

## Review Article

## Feral ferrets (*Mustela furo*) as hosts and sentinels of tuberculosis in New Zealand

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### Abstract

The control and eventual eradication of bovine tuberculosis (TB) poses major challenges in New Zealand, given the variety of wildlife species susceptible to TB, many of which are capable of onwards transmission of *Mycobacterium bovis* infection. Here we discuss the role of feral ferrets (*Mustela furo*), focussing on potential transmission or risk pathways that have implications for management of TB. Firstly inter-specific transmission to ferrets. Ferrets scavenge potentially infected wildlife, including other ferrets, thus prevalence of TB can be amplified through ferrets feeding on tuberculous carcasses, particularly brushtail possums (*Trichosurus vulpecula*). Secondly intra-specific transmission between ferrets. The rate of ferret-ferret transmission depends on population density, and in some places ferret densities exceed the estimated threshold for disease persistence. TB can therefore potentially be maintained independently of other sources of infection. Thirdly transmission from ferrets to other wildlife. These include the main wildlife maintenance host, brushtail possums, that will occasionally scavenge potentially tuberculous ferret carcasses. Fourthly transmission from ferrets to livestock. This is considered to occur occasionally, but the actual rate of transmission has never been measured. Fifthly geographical spread. *M. bovis*-infected ferrets can travel large distances and cause new outbreaks of TB at locations previously free of TB, which may have caused an expansion of TB-endemic areas.

Ferrets play a complex role in the TB cycle in New Zealand; they are capable of contracting, amplifying and transmitting *M. bovis* infection, sometimes resulting in ferret populations with a high prevalence of TB. However, ferret population densities are usually too low to sustain infection independently, and transmission to other wildlife or livestock appears a rarer event than with

possums. Nevertheless, management of ferrets remains a key part of the National Pest Management Strategy for TB. Control is prudent where *M. bovis*-infected ferret populations exist in high numbers, to reduce the onward transmission risk of any self-sustained infection to livestock. When ferret numbers are well below the theoretical disease maintenance threshold, ferret control is still sometimes warranted because of the animals' ability to acquire infection when young and, through dispersal, transport it outside TB-endemic areas. Ferrets can also be used as disease sentinels for TB, especially in areas where alternative sentinel species are rare or expensive to survey, and when sampling of possums is not cost-effective.

**KEY WORDS:** *Disease surveillance, disease sentinel, ferret, Mustela furo, tuberculosis, Mycobacterium bovis, New Zealand, pathogen, wildlife*

### Introduction

Estimating the extent of transmission of *Mycobacterium bovis* within and between wildlife hosts is a prerequisite for effective control of bovine tuberculosis (TB) in livestock (de Lisle *et al.* 2005a; Gavier-Widen *et al.* 2009; Palmer *et al.* 2012). In New Zealand, concerns centre primarily on four species, all of which are introduced: brushtail possums (*Trichosurus vulpecula*), wild deer (mainly *Cervus elaphus*), wild (originally feral) pigs (*Sus scrofa*) and feral ferrets (*Mustela furo*) (Nugent 2011). All four species are frequently infected and play varying roles in both the persistence and spread of disease at a landscape scale (Nugent 2011; Anderson *et al.* 2013; Yockney *et al.* 2013), thus complicating efforts to control and ultimately eradicate TB from agricultural systems. Understanding the routes and frequencies of *M. bovis* transmission among these species is crucial in designing efficient and effective programmes for disease management (Renwick *et al.* 2007; Yockney *et al.* 2013).

