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**Risk of Tb Persistence and Spread: the Role of Deer, Pigs, and
Ferrets**

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Summary

Project and Client

The role of wild deer, feral pigs, and ferrets in maintaining and spreading bovine tuberculosis (Tb) was assessed by Landcare Research, Lincoln, for the Animal Health Board (Project R-10576) between July 2002 and December 2003.

Objectives

To assess the risk of Tb persistence and spread (despite possum control) by wild deer, pigs, and ferrets, and identify information and strategies needed to prevent this, by:

- synthesising current information on dispersal, demography, Tb prevalence, and transmission routes and frequencies in wild deer, pigs, and ferrets;
- developing a probabilistic model linking the risk of Tb persistence and spread to deer, pig, ferret, and possum density, to help evaluate the effectiveness of potential strategies for management of tuberculous deer, pigs, and ferrets;
- identifying knowledge gaps that might preclude development of practical containment strategies.

Main Findings

Deer: The densities of wild deer in New Zealand are well below the threshold for Tb persistence. Reduction of wild deer density is therefore not an essential part of Tb eradication. However, wild deer are a medium-term reservoir of Tb that can potentially infect other species and, very occasionally, other deer. That risk remains for 10–15 years after possum control. The risk is highest inside Vector Risk Areas, but exists at declining levels for up to 30 km beyond.

There is enough global information and understanding on deer movements, age structures, and the pathogenesis of Tb to be able to model and broadly predict the risk wild deer pose as a host in maintaining and spreading Tb. The major information gap is robust data to quantify the risk of transmission to possums, either directly or via other species. More information on the fate of infected deer would also help clarify how long such deer are likely to remain a threat to Tb eradication.

Pigs: In general pigs do not contribute to the maintenance of Tb within an area. The main unknown is whether in areas where both ferret and pig densities are high, co-scavenging between the two species amplifies and maintains the infection longer than would otherwise be the case.

There is good evidence that pigs spread Tb more widely than do deer. Better information on how often pigs are translocated (both alive and dead) by hunters and the fate of those animals would also help characterise the likelihood of Tb spread by pigs.

Pigs provide a source of infection not only to other scavengers such as ferrets, but also, occasionally, for possums. Again the main information gap is data on how often this occurs in different habitats and with different mixes of species at different densities.

Ferrets: Ferrets at high densities may be maintenance hosts, requiring control to reduce their densities. Where ferrets densities are naturally low, or are held low by control, Tb is unlikely to persist more than a few years in ferrets. Provided vector control reduces the transmission of Tb to ferrets to zero or near zero, and assuming almost all dispersal is by juveniles, the risk of Tb spread by ferrets once intensive vector management has begun appears to be quite low. Offsetting that, ferrets are more likely to disperse, and to disperse longer distances than deer, and possibly also pigs.

The frequency with which dispersing infected ferrets establish new foci of infection is not known, either in absolute terms or relative to pigs and deer. As with pigs and deer, the main information gaps are how often infected ferret carcasses are scavenged by pigs, possums, and other ferrets and how often such scavenging causes infection. It is also not clear whether a ferret–pig loop, not requiring possums for maintenance of Tb, exists in some dryland areas of New Zealand.

Recommendations

Management

Deer control should not routinely be included as part of vector control operations within VRAs, except perhaps where vector control is being initiated in areas with both high prevalences of Tb and high densities of deer, and in the outermost 10 km of a VRA (for red deer; 5 km for fallow).

The AHB should, as a precautionary measure, increase efforts to prevent the spread of Tb by pigs, by both educating hunters about the Tb risks and by a programme of intensified surveillance doubling also as partial pig control in areas just outside known areas of infection.

Within VRAs, there is generally little need for pig control to reduce Tb-persistence. One exception may be in areas where both pig and ferret densities are high, because here pig control could help speed the reduction in reactor rates by removing some of the reservoir of infection in the area.

Where ferret densities are high enough to sustain Tb, ferret control is essential. At densities below that, complementing possum control with ferret control will reduce reactor rates more quickly (particularly if combined with pig control where necessary). As with pigs, a surveillance buffer (doubling as moderate control) of 10–20 km should be maintained around the boundary of established infection.

The AHB should consider increasing the width of buffer already incorporated in VRAs to at least 10 km, especially where the density of at least one of the spillover hosts is high.

Research Priorities (not in order)

- How often possums acquire Tb by scavenging/contacting infected possum, deer, pig, and ferret carcasses
- Dispersal of pigs in continuous native forest
- Effect of Tb infection on ferret survival and, also, validate the Caley (2001) threshold
- Likelihood of Tb re-establishing in a recovering possum population after cessation of possum control, and modelling the role of possums in spreading Tb
- Identification, via social science, of how to prevent hunters potentially spreading Tb.
- Likelihood of Tb re-establishing in a recovering possum population after cessation of possum control
- Model the role of possums in spreading Tb

1. Introduction

The role of wild deer, feral pigs, and ferrets in maintaining and spreading bovine tuberculosis (Tb) was assessed by Landcare Research, Lincoln, for the Animal Health Board (Project R-10576) between July 2002 and December 2003.

2. Background

Wild deer, feral pigs, and ferrets are frequently infected with Tb in areas where the disease is also established in possums, but most of the infection in these three species is thought to arise from transmission from sympatric Tb-infected possum populations. Despite being predominantly spillover hosts, these three species pose a potential threat to the *National Pest Management Strategy for Bovine Tb* (NPMS; Animal Health Board 2001), which aims to eradicate Tb from wildlife, largely by control of possums. The risk is that, as long-lived and/or wide-ranging species, deer, pigs and ferrets may carry Tb through both time and space, and eventually establish infection in possums in areas not previously infected, or reinfect possums in areas where possum control has been stopped after apparently successful elimination of Tb.

In this report we attempt to assess the nature and importance of these risks, and the threat they pose to the AHB's goal (official freedom from Tb by 2013), by summarising currently available data on dispersal and movement patterns, population density, demography, disease dynamics, and the route and frequency of intraspecific and interspecific transmission for each of the three species. We then attempt to link the information on movements, prevalence, and transmission in simple models aimed at predicting the likelihood that each of these species contributes significantly to the persistence and spread of Tb.

3. Objectives

To assess the risk of Tb persistence and spread (despite possum control) by wild deer, pigs, and ferrets, and identify information and strategies needed to prevent this, by:

- synthesising current information on dispersal, demography, Tb prevalence, and transmission routes and frequencies in wild deer, pigs, and ferrets;
- developing a probabilistic model linking the risk of Tb persistence and spread to deer, pig, ferret, and possum density, to help evaluate the effectiveness of potential strategies for management of tuberculous deer, pigs, and ferrets;
- identifying knowledge gaps that might preclude development of practical containment strategies.

4. Methods

4.1 Literature review and synthesis

We reviewed the scientific literature (and known unpublished data) on movement patterns, population density, disease dynamics, and the route and frequency of intraspecific and interspecific transmission, including three reports recently completed for the AHB, covering the role of deer (Nugent & Whitford 2003), pigs (Nugent et al. 2003), and ferrets (Ragg & Byrom 2002) as hosts of Tb.

We characterised qualitatively the ability of each species to maintain and spread Tb by considering factors such as the frequency of becoming infected, the likely or potential intraspecific and interspecific transmission mechanisms and pathways, longevity, home range size and dispersal patterns, and population density.

4.2 Modelling persistence and spread

We attempted to formalise the knowledge and insight summarised in this review by building probabilistic models of the relative risk of persistence and spread of Tb posed by each species. Key starting assumptions were that deer, pigs, and ferrets are all spillover hosts, and that possum densities within Vector Risk Areas (VRAs) are kept very low so that the disease will eventually disappear from spillover host populations. If this assumption does not apply (e.g. if ferrets are at densities sufficient for them to act as maintenance hosts both inside and outside VRAs), then the strategic solution currently applied to possums will be required for ferrets. That will, in turn, require a different epidemiological model than the one presented here.

The simulation models linked an assumed ‘typical’ density of each species within an infected area (i.e. a VRA) to an assumed population age structure, survival rate, within-species Tb transmission rate, and initial Tb prevalence, and a species-specific level of Tb-induced mortality. To model persistence we assumed possum control was applied at the start of the simulated period, and immediately reduced transmission from possums to zero. The persistence of Tb in each age class of the spillover host population was then calculated for each subsequent year. We assumed that ongoing recruitment maintained densities at a constant level. To assess Tb spread, we arbitrarily defined a ‘typical’ large VRA, and used age- and sex-specific dispersal data, where available, to describe dispersal from the VRA in relation to spillover host density within the VRA. In the absence of any quantitative data on the rate of transmission from any of these hosts to possums, we use modelled predictions about numbers of infected dispersers to estimate the minimum rate of interspecific transmission to possums that would be of significance in the spread of Tb. Because the models differed for each species, the details of the species-specific assumptions are provided in the results section.

5. Main Findings

5.1 Wild deer as hosts and spreaders of Tb

Population density and distribution of wild deer in New Zealand

The total population of wild deer in New Zealand in the 1980s was estimated to be about 250 000 (Nugent & Fraser 1993), mostly confined by hunting to about 60 000 km² of taller forest (Nugent 1992a). Red deer (*Cervus elaphus*), sika (*C. nippon*), and fallow deer (*Dama dama*) comprise over 85% of the deer within New Zealand (Nugent 1992b). Deer carrying capacities were thought to be 20–30 deer/km² in forested areas, but commercial hunting pressure has kept red deer densities mostly in the 2–5/km² range in the South Island and 5–15/km² in the North Island (Nugent 1992a). Sika and fallow densities tend to be higher, with fallow deer densities up to 40/km² recorded in parts of the Blue Mountains, Otago (Nugent & Yockney 2001). Deer continue to expand their range, mostly as a consequence of farm escapes or illegal liberations (especially of fallow and sika) (Fraser et al. 2000).

Home ranges, dispersal and movement patterns of wild deer

Home range size varies according to species, extent of forest cover, level of disturbance, and seasonal availability of food. Home ranges of males tend to be substantially larger and less stable than those of females (Table 1). Deer do not have exclusive territories (other than for males during the rut).

The rate at which deer dispersed from initial liberation points into previously unoccupied areas of New Zealand was highest for red and sika deer (1.6 km/yr) and lower for fallow, sambar, rusa and wapiti (Caughley 1963). The few historical data on deer movement patterns in New Zealand were summarised by Nugent (1993), and further assessed in two studies (G. Nugent, unpubl. data; Knowles 1997).

1. In mixed forest in Westland, home range size of six young female red deer for which there was enough data to calculate a home range size varied between 171 ha and 382 ha with substantial overlapping of the ranges. At the end of the 2.5-year study all but one animal was within 2.5 km of its initial capture location. Another female deer monitored in this study moved 6 km in the 11 days immediately after capture, and then was not found again, suggesting that capture had induced its dispersal. The only two males in this study had much larger range sizes (2106 ha and 2600 ha) than the females, but were both back within 3 km of their initial capture location by the end of the study (Table 1; Nugent, unpubl. data).
2. In largely tussock land in Otago, Knowles (1997) recorded home range sizes of 100–750 ha for four females, but two other females moved up to 19.5 km between successive re-locations and both had home range sizes of over 3000 ha. Maximum distances moved by the three males studied ranged from 9.3 to 22.4 km (Table 1) and home range sizes were between 2000 and 11 000 ha.

Red deer in New Zealand forests do not migrate seasonally, except perhaps where heavy snow forces them to lower altitudes in winter (Nugent 1993). Overseas data indicate that red deer with adequate resources and cover have home ranges of typically 200–300 ha for females, and at least 2–3 times larger for males, and that both male and female ranges are much larger where cover or food is limited.

Table 1 Home range sizes, and mean and maximum recovery distances of wild deer in New Zealand. The three most recent studies used radio telemetry to obtain multiple locations for each animal, while the early studies used self-attaching snare collars that gave only first and final locations. The mean and maximum distances are the distances between the first and final locations recorded for each deer, except for the Otago study (marked with an asterisk) where the maximum distance is the greatest distance between any two locations.

Area	Deer species	Mean or range of home range sizes (km ²). Sample sizes in parentheses		Mean distance (km)		Maximum distance (km)		Habitat and study method	Authors
		M (n)	F (n)	M	F	M	F		
Westland	Red	23.5 (2)	2.46 (6)	2.1	0.9	2.9	2.5	Mixed beech/podocarp; Radio-telemetry	G.Nugent (unpubl. data)
Otago	Red	20.0–110.0 (3)	1.0–7.50 (4) 33 (2)	-	-	22.4	4.2* 19.5*	Tussock: Radio-telemetry	Knowles 1997
Nelson	Red	-	-	3.2	1.4	32.2	6.4	Beech/ tussock; Snare collars	Gibb & Flux 1973 R.Taylor & B.Thomas, (unpubl. data)
Avoca, Canterbury	Red	5.2 (3 adult) 9.9 (8 subadult)	5.5 (48)	5.8	2.6	20.1	19.9	Beech/tussock: Snare collars	R.J. Henderson (unpubl. data)
Kaimanawa	Red	-	-	3.0	3.4	8.5	8.5	Beech/grassland/manuka; Snare collars	Davidson 1979
Kaimanawa	Sika	-	-	2.4	1.7	17.7	6.0	Beech/grassland/manuka; Snare collars	Davidson 1979
Blue Mountains	Fallow	2.1 (19)	9.9 (19)	0.9	0.5	2.5	4.2	Mixed pine and native forest; Radio-telemetry	Nugent 1994b

Red deer fawns begin to disperse from the maternal range within 6 months of birth, and continue to do so for up to 4 years (Staines 1974). Female deer often adopt ranges that overlap with those of their mothers, leading to the formation of matriarchal groups, but males usually leave the natal range completely. In non-migratory populations, few female red deer appear to disperse more than 4 km in their lifetimes, and very few shift more than 10 km (Fig. 1a). Males, particularly subadults or young adults, disperse more widely than females on average, with about 5% moving more than 10 km.

Fallow deer (Nugent 1994a, 1994b; Statham & Statham 1996; Dolev et al. 2002) generally have home range sizes and dispersal distances that are substantially smaller than those reported for red deer (Table 1; Fig. 1b), with measures for sika intermediate between those of fallow and red deer. (Table 1; Fig. 1c; Maruyama et al. 1978; Davidson 1979; Feldhamer et al. 1982; Borkowski & Furubayashi 1998).

Wild deer as hosts of Tb

Tuberculosis was first recorded in wild deer in New Zealand in 1956 on the West Coast (de Lisle et al. 2001), and has since been recorded in all of the larger VRAs (de Lisle & Havill 1985; Mackintosh & Beatson 1985; Nugent & Lugton 1995; Lugton et al. 1998; Nugent & Whitford 2003) and in red, sika and fallow deer (Cooke et al. 1999).

