The selection and breeding of Merino sheep for footrot resistance

RODNEY AND HELEN PATTERSON

"Longview", 5K R.D., Oamaru, North Otago, New Zealand.

The practical side of selecting and breeding Merino sheep for footrot resistance works in practice but does it work in theory? Our aim is to breed high income producing Merinos in terms of clean wool weight, micron, and number as well as weight of lambs weaned under the environmental conditions in which we want to run them in order to achieve maximum short term as well as long term nett profitability and capital gain.

Footrot is a significant, severely debilitating disease particularly of Merino sheep and indirectly man, being an economic, emotional and social burden. It is one of the most feared and dreaded of all common sheep diseases presently in New Zealand. Significant and continuing financial losses are incurred by the sheep industry through production foregone and costs of treatment and prevention (Skerman *et al.*, 1988). Despite all the claimed advances in footrot treatment the incidence today is probably as high as it has ever been.

The survival of any species in a competitive and ever changing environment is dependant on sufficient genetic variation and adaptability to meet the challenges. A successful species not only achieves this, but thrives ready to surmount the next challenge e.g. the presence of serious diseases such as bacterial wilt and stem nematode in lucerne doesn't mean we persist with the cultivars bred in my grandfather's time. We assess what the local disease problems are and are likely to be and choose the cultivar accordingly. Resistant lucerne cultivars persist and produce where susceptible cultivars are eliminated (see Table 1) (Dunbier and Easton, 1982). At present the options for doing this within and between sheep breeds are rather limited, although breeding for footrot resistance in some Romneys and Corriedales is well established.

It is essential in any selection programme for disease resistance to have a base population with wide genetic diversity that has sets of broadly adaptable fitness genes and locally adaptable fitness genes. For example, white clover (Williams, 1987) and Merino sheep can both be found from the sub-tropics to the sub-Antarctic with their specialised locally adapted strains being a characteristic of their outstanding success. This is why there is such a strong emphasis on cultivars and bloodlines for particular environments. During the next stage of the programme it is essential to create a highly repeatable and reliable selection differential to achieve the desired end results through time.

TABLE 1 Resistance in susceptibility of different lucerne cultivar.

Cultivar	Bacterial Wilt	Stem Nematode	Aphids
Wairau	S	S	S
WL 320	R	R	R & S
AS 13 R+	R	R	R & S
Otaio	R	R	R

Through the naturally occurring genetic variability present in Merino populations we can select for desirable traits. Natural selection for neophobic and poison-shy rabbit populations, drench resistant worms and dip resistant flies in sheep have occurred over the past few decades. Footrot has been associated with sheep for a much longer time so it is reasonable to expect varying degrees of susceptibility and resistance to be present in the population. Despite the claimed purity of the Merino breed the modern Australian Merino is in fact descended from over 20 historical breeds (Massy, 1990). This ancestral variability is reflected in the susceptibilities of strains or bloodlines to footrot ranging from almost totally susceptible to those exhibiting a reasonable degree of resistance. We have screened a wide selection of the main Australian derived New Zealand bloodlines, and one Australian bloodline directly but most aren't represented in our

selected flock.

Rather than continually using chemical treatments to relieve disease symptoms in our 28,000 Merinos, the future direction and challenge is to select and breed from sheep that as well as being outstanding in terms of wool production and lambs weaned will naturally live in harmony with their environment be it gastro-intestinal nematode resistance, flystrike resistance, footrot resistance (it is interesting to note that all three are effectively skin diseases) or preferably all three plus possibly others such as facial excema resistance. Animals with the above debilitating and performance reducing diseases will never perform to their full potential, so we see little point in trying to increase this potential in isolation from disease resistance if it isn't going to be fully realisable in the commercial flock situation. In fact on most farms the annual feeding level isn't sufficient for this potential to be fully expressed anyway. What we want to breed are high performing, competitive, easy care, low input Merinos. We consider that current research should be directed towards breeding this type of sheep for the benefit of the whole commercial Merino industry.

The reasons and thinking behind why we decided to follow the selection and breeding path to footrot control along with the strengths and weaknesses of the traditional approaches to control measures have been discussed in an earlier paper (Patterson & Patterson, 1989). We have learnt and appreciated more about the vagaries of footrot by actively trying to encourage it than we ever learnt trying to become footrot free again. The incidence, severity and duration of footrot can be easily recorded in individual Merino sheep and is highly repeatable through time. We believe that footrot resistance is inherent or innate, as fully resistant animals never show any sign or symptom of the disease. There is no evidence in our observations that resistance is acquired i.e., once having had footrot Merinos do not then develop short-term, let alone long-term immunity. Skerman *et al.* (1988) also noted this in their work at Wallaceville. In fact animals that have had footrot previously are difficult to cure completely and are virtually always the most susceptible and have the longest and most severe infections following a new challenge.