In this paper we first review historical data and research on the role of ferrets as a host and potential vector of TB in New Zealand. This includes a synthesis of relevant information about the ecology of ferrets relevant to TB management, and a summary of what is known or suspected about the various pathways for intra- and inter-specific transmission and geographic spread of TB involving ferrets, placing particular emphasis on ferrets as

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NVL	No visible (TB) lesions
TB	Tuberculosis due to <i>Mycobacterium bovis</i>
TCI	Trap-catch index
VRA	Vector risk area

part of a multi-host complex. We evaluate the importance of these pathways in terms of the main risks or impacts posed by ferrets in relation to the management needs for TB control in New Zealand. Finally we evaluate the potential use of ferrets as sentinels for disease surveillance to support TB management. Where considered necessary to address the aims of this paper, we present previously unpublished research contracted by New Zealand's TB management agency (TBfree New Zealand).

## The epidemiology of TB and its relationship to ferret ecology

### History and prevalence of TB in ferrets

Tuberculosis in feral ferrets was first recorded in Taumaranui, in the Manawatu-Wanganui region of New Zealand, in 1982 (de Lisle *et al.* 1993). In the late 1980s, TB was recorded in ferrets from the Marlborough region (de Lisle *et al.* 1993) and in 1990 in the Mackenzie Basin in the South Island (Walker *et al.* 1993). By 1993, it was considered by New Zealand's Chief Veterinary Officer that there was increasing epidemiological evidence that, in rabbit-prone areas, ferrets were acting as vectors of TB for cattle and farmed deer, and they were implicated in the rapid expansion of the TB-affected area in North Canterbury (O'Hara 1993). Subsequent observations indicated that wherever ferrets overlapped with tuberculous cattle and possums, the disease was also present in the ferrets (Ragg *et al.* 1995a; Ragg and Walker 1996; Caley 1998). It soon became apparent there was a strong relationship between disease prevalence in ferrets and the density of tuberculous possum populations (Caley *et al.* 2001). Beginning in the early 1990s, research into the basic population ecology, behaviour and disease status of ferrets began to elucidate their role in TB transmission (Ragg 1997; Morley 1999; Byrom 2001). Early reviews of epidemiology of TB in wildlife in New Zealand provided some insight into the nature of the disease not only in possums but in other wildlife hosts including ferrets. (Morris *et al.* 1994; Morris and Pfeiffer 1995)

Historically, the prevalence of macroscopic TB lesions in ferrets ranged from 0 to 66% across several sites in New Zealand (Caley 1998; Caley *et al.* 2001; Caley and Hone 2005). However, before instigation of intensive lethal control of possums (and ferrets) for TB management, gross lesion prevalences between 15% and 20% appear to have been the norm for ferret populations in TB-endemic regions of the South Island (Ragg *et al.* 1995a; Byrom 2004; Byrom *et al.* 2008). As with most wildlife cross-sectional surveys based on visual necropsy data alone, gross diagnosis generally underestimates the true prevalence of *M. bovis* infection in ferrets, and estimates of prevalence based on bacterial culture of key tissues from necropsied ferrets tend to be substantially higher than those based on visual diagnoses alone. In samples where both culture and lesion status have been reported, 27–90% of *M. bovis* culture-positive ferrets have been cited as showing no visible lesions (NVL) (Lugton *et al.* 1997a; Caley *et al.* 1998; Byrom *et al.* 2008). High proportions of culture-positive ferrets with NVL may reflect cursory necropsies being undertaken in studies where all samples were being submitted for bacteriological culture regardless of lesion status. In general, feral ferret surveys reported in New Zealand over the last decade have often recorded a high proportion of individuals with NVL among culture positive cases (de Lisle *et al.* 2005a, b, 2008). By contrast to the New Zealand situation, there are few data available for the progenitor wild species in Europe; the polecat (*Mustela putorius*). Just a

single culture-positive TB case with NVL was recorded among 24 polecats surveyed from TB-endemic regions of Great Britain by Delahay *et al.* (2007).

