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Potential transmission routes of rabbit haemorrhagic disease: consequences for epidemiology of RHD in wild rabbits

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INTRODUCTION

Rabbit haemorrhagic disease (RHD) was released in New Zealand in the spring of 1997 and has subsequently spread naturally and with the assistance of people to most, if not all, rabbit populations in the country (Parkes *et al.*, 2001). The disease and its impact on rabbit numbers have been described for the initial epidemics in Otago rabbit populations (O'Keefe *et al.*, 1999), in ongoing cross-sectional studies at 10 sites in Canterbury and Otago (Parkes *et al.*, 2001), and in a single longitudinal study in the Manawatu (Henning *et al.*, 2000).

In this paper we wish to discuss alternative ways the RHD virus (RHDV) might be transmitted to rabbits, report on the evidence from field and pen trials on various transmission routes, and speculate on the possible routes that fit the observed epidemiological data from our study sites in Otago and Canterbury.

RESULTS

Direct transmission

RHD V is present in all body secretions of infected rabbits, and susceptible rabbits can be infected orally, nasally, through the eyes, or via wounds (Westbury *et al.* 1994). Therefore, it is likely that direct transmission from infected to susceptible rabbits occurs in the wild. One might think that the rate of transmission by this mechanism would depend on the contact rate between rabbits and so on rabbit density (e.g., Barlow & Kean, 1998). Parkes *et al.* (1999) presented data purporting to show an effect of rabbit density on the efficacy of RHD, with consistently high mortality at high rabbit densities but variable mortality rates at low densities. This may indeed be so, but more careful analysis of the data showed the conclusion was too bold as the methods used to estimate mortality rates (spotlight count indices) are imprecise at low rabbit densities (Fletcher *et al.*, 1999; Parkes *et al.*, 2001). Further, there are some study sites, e.g., at part of Earnsclough Station in central Otago, where the disease has persisted and continued to suppress rabbit numbers despite very low rabbit densities (Parkes *et al.*, 2001).

Rabbits infected with RHDV have been reported with sub-acute and chronic forms of the disease, although no details about these states were reported, particularly if they involved infectious animals (Marcato *et al.*, 1991). However, Zheng *et al.* (2001) did report the presence of RHDV in the tissues of antibody-positive rabbits in New Zealand, suggesting that an infectious state may be possible.

Indirect transmission

Free-living RHDV can survive in the environment for

many weeks at temperatures of up to 20°C (Rodak *et al.*, 1991), and has been found in rabbit carcasses (Cooke, 1999). It is excreted by rabbits in their urine and faeces leading several researchers to conclude that virus may persist in the environment (e.g., Simon *et al.*, 1998; Marchandeanu *et al.*, 1998; Cooke, 1999).

Victims of RHD often die above ground and many are then scavenged by feral cats, ferrets and harrier hawks. These species also become seropositive to RHDV (Parkes *et al.*, unpubl. data), although to date there is no convincing evidence that the virus can replicate in anything but the rabbit. Nevertheless, it is known that foxes and dogs that eat viremic rabbits shed viable virus in their faeces (Simon *et al.*, 1998), and so the New Zealand vertebrate scavengers may spread virus by contaminating the environment.

A more likely route of contamination to the environment and so to susceptible rabbits is from the scavenged or infectious rabbits via blowflies and other insects in contact with carcasses or infectious secretions. It is known that RHDV occurs in the gut of insects, such as flies attracted to dead animals, mosquitoes, and rabbit fleas (Cooke, 1999), that the virus is viable for up to 9 days in insects, and that there is enough virus in fly spots on vegetation to infect rabbits that eat a spot (Asgari *et al.*, 1998).