After observing and recording natural outbreaks when some rams always had footrot and others never succumbed, we then challenged our rams by deliberately attempting to give them all footrot. Even after four years of trying we haven't yet managed to accomplish this. Production traits don't appear to be genetically correlated with susceptibility to footrot and we progeny test resistant rams with the highest clean fleece weights and lowest microns.

Culling only ewes is a highly successful way of reducing footrot, but little will be gained long term if susceptible rams continue to be used. The commercial ewes in our flock aren't deliberately challenged but are seldom treated, and those showing any sign of footrot are culled from the selection programme. This results in a culling margin of up to 50% of the ewes for footrot when they enter the programme, and continues on with a further 20-40% from the original population, with intermediate resistance, being culled before they reach the end of their productive life.

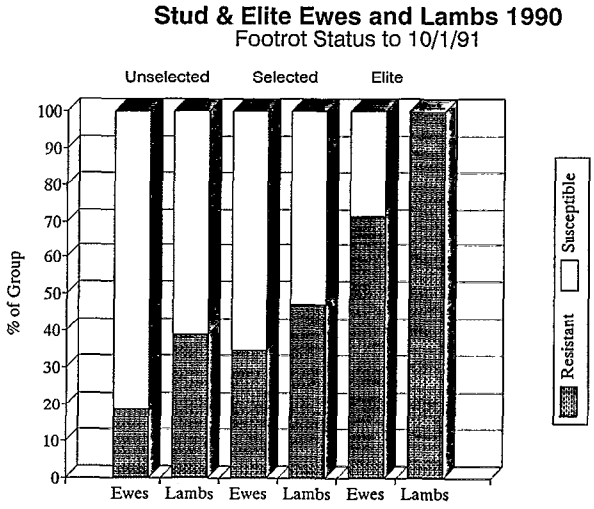

FIG 1 Comparison of footrot status of ewes and lambs (up to 18 weeks old) at weaning, run in the same mob from 7 weeks of age.

All progeny however are deliberately continually challenged under irrigation and we are slowly building up a resistant flock of elite rams and ewes. In future the progeny out of these highly selected parents will form the basis of our flock. Although this is the first season that we have produced these elite Merinos (that is

progeny from two selected parents) they are exhibiting a high degree of resistance and the next stage is to cross these with susceptible, intermediate and resistant groups to test the overall effectiveness of our programme before commercially releasing our rams as being bred for footrot resistance. (See Fig. 1)

We find under deliberate challenge to footrot that the most susceptible animals have the greatest incidence, severity and duration. As the challenge progresses the intermediate group succumb to mild footrot in one or two feet with natural remission often evident especially if conditions become less favourable for footrot spread by becoming either too dry or too cold. Intermediate levels of resistance are reflected firstly in the time taken by different groups of sheep to become affected after exposure and secondly, to heal after becoming infected either naturally or following treatment (Egerton *et al.*, 1983). The intensity and duration of the challenge determines the progression of infection. A severe and ongoing challenge is essential to be able to delineate between the intermediate and the resistant group. The resistant group shows no signs or symptoms of footrot.

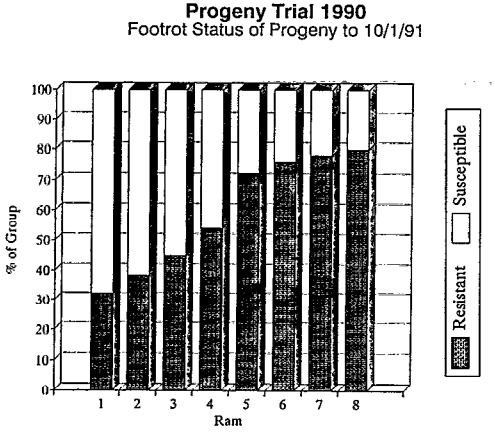

FIG 2 Footrot status of lambs at weaning up to 18 weeks old, run in same mob from 4 weeks old. Rams 1-8 exhibited increasing levels of footrot resistance.

The footrot history of 8 sires was correlated to the footrot status of the progeny (See Fig. 2). In our experience if a susceptible to intermediate ram from a family that has generally shown high degrees of resistance is progeny tested it is likely to leave significantly more resistant progeny than an unrelated ram of equivalent footrot history or status. This coupled with the strong family influences that we have recorded indicate to us that the susceptibility to footrot is genetically controlled. The available evidence indicates that resistance to footrot is multigenic as its inheritance certainly doesn't follow simple Mendelian genetic principles.