In New Zealand, possum control (even without ferret control) has been reported to reduce TB infection among sympatric ferret populations: Caley *et al.* (2001) recorded an 80% reduction in *M. bovis* infection among ferrets in the years immediately following localised possum control alone (compiled results from several study sites). In line with that, the imposition of large-scale possum control nationally since the late 1990s (either with or without concurrent effort to control ferrets) has seen major reductions in *M. bovis* infection in ferrets overall, from the 15% to 20% noted above as the norm in TB-endemic areas in the early 1990s, to <1% now (33 infected ferrets from 3,782 surveyed in 2010/11; Anonymous 2011a). As an example from one of the areas in which TB in ferrets has been most studied, the Mackenzie Basin in the central South Island, prevalence of TB in ferrets has fallen to near zero over the last two decades since active management of TB in wildlife was instigated. The Mackenzie Basin comprises semi-arid grassland and tussock habitats with, in the 1990s, a high density of rabbits (up to 1,900/km<sup>2</sup>; Moller *et al.* 1997) consequently supporting a high density of ferrets (up to 5/km<sup>2</sup>; Moller *et al.* 1996). At that time, prevalence of TB among ferrets was high, with more than 15% of animals with culture-confirmed *M. bovis* infection (Walker *et al.* 1993). Since then, sustained and intensive possum control has been imposed over an area of 5,000 km<sup>2</sup>. In 2010/11 over 1,000 ferrets were necropsied but only one *M. bovis* culture-positive ferret was recorded (Anonymous 2011a). However, it is not possible to definitively attribute that decline solely to possum control, because some modest ferret control and/or TB surveillance of ferrets was imposed in conjunction with possum control, and because rabbit numbers declined greatly after the introduction of a rabbit calicivirus in 1997, which by itself is likely to have resulted in lower ferret densities overall (Norbury *et al.* 2003).

The prevalence of TB among ferret populations in TB-endemic areas appears to increase with ferret age (Ragg 1997; Lugton *et al.* 1997b; Byrom *et al.* 2008) (Figure 1). The youngest known infected ferrets are about 1.75 months old, with models fitted to the age-specific prevalence data suggesting a low or zero prevalence in newborn kits (Caley and Hone 2002, 2004). In TB-endemic areas not subject to intensive population control of possums, *M. bovis* culture-positive prevalence among adult ferrets can reach 100% by 12 months of age (Caley and Hone 2002). Overall, disease is more prevalent among male ferrets than females (Ragg 1997; Lugton *et al.* 1997b; Caley *et al.* 1998). The instantaneous rate of new *M. bovis* infections in ferrets in TB-endemic areas has been estimated to vary from 0.09 to 7.9 per year (Caley and Hone 2002), with male ferrets acquiring new *M. bovis* infections at 2.2 times the rate of females. Sex-specific differences in prevalence of TB are most likely due to male ferrets being more likely to find and/or scavenge carrion than females, due to their larger home ranges (Lugton *et al.* 1997b; Ragg 1998).

### Disease characteristics in ferrets

In feral *M. bovis*-infected ferrets, gross tuberculous lesions occur most commonly in lymph nodes draining the alimentary tract (34.5%) but rarely in the lungs (2.9%) (Ragg *et al.* 1995b), indicating relatively little pulmonary pathology in this species. This indicates that ferrets probably acquire infection primarily via the oral route under natural conditions, most likely through ingestion of infected material (Lugton *et al.* 1997a, b). In support of this,



**Figure 1.** Estimated age-specific prevalence of tuberculosis (TB) in a sample of 407 ferrets (*Mustela furo*) from Molesworth Station in the Marlborough region of New Zealand. Age classes are monthly below 1 year, then pooled for 16–18 months old and >23 months. Age-specific prevalence of TB was estimated by ageing all infected ferrets and a random selection of uninfected ferrets, using the latter to estimate the age distribution for all uninfected ferrets (from Nugent and Whitford 2008).

experimental infection studies have confirmed ubiquitous development of *M. bovis* infection and representative TB pathogenesis following oral exposure of captive ferrets to inocula of virulent *M. bovis* (Cross et al. 2000a, b). In the wild, ferrets most likely contract *M. bovis* infection through scavenging tuberculous carrion (particularly possum carcasses) and/or by killing *M. bovis*-infected prey from the age of weaning (Smith et al. 1995; Ragg et al. 2000; Caley and Hone 2002). Ferrets are known to cannibalise, so ferret-to-ferret (intra-specific) transmission is also possible via that route (Ragg et al. 2000; McAuliffe 2001; Byrom 2004).