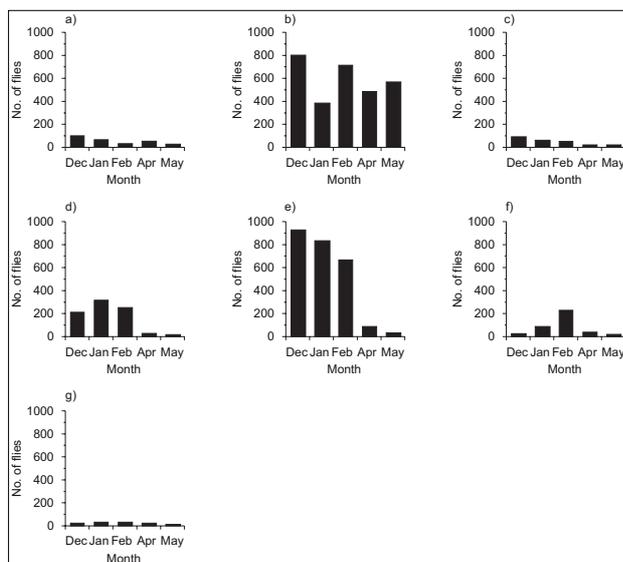
A review of potential insect vectors of RHD in New Zealand (Crosby & McLennan, 1996) suggested a variety of flystrike and carrion-feeding blowflies and a fleshfly (the striped dung fly *Hybopygia varia*) might act as vectors of RHDV. Direct vectors are flies that land on infected rabbits, pick up the virus, and then land on susceptible rabbits. Indirect vectors are flies that visit contaminated rabbits (probably mostly carcasses) passing virus on to food that is then eaten by a susceptible rabbit. There is circumstantial evidence from New Zealand trials, which allowed or excluded flies from caged susceptible rabbits, that insects transmit the disease (Barratt *et al.*, 1998). Crosby & McLennan (1996) thought that free-living biting insects, such as mosquitoes or sandflies, would be unlikely vectors, although they apparently underestimated the time the virus might be viable in the gut of insects. Parasitic biting insects such as fleas are known to transmit RHD in Australia (Cooke, 1999). However, rabbit fleas are absent from New Zealand, although a survey done in the 1950s (Bull, 1953) showed a mite (*Listrophorus gibbus*) and a louse (*Haemodipsus venticosus*) were widespread in wild rabbits in New Zealand.

In New Zealand, five species of blowflies have been shown to carry RHDV. Heath *et al.*, (1999) found RHDV in the native bluebottle (*Calliphora quadrimaculata*), the golden brown blowfly (*C. stygia*), the European blue blowfly (*C. vicina*), and the European sheep blowfly

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FIGURE 1: Monthly abundance of flies caught in baited traps at Earnsclough and Matangi in Central Otago. From top left across: a) *Calliphora stygia*, b) *C. vicina*, c) *C. quadrimaculata* (x10), d) *Lucilia sericata*, e) *Xenocalliphora hortona*, f) *Hybopygia varia*, g) *Hydroteia rostrata* (x 10).



(*Lucilia sericata*), and Barratt *et al.* (1998) found RHDV in the striped dung fly. Figure 1 shows the abundance of seven species of flies sampled in traps baited with dead rabbit at two sites in central Otago between December 1998 and May 1999. *Calliphora vicina* was the only species abundant in all months, and the small blue carrion blowfly, *Xenocalliphora hortona*, was abundant until February. *Lucilia sericata* appeared to vanish after about February, and *H. varia* only appeared in abundance in January and February, although it was very abundant in samples taken in March 2000 in North Canterbury (J. Parkes, unpubl. data). The other species were rarely caught in the traps in Otago. The disease was active in Otago during the late summer and early autumn of 1999 (Parkes *et al.*, 2001).

Route of infection and the fate of young rabbits

Nine-week-old rabbits orally dosed with RHDV in a New Zealand trial all survived the disease, only 1 of 12 produced antibodies, and most (16 of 18, i.e., the 12 dosed and 6 control rabbits) died when orally redosed at 17 weeks (Parkes *et al.*, 2001).

In contrast, Robinson *et al.* (2001) showed that young Australian rabbits (5 to 11 weeks old) generally also survived inoculation with RHDV, but usually seroconverted and were presumably immune to further challenge.