Three years ago we progeny tested 7 rams across 550 ewes, the following year 8 rams across 950 ewes and this current season 32 rams across 2,500 ewes. 51 rams across 4,000 ewes will be progeny tested in the coming season, *including the same selected ram against selected and unselected ewes.* Susceptible animals are foot trimmed and then treated weekly because the physical condition of most untreated animals deteriorates rapidly. A few seem to be little affected and exhibit high degrees of tolerance. Most of those susceptible animals are disposed of to the freezing works. Vaccinating isn't considered an option as it isn't cost effective due to the short term protection it gives and its low success rate. The resulting prolonged abscesses which may last for several years automatically ensure a financial penalty at the freezing works.

A question that we are often asked is, "Why are you bothering to go to all this trouble? Is it worth it?" The fact is we enjoy running Merino sheep intensively under high rainfall on improved pastures for their inherent productivity and profitability, but don't like footrotting. Footrotting must be one of the worst jobs on any farm. It is rewarding however to tip up Merinos that have been challenged for long periods of time and find that they are resistant. Having, or claiming, a footrot-free property under low rainfall means little if Merinos are shifted onto improved pastures, higher rainfall areas or irrigation, as most older Merinos are sooner or later. Being footrot-free is a temporary rather than long term state on high rainfall farms with oversown and topdressed pastures, as conditions can be so favourable for explosive spread from either internal or external sources. In our programme every sheep culled with footrot is a positive step in the right direction, as we

are left with a more resistant population. The change to another breed of sheep that is more immune to footrot doesn't make economic sense especially when that immunity is already present in the Merino breed. It is simply a matter of identifying those animals with the necessary combination of genes and then breeding from them.

We are sure that eventually even in the conservative, traditional and vested interest Merino breeding industry that ram breeders will have to offer highly productive animals selected on genetic merit in the commercial situation, that will live in harmony with their environment. The main barrier to any selection programme, be it for objective measurement or disease resistance, is that immediately half the flock are placed below average and it's difficult to convince anyone that they have below average sheep in their flock. In the past, the art of successful ram breeding has often been to sell 100% of the previous years male lamb crop for $500 instead of culling inferior males as $30 wethers.

Even though we have a Merino stud the available gene pool is too small and the expense too great to make as much progress for footrot resistance or performance breeding as we can make in our commercial sheep.

Progeny from subjectively assessed parents have little relevance today in industries such as the evolving and advancing dairy, pork and poultry industries which once in world-wide terms had stud industries many times larger than the present day Merino stud industry. The previous reliance on blue-blood pedigrees from small and deliberately narrowly based gene pools and selection on subjective non-performance related characteristics rather than objectively measured high performing ones has gone the way of other dinosaurs. In order to be competitive long term it is essential to evolve and adapt to thrive.

There is more to life than the tiresome burden of continually treating susceptible Merino sheep for footrot, even if they have blue blood in their veins, when through the above approach of selection and breeding its incidence, severity and duration can be reduced then minimised in a cost-effective and rewarding manner.

The future challenge is to establish a reliable blood test that will accurately predict the disease susceptibility of any sheep, rather than just emphasising the potential and promise of biotechnology. We would appreciate practical results rather than theoretical hype and mumbo-jumbo so that sheep with innate disease resistance can be accurately identified prior to challenge and thus the impact of diseases minimised in the commercial farming situation.

REFERENCES

Dunbier, M.W. and Easton, H.S. 1982. Longer stand life with new cultivars. *Agronomy Society of New Zealand Special Publication No. 1*: 121-6.

Egerton, J.R., Ribeiro, L.A., Kieran, P.J. and Thorley, C.M. 1983. Onset and remission of ovine footrot. *Australian Veterinary Journal 60*: 334-6.

Massy, C.J. 1990. The Australian Merino. Viking O'Neill, Penguin Books Australia Ltd.

Patterson, R.G. and Patterson, H.M. 1989. A practical approach to breeding footrot resistant Merinos. *Journal of New Zealand Mountain Lands Institute 46*: 64-75.

Skerman, T.M.; Johnson, D.L.; Kane, D.W. and Clarke, J.N. 1988. Clinical footscald and footrot in a New Zealand romney flock: Phenotypic and genetic parameters. *Australian Journal of Agricultural Research 39*: 907-16.

Williams, W.M. 1987. Adaptive variation. In: *White Clover*, Eds. Baker, M.J. and Williams, W.M. *C.A.B. International*, United Kingdom. pp 299-321.