The main sites of *M. bovis* pathology in feral ferrets are the retropharyngeal and mesenteric (jejunal) lymph nodes, associated with the upper- and mid-alimentary tract, respectively. This pattern of lesion distribution is analogous to that observed in similarly sized carnivorous/scavenging meerkats (*Suricata suricatta*) in Africa (Drewe et al. 2009), although in the absence of the pulmonary disease observed in meerkats. In ferrets, macroscopic alimentary tract TB lesions are usually characterised by enlargement and erythema of the nodes, with gross lymphadenopathy and extensive liquefactive necrosis a feature of advanced disease (Lugton et al. 1997a). These clinical features have been successfully emulated in captive ferrets by feeding them small (<1 cm<sup>3</sup>) offal samples containing high doses of virulent *M. bovis* (i.e. >10<sup>6</sup> bacteria) (Qureshi et al. 1997, 1999), reinforcing the suggestion of ingestion of material bearing large numbers of bacilli (such as tuberculous carrion) as the predominant mode of infection in the wild.

Infected ferrets excrete *M. bovis* bacilli more readily than pigs or deer, most commonly via the oral cavity, with 23% of oral swabs culturing positive, and tracheobronchial swabs, faeces, urine and mammary glands also being positive (Lugton et al. 1997b; Qureshi et al. 2000). In experimental infection studies, tuberculous ferrets survived for at least 20 weeks post-infection, despite substantial loss of bodyweight and condition (Cross et al. 2000a, b). Progression of the disease can be prolonged in ferrets and it may eventually contribute to mortality in the wild (Lugton et al. 1996). However, because of their inherent high annual mortality rate, which can average 1.1 per year for

females (Caley and Morriss 2001), there remains uncertainty over whether tuberculous ferrets suffer increased mortality due to disease advancement (Caley and Hone 2002).

In common with the other major wildlife hosts in New Zealand and elsewhere, protective vaccination against *M. bovis* has been investigated in ferrets as a potential means of controlling TB. Vaccination using *M. bovis* bacille Calmette–Guérin has been shown experimentally to confer some protection to captive ferrets against pathogenic strains of the bacillus (Qureshi et al. 1999; Cross et al. 2000b); however, lethal population control currently remains the tool used for control of TB in feral ferrets. As with the other mammalian wildlife hosts of TB in New Zealand, the wide-scale use of lethal control is facilitated because ferrets are an introduced invasive pest species.

### Ferret population demographics and ecology

Ferrets prefer pasture, rough grasses and scrubland (Clapperton and Byrom 2005) and there are good correlations between ferret density and the abundance of rabbits, their primary prey (Mills 1994; Ragg and 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 10.1 per km<sup>2</sup>, with densities typically lower at North Island sites (Table 1). These habitats therefore hold the greatest risk of persistence of TB in ferrets independent of other wildlife hosts. An extensive investigation of field techniques to assess ferret relative abundance, and calibrating these to absolute densities, was undertaken by Moller et al. (2002) and concluded that, while no relative abundance measurement technique was particularly precise, a trap-catch index (TCI) offered the most reliable measure. The authors suggested that standardised levels of residual trap catch indices could be set to guide wildlife management decisions around ferret populations, similar to the way these are used by possum TB managers (Anonymous 2011b).