The susceptibility of deer to Tb varies among individuals and is influenced by climate, nutrition, age and genetic factors (Griffin & Buchan 1994; Mackintosh et al. 2000). Unlike cattle, deer rarely exhibit clinical signs of the disease until the last 1–2 weeks of life (Beatson 1985; Griffin & Buchan 1994). Some wild deer undoubtedly die of Tb, but Nugent & Whitford (2003) deduced from the increase in prevalence with age that Tb-induced mortality was low. Using these data, we estimate that less than 10% of infected deer actually die of Tb in regularly hunted areas.

High prevalences (>30%) of infection in some wild red deer populations in New Zealand (Nugent & Lugton 1995; Lugton et al. 1998), and sometimes-explosive outbreaks of Tb on farms (Griffin et al. 1998), led to speculation in the early 1990s that wild deer could also be maintenance hosts of Tb (Morris & Pfeiffer 1995). The establishment of Tb in wild white-tailed deer (*Odocoileus virginianus*) in Michigan, USA (Schmitt et al. 1997; O'Brien et al. 2002) confirms that possibility, but Hickling (2002) predicts that in the absence of the enhanced aggregation and interaction induced by supplemental feeding, Michigan deer would maintain Tb only when their densities exceeded about 12/km².

In New Zealand, the prevalence of Tb in wild deer populations rarely exceeds 6% unless infection has been present in the sympatric possum population for many years (Nugent 1998). Where infection in possums is longstanding, there is a positive relationship between the average area-wide possum density and Tb prevalence in deer (Nugent & Whitford 2003).

Wild fawns less than 10 months of age are seldom infected, even when a high proportion of their mothers have Tb, indicating that intraspecific transmission is rare (Lugton et al. 1998; Nugent & Whitford 2003). After about 10 months of age, however, Tb prevalence increases linearly with age for about 3 years, and faster in males (Nugent & Whitford 2003), suggesting that the risk of infection is largely constant. After about 4 years, however, prevalence stabilises and then declines. The decline in older deer is thought to reflect Tb-induced mortality and some loss of infection through resolution of the disease (Nugent & Whitford 2003). Consistent with that, a lower proportion of infected adult deer have typical Tb lesions than subadult deer.

O'Brien et al. (2002) found that in Michigan male white-tailed deer ≥ 2 years were more likely to be infected than females of similar age. In New Zealand, the overall prevalence in males and females is similar but this is possibly because a greater frequency of infection in young males is offset by a greater mortality due to infection in older males (Nugent & Whitford 2003).

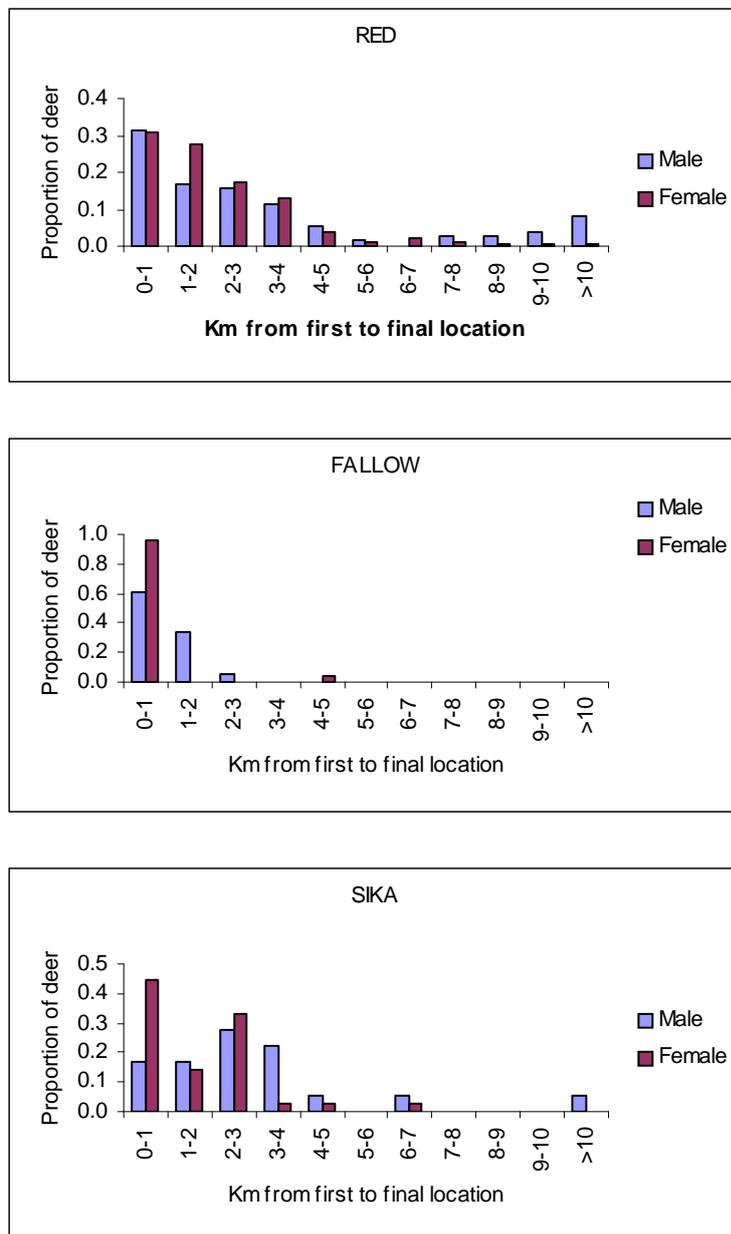


Fig. 1 Proportion of deer (by sex) for each first-to-final-location distance class for red deer from four areas (Nelson Lakes, Avoca, Kaimanawa Range and Westland (146 females, 108 males); fallow deer from the Blue Mountains (20 f, 33 m); and sika deer from the Kaimanawa Range (36 f, 18m). Adapted from Nugent (1993).

Transmission of Tb to and from wild deer

Transmission to deer: Infection via the tonsils is considered to be the primary route of transmission to both wild and farmed deer (Lugton et al. 1997) because infection is found most frequently in the tonsils (Lugton 1997) or in the retropharyngeal lymph nodes that drain the tonsils (Lugton 1997; Lugton et al. 1998). Supporting that, inoculation of the tonsils in experimental infection studies produces a pattern of infection similar to that of natural infection (Mackintosh et al. 1995). Tonsillar infection may occur when the animal is eating, drinking, licking, muzzling or grooming (Mackintosh et al. 1995). Aerosol transmission is thought to be of minor importance, because lung lesions are uncommon (seen in only 13% of infected wild deer; Nugent & Whitford 2003) and [\(in the early days of the Tb eradication programme, when the disease was able to progress far further than is possible today\)](#) coughing [wais](#) rarely seen in infected farmed deer, in contrast to cattle where thoracic lesions predominate and coughing [wais](#) a common feature of the disease (Mackintosh et al. 1995; Lugton 1997). Wild deer excrete few Tb bacilli for most of the time that they are infected (Lugton et al. 1998).

Intraspecific transmission of Tb has been demonstrated in captivity, between experimentally infected white-tailed deer and uninfected deer, both when they were in direct contact, and when they were not but shared the same housing facilities (Palmer et al. 2001). It has also been shown between experimentally infected red deer and uninfected deer held in a paddock (e.g. Mackintosh & Griffin 1994).

A few infected deer become highly infectious through development of severe respiratory infection, or open sinuses that drain bacilli onto the skin (Lugton 1997; Griffin & Mackintosh 2000). Rapid spread of Tb amongst farmed deer appears to always involve such severely infected individuals and probably occurs through direct contact with bacilli-laden discharges (Lugton 1997; Lugton et al. 1998). Lugton (1997) suggested that one possible transmission pathway is through the victimisation of weaker, diseased animals (by biting, for example) by other members of the herd; something that would be rare in wild deer, where sick animals have far greater scope to isolate themselves from other deer. Pseudo-vertical transmission is another potential pathway for intraspecific transmission, as fawns can become infected by ingesting contaminated milk (Palmer et al. 2002). However, no cases of grossly lesioned mammary tissue have been reported from wild deer in New Zealand. As noted above, very low infection rates in fawns <10 months old indicates deer-to-deer transmission is rare in the wild and also indicates that transmission via shared foodstuffs or environmental contamination is rare.

The other likely sources of infection for wild deer are cattle, ferrets, pigs and possums. Most infected deer are found in 'deep' forest, where they have little or no opportunity to interact with ferrets or livestock, eliminating those species as the major source of infection. Reducing possum density, but not pig or deer density, drastically reduces the rate of transmission to wild deer (Nugent & Whitford 2003), indicating that:

- 1) pigs and other wild deer are not the main source of infection for deer;
- 2) possums are the main source of infection; and
- 3) transmission is via some direct possum–deer interaction

Transmission from deer: Indirect transmission to possums and other herbivores (including livestock) via shared foodstuffs or environmental contamination is likely to be rare given transmission to other deer by this mechanism is rare. However, farmed deer have infected cattle (Hennessey et al. 1986). Transmission apparently occurred from pasture contamination in three cases and across a fence in another case. In all four cases, there was a high

prevalence of infection in the deer herds. By default, the main route of interspecific transmission from infected wild deer is by scavenging of their carcasses, mainly by pigs, ferrets, and possums.

Pigs have been videotaped feeding on deer carcasses. In one instance, a large boar ate the intestinal tract of a deer in a single feed, while it was still relatively fresh (G. Nugent, unpubl. data). In August 1999, a family of piglets 3–6 months old was observed feeding on the carcass of a female deer in the central Hauhungaroa Range, and ate most of the edible tissue before it completely putrefied (G. Nugent, per. obs.). Tb was cultured from lesions in a heavily infected deer carcass 6 weeks after it had been shot (and left in the field out of reach of pigs) in this area in August 1999 (J. Whitford, unpubl. data). Together, such observations indicate that pigs will readily eat large quantities of potentially infected carrion, often in large family groups or even groups of families feeding on the same carcass (G. Nugent, pers. obs.), so that all of the potentially infected tissue is consumed. Although there is anecdotal evidence that pigs do not begin to feed until the carcass has begun to putrefy, some Tb bacilli are likely to still be viable by the time the carcass is scavenged.

Ferrets frequently scavenge other dead animals (Ragg et al. 2000) and have been videotaped feeding extensively on pig carcasses (Yockney & Nugent 2003), so presumably would also scavenge deer carcasses. As a much smaller animal than a pig, the likelihood that a ferret would feed on the small amount of infected tissue in a whole deer carcass in any one feed will be lower than the likelihood of pigs doing so. However, for hunter-killed deer in particular, where only the head and offal is normally left in the field, that likelihood will be significantly increased. Intuitively, we believe the likelihood of ferrets ingesting Tb is lower (per carcass encountered) than for pigs but that lower rate might be partly offset by a greater susceptibility of ferrets becoming infected per bacilli ingested and by their greater propensity to transmit bacilli to other hosts.

Possums occasionally scavenge meat and carcasses (Ragg et al. 2000; Caley 1998), and they have been videotaped feeding on pig and deer remains on several occasions (Yockney & Nugent 2003). Although a few extended bouts of scavenging by possums have been recorded, most encounters with deer or pig carcasses are brief, so the likelihood of ingesting Tb bacilli per encounter will be much lower for possums than for pigs or ferrets. Nonetheless, there are some observations that suggest transmission from deer to possums. In 1997 we recorded Tb in a single large mesenteric lesion in a possum trapped near the site where infected deer carcasses were left several months previously (G. Nugent, unpubl. data). In Waipawa, Hawkes Bay, a new Tb outbreak involved transmission from farmed deer to possums (Mackereth 1993), though the mechanism is not known.

Another suggested pathway for deer-to-possum transmission is from the investigation of, and direct contact with, healthy possums by tuberculous deer, i.e. the reverse of the route by which possums are thought to infect deer (Lugton 1997). The low number of bacilli excreted by most infected deer (Lugton 1997) makes this seem unlikely.

Overall, wild deer are most likely to transmit Tb mainly to scavenging wildlife, and only rarely to other herbivores (including livestock). Of the wildlife species at risk, pigs have the greatest likelihood per carcass encounter, and possums the least, but risks will depend on the relative density and susceptibility to infection of the particular scavengers, with the higher density of possums typical of much of the forested deer habitat greatly offsetting the lower risk per possum-carcass encounter.

Persistence of Tb in deer

Tuberculosis persists in deer populations that share their habitat with infected possums, or when their densities are above the as yet unknown threshold for disease maintenance. In the absence of spillover from possums, however, persistence of infection will depend on the typical survival rate of uninfected deer, and the general pattern of disease progression (or resolution) with age. In hunted deer populations, few males survive more than 5 years and very few exceed 10 years (Nugent et al. 2001). Survival rates for females are higher, and occasionally they may survive to 15–20 years. Tb is known to have persisted in already infected deer for at least 8 years after possum control had reduced transmission from possums to near zero (as shown by the almost complete absence of new infection in deer born after possum control, compared with the continued high prevalence in those born before control; Nugent & Whitford 2003). Tb has also persisted as a self-sustaining disease in a high-density population of white-tailed deer in Michigan, USA, 18 years after it was eradicated from cattle (O'Brien et al 2002). Likewise, Tb re-emerged briefly on Molokai, Hawaii, 10 years after all cattle were removed (USAHA 2001). In light of our New Zealand results, and the comparatively short life span of pigs and the mongooses present, we speculate that axis deer (*Axis axis*) on Molokai are the species most likely to have carried the disease undetected over the 10-year gap.

Geographic spread of Tb by deer

Infected deer have the potential to carry Tb with them when they disperse, which for young males, in particular, may be up to 30 km. However, few deer disperse more than 10 km, and most <5 km (Fig. 1). All the empirical data in New Zealand on Tb spread by deer (from the Hauhungaroa Range) are consistent with such dispersal. Possum control in 1994 appeared to largely eliminate transmission from possum to deer born after 1994 in the eastern part of the area, but some cases of new infection were recorded in female deer shot within 3 km of uncontrolled possum populations, and in males up to 5 km away (Nugent & Whitford 2003; G. Nugent, unpubl. data). Other more circumstantial examples of long-distance spread of Tb by deer include:

- 1) an outbreak of Tb in cattle in northern Rangiteiki, in which the only infected wildlife found was a fawn (born after the removal of all infected cattle) (Nugent & Mackereth 1996). As the infected fawn remains the only infected wild deer of this age ever recorded in New Zealand, the result must now be viewed with some scepticism.
- 2) an outbreak of Tb in cattle at Waikaka, King Country, where one of 50 wild deer had Tb, while there was no evidence of infection in the many possums examined (G. Cochrane, pers. comm.).
- 3) the discovery of two infected deer (one male, one female) in 2001 in the Urewera area approximately 20 km from the nearest known source of infection (G. Corbett, Environment Bay of Plenty, pers. comm.).

Deer may also spread Tb via their translocation and illegal liberation by hunters intent on establishing a new or improved hunting resource. However, this is most likely to involve liberation of farmed (rather than wild-caught) deer, so the frequency with which this will involve infected deer is likely to be negligible.

