**Genetics of scrapie**

Genetic selection studies in Cheviot, Herdwick and Swaledale sheep have been used to create flocks with different susceptibilities to experimental scrapie infection. From these it has been concluded that the incubation period of scrapie is controlled by one major gene (Sp [scrapie incubation period]) with two alleles (S and pA). Sp SA homozygotes have the shortest incubation period, Sp pAPa heterozygotes have a longer incubation period and Sp pA homozygotes have extremely long incubation periods and are relatively resistant to clinical scrapie. Further studies in Suffolk sheep indicate that the genetic control of natural scrapie infection is similar.

Studies in sheep and mice suggest that following infection the scrapie agent develops and persists in extra-neural tissues including the spleen and lymph nodes and that clinical scrapie only develops following spread to the central nervous system (CNS). In mice, the Sinc gene controls multiplication of scrapie in the CNS, but not in the lymphoreticular system. There is evidence that the Sp gene in sheep acts in the same way; therefore, while clinical disease may not develop in Sp pA homozygotes, these animals may be carriers of the disease in extra-neural tissues and a source of environmental contamination or transplacental infection.

PrP is coded for by a single PrP gene, which is present in all mammalian species. Specific mutations in the human PrP gene are linked with increased susceptibility to familial Creutzfeld-Jakob disease. There is growing evidence that the supposed Sp gene is the same as the PrP gene and that mutation in the sheep PrP gene is a fundamental component in the pathogenesis of scrapie.

The PrP gene can be extracted from EDTA blood samples and amplified by polymerase chain reaction to read the DNA sequence. Sites of polymorphism in the PrP gene have been identified at amino acid loci 136, 154 and 171. In Swaledale sheep, all scrapie cases have glutamine/glutamine at site 171 (GLN:GLN 171), while only 50 per cent of unaffected sheep have GLN:GLN 171 (others have arginine: arginine [ARG:ARG 171] or ARG:GLN 171). Assuming that the PrP gene is the same as the supposed Sp gene, it is reasonable to assume that the incidence of scrapie in the progeny of ARG:ARG 171 rams will be low.

for several generations. Using different lambing fields in subsequent years may reduce pasture contamination.

The use of rams for one year and subsequently only after a period of two to three years – when the scrapie prevalence in their progeny has been determined – is an economically inefficient control measure and likely to be surpassed by DNA sequencing (see box above). Identification of ARG:ARG 171 rams and their use in closed flocks with a high incidence of scrapie should result in none of the progeny having GLN:GLN 171 and consequently reduce the incidence of scrapie. This strategy is the basis of the Swaledale Sheep Breeder Scheme, currently under evaluation.

**ERADICATION**

Eradication by slaughter has been unsuccessfully attempted in Iceland and the USA. In the absence of a serological diagnostic test in the live animal, a whole flock eradication policy is required. Unfortunately, persistent environmental contamination and the inability to guarantee scrapie-free status in replacement stock, makes freedom from scrapie following repopulation impractical in most circumstances.

**Further reading**


**Answers to CROSSWORD**

| Across | 5 Mitochondria 6 Zein 7 Epiphora 8 Vizsla 9 Minim 11 Visna 13 Cortex 15 Gracilis 16 Ossa 17 Fibrillation |
| Down | 1 Stenosis 2 Sclera 3 Corium 4 Migram 5 Maedi 10 Narcosis 11 Vermis 12 Allele 13 Costal 14 Eosin |

**CORRECTION**

Poisons: Alphachloralose (In Practice, September 1995, p 381)

The box listing the clinical signs of alphachloralose poisoning in the cat and dog should have included hypothermia, and not hyperthermia.
Poisons: Alphachloralose

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doi: 10.1136/inpract.17.10.469

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