Life expectancy of juvenile ferrets once they emerge from their natal den typically ranges from 1 to 2 years (Caley and Morriss 2001; Byrom et al. 2008), although survival up to 3.5 years has been recorded (Nugent and Whitford 2008). Caley et al. (2002) estimated a survival probability of 25% during the first year of life, rising to 55% thereafter, resulting in a skewed age distribution towards young individuals in any population. In the Mackenzie Basin, survival of juveniles varied from 19% to 100% in the 4 months after emergence from the natal den, decreasing to 0–60% by the end of the first winter, depending inversely on ferret density (Byrom 2002). Annual survival rates of 15–65% have been reported for feral ferrets in New Zealand (Norbury and Heyward 1997; Morley 2002; Byrom et al. 2008), similar to reported rates of 32–67% for the polecat in Europe (Kristiansen et al. 2007). Due to the high annual mortality rate among ferrets, they are regarded as less likely to contribute to risk of TB persistence than longer-lived wildlife hosts of TB in New Zealand, such as wild deer (Barron et al. 2013; Nugent et al. 2015b).

### Home ranges and movement patterns

Movements and home ranges of wildlife hosts provide an indication of the potential for both disease spread (Byrom 2004) and for surveillance utility (Anderson et al. 2013; Yockney et al. 2013). Home range size of ferrets varies between habitats, but is typically around 140 hectares for males and 100 hectares for females (Table 2). Male and female home ranges overlap, both



**Table 1. Estimates of feral ferret (*Mustela furo*) densities (per km<sup>2</sup>) for different habitats in New Zealand.**

Study site	Estimate	Time of year	Habitat	Technique <sup>a</sup>	Reference
North Island sites					
Hohotaka, Manawatu-Wanganui	3.1	Summer	Pasture	Removal	Caley and Hone (2005)
Rangitikei, Manawatu-Wanganui	2.0	Summer	Pasture	Removal	Caley and Hone (2005)
Waipawa, Hawkes Bay	1.2	Summer	Pasture	Removal	Caley and Hone (2005)
Castlepoint, Wellington	1.1	Summer	Pasture/Scrub	Removal	Caley and Hone (2005)
Cape Palliser, Wellington	0.6	Summer	Pasture/Scrub	Removal	Caley and Hone (2005)
South Island sites					
North Canterbury	5.3–7.3	All year	Pasture	Trap catch index	Caley <i>et al.</i> (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 and Morriss (2001)
Mackenzie Basin/Central Otago	2–5	All year	Semi-arid tussock	Minimum left alive	Moller <i>et al.</i> (1996)
Palmerston, East Otago	5.3–6.3	Autumn	Pasture	Removal trapping/mark-recapture	Cross <i>et al.</i> (1998)
Macraes, Central Otago	4.4	Summer/autumn	Tussock grassland	Removal trapping	Middlemiss (1995)
Central Otago	1.6–10.1	Summer/autumn	Semi-arid tussock	Mark-recapture	Norbury <i>et al.</i> (2004)

<sup>a</sup> Standard methods for estimating mammalian abundance, as described in Seber (1973); and Krebs (1998)

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.* 1998a; Moller and Alterio 1999). Ferrets increase the size of their home ranges at times of low food availability, for example after rabbit population control (Norbury *et al.* 1998b).

Male 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 and Morriss 2001; Byrom 2002). Most dispersal occurs during late summer (late February and early March), with no sex difference in mean dispersal distance (Caley and Morriss 2001; Byrom 2002; Byrom *et al.* 2008). Three straight-line dispersal distances were recorded of juvenile female ferrets of 12, 20 and 27 km in the Maniototo district of the Otago region by Ragg (2002).

Caley and Morriss (2001) reported that ferrets dispersed non-directionally across pasture habitat in North Canterbury, with a mean dispersal distance of 2.1 (SE 1.0) km; whereas in the Mackenzie Basin, dispersal has been reported to be directional, following braided riverbed channels. Mean dispersal distances were longer and were recorded as 6.7 (SE 1.6) km for males and 11.8 (SE 3.4) km for females (Byrom 2002). In a mix of pasture, scrub and riverbed habitat in inland Marlborough, maximum distances travelled by ferrets of both sexes were 10–27 km (Byrom 2004; Byrom *et al.* 2008).