Summary and information gaps: deer

Deer are currently considered density-dependent maintenance hosts of Tb, with the density threshold for disease persistence through deer-to-deer transmission alone being above the densities at which deer routinely occur in the wild in New Zealand (Lugton et al. 1998;

Nugent & Whitford 2003). Reduction of wild deer density is therefore not an essential part of Tb eradication.

As spillover hosts infected via interspecific transmission from possums (Nugent & Whitford 2003), infected wild deer constitute a medium-term reservoir of Tb that may infect other species and, very occasionally, other deer. Farmed deer have been implicated in the introduction and establishment of new foci of infection in possum and ferret populations (Mackereth 1993; Morris et al. 1994; de Lisle et al. 1995; Morris & Pfeiffer 1995). For wild deer, the risk of that occurring is highest inside VRAs where Tb prevalence in deer is highest. Although some risk exists of deer spreading Tb up to 30 km, the Hauhungaroa data show most of the risk is confined to within about 4-5 km of infected possum populations. As VRAs typically include an 'uncertainty' buffer of at least this width around all known foci of infection, possum control within VRAs means the likelihood of deer spreading Tb beyond the VRA into areas with high-density uncontrolled possum populations is low (but not zero).

Although the New Zealand data are relatively sparse, there is enough global information on deer movements, age structures, and the pathogenesis of Tb to be able to model and broadly predict the risk wild deer pose as a host in maintaining and spreading Tb. There are also enough observations to qualitatively characterise some of the routes by which deer may transmit Tb to possums.

The major information gap is the absence of robust data to quantify the risk of transmission to possums, either directly or via other species. Two major pieces of information are needed: the frequency and predictability with which deer carcasses are scavenged by possums, and the percentage of scavenging events of infected deer that result in infection in possums. Because most deer are killed by hunters, the focus needs to be on the carcass remnants of infected deer likely to be left in the field (or elsewhere) by hunters.

More information is also needed on the fate of infected deer not killed by hunters (resolution, stasis, or rapid progression as the animal reaches old age), and on the behaviour and Tb-excretion characteristics of infected wild deer in the terminal stages of illness, particularly duration of infectivity. This would help clarify how long such deer are likely to remain a threat to TB eradication.

5.2 Feral pigs as hosts and spreaders of Tb

Population density and distribution of feral pigs in New Zealand

Feral pigs are widely but patchily distributed over 35% of New Zealand (93 000 km²; Fraser et al. 2000). Their distribution has expanded in recent decades mainly due to their illegal liberation. Hunting pressure and habitat constraints usually limit pig numbers, with densities of up to 8/km² recorded in favourable habitat in heavily hunted areas and 12–43/km² in unhunted areas (McIlroy 1990). Nugent et al. (1996) suggested a national population of approximately 100 000 feral pigs, roughly 1 pig/km² for their current range.

Feral pigs have a shorter life span than wild deer, particularly in heavily hunted populations, where the mean age at harvest can be less than 1 year (Dzieciolowski & Clarke 1989).

Home range size and dispersal of feral pigs

Home range size of feral pigs varies with resource abundance, population density and body weight (Singer et al. 1981; Baber & Coblenz 1986; Saunders 1988; Caley 1993; Dexter 1999; Saunders & McLeod 1999; McIlroy 2001). Adult feral pigs in New Zealand appear

relatively sedentary when food and cover are adequate (Martin 1975; McIlroy 1989; Nugent et al. 2003), but may be more nomadic in areas with sparse cover (e.g. Knowles 1994). Mean home range sizes of pigs in five New Zealand studies in largely forested areas range from 70 up to 1170 ha (Table 2), whereas Knowles (1994) recorded much larger ranges for a few (possible nomadic) pigs in largely tussock habitat in Central Otago. Home ranges are generally, but not always, larger for boars than for sows (Table 2). For 16 feral pigs released in West Coast forest, a 6-km radius around the location of each pig at any one time encompassed about 95% of all of its previous known locations (Nugent et al. 2002).

There are few data on pig dispersal in New Zealand. In a piglet movement study at Mt White, Canterbury, no large-distance dispersal occurred, home range size ranged from 10 to 1490 ha, and the maximum distance between locations of any juvenile was 5.8 km (K. Barber, unpubl. data; Table 2). In contrast, Knowles (1994) recorded much larger distances (up to 30 km) between pig locations in central Otago. Nugent et al. (2002) recorded a shift of 35 km by a sow, apparently after it was chased by dogs, and shifts of up to 55 km have been recorded in Australia (Table 2).

Overall, pig home range sizes and dispersal patterns appear to be larger and less stable than those of deer, particularly in habitats with limited cover.

Table 2 Home range estimates (minimum convex polygon) for feral pigs in New Zealand and Australia, including study duration, average number of locations and the maximum distance between known locations for any individual pig. – = data not presented. Released-pig studies are indicated by *

Study region		Average number of relocations per pig	Home range (km ²)		Female		Maximum distance_moved (km) ♂/♀	Reference
			Male		Mean (n)	Range		
Lees Valley, North Canterbury	NZ	–	– (10)	–	– (12)	–	13.0 ♂, 0.8 ♀	Martin 1975
Mount Harte, Murchison	NZ	71	0.89 (4)	0.38–2.09	0.70 (3)	0.28–1.17	>2.4 ♂	McIlroy 1989
Pisa Range, Central Otago ^a	NZ	<12	– (6)	22.57–157.17	– (8)	5.43–23.41	29.8 ♂, 9.3 ♀	Knowles 1994
Springfield, Canterbury*	NZ	9	0.66 ^b (9)	0.11–1.63	–	–	3.9 ♂	Landcare Research unpubl.
Mokai, Western Taupo*	NZ	8	3.23 ^b (8)	0.27–12.83	–	–	4.8 ♂	Landcare Research unpubl.
Mokai, Western Taupo* ^c	NZ	8	0.79 ^b (8)	0.23–2.33	–	–	2.5 ♂	Landcare Research unpubl.
Mt White, Canterbury ^d	NZ	22	4.53 (12)	0.10–14.85	4.87 (12)	0.58–11.79	5.8 ♂, 4.9 ♀	K. Barber, unpubl.
Hochstetter Forest, Central Westland*	NZ	10	11.35 (4) 7.36 ^b (4)	4.76–20.34 3.57–12.39	11.74 (9)	3.38–26.44	35 ♀ ^e	Nugent et al. 2002
Western NSW	AUS	–	43	–	6.2	–	23	Giles 1980
Namadgi National Park, ACT	AUS	35	4.94 (25)	1.0–22.6	4.23 (17)	0.7–8.5	>5	McIlroy et al. 1989
Sunny Corner, NSW	AUS	204	10.7 (7)	4.1–21.8	4.9 (5)	3.5–6.8	>10	Saunders & Kay 1991
Kosciusko National Park, NSW	AUS	28	34.6 (12)	13.2–70.1	10.21 (8)	3.7–19.9	18	Saunders & Kay 1996
Douglas-Daly area, NT	AUS	>150	33.5 (6)	–	24.1 (6)	–	23	Caley 1997
North-west NSW	AUS	–	7.9–11.6 ^f	–	4.2–8.0 ^f	–	50 ♂	Dexter 1999

^a home range assumed to be calculated as minimum convex polygon ^d juvenile pigs (<15 kg start of radio tracking)

^b males castrated

^e individual recovered 15 months after release

^c domestic pigs

^f seasonal means

Feral pigs as hosts of Tb

Tuberculosis was first identified in a feral pig in New Zealand in 1962 (Allen 1991), and has since been reported from pigs in most areas in which Tb is endemic in wildlife (de Lisle 1994). Prevalence of Tb in feral pigs varies immensely, ranging from zero to close to 100% in adults (Appendix 1). Highest prevalences occur where infection in other wildlife is common and there are no areas where the prevalence of Tb is high in pigs but low or zero in other sympatric or nearby hosts (Nugent et al. 2003).

Of all the domestic livestock species, the pig is considered to be the most susceptible to infection with Tb (Chause 1915, cited in Francis 1958). Young pigs rapidly develop severe lesions in tonsils, head lymph nodes, lungs and abdominal organs (Griffith 1907, cited in Lugton 1997). With time, however, progressive fibrosis and calcification occurs (Lugton 1997), and lesions may even regress and resolve into fibrous tissue (Ray et al. 1972; Bollo et al. 2000). Numbers of bacilli in lesions in pigs appear generally to be low, especially compared to those in possums and ferrets (Cooke et al. 1999).

Of the large mammalian hosts, pigs appear to be best overall at coping with Tb infection, at least in adults. Consistent with that, pigs rarely show clinical signs of the disease – in one study in Hochstetter Forest, 16 released pigs that became infected within 2 months of exposure (Nugent et al. 2002) were all in good condition with no external visible signs of infection when killed up to 21 months later. The implication is that Tb-induced mortality is usually low, at least within the first year of becoming infected, and as a consequence, prevalence in pigs should increase with age. This is borne out by field data. No infection has yet been found in newborn piglets in New Zealand. In a high-prevalence area (western Hauhungaroa Range), only 20% of pigs <6 months were infected compared with 60% of pigs >6 months (Nugent et al. 2003), for example.

Transmission of Tb to and from feral pigs

Transmission to pigs: Although intraspecific transmission occurs in captivity (Ray et al. 1972), transmission between feral pigs must be rare, as there are few places in the world has Tb persisted in feral pigs in the absence of infection in other species (Nugent et al. 2003). The clearest evidence for that is the decline in prevalence in pigs following the removal of infected cattle and buffalo in the Northern Territory of Australia (McInerney et al. 1995).

Pseudo-vertical transmission has been suggested as an explanation for clustering of infection in family groups of piglets (Knowles 1994; Lugton 1997). However, a litter of piglets 1–2 months old in Hochstetter Forest were all infected with the same Tb strain, while their mother was infected with a different strain (Nugent et al. 2003), indicating that some other horizontal mechanism was involved, at least in that instance.

Pigs may also become infected via environmental contamination (Albiston & Pullar 1954, cited in Knowles 1994), but the rarity of infection in moderately susceptible and commonly sympatric species such as sheep and rabbits suggest such contamination is rare (Lugton 1997).

The high prevalences observed in some feral pig populations must therefore result from interspecific transmission, and there is general agreement that the predominance of lesions in the head and mesenteric lymph nodes in feral pigs in New Zealand results from scavenging tuberculous carrion (Nuttall 1986; de Lisle 1994). Lugton (1997) considered oral ingestion via the tonsils to be the primary mechanism by which feral pigs became infected.

All of the four main hosts of Tb in New Zealand (deer, possums ferrets, and pigs) provide potential sources of infected carrion. Possums are likely to be the greatest source of infection, as they are usually much more abundant than the other three hosts, and their carcasses are readily scavenged by pigs (Thomson & Challies 1988) even when freshly killed (G. Nugent, pers. obs.). Data on the fate of terminally ill possums (Ramsey et al. 2001) suggests that about 80% of Tb-infected possums die in places accessible to pigs, and preliminary results from a current study suggests that pigs find about three-quarters of the possum carcasses in their home ranges (G. Nugent, unpubl. data). Taken together, these data suggest that pigs will scavenge at least half the infected possums that die within a thier home range.

Deer carcasses are also readily scavenged by pigs (see Section 5.1). However, pigs appear less likely to cannibalise the carcasses of other pigs, or feed on ferret carcasses. Such cannibalism does occur, as one pig carcass left in the eastern Hauhungaroa Range in winter 2002 had been completely scavenged by pigs when it was revisited 6 weeks later (J. Whitford, pers. obs.). However, seven carcasses left in the same area in winter 2003 were untouched 1 month later. Likewise, none of 23 sets of pig remains videotaped in the South Island high country in 2003 to determine their fate were fed on by pigs (Yockney & Nugent 2003) and none of several pigs that died in the large pens in that trial were scavenged by their well-fed pen mates. In a related trial, no ferrets placed in pig habitat were scavenged by pigs in summer (A. Byrom, unpubl. data).

Transmission from pigs: Although solitary pigs have sometimes been observed associating closely with cattle, there is little evidence in the scientific literature that behavioural interaction between live pigs and other hosts is likely to be an important route of transmission (as has been suggested as a transmission pathway between terminally ill Tb possums and cattle and deer; Morris & Pfeiffer 1995).

The typically very low numbers of bacilli and the relatively low level of purulent infection seen in most older pigs (Francis 1958) suggest they are unlikely to cause significant environmental contamination. Lugton (1997) found no evidence of excretion of bacilli via urine, faeces or the nasal cavity in four pigs with widespread and especially florid lesions, but there are occasional reports of grossly infected pigs with draining abscess under the lower jaw and elsewhere (e.g. Lugton 1997). Much lower rates of infection in livestock on farms in the North Canterbury high country, compared with >50% infection rates in sympatric pigs, indicate that pig–livestock transmission rates there must be low. Again, the rarity of infection in susceptible and commonly sympatric species such as sheep and rabbits suggests transmission via contaminated pasture must be rare (Lugton 1997). However, pigs in forest frequently use hollow logs and tree trunks as resting places, and the same places are sometimes used by possums as dens (G. Nugent, pers. obs.). Contamination of such sites by pigs with draining lesions is therefore a potential mechanism for transfer to possums.

As with deer, however, the main route of transmission of Tb from infected pigs will be via carcasses of pigs that die naturally, or are killed by hunters. Ferrets are likely to be the most at-risk species, as in the fate-of-pig-carcasses trial described above, three-quarters of 13 sets of pig remains were scavenged by ferrets in summer, but possums were also recorded feeding (briefly) on pig mesenteric tissue (Yockney & Nugent, 2003). Combining data from that trial and a preceding trial monitoring the fate of deer carcasses, possums have been seen to feed on about a fifth of the c. 40 pig or deer carcasses monitored so far, and on more than a quarter of those actually encountered by possums. This indicates that although such feeding behaviour is not universal, neither is it rare.

Persistence of Tb in pigs

Pigs are not seen as maintenance hosts, despite their high susceptibility and the extremely high prevalences of Tb sometimes recorded in pig populations. Tb will therefore not persist in pig populations as a result of intraspecific transmission between live pigs. However, occasional scavenging of pigs by pigs might lead to the persistence of the disease for more than one generation. This could also occur via a pig–ferret–pig scavenging cycle. It is not known whether either of these mechanisms happens often enough to substantially extend the length of time Tb persists in pigs once other sources of infection have been removed.

That possibility aside, the persistence of Tb in pigs appears likely to be linked mainly to the longevity of infected pigs. Although pigs may survive for 10 or more years, as already noted the high turnover in New Zealand's heavily hunted populations will usually reduce mean survival times to less than 2 years. In addition the few infected pigs that survive for 5 years or more could well resolve the infection during that time (presuming all further challenges from external sources have been eliminated). Our assessment is therefore that persistence of Tb in pigs through survival of infected animals is likely to be of substantially shorter duration than in deer.

