication would exceed that which could be directly attributed to the disease if left untreated.
- The cost of virulent and intermediate footrot could be halved through conventional control techniques but would be recurrent from season to season.
- The cost of virulent or intermediate footrot under a control management option is still greater than that of uncontrolled benign footrot.

Breeding strategies should aim to reduce the impact of virulent or intermediate footrot to that of benign footrot so that no specialised control strategies are warranted and the disease has minimum impact on production. An alternative strategy could be to improve the responsiveness of sheep to vaccination so that footrot could be managed similarly to the clostridial diseases, with annual booster vaccinations offering long-term effective protection.

On the basis of observations by Egerton et al. (1983), Raadsma et al. (1990) listed the key indicators of resistance to footrot as:
- failure to develop clinical footrot following challenge
- ability to show spontaneous healing
- accelerated healing after therapeutic vaccination
- failure to develop footrot following vaccination.

**Genetic variation in resistance to footrot**

Sources of genetic variation in resistance to footrot have recently been reviewed by Egerton and Raadsma (1991). In some cases large differences have been reported between breeds, strains and
flocks within strains (in the case of Merinos), but most reports were based on inadequate numbers of animals and/or description of footrot resistance indicators. Skerman and Moorhouse (1987) report on the development of bloodlines in the Romney Marsh and Corriedale breeds with increased resistance to footrot. Both bloodlines evolved through direct selection under natural outbreaks of footrot, and both studs claim to have reduced footrot to an insignificant problem.

Only a limited number of investigations report on genetic variation in resistance to footrot between sheep within-flocks. In New Zealand, Alwan (1983), Baker et al. (1986), and Skerman et al. (1988) report a moderate (0.28–0.55 ± 0.26) heritability for liability of footrot derived from the expression of affected and non-affected sheep. When considering only sheep affected with severe lesions (underrun) and sheep not affected with severe lesions, the heritability appears to be lower (0.07–0.17 ± 0.29). The latter distinction is important, since the interest is to reduce the number of sheep with severe lesions in the flock.

For Merino sheep, Raadsma et al. (1990) outlined a study which was specifically designed to obtain estimates of the heritability of resistance to footrot for a range of conditions. These include the development of footrot following experimental challenge, natural exposure on pasture, and following vaccination. From results in the first year of the experiment it was evident that not all sheep succumbed to footrot after initial challenge. Some sheep remained free from footrot for the duration of the experiment, some sheep showed clinical signs of severe footrot at all inspections, while others contracted footrot but were able to overcome the disease through self-cure or after vaccination. The heritability of footrot severity score was 0.14 ± 0.12 during challenge, and 0.22 ± 0.14 after vaccination.

Following the introduction of infected donor sheep, a natural outbreak occurred over a 4-month period in the same group of animals. The heritability of resistance to natural footrot was 0.34 ± 0.15 for footrot scored during transmission, after subsequent vaccination, the heritability of footrot score was 0.17 ± 0.14. The genetic correlation for resistance to induced footrot and natural footrot was 0.86 ± 0.34. It is intended to repeat this study for a further 2 years to screen another 800 progeny from a further 80 sires to obtain accurate estimates of the relevant genetic parameters.

Indirect selection for resistance to footrot

The option of exploiting genetic differences in footrot through direct selection is clearly not practical as it would require challenging all animals before selection. We are therefore examining the possibility of using indirect selection strategies. To minimise the effect of challenge on vaccine responsiveness, we are monitoring serological responses in non-challenged progeny group following a full course of vaccination with a decavalent vaccine. Routine serological procedures based on K-agglutination and ELISA techniques form the basis of this screen. The other options for indirect selection strategies include the use of genetic markers linked to resistance, including RFLP analysis (See Raadsma et al., these proceedings).

Resistance to footrot and other breeding objectives

It is unlikely that resistance to footrot will be the sole breeding objective in well-designed programmes. It is therefore important to have accurate information on resistance to footrot and all the major production traits which are recommended as breeding objectives and/or selection
criteria for sheep. In high rainfall areas where resistance to other important diseases such as flystrike and internal parasites may also need considered, relationships with resistance to footrot need to be known. No estimates of the relevant phenotypic or genetic parameters are available for any of these diseases or their relationship to production traits. The study described by Raadsma et al. (1990) seeks to obtain estimates for footrot, fleece rot, flystrike and resistance to dermatophilosis, as well as all major production traits.

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REFERENCES