#### Control, capture and survey methods

Control of ferrets is conducted primarily by trapping (Spurr *et al.* 2001), usually using traps baited with carrion, e.g. rabbit meat,

**Table 2. Home range sizes (hectares) of male and female ferrets (*Mustela furo*) in the South Island of New Zealand.**

Study site	Season	Mean (min, max) home range and sample size		Reference
		Males	Females	
Mackenzie Basin	Varied	288 (190, 372) (n=5)	111 (44, 225) (n=7)	Pierce (1987)
East Otago	Autumn/early winter	86 (39, 131) (n=7)	45 (18, 89) (n=10)	Ragg (1997)
Otago/Mackenzie Basin	All year	102 (19, 316) (n=34)	76 (16, 240) (n=28)	Norbury <i>et al.</i> (1998b)
Otago Peninsula	Spring/autumn	163 (95, 220) (n=6)	135 (73, 206) (n=10)	Moller and Alterio (1999)
North Canterbury	Winter/spring	194 (60, 320) (n=5)	99 (50, 150) (n=10)	Spurr <i>et al.</i> (1997)
North Canterbury	Summer/autumn	139 (2, 220) (n=4)	151 (62, 314) (n=4)	Young (1998)
North Canterbury	Summer/autumn	80 (n=1)	128 (28, 265) (n=11)	Young (1998)
North Canterbury	Summer/autumn	760 (n=1)	230 (n=5)	Caley and Morriss (2001)
Central Otago	Autumn/winter/spring	94 (68, 120) (n=3)		Baker (1989)
Central Otago	Autumn/winter/spring		59 (40, 79) (n=8)	Ragg (2002)
			(fully described ranges)	
Otago Peninsula	Winter	107 (n=1)		Dymond (1991)
Inland Marlborough	All year	178 (63, 294) (n=5)	52 (13, 91) (n=7)	Yockney <i>et al.</i> 2013

(adapted from Byrom *et al.* 2008)

chicken necks (Norbury *et al.* 2004). Poisoning is rarely used, although sometimes fish-based bait containing diphacinone can be deployed (Spurr *et al.* 2001). Ferret control is done mostly in late summer/autumn and early winter, because control at that time will have the greatest impact on the population (Barlow and Norbury 2001), not least because juvenile dispersal and low adult trap-shyness at these times makes ferrets easier to target. Most trapping for surveillance purposes is also conducted at that time, mostly to maximise the numbers of ferrets obtained by avoiding times of low trappability.

Ferrets can also be controlled through secondary poisoning, particularly of rabbit populations, although kill rate is highly variable depending on the toxin used. Heyward and Norbury (1999) recorded approximately 10% incidental ferret mortality as a result of rabbit poisoning using carrots coated with 0.02% w/w sodium fluoroacetate (1080). In contrast, Alterio (1996) recorded a 100% mortality in five ferrets following a rabbit poisoning operation using broadifacoum. The recent emphasis on ferrets as sentinels means that trapping is still the preferred method of control as it enables carcass recovery.

As alluded to above, capture rates and bait-take by ferrets decline during mid- to late-winter and into spring, coincident with the breeding season (Clapperton and Byrom 2005). Several hypotheses have been proposed to explain this (Spurr *et al.* 2001), including a preference for live prey as juvenile rabbits become more common at this time (Byrom *et al.* 2003), reduced home range sizes and reduced food consumption, and/or seasonal neophobia (i.e. a transient aversion to novel objects in the environment). However, home ranges appear to change little during the breeding season and ferrets may actually increase their activity levels within their range during that time (Ragg 2002), while the hypothesis of reduced food intake has been only weakly supported in empirical studies (Spurr *et al.* 2001). Of the above theories, neophobia appears the most plausible explanation to explain changes in seasonal trappability (Hammond 1996; Byrom *et al.* 2002). Attempts to increase the trappability of ferrets during late winter and spring (e.g. using scented lures) have met with only limited success (Clapperton 1996; Spurr *et al.* 2001).