Spread of Tb by pigs

As with deer, the risks of pigs contributing to new outbreaks of Tb are greatest within a few kilometres of areas already containing infected possums. The less stable home ranges of pigs and their greater propensity to shift long distances suggest that the risk radius is greater for pigs than deer. The best evidence for that comes from the Hauhungaroa Range, where possums on the eastern side were poisoned in 1994, so the main source of infection for deer and pigs was the uncontrolled infected possum population remaining on the western side of the range. Prevalence in older pigs (>6 months old) remained high (but variable) over the next 6 years throughout the 7-km wide eastern side, whereas prevalence in younger pigs declined with increasing distance eastward (Nugent et al. 2003). The implication is that all older pigs, even those shot up to 7 km away from the western side had at some time in their lives ranged far enough to the west to encounter infected possums. In contrast, new infection in deer was mostly confined to within 3 km of the western side (Nugent & Whitford 2003).

Clear evidence of a role for pigs in Tb spread is provided by a released female pig shot with three of her offspring over 35 km from their release point in Hochstetter Forest. The sow was infected, with a high probability it had become so near the release site and taken Tb with it when it shifted (Nugent et al. 2002). Other examples of possible Tb spread are an infected pig in the Richmond Range, Nelson, 35 km outside the nearest VRA boundary, and an infected pig on Arapawa Island (M. Mitchell, pers. comm.). More recently, infected pigs have been found in the eastern Urewera Ranges more than 10 km from the nearest known source of possum Tb (G. Corbett, Environment Bay of Plenty, pers.comm.).

More circumstantially, the absence of pigs and the absence of Tb appear to be linked, implicating pigs in Tb spread. Areas with few pigs such as the Ruahine and central Tararua ranges do not appear to contain infected wildlife. Tb does not appear to have spread eastward through the Kaimanawa and Kaweka ranges (where there are few pigs, but a widespread presence of possums and high densities of deer). It has, however, spread north from Turangi through native and pine forest that does contain pigs, and some of the earliest indications of that spread were hunter reports of diseased pigs (Nugent & Proffit 1994). Against this, pigs are uncommon or absent over much of Westland where Tb is most abundant in wildlife.

Adding to the risk posed by natural dispersal, both live pigs and pig carcasses are often moved long distances by hunters. The release of potentially infected pigs in Tb-free areas is a plausible explanation for some of the Tb outbreaks well outside known infected areas. For pig carcasses, this risk appears greater than for deer, because pigs are usually transported with the head on, which is then later discarded, sometimes in areas freely accessible to possums and ferrets (Nugent et al. 2003; Yockney & Nugent 2003).

Summary and information gaps: pigs

In summary, there is no strong basis for expecting pigs alone to contribute to the maintenance of Tb within an area beyond their normal longevity under the local hunting regime. The main unknown is whether in some areas, where both ferret and pig densities are high, co-scavenging between the two species amplifies and maintains the infection longer than would otherwise be the case.

There is much stronger evidence that pigs spread Tb more widely than deer, and are capable of readily transporting Tb across possum control buffers at least 7 km wide (as appears to be the case in the eastern Hauhungaroa Range). Stronger empirical evidence is needed to verify and support this conclusion. Better information on how often pigs are translocated, both alive and dead, by hunters and the fate of those animals would also help characterise the likelihood of Tb spread by pigs, but collecting such data will be difficult.

There are now good grounds for believing that pigs provide a source of infection not only for other scavengers such as ferrets, but also, occasionally, for possums. Again the main information gap is quantitative data on how often this occurs in different habitats and with different mixes of species at different densities.

5.3 Feral ferrets as hosts and spreaders of Tb

Population density and distribution of ferrets

Ferrets prefer pasture, rough grasses and scrubland, and are relatively rare in continuous forests and wetter areas (Clapperton 2001). There is a good correlation between ferret density and the abundance of their primary prey, rabbits (Mills 1994; Ragg & Walker 1996; Norbury et al. 2002). As a result, ferret densities are highest in semi-arid rabbit-prone grasslands and braided river areas of the eastern South Island, where estimated population densities range from 2.9 to 8.2 per square kilometre (Table 3).

Life expectancy of juvenile ferrets once they emerge from their natal den is estimated at 1–2 years (Ragg 1997; Caley & Morriss 2001). Caley et al. (2002) estimated a survival probability of 25% during the first year of life, rising to 55% thereafter. Annual survival rates of between 19% and 54% have been reported from other study sites in the South Island (Norbury & Heyward 1997; Morley 2002). In the McKenzie Basin, survival of juveniles varied between 19% and 100% in the first four months after emergence from the natal den, dropping to 0–60% by the end of the first winter, depending inversely on ferret density (Byrom 2002).

Table 3 Density estimates (ferrets per km²) for different habitats in New Zealand

Study site	Density estimate	Time of year	Habitat	Technique	Reference
Palmerston, East Otago	2.9–8.2	All year	Pasture	Mark-recapture	Ragg 1997
Macraes, Central Otago	4.4	Feb–Apr	Tussock grassland	Removal trapping	Middlemiss 1995
North Canterbury	5.3–7.3	All year	Pasture	Trap catch index	Caley et al. 1998
North Canterbury	0–6.39	All year	Pasture	Mark-recapture	Morley 1999, 2002
North Canterbury	1.5–3.1	Summer/autumn	Pasture	Petersen estimate	Caley & Morriss 2001
Mackenzie Basin / Central Otago	2–5	All year	Semi-arid tussock	Minimum left alive	Moller et al. 1996
Central Otago	1.6–10.1	Summer/autumn	Semi-arid tussock	Mark-recapture	Norbury unpubl. data (AHB project R-10592)

Home ranges, dispersal and movement patterns of ferrets

Home range size varies between habitats, but is typically around 140 ha for males and 100 ha for females (Table 4). Male and female home ranges overlap, both within and between the sexes, especially in areas with high densities of ferrets resulting in high levels of social contact (Ragg 1997; Norbury et al. 1998b; Moller & Alterio 1999). In areas of low prey availability, ferrets of the same sex appear to maintain exclusive areas (Moors & Lavers 1981). Ferrets increase the size of their home ranges at times of low food availability (Norbury et al. 1998a).

Table 4 Home range sizes (ha) of ferrets in the South Island, New Zealand.

Study site	Season	Mean, range (sample size)		Reference
		Male	Female	
Mackenzie Basin	Varied	288 190–372 (5)	111 44–225 (7)	Pierce 1987
East Otago	Autumn/early winter	86 39–131 (7)	45 18–89 (10)	Ragg 1997
Otago/Mackenzie Basin	All year	102 19–316 (34)	76 16–240 (28)	Norbury et al. 1998b
Otago Peninsula	Spring/autumn	163 95–220 (6)	135 73–206 (10)	Moller & Alterio 1999
North Canterbury	Winter/spring	194 60–320 (5)	99 50–150 (10)	Spurr et al. 1997
North Canterbury	Summer/autumn	139 2–220 (4)	151 62–314 (4)	Young 1998
North Canterbury	Summer/autumn	80 (1)	128 28–265 (11)	Young 1998
North Canterbury	Summer/autumn	760 (1)	230 (5)	Caley & Morriss 2001
Central Otago	Autumn, winter and spring	94 68–120 (3)		Baker 1989
Otago Peninsula	Winter	107 (1)		Dymond 1991

Males and female ferrets are equally likely to disperse, and 50% of juvenile ferrets move more than 2–5 km from their place of birth (Caley & Morriss 2001; Byrom 2002). Most dispersal occurs during late February and early March, with no sex differences in mean dispersal distances (Caley & Morriss 2001; Byrom 2002). In North Canterbury ferrets dispersing across ‘undirectional’ pasture habitat had a mean dispersal distance of 2.1 ± 1.0 (SE) km (Caley & Morriss 2001) whereas in the Mackenzie Basin, ferret dispersal was much more directional, following the braided riverbed habitat, and resulting in a much greater mean dispersal distance (males 6.7 ± 1.6 km; females, 11.8 ± 3.4 km; Byrom 2002).

Ferrets as Tb hosts

In New Zealand, Tb was first recorded in wild-caught ferrets from Taumaranui in 1982 (de Lisle et al. 1993), and since then has been found in most areas with Tb well established in cattle or possums (Walker et al. 1993; Ragg et al. 1995b; Ragg & Walker 1996; Caley 1998). The instantaneous rate at which ferrets acquire Tb infection at eight sites in the North and South Islands sampled at various times between 1994 and 1998 varied between 0.09 and 7.9

per year (Caley & Hone 2002). Male ferrets encountered infection at 2.2 times the rate of females.

In wild infected ferrets, Tb lesions occur most commonly in the mesenteric (jejunal) lymph node (34.5%) but rarely in the lungs (2.9%; Ragg et al. 1995b). In a sample of 38 diseased ferrets with primary single lesions, 79% of the lesions were associated with the alimentary tract, with the remainder in peripheral body nodes.

Infected ferrets excrete Tb more readily than pigs or deer, most commonly via the oral cavity, with 23% of oral swabs culturing positive, and with tracheobronchial swabs, faeces, urine and mammary glands also being positive (Lugton et al. 1997b).

For ferrets, survey results are often reported from gross diagnosis only. This underestimates the true prevalence of the disease, as 27–50% of culture-positive ferrets in some intensively studied samples have shown no visible lesions (Lugton et al. 1997a; Caley et al. 1998). Reported prevalences range from 0 to 32% (Table 5).

Table 5 Surveys of Tb infections in ferrets. All surveys (except Lugton et al. 1997a) were from gross diagnosis at necropsy, with only gross or suspicious lesions submitted for culture and/or histopathology.

Area	% Prevalence (95% CI)	Diagnosis technique	Reference
Reporoa	0 (0–1.8)	Gross diagnosis at necropsy, 6 ferret samples submitted for culture	Paterson & Cowan 1994
Hohotaka	4.3 na	Gross diagnosis at necropsy then culture	Caley 1996
Waitarere	0 (0–8.1)	Gross diagnosis at necropsy, 15 ferret samples cultured	Coleman et al. 1993
Wairiro	10 (3.4–22.2)	Unknown	Atkinson & Cowan 1994
Pooled North and South Island study	32 (26–38)	Gross, histo and culture of all samples	Lugton et al. 1997a
Amuri	7.9 (1.6–17.8)		Walker et al. 1994
Tiromoana	3.8 na	Gross diagnosis at necropsy then culture	Caley 1995
Scargill	10.7 na	Gross diagnosis at necropsy then culture	Caley 1995
Hakataramea Valley	1.7 (0.5–8.7)		Walker et al. 1994

Area	% Prevalence (95% CI)	Diagnosis technique	Reference
Mackenzie Basin	15.5 (8.5–25.1)	Gross diagnosis at necropsy then culture	Walker et al. 1993
Otago and Southland	17.6 (14.9–21.5)	Gross diagnosis	Ragg 1995a

Tuberculosis is more prevalent in male ferrets than in females (Lugton et al. 1997b; Ragg 1997; Caley et al. 1998), possibly because male ferrets are more likely to find and/or scavenge possum carrion than females (Ragg 1998; Lugton et al. 1997b). The age-specific prevalence of Tb in ferrets is low or zero in newborn kits, but increases sharply in ferrets from the age of 1.75 months (the age at which they begin to fend for themselves; Caley & Hone 2002) and is typically higher in adults than in juveniles (Lugton et al. 1997b; Caley et al. 1998).

Transmission to and from ferrets

Transmission to ferrets: There has been considerable debate over the extent to which intraspecific transmission occurs in ferrets (Byrom 2001). There is a positive correlation between ferret Tb prevalence and possum abundance (Caley 1998; Caley et al. 2001), which suggests that most Tb in ferrets comes from possums. However, Caley (2002) has shown that reducing ferret, but not possum, numbers reduced the force of infection in ferrets, indicating ferret-ferret transmission. Caley (2003) postulated that ferrets would have to reach a peak density of ≥ 5.3 ferrets/km², and a year-round density of ≥ 2.9 ferrets/km², to maintain Tb independent of other wildlife sources, but these figures have not been validated and have wide confidence limits, so the host status of ferrets remains unclear.

In support of intraspecific transmission, Qureshi et al. (2000) demonstrated horizontal transmission of Tb among ferrets housed at close quarters in a pen trial, through behaviours such as den sharing, sniffing of orifices and faeces, cannibalism and aggressive breeding behaviour. Three ferrets were experimentally infected with three different strains of Tb and housed with ten non-infected ferrets. Eight of the 10 non-infected ferrets became infected, with one ferret being infected with two different strains. Also, wild ferrets in New Zealand are known to share dens (Ragg 1998; Norbury et al. 1998b).

As with pigs, the route of infection of bovine Tb in wild ferrets appears to be via the alimentary tract, most likely through scavenging or predation of Tb-infected food sources from the age of weaning (Lugton et al. 1997a, 1997b; Ragg et al. 1995b), rather than from pseudo-vertical transmission, routine social contact such as mating or fighting, or other environmental factors (Caley & Hone 2002). This could still involve ferret-ferret transmission as ferrets eat other ferrets (Ragg et al. 2000; McAuliffe 2002; A. Byrom, unpubl. data). However, the frequency of cannibalistic transmission has yet to be established.

Transmission from ferrets: Potential routes of transmission of bovine Tb from ferrets to domestic stock are via the mouth or by passing organisms into the environment with the faeces (Ragg 1995b; Ragg et al. 1995a, b; Lugton et al. 1997a, b). Because cattle and deer showed little exploratory behaviour towards ferrets in a behavioural monitoring study, it is thought unlikely that direct transmission from ferrets is a major source of Tb-infection of livestock (Sauter & Morris 1995). However, mule deer (*Odocoileus heimionus*) have been

known to kill quite large predators (e.g. coyotes; Wilkinson & Douglass, 2002) so red deer females with fawns may respond aggressively to a terminally ill ferret caught in pasture. In addition ferret control reduces the incidence of Tb in livestock Caley et al. (1998), indicating that such transmission must occur at some low frequency.

Very little is known about pathways of transmission from ferrets to wildlife hosts other than ferrets. Ferrets are often implicated in unexplained Tb outbreaks and the creation of new foci of infection, which implies that live or dead ferrets are capable of transmission of Tb to other wildlife sources. The mechanism for this is still not clear, but Ragg et al. (2000) observed one possum scavenging on a ferret carcass and McAuliffe (2002) observed hedgehogs feeding on ferret carcasses. Both Ragg et al. (2000) and more recently A.Byrom (unpublished) observed feral cats and harrier hawks scavenging on ferret carcasses.

Persistence of Tb in ferrets

Of the three largely spillover hosts considered here, ferrets appear most likely to occasionally reach densities at which intra-specific transmission contributes to Tb maintenance. Such areas aside, the generally lower prevalences in ferrets relative to pigs and their somewhat shorter life span indicate that persistence of Tb through survival of infected ferrets is unlikely to span more than 2–3 years.