DISCUSSION

The results from 10 South Island sites allow two general conclusions to be made (Parkes *et al.*, 2001). Firstly, the disease has persisted at all study sites, although not in every year since 1997. Often, but not always, there is an outbreak of disease in the spring, affecting rabbits born in the previous spring and earlier, and/or a second epidemic in the summer or autumn when young rabbits born during the spring recruitment pulse (Gibb & Williams, 1994) are old enough to be infected; rabbits younger than about 10 weeks being resistant to RHD (e.g., Morisse *et al.*, 1991). Incidentally, part of the mechanism of this resistance appears to involve

the maturation of ABH blood antigens on the surface of epithelial tissues in the young rabbits, which do not permit the virus to bind and enter the cells until the A and H antigens mature (Ruvoen-Clouet *et al.*, in press), i.e., young rabbits are not effectively challenged by the oral route, as we found in the pen trial reported above. These ABH antigens are common to all vertebrates and it is interesting that we found no antibodies in young ferrets despite the presence of antibodies in adult ferrets from the same populations sampled in areas where RHD was active in rabbit populations (Parkes *et al.*, unpubl. data). However, inoculation that bypasses this barrier raises antibodies but fails to kill young rabbits (Robinson *et al.* 2001) so there are clearly mechanisms or sites other than that described by Ruvoen-Clouet *et al.* (2001) that are involved with juvenile rabbit resistance to RHDV.

The second general conclusion is that there appear to be two broad outcomes of the disease. One outcome results in a sustained (so far) reduction in rabbit densities, typically leaving about 30%, on average, of the rabbits that survived the epidemics with antibodies to RHD, but with no relationship in seroprevalence between successive generations of rabbits. The other outcome results in no reduction in rabbit numbers, up to 90% of the rabbits with antibodies, and a direct relationship between the proportion of seropositive animals in successive generations, i.e., suggesting 'longitudinal' transmission of immunity at these sites; although whether this is from mother to young or just the outcome of some factor common to the site between years is unclear (Parkes *et al.*, 2001).

The first outcome (high mortality and modest antibody prevalence with no inter-generational effects) casts no particular light on likely transmission routes. However, the second outcome (low mortality and related and often high antibody prevalence in all age cohorts) suggests two transmission routes might be most likely; if the phenomenon is caused by the way the virus is transmitted. One possible route is via inoculation by biting insects. If biting insects were present at these sites (but not at those where we see the first outcome), it is possible that young rabbits inoculated by insects carrying RHDV could survive and be immunised using the mechanism demonstrated by Robinson *et al.* (2001).

Another possible route is from mother to young. If seropositive rabbits at these sites remained infectious, mothers could infect their offspring when the latter were at just the right age to seroconvert and survive. The problem with this explanation is if the route from mother to young was oral, we would expect the young to survive and not seroconvert. So we would have to speculate either that a right dose delivered at just the right age (somewhere between 9 and 17 weeks) would be enough to overcome the ABH antigen barrier but not enough to be fatal, or that the route might be through the eyes and behave differently, or via broken skin and scratches and act as an inoculum.

Since the major biociding conducted in the initial releases of RHDV in 1997, anecdotal evidence and some trials in 2001 (B. Reddiex, pers. comm.) suggest some attempts to start epidemics by local baiting with a commercially available bait have failed. Further, epidemics have recurred in areas with very low rabbit densities (Parkes *et al.*, 2001) suggesting that direct rabbit-to-rabbit

transmission is not essential, and that indirect agents of spread, such as flies, might be important. There is good circumstantial evidence that flies do transmit RHDV, however identifying which species are most important would require much more detailed observations to match the start and end of epidemics with the increase and decline in abundance of different fly species observed to carry RHDV. Our data on all these factors were not collected frequently enough to determine any coincidences, and there is no published information on the seasonality of fly abundance in our study regions. Studies on the seasonality of fly abundance in the North Island (e.g., Murray, 1956; Dymock *et al.*, 1991) show considerable variations between years, habitats, and trapping methodologies, suggesting that field studies to understand the timing of epidemics would not be simple.

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