Spread of Tb by ferrets

Young ferrets will scavenge and are therefore likely to begin acquiring Tb soon after emerging from the natal den (Caley & Hone 2002). However, they don't disperse until about 57 days on average after emergence (Byrom 2002). Because they do not disperse for several weeks after they emerge from the natal den, juvenile ferrets may become infected before dispersing. The likelihood of that is highlighted by observations of whole litters feeding simultaneously on pig carcasses (Yockney & Nugent, 2003). But this risk is likely to be small, especially if the amount of Tb-infected carrion is low (as in intensively managed VRAs).

Our assessment is that the greater proportion of ferrets that disperse at least 5 km, especially females, and the very long dispersal distances sometimes recorded, suggest that this species has greater potential to spread Tb than deer. However, there is less likelihood of ferrets being moved, alive or dead, by humans, so the risk of Tb spread by ferrets is likely to be lower than for pigs.

The relative risk of infected ferrets causing new Tb outbreaks is less clear. Ferret lesions are perhaps more infectious (greater numbers of Tb bacilli) but the quantity of infective material per carcass is possibly lower in pigs or deer. Whether scavenging of ferret carcasses by other wildlife Tb hosts is as common as for deer and pig carcasses is unclear.

Summary and information gaps: ferrets

Ferrets may sometimes be maintenance hosts, requiring control to reduce their densities. The areas where this applies need to be better defined, which requires field validation of the threshold density tentatively identified by Caley (2001). Regardless of how they become infected, ferrets transmit Tb to cattle at some low frequency, so ferret control is likely to reduce reactor rates (Caley et al 1998).

Where ferrets densities are naturally low, or are held low by control, however, Tb is unlikely to persist for more than a few years in ferrets through survival of already infected animals. Provided vector control reduces the transmission of Tb to ferrets to zero or near zero, and

assuming almost all dispersal is by juveniles, the risk of Tb spread by ferrets once intensive vector management has begun appears to be comparatively low. Offsetting that, ferrets are more likely to disperse, and to disperse long distances, than deer and possibly also pigs.

The frequency with which dispersing infected ferrets establish new foci of infection is not known, in either absolute terms, or relative to pigs and deer. As with pigs and deer, the main information gaps are how often infected ferret carcasses are likely to be scavenged by pigs, possums, and other ferrets, and how often such scavenging causes infection. It is also not clear whether a ferret–pig loop, not requiring possums for maintenance of Tb, exists in some dryland areas of New Zealand.

6. Modelling Persistence and Spread of Tb by Spillover Hosts

6.1 Modelling risk: wild deer

Model inputs

We estimated the age-specific decline in prevalence for deer assuming no possum–deer or deer–deer transmission of Tb, using prevalence data from the Hauhungaroa Ranges (Nugent & Whitford 2003), and calculated annual survival rates from the age structure data of those deer. We assumed no additional mortality due to Tb infection, and a stable density of deer at 6/km².

The likelihood of natural dispersal of red deer was modelled using the distribution of distances between initial and final locations of marked animals in the Nelson Lakes area (Table 1), as an approximation of the distribution of dispersal distances. Spread from an infected area was modelled assuming a large (1 million ha) circular VRA, with deer randomly distributed throughout at an arbitrary density of 6/km². Deer were assumed to disperse in a random direction for a distance randomly selected from the assumed distribution of dispersal distances. The density of deer arriving in each successive 3-km annulus beyond the outer edge of the VRA was then calculated. The edge of the VRA was (simplistically) assumed to exactly describe the outer limit of infection in deer, with all deer initially located within the VRA subject to the same historical force of infection (i.e. there was no tapering off of prevalence toward the boundary).

Results

Persistence within VRAs: A simple constant hazard model provided a reasonable fit to the age structure of the deer in the Hauhungaroa dataset ($\chi^2 = 3.93$, 6 d.f., $P = 0.69$), suggesting a finite survival rate of 0.67/year from age 1 onwards. A simple model fitted to the prevalence data for those deer (Prevalence = $(1 - e^{\lambda \cdot \text{age}})$) suggests that in the absence of possum control the force of infection (λ) was 0.178 per year. We assumed no difference between males and females. The resulting age-specific prevalence (Fig. 2) combined with the predicted survival rate produces the stable age structure and densities of Tb-infected deer in each age class shown in Table 6. This model predicts an overall prevalence of 37% if fawns are excluded, and 25% if they are included.

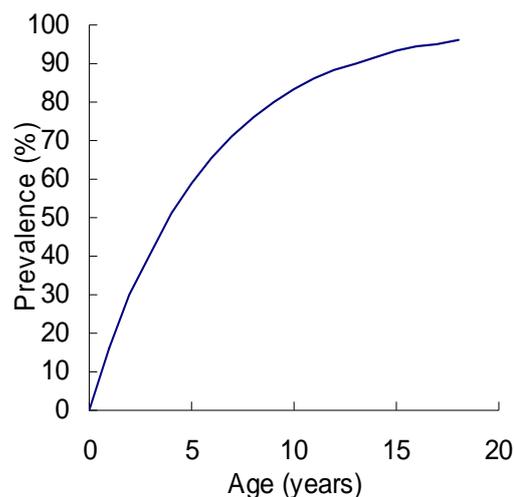


Fig. 2 Age-specific prevalence of Tb based on a constant force of infection $\lambda = 0.178$ prior to possum control.

Table 6 Assumed stable age structure of the Hauhangaroa deer population. Age 0 refers to new fawns. Density is based on an overall population density of 6/km².

Age (Years)	Proportion of population	Density of infected deer (per km ²)
0	0.33	-
1	0.22	0.036
2	0.15	0.044
3	0.10	0.041
4+	0.20	0.127

These results suggest the density of Tb-infected deer declines by 90% to 0.13/km² after just 6 years, but that Tb would not disappear completely from deer until about 15 years after transmission stopped (Fig. 3). These predictions are consistent with empirical data showing declining overall prevalence but continued persistence of Tb in older deer in the eastern Hauhangaroa and Umukarikari ranges up to 8 years after control (Nugent & Whitford 2003; G. Nugent, unpubl. data).

The key implication is that possum control alone will greatly reduce the risk posed by deer, but that low possum densities must be maintained for 15 years to avoid any risk of deer–possum transmission re-establishing infection in a recovering possum population. Reducing the deer population by applying deer control that decreased the survival rate of deer from 67% per year to 50% per year reduces the risk period to c. 6–12 years (Fig. 3b).

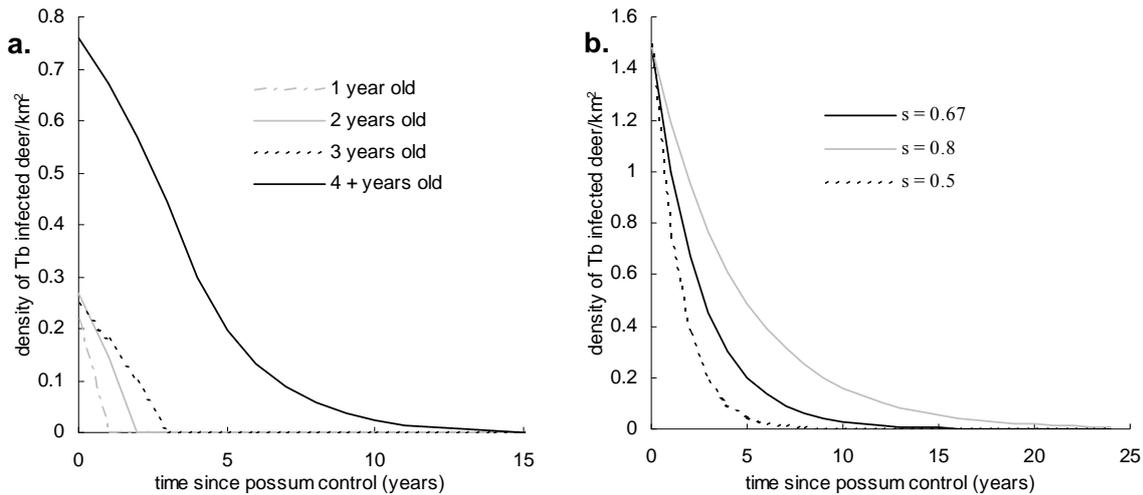


Fig. 3 Decline in density of Tb-infected deer after possum control, assuming a population density of 6 deer/km². **a.** Age-specific decline assuming finite survival (s) = 0.67/year. **b.** Total population decline for three different survival rates.

Spread from VRAs: Of 56 female red deer marked for >9 months and recovered as adults, the largest distance between initial and final capture was only 6.4 km. We therefore focused on males as likely to spread Tb the furthest (Fig. 4a). A simple exponential model was fitted to the combined data for all males ($\chi^2 = 3.7$, 5 d.f., $P = 0.59$), described by the probability density function $f(x) = 0.3e^{-0.3d}$, where d is distance between first and final location. This gives an average distance between first and final location of 3.3 km, and provided a reasonable match between observed and predicted data (Fig. 4a). The density of male deer predicted to disperse beyond the VRA over the period of one year declines exponentially to near zero at about 15 km (Fig. 4b).

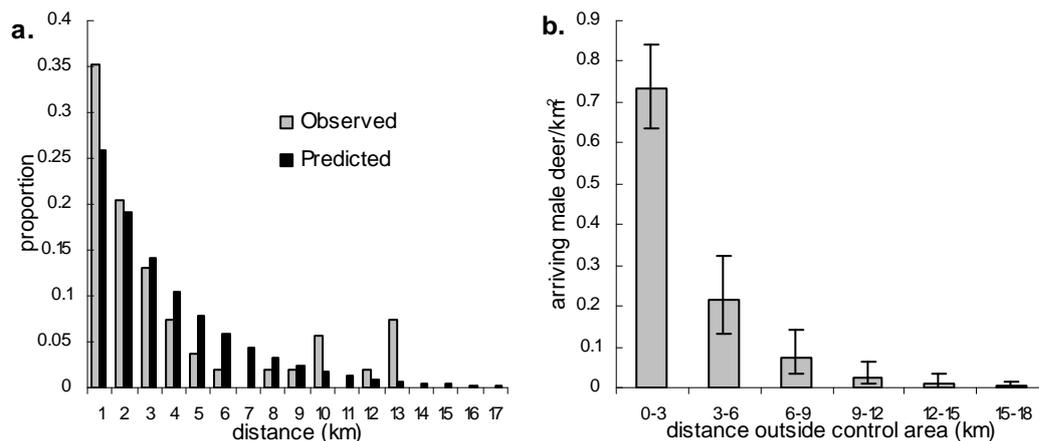


Fig. 4 a. Observed and model-predicted distance between first and last capture for male deer at Nelson Lakes. **b.** Density of male deer arriving from VRA (male density within the VRA was assumed to be 3/km²). 95% confidence interval shown.

Prior to possum control, the prevalence for all male deer (excluding new fawns) was 37%, so the density of infected deer dispersing outside the VRA is obtained by multiplying the values on the y-axis in Fig. 4b by 0.37. Looking at the strip 9–12 km outside the VRA, as an

example, and taking the upper bound of the confidence interval, the density of Tb-infected deer arriving in this area would be 0.024 deer/km², a total of approximately 30 infected deer for the entire 1200-km² annulus around a VRA of this size. Another way of expressing this result is that there would be an average of roughly one dispersing deer carrying Tb more than 10 km for every 20 km of VRA boundary each year.

What risk do these emigrants pose? Although observations of possums scavenging on deer carcasses indicate that direct transfer to possums is possible, there are no data with which to assess its likelihood. If 1% of infected emigrants caused a new outbreak of Tb, the total of >30 such emigrants carrying Tb more than 10 km outside the 10 000 km² VRA would produce one new outbreak at this distance every three years. If, however, only 0.1% of infected deer produced new outbreaks in possums, then there would be only one deer induced outbreak every 30 or so years.

Because about a quarter of the c.40 pig and deer carcasses that have been video-monitored to date have been fed on by possums (Yockney & Nugent 2003) the contact/scavenging rate is probably higher than 10%. The key unknown is therefore the percentage of occasions on which possums feed on, or contact, infected deer that actually result in Tb transmission. If this rate is also greater than 10%, then deer-induced outbreaks will occur with some regularity (i.e. at least every 2–3 years) more than 10 km beyond the area with established infection in possums, if the total VRA boundary adjacent to suitable deer habitat is about 300 km.

An important point is that dispersing infected deer may carry Tb well outside the VRA but not transmit it until it dies years later – i.e. each VRA may be surrounded by a latent infected area up to about 15 km across.

The reduction in prevalence in deer following possum control (Fig. 3b) also reduces the risk of spread by deer proportionately. As almost all long-distance deer dispersal is by young males, and because only a few males live to more than 5 years, the reduction in risk is likely to be greater still for areas furthest from the VRA. In fact, if few or no deer more than 4–5 years of age disperse, then the risk of Tb spread by deer should be largely eliminated within 5 years of the initiation of possum control.

6.2 Modelling risk: feral pigs

Model inputs

We estimated the age-specific change in prevalence for pigs by assuming no pig-to-pig transmission of Tb. Yearly survival rates for pigs were estimated by Caley (unpubl. data) using age structure data from Dzieciolowski & Clarke (1989). We again assumed no additional mortality due to Tb infection.

The density of pig populations was assumed to be 8/km² (i.e. a hunted population), because the age-structure data on which the survival estimates were based came from hunted populations.

Because local Tb prevalence in pigs varies widely, we modelled both an extremely high and a low force of infection prior to possum control, to cover two extreme situations.

Too few data were available to quantify the dispersal patterns of pigs. We therefore rely on the results for deer (see above) as being sufficiently similar to those for pigs.

Results

Persistence within VRAs: Assuming an annual birth pulse and an instantaneous rate of increase of 0, the pig population modelled would have a stable age structure (Table 7). Assuming further that piglets do not contract Tb from possums until after they are weaned, the force of infection required to produce a prevalence of 96% is about 8/yr (from a simple exponential model of prevalence = $(1 - e^{-(age-0.167)*\lambda})$). To produce an overall prevalence of 31% the force of infection required under these assumptions is approximately 0.37/yr. The predicted age-specific prevalence, and corresponding density of Tb-infected pigs, is shown in Table 7.

Table 7 Assumed stable age structure of a feral pig population, with predicted age-specific prevalence and densities of infected pigs under two different forces of infection. The density estimates assume an overall population density of 8/km².

Age (Years)	Proportion of population	Age-specific prevalence	Age-specific prevalence	Density Tb infected/km ²	Density Tb infected/km ²
		$\lambda = 8/\text{yr}$	$\lambda = 0.37/\text{yr}$	$\lambda = 8/\text{yr}$	$\lambda = 0.37/\text{yr}$
0.5	0.56	93	11	4.17	0.49
1.5	0.19	100	39	1.52	0.59
2.5	0.11	100	58	0.88	0.51
3.5	0.06	100	71	0.48	0.34
4.5+	0.09	100	80	0.72	0.58

Assuming no pig-to-pig transmission, and no possum-to-pig transmission following the commencement of possum control, there would be <1 infected pig/km² 4 years after possum control commenced regardless of the starting prevalence (Fig. 5). Tb is predicted to persist for less than 10 years under any scenario. These are worst-case scenarios with comparatively high pig densities (compared to the broad-scale national average of 1/km²), and a high prevalence of Tb in older pigs prior to possum control. Lowering pig density (by control) will reduce this persistence period.

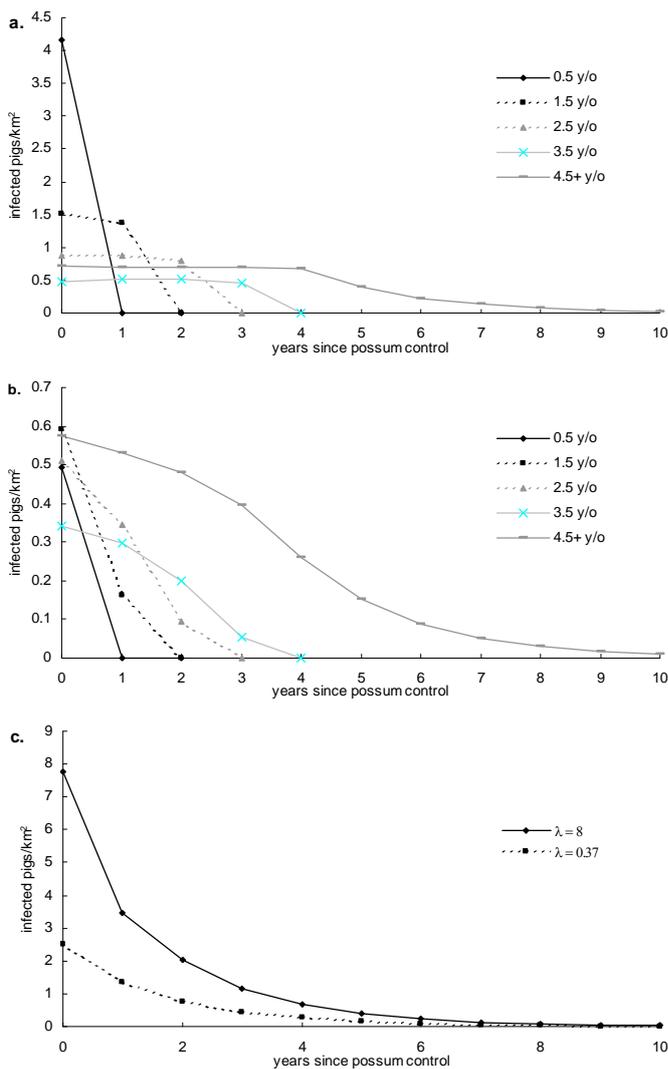


Fig. 5 Age-specific decline in density of Tb-infected pigs assuming total density of 8/km² and **a.** a force of infection prior to possum control of 8/yr, or **b.** a force of infection prior to possum control of 0.37/yr. The decline in total density of Tb-infected pigs (**c.**) is also shown.

Spread from VRAs: Although not specifically modelled, pigs are highly likely to display much the same pattern of declining density of infected animals with increasing distance from the VRA as that shown by deer (Fig. 4), on much the same spatial scale as deer in good pig habitat, but at perhaps twice the scale in poorer habitat. The slightly higher density assumed for pigs, and the near total initial prevalence would increase the density of infected pigs at each distance proportionately with respect to the predicted values for deer (i.e. would be about 2.5 times higher if the dispersal patterns were the same). If dispersal distances were greater, the greater number of infected dispersers (compared to deer) would be spread more widely, but even close to the VRAs the density of infected dispersers under the high-prevalence scenario would still be higher than for deer at the modelled densities.

As with deer, the risk of spread is predicted to decline quickly after the initiation of possum control, provided most dispersal is by younger pigs. Assuming, however, that older boars are semi-nomadic (see Knowles 1994), the risk of spread would decline to a rate closer to the rate of decline in prevalence within the VRA.

Again, the models predict that there are enough pigs carrying Tb >10 km outside VRAs to cause new outbreaks of Tb with some regularity even if only 1% of infected dispersers eventually transmit Tb to possums.

6.3 Modelling risk: ferrets

Model inputs

We estimated the age-specific change in prevalence for a population of ferrets assuming a density of 2.6/km², which is just below the proposed maintenance host threshold of 2.9 ferrets/km² (Caley 2001). Again we assumed no transmission to ferrets after the initiation of possum control. There is no reliable empirical information on the decline in prevalence in ferrets after possum (but not ferret) control because it has not been possible to determine trends in prevalence in ferrets without also significantly affecting ferret density.

Ferret populations were modelled assuming mortality rates as presented in Caley (2001):

Juvenile instantaneous mortality rate (d_j) = 1.44/year

Adult instantaneous mortality rate (d_a) = 0.56/year.

We calculated a stable age distribution in February assuming all age classes were capable of producing the same number of young (Table 8), and explored three different initial age-specific prevalence patterns, calculated from the model in Caley & Hone (2002) (Table 9). The number of infected ferrets/km² in each age class was then calculated approximately as: (proportion of the population in an age class) × (age-specific prevalence) × (population density). Additional mortality due to disease (α) was set at either zero or at 1.4/year. Caley (2001) estimated the transmission rate per ferret (β) to be 0.55, and we calculated the force of infection at the start of each year as $\lambda = \beta \times$ density of infected ferrets. The number of newly infected animals in the 1–4-year-old age classes was based on the density of susceptible animals in the previous year picking up the infection, as follows:

Density of newly infected 2 yo ($t + 1$) = density of susceptible 1 yo (t) × $(1 - e^{-\lambda}) \times d_a$, where the density of susceptible one year olds the previous year = 0.16 (age structure) × 2.6 – density of infected 1 yo.

For example, the total density of infected two year olds would be calculated thus: if the density of infected one year olds in one year was 0.0855/km², then the density of infected two year olds the following year would be $0.0855 \times (d_a + \alpha) + (0.16 \times 2.6 - 0.0855) \times (1 - e^{-\lambda}) \times d_a$.

The prevalence in the '0' year olds is based on the new year's force of infection (i.e. number of currently infected 1,2,3 and 4 year olds, rather than the previous year used for the above calculation) applied for 1 month.

Table 8 Assumed age distributions of ferrets in February.

Age class (years old)	Proportion of population
0 (newly emerged young)	0.67
1	0.16
2	0.09
3	0.05
4	0.03

Table 9 Initial age-specific prevalence patterns of ferrets with a varying force of infection.

Age class (years)	Proportion of population	Prevalence pre possum control		
		$\lambda = 1.02$ (Scargill)	$\lambda = 3.4$ (Awatere)	$\lambda = 5.77$ (Castle Point)
0	0.67	0.08	0.22	0.68
1	0.16	0.48	0.73	0.97
2	0.09	0.59	0.80	0.98
3	0.05	0.65	0.82	0.98
4	0.03	0.68	0.83	0.98

The few data available on dispersal of older ferrets suggest that although some older ferrets may move sufficient distances to take them out of VRAs, this probably only occurs when rabbit populations are drastically reduced (Norbury et al. 1998a). We therefore modelled spread of Tb by ferrets assuming that juveniles were by far the most important vehicle. We used the same-sized 10 000-km² circular area as for deer, with juvenile ferrets randomly distributed within the area at a density of 1.75/km².

Two dispersal scenarios were modelled using (1) a Scargill, North Canterbury dataset (Caley & Morriss 2001) for which juvenile dispersal can be described by the probability density function $f(x) = 0.455e^{-0.455d}$ where d is distance, and the average dispersal distance is 2.2 km; and (2) a MacKenzie Basin dataset (Byrom 2002) for which the probability density function is $f(x) = 0.106e^{-0.106d}$, and the average dispersal distance of 9.47 km.

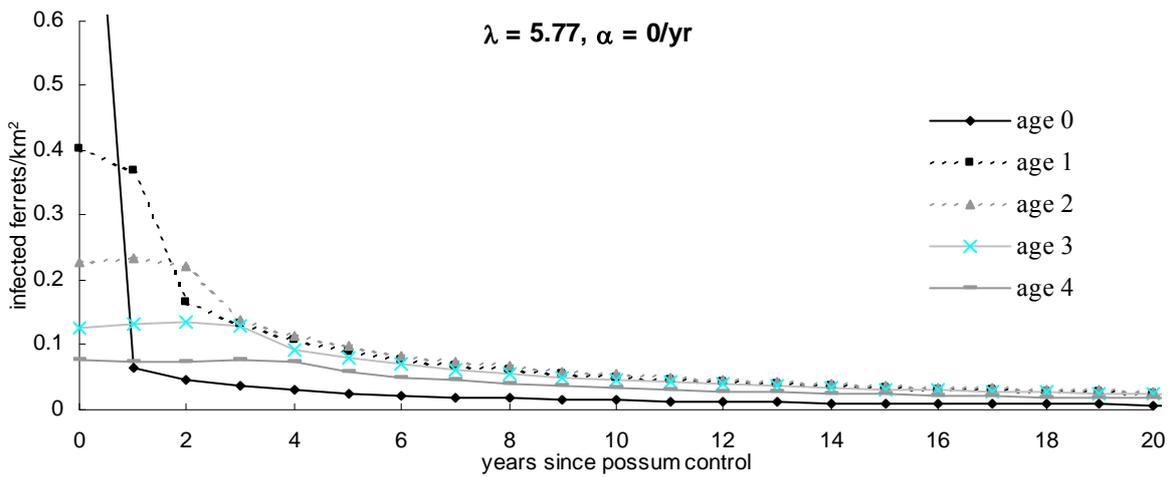
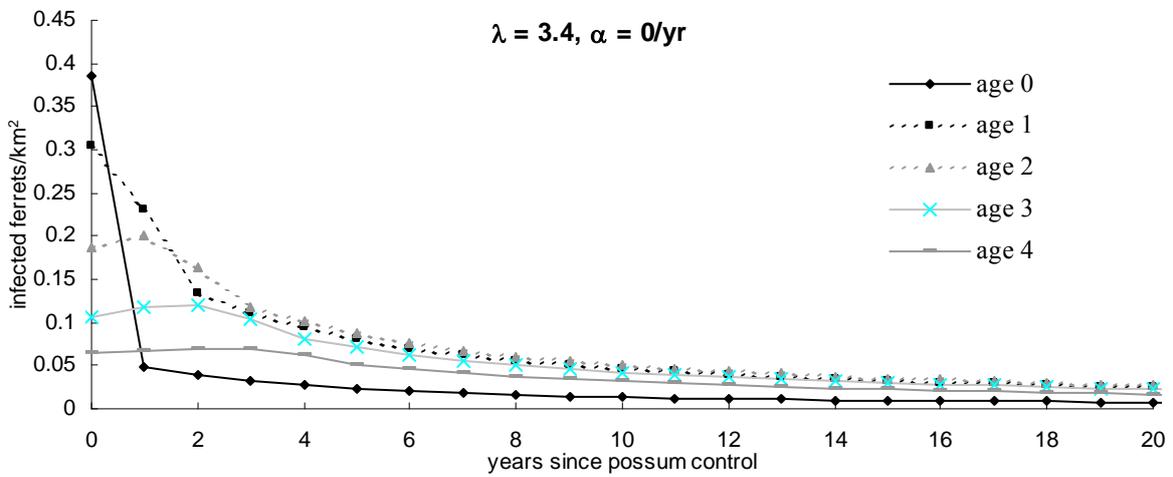
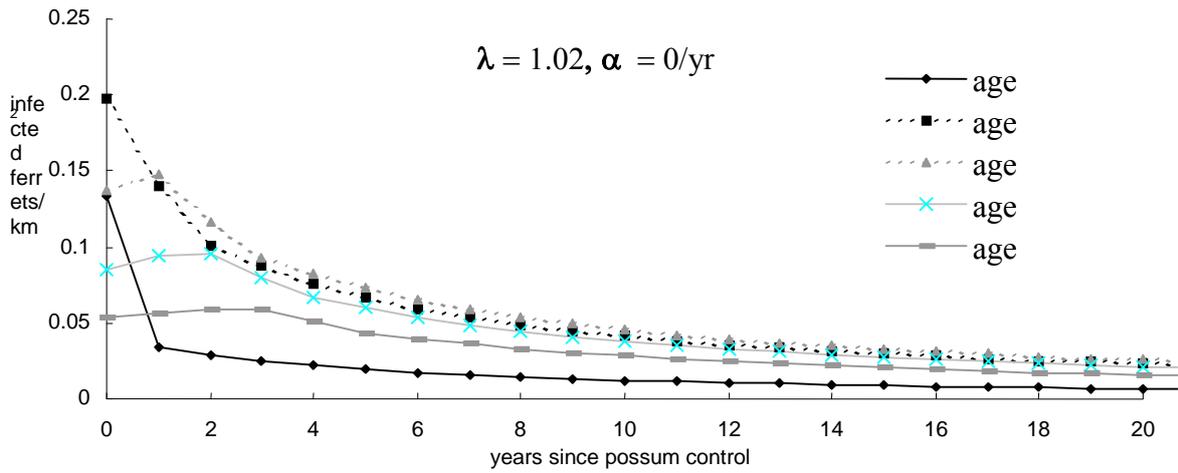
Juvenile ferrets were assumed to disperse in random directions, and to travel for a distance selected at random from the probability distributions above. This was probably reasonable for the Scargill data, but may not be reasonable for the Mackenzie Basin data where ferrets dispersed along riverbeds. The density of dispersed ferrets in strips outside the VRA was then calculated as for deer, as was the combined risk at various distances outside the VRA.

Results

Persistence within VRAs: When Tb-induced mortality is set to zero, the model predicts persistence of Tb in ferrets for at least 20 years, longer than for pigs and deer. In contrast to those species, however, the persistence is the result of inter-generational transmission, rather than purely the survival of already infected individuals. This persistence will obviously be

substantially reduced if ferret density is reduced (i.e. ferret control is imposed). However, the model also predicts that persistence of Tb will also be greatly reduced (to less than 8 years) if Tb significantly increases ferret mortality (Fig. 6 d–f). This highlights the importance of the uncertainty over whether infected ferrets suffer increased mortality due to Tb, with the best current estimate having a 95% confidence interval that includes 0 (Caley & Hone 2002).

As the modelled density is just below the maintenance threshold, these predictions are worst-case scenarios of situations not actually requiring ferret control to eliminate Tb from ferrets. Increasing ferret mortality by imposing ferret control will substantially reduce persistence times. [Conversely any continued transmission of Tb from possums to ferrets would extend persistence times](#)



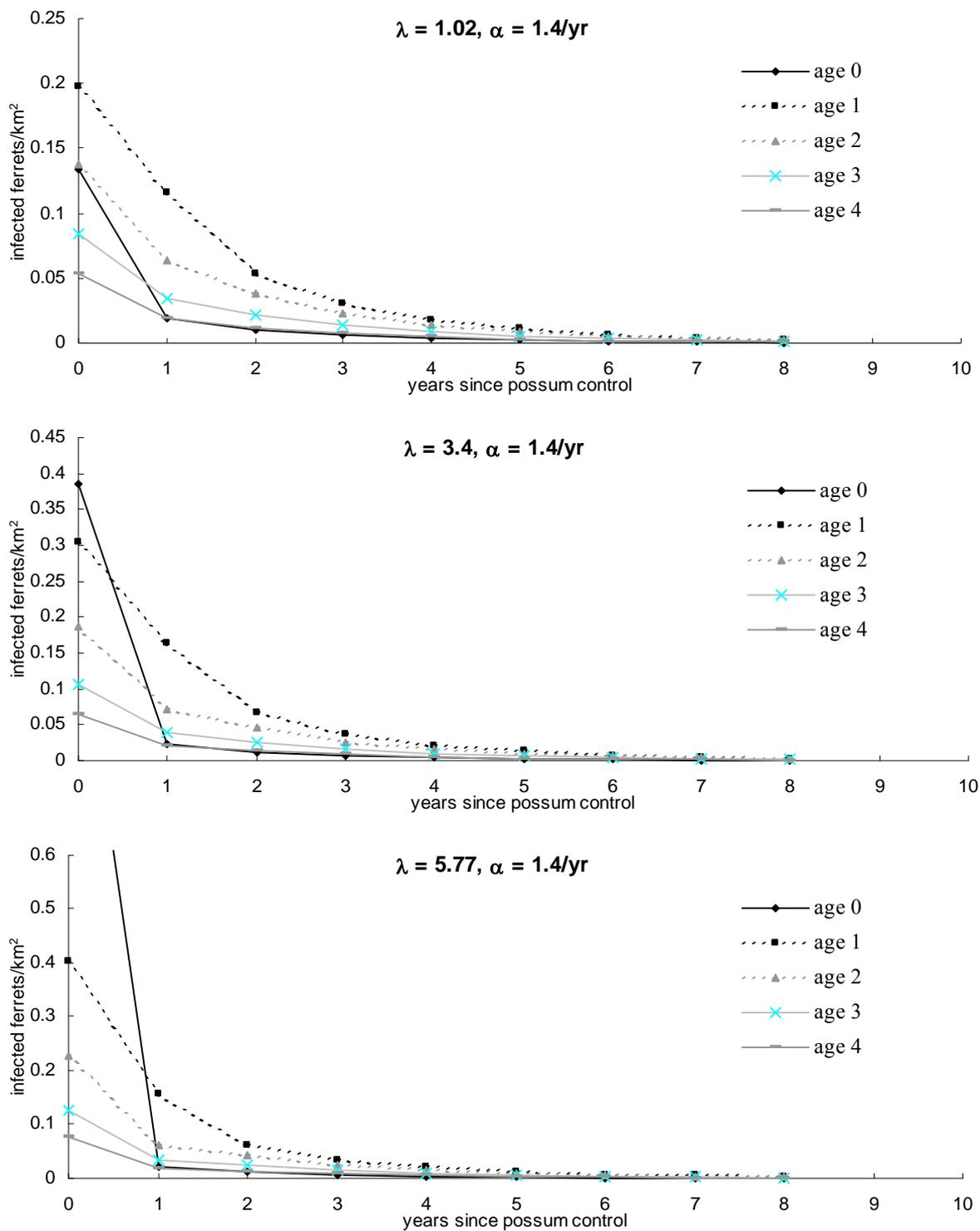


Fig. 6 Age-specific decline in the density of infected ferrets following possum control assuming no further infection from possums at different starting prevalence, and either with or without additional mortality due to disease. A total ferret density of $2.6/\text{km}^2$ was assumed. Note the different scales on both axes.

Spread of Tb by ferrets: Using the Scargill data, the model predicts few juveniles would disperse long distances. An important caveat with this dataset is that the ferrets monitored were not trapped until they had become free-ranging juveniles. This may mean the sample was biased against ferrets dispersing long distances, i.e. it would be more likely to catch ferrets remaining in the study area in the first place. In the Mackenzie Basin, ferrets dispersed much further. Those ferrets were radio-collared after being dug from the natal den and hence the dispersal profile may be less biased. Given that dispersal in the Mackenzie system generally followed riverbeds, the local density at various distances outside the control area may be higher in some places (riverbeds) than indicated in the model and lower in others, as we assumed ferrets dispersed in random directions. However, both profiles suggest that proportionately few ferrets disperse more than 10 km compared with deer (Fig. 7).

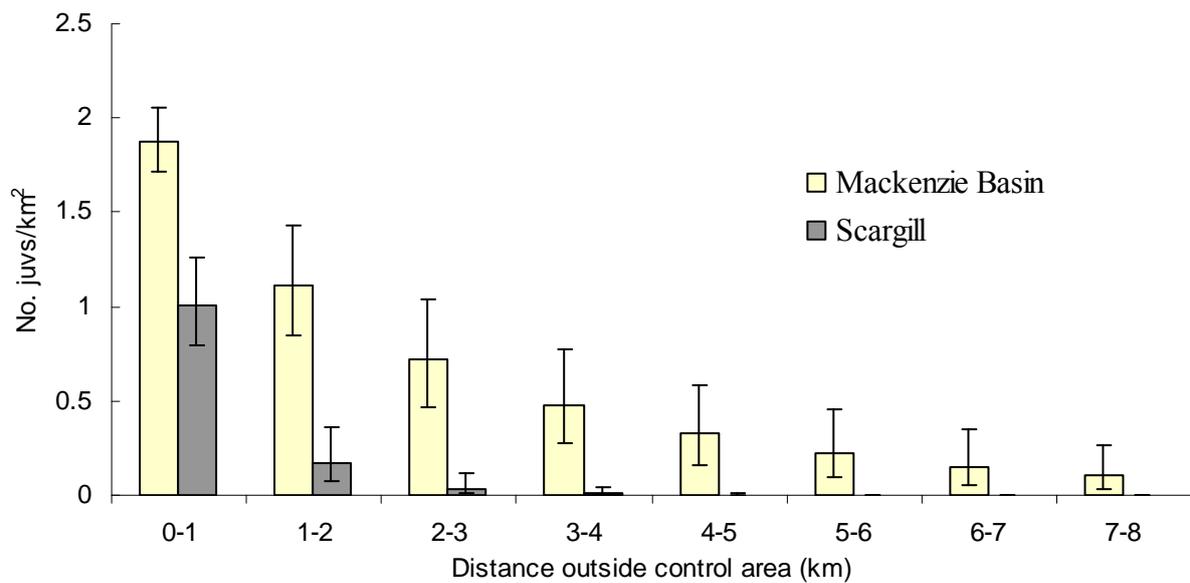


Fig. 7 Density of dispersed juvenile ferrets in successive 1-km strips outside a VRA following dispersal in February/March.

Under either dispersal scenario, the density of diseased juvenile ferrets settling outside a possum control area, prior to possum control being undertaken within the VRA, can be quite high (Fig. 8), especially where the force of infection is high. With a McKenzie Basin dispersal profile, and $\lambda = 5.77$, one infected juvenile would arrive in the 1-km strip immediately outside the VRA for every kilometre of VRA boundary. Under the Scargill dispersal model, a similar density of infected ferrets occurs immediately outside the VRA, but this drops off rapidly at distances >1 km.

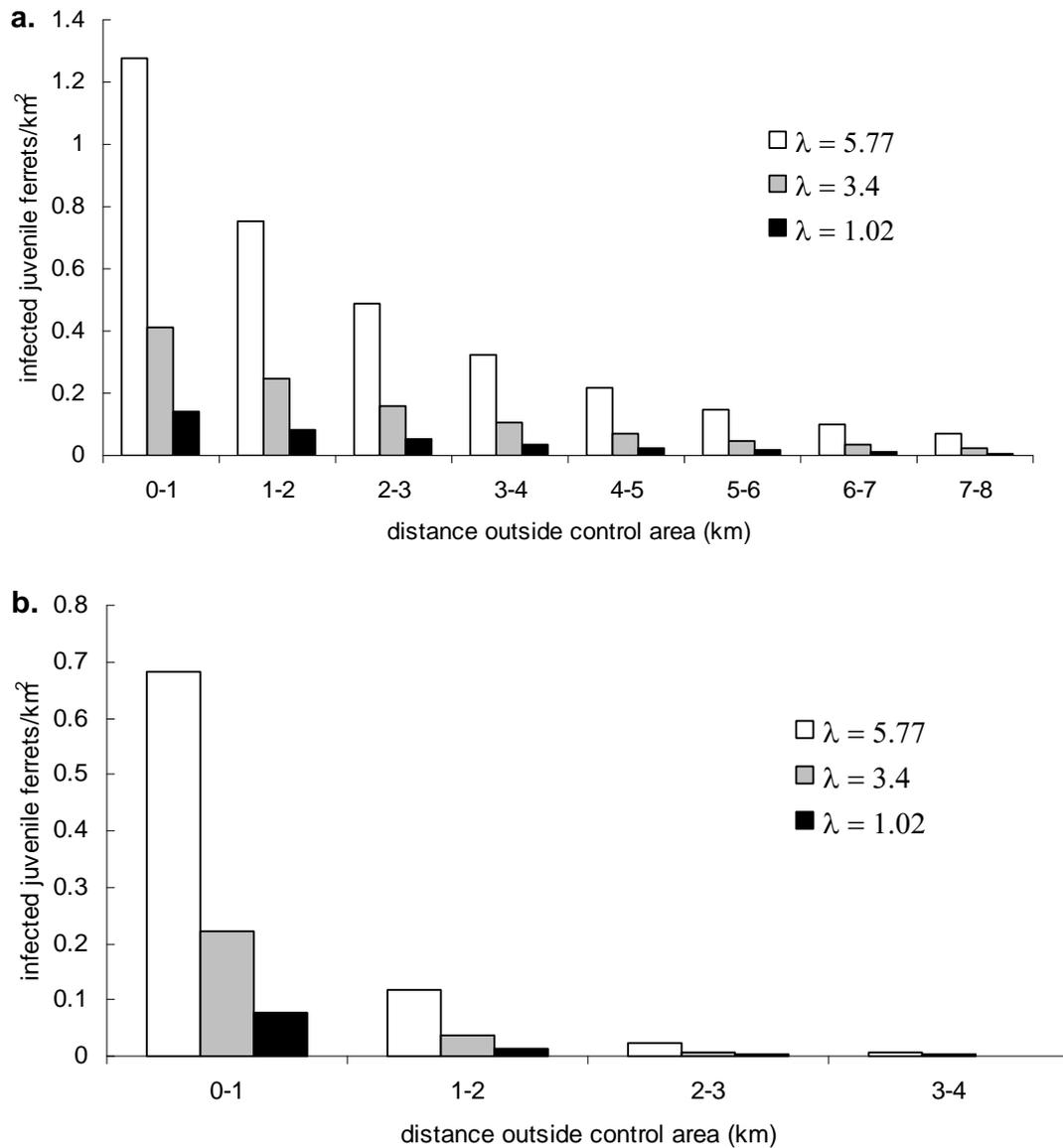


Fig. 8 Density of diseased juvenile ferrets settling outside a possum control area prior to possum control being undertaken within a control area, assuming the dispersal profile seen in **a.** Mackenzie Basin; **b.** Scargill.

Possum control quickly reduces the potential for Tb spread by ferrets. Under the worst-case scenario noted in the preceding paragraph, the density of juvenile infected ferrets predicted to arrive in the 1-km strip just outside the VRA reduces from one per 1 km of VRA boundary to one per 15 km of VRA boundary (Fig. 9).

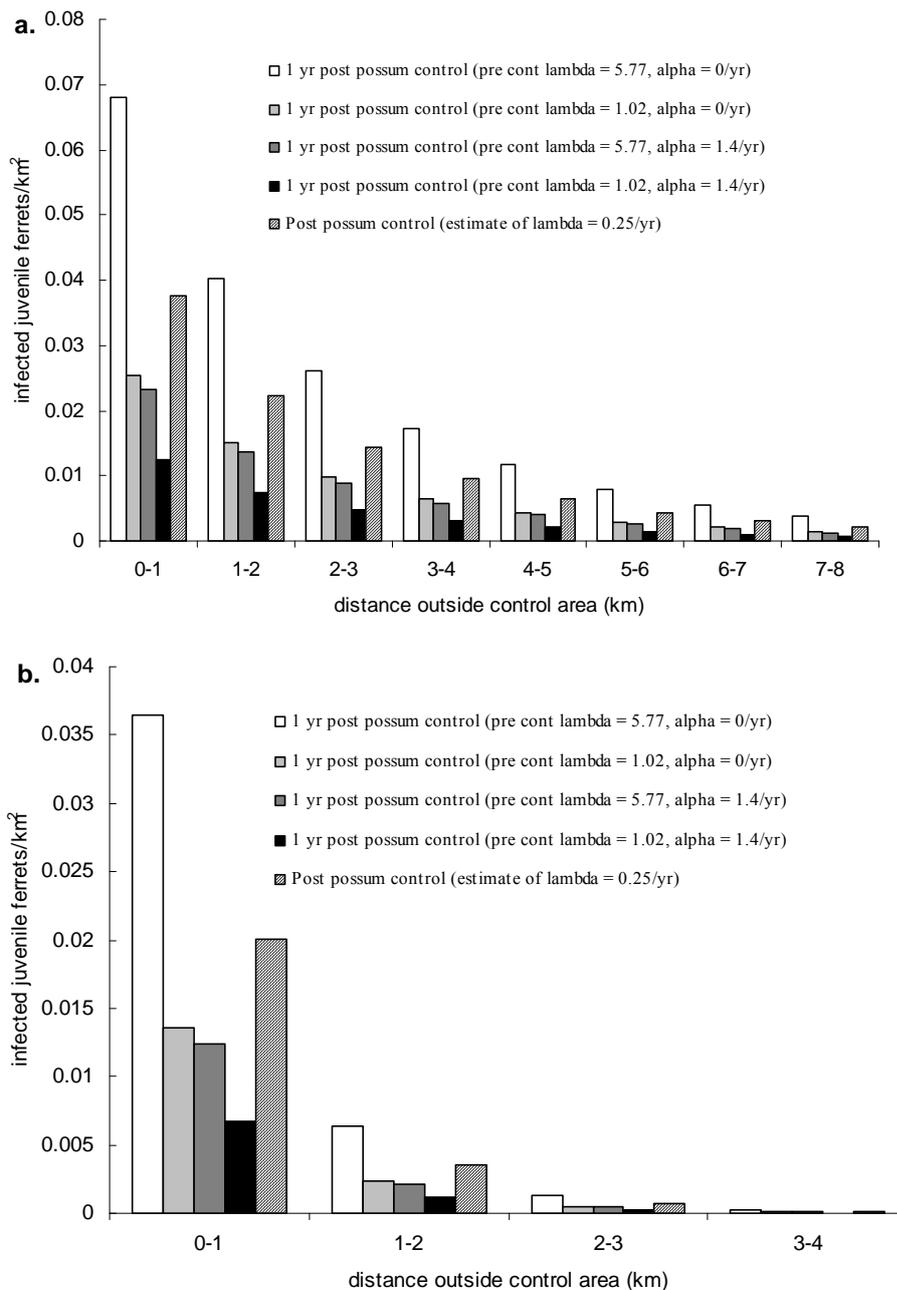


Fig. 9 Density of infected juvenile ferrets outside VRAs following dispersal. The profile is based on a situation inside the VRA where ferret density is $2.6/\text{km}^2$, i.e. just below the suggested maintenance host threshold. **a.** Dispersal profile based on Mackenzie Basin. **b.** Dispersal profile based on Scargill.

Whether these infected juveniles pose a Tb risk again depends on the frequency with which they establish new infection at the settlement site. Extrapolating from Fig. 8, before possum control, roughly one infected juvenile ferret per year is likely to carry Tb more than 10 km outside the VRA per 20 km of VRA boundary – much the same as for deer. The risk to Tb-free possum populations will therefore be much the same as for deer, unless either the ferret-carcass-scavenging rate by possums or infectivity to possums from ferrets is markedly different than for pigs or deer. Because ferrets are smaller, and therefore likely to have more

infectious tissue per carcass, the risk may well be higher for ferrets than for the other two species.

In areas where ferret density within VRAs is just below the estimated maintenance threshold, older ferrets may provide a reservoir of Tb following possum control for a number of years, particularly if there is no additional mortality due to disease. As residents, they pose little risk of moving outside VRAs unless there is a sudden reduction in rabbit numbers (Norbury et al. 1998a). One implication is that intensive rabbit control should, if possible, not be initiated at the same time as possum control is started.

7. Conclusions

7.1 Tb persistence

Risk and information gaps

All three spillover hosts are likely to contribute to the temporal persistence of Tb within VRAs (using the term to mean the area with established infection in possums). Deer, by virtue of the medium-term survival of a few females in particular, and because the level of Tb-induced mortality is low, pose the greatest threat in this regard, possibly carrying the disease for 10–15 years. That risk would be reduced if infected deer die earlier, or if some of them completely resolve the disease. Improvement of the predicted persistence times therefore requires better information on these two epidemiological parameters.

The risk posed by pigs is of lesser duration than deer, mainly because of their shorter life span, especially in hunted populations. We consider it even more likely that the risk of persistence in pigs may be attenuated by disease resolution in older animals, once the cycle of continual reinfection from possums is broken.

Tuberculosis may also persist in ferret populations for more than a decade, but only if there are significant levels of intraspecific transmission, and no impact of Tb on ferret mortality. If either of these is not true, then Tb is unlikely to persist in ferret populations for more than 7–8 years (Fig. 6). Proving continued ferret–ferret transmission after possum density (but not ferret density) is reduced is likely to be expensive, so determining the effect of Tb on ferret mortality appears to be the simplest way of determining whether persistence in ferrets is a significant risk. Because we modelled a worst-case scenario, and because some level of ferret control is likely to be imposed in most if not all areas with ferret densities close to the Caley threshold estimate (if only for surveillance purposes or to reduce the risk of ferret–cattle transmission), we consider that persistence in ferrets is unlikely to be a major problem.

Management implications

For all three species, the risk of persistence is much reduced if their densities are also reduced at the time possum control is carried out. This is particularly so for ferrets where ferret–ferret transmission is the driver of Tb persistence. The risk will be reduced further still if low densities of spillover hosts are maintained after initial control, particularly for ferrets.

Obviously, controlling four host species (i.e. possums plus the three spillover hosts) will be more expensive than controlling just possums, except perhaps where aerial 1080 poisoning is

used. Even with 1080 poisoning, deliberate targeting of pigs and deer as part of vector control will be problematic because of hunter opposition. Although control of a spillover host would speed the eradication of Tb, it is only necessary if persistence in the particular species lengthens the time over which possum control is required. At present few if any large long-infected areas appear likely to be declared Tb-free within 10 years of the initiation of vector control. In addition, the very low possum densities now routinely achieved and maintained as part of vector control mean it will also take several years after the cessation of control for possum numbers to rebuild and become capable of again sustaining infection. It therefore seems likely that only deer and high-density ferret populations are likely to require extended possum control for longer than the minimum required to clear Tb from possums alone.

7.2 Tb spread

Risk and information gaps

All three species are also likely to carry Tb well outside VRAs through natural dispersal. For pigs in particular that risk is greatly amplified by the transport of live pigs by hunters and their subsequent release in Tb-free areas, and by the transport of whole pig carcasses from hunting grounds inside VRAs to distant Tb-free areas. The latter risk is smaller for deer because far fewer released wild deer are likely to have been captured alive, and because hunters far less frequently carry the heads of deer (the most frequently infected part of deer; Lugton et al. 1998) away from the kill site.

Because the densities of each host, their dispersal profiles, and the overall Tb prevalences assumed for each species in our models were not greatly different (i.e., were of the same order of magnitude), the numbers of infected dispersers of each species were broadly similar – some tens of infected individuals each year carrying Tb more than 10 km from the one-million-hectare VRA modelled. The biggest information gap was the lack of a robust dispersal profile for feral pigs. That gap is unfortunate, as there is qualitative data (particularly Knowles 1994, and the more widespread distribution of infected pigs than infected deer in the eastern Hauhungaroa (Nugent & Whitford 2003)) indicating that feral pigs may carry Tb further than deer and (from Section 6.3) ferrets.

The risk posed by infected dispersers is unclear. There are now sufficient published (Ragg et al. 2000) and unpublished data to indicate that the percentage of infected carcasses encountered, contacted, and sometimes scavenged by possums is likely to be at least 10–20%. What remains completely unknown is what proportion of such interactions results in transmission of Tb, whether that proportion differs depending on the species and the type of remains, and what proportion of spillover-infected possums then transmit Tb to other possums and cause a new outbreak. However, we again note that even if only 10% of such interactions by possums with deer, pig, or ferret remains result in possum infection, this is likely to translate into one new outbreak >10 km from the VRA boundary being caused by each species every 3 years or so for a one-million-hectare VRA (if it were entirely surrounded by pig, deer, and ferret habitat).

Adding to the risk of direct transmission to possum through scavenging is the possibility of transmission to ferrets, either directly or through pigs, and then from ferrets to possums. Obviously this risk is highest wherever high-density ferret populations adjoin VRAs, partly because the likelihood of a carcass being scavenged by ferrets is higher, and partly because ferret–ferret transmission may then amplify the disease. The complexities of the potential pathways to new outbreaks under these circumstances are beyond the scope of this review, if

only because of the paucity of data on the routes and frequencies of interspecific and intraspecific transmission of Tb.

A key component of the risk of Tb spread that is not covered by this review is the risk of spread by possums. The importance of that risk relative to that posed by the three spillover hosts covered here needs to be determined to help assess management priorities. Likewise, an indication of the density at which a recovering possum population (following cessation of control) is capable of again sustaining Tb is required.

Management implications

There are two practical options for reducing the risk of Tb spread by spillover hosts: reduction of spillover host density within the VRA and/or reduction of the possum density outside the VRA. The most conservative solution is to implement both.

One corollary of the dispersal pattern depicted above for ferrets and deer is that most of the dispersers that pose the greatest threat of long-distance spread are likely to leave from within 10 km of the VRA boundary. Reducing spillover-host density within the outermost 10 km of the VRAs when possum control is first initiated would reduce the risk of Tb spread in proportion to the reduction in spillover host density.

The width of possum-control buffers needed around VRAs is unclear, because the risk cannot be quantified in the absence of data of interspecific transmission rates. The no-risk approach is to define buffers purely on the basis of the movement of deer, pigs, and ferrets from within VRAs (Fig. 4b); i.e. set a VRA buffer width of about 15 km. In practice that is likely to be close to what is currently done anyway, at least along waterways, because the actual distribution of Tb in wildlife is rarely known with precision, and managers must err on the side of caution. Provided the possum control in this buffer immediately beyond the infected possum population is effective, there seems little need for deer or pig control in this zone, as they are unlikely to be infected or to transmit Tb to other pigs or deer that then also disperse a long distance in the same direction. However, if intensive Tb-surveillance of pigs (and to lesser degree, of deer) was implemented in this zone (to more accurately define the geographic limits of infection), that would reduce this small risk even further. Ferret control within the buffer, however, seems advisable partly because there is evidence that the resulting low density may induce dispersing ferrets to settle there (Byrom 2002), and partly because ferret–ferret transmission may amplify Tb.

8. Recommendations

8.1 Management

Wild deer

We largely reaffirm Nugent & Whitford's (2003) recommendation that the AHB should not routinely include deer control as part of ongoing vector control operations within VRAs, except perhaps where vector control is being initiated in areas with high prevalences of Tb wildlife and high densities of deer. Further, deer control should be considered where Tb is still prevalent in deer and there is an expectation that vector managers are likely to want to cease possum control within 10 years. We also suggest that deer control be a particular

priority if deer densities are high, and Tb prevalence in deer is also moderate or high in the outermost 10-km-wide zone of a VRA. A control zone of 5 km is likely to be sufficient for fallow deer. Provided that the VRA already includes a possum control buffer of 10–15 km, we consider deer control outside the infected area is not warranted.

Feral pigs

Because prevention of Tb spread in wildlife is central to the goals of the National Pest Management Strategy, we consider that the AHB should, as a precautionary measure, increase efforts to prevent the spread of Tb by pigs, at least until it is confirmed that actual transmission of Tb to possums is far less frequent than the moderate frequency with which it appears possums scavenge pigs guts. As recommended by Nugent et al. (2003), this should include both educating hunters about the risk posed by transporting pig carcasses (and live pigs for release) to Tb-free areas, and a programme of intensified surveillance doubling also as partial pig control in areas just outside the known areas of infection.

The surveillance programme should be most intense within 15–20 km of the known ‘infection boundary’ (as defined by the outermost cases of ‘established’ infection in livestock, possums, or female wild deer, but to some degree excluding isolated outlying occurrences in wild ferrets, male deer, and pigs). For areas 20–30 km beyond that that also have few livestock available for Tb-testing, less intensive surveillance based on wild pigs (or ferrets) should be used to enhance the prospects of early detection of the occasional long-distance spread of Tb.

Within VRAs, there generally seems to be little need for pig control to reduce Tb-persistence. One exception may be in areas where pig and ferret densities are both high, where pig control could help speed the reduction in reactor rates by removing some of the reservoir of infection in the area.

Ferrets

Nothing in our review or modelling suggests that the AHB should re-evaluate the current level of emphasis on ferret control within VRAs. Where ferret densities are high enough to sustain Tb, then obviously ferret control is essential. At densities below that, complementing possum control with ferret control should help to reduce reactor rates more quickly (particularly if combined with pig control where necessary).

With respect to spread of Tb, there are two issues with ferrets. First is the likelihood of spread by ferrets, and as with pigs we suggest that a surveillance/moderate control buffer of 15–20 km be maintained around the boundary of established infection, with lower-intensity surveillance extending well beyond that where herd-testing coverage of ferret habitat is light. The second issue is the likelihood of ferret–ferret transmission in the settlement areas of natural dispersers (whether they be pig, possum, deer, or ferret) – the AHB needs to intensify the suggested surveillance/control programme in areas where local ferret densities are high enough for Tb to spread within the ferret population.

Overall

Because the distribution of Tb in the maintenance host populations is seldom known with any precision, the current c. 5km buffer widths already incorporated in VRAs are likely to be prevent most, but not all, nominally infected deer, ferrets, and pigs from settling outside a VRA. Consideration should be given to increasing this ‘incorporated-buffer’ width to at least 10 km. The aim of the surveillance recommended above is to improve the reliability and timeliness with which buffers are defined.

8.2 Research

The priorities (in no particular order) for research are to:

- Determine how often possums acquire Tb by their scavenging or coming in contact with infected possum, deer, pig, and ferret carcasses.
- Characterise the dispersal profile for pigs in continuous native forest
- Identify, via social science, how to prevent hunters potentially spreading Tb.
- Determine the effect of Tb infection on ferret survival, and, also, validate the Caley (2001) threshold
- Determine the likelihood of Tb re-establishing in a recovering possum population after cessation of possum control
- Model the role of possums in spreading Tb

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11. Appendix

Appendix 1. Summary of recent operational surveys conducted mostly for surveillance purposes or to define the outer geographic limits of Tb infection in feral pigs. Source: Nugent et al. (2003).

Survey	VRA	Prevalence (%) (Sample size)	Possums (P) & Ferrets (F) Populations controlled	Densities	Infection in possums or ferrets	Source
Ure/Medway, Marlborough	Yes	3.7 (82)	Yes	Low P & F	Yes	Marlborough Regional Council
Richmond/Rai, Marlborough	Yes	0 (186)	No	Low P	?	Marlborough Regional Council
Upper Waihopai, Marlborough	Yes	14.3 (7)	No	Low P & F	No	Marlborough Regional Council
Acheron Catchment, Marlborough High Country		85.2 (61)	No		Yes	Marlborough Regional Council
GoldenDowns, Tasman		1.6 (315)		Moderate/high P	Yes?	Southern Pest Management
Hokonui Hills, Southland	Yes	0 (7)	Yes	Low P	Yes	Southland Regional Council
Taringatura, Southland	Yes	0 (21)	Yes	Low P	No	Southland Regional Council
Tomogalak, Southland		0 (59)	Yes	Low P	Yes	Southland Regional Council
Whirinahi-Waipunga, Southern Ureweras	No	25 (8)	No		No	Bay of Plenty Regional Council
Maungataniwha, Hawke's Bay	No	0 (130)	No	High P	No	Hawkes Bay Regional Council
Hawke's Bay Region	No	0 (134)			No	Hawkes Bay Regional Council

Survey	VRA	Prevalence (%) (Sample size)	Possums (P) & Ferrets (F)		Infection in possums or ferrets	Source
			Populations controlled	Densities		
Weber, Manawatu/Wanganui	Yes	2.1 (47)	No	High P	No	Horizons, Manawatu/Wanganui
Taunoka (1998), Manawatu/Wanganui		4.8 (21)	No	High P	Unknown	AgriQuality , Wanganui
Taunoka, Manawatu/Wanganui		3.2 (31)	No	High P	Unknown	AgriQuality, Wanganui
Waitotara, Manawatu/Wanganui		0 (42)	?			AgriQuality, Wanganui
Mangaporau, Manawatu/Wanganui		0 (46)	Yes	Moderate P	Unknown	AgriQuality, Wanganui
Taranaki Region		0.3 (288)	?		No	Taranaki District Council