## Risk factors posed by ferrets to TB management

### Inter-specific transmission of *M. bovis* to ferrets

Quantifying the relative risk of infected wildlife such as pigs, deer and possums causing new TB outbreaks in ferrets is important in characterising potential routes and rates of transmission within the New Zealand wildlife host complex (Caley and Hone 2004). As noted above, experimentally reducing possum (but not ferret) abundance can reduce prevalence of TB in ferrets (Caley *et al.* 2001). That research identified that possum-to-ferret transmission was common, and indicated that most *M. bovis* infection observed in ferrets had resulted from transmission from infected possums (Caley and Hone 2005). In support of this, Ragg *et al.* (2000) and McAuliffe (2001) used night-time video surveillance of freshly killed possum carcasses to characterise visits by ferrets to carrion in South Island farmland settings. The two studies found that 77% and >20%, respectively, of ferret encounters with possum carcasses resulted in feeding.

Ferrets also scavenge on the carcasses of other hosts of TB. Yockney and Nugent (2003) investigated the rate of scavenging of potentially infectious pig carcasses by ferrets in a mosaic of scrub, river flats, and farmland in the Marlborough region, also using detailed video surveillance. Ferrets visited 10/13 (77%) pig offal piles in summer, but none in winter. Probable family groups of ferrets were observed feeding together, often spending long periods of time at a carcass and clambering over each other and the carcass to gain access to soft visceral tissues (Yockney and Nugent 2003). In the four instances in which a whole pig carcass was scavenged, ferrets were responsible for the initial opening of the carcass, leading the authors to conclude that the remains of just one infected pig could theoretically amplify the numbers of tuberculous wild animals locally. Ferrets have also been observed feeding on the carcasses of deer (Yates *et al.* 1995).

When coupled with the predominance of alimentary tract lesions, these studies and observations strongly implicate scavenging on the lesioned carcasses of other hosts of TB (particularly possums) as a likely mechanism of infection for ferrets, suggesting that they might be amplifier hosts of infection, i.e. one tuberculous carcass producing many new infected ferret cases. Through amplification of the total burden of infection in the environment, ferrets therefore have the potential to deepen and broaden the risk pathways that could generate new outbreaks of TB. What is not known with any accuracy or precision is the rate at which ferrets become infected from other infected wildlife. Nevertheless, to enable quantitative use of ferrets as sentinels in predictive epidemiological modelling frameworks, Anderson *et al.* (2013) proposed and used a probability value of 0.08 to describe the possible transmission rate to ferrets arising from a single infected possum in cases involving home range overlap.

### Intra-specific transmission; infection of ferrets by ferrets

Ferrets have the potential not only to amplify *M. bovis* infection but to independently sustain it. There has been considerable debate over the route and extent of intra-specific transmission (Caley and Hone 2004, 2005).

Several possible routes by which intra-specific *M. bovis* transmission could occur among ferrets in the wild have been postulated, including cannibalism (Ragg *et al.* 2000; McAuliffe 2001; Caley and Hone 2005), pseudo-vertical transmission between mothers and their dependent offspring, routine social contact such as mating or fighting (Caley and Hone 2002), or other environmental factors such as animals examining latrine sites. Qureshi *et al.* (2000) demonstrated horizontal transmission of three strains of *M. bovis* among captive ferrets housed at close quarters in a pen trial, although by an undefined mechanism. Other observed risk behaviours included den sharing, sniffing of orifices and faeces, cannibalism and aggressive mating behaviour. Infection through cannibalism is possible as McAuliffe (2001) observed that ferrets were more than twice as likely to visit ferret carcasses than hedgehog or possum carcasses in a field study in North Canterbury farmland in autumn-winter, and >10% of these visits resulted in scavenging. Other studies have reported scavenging rates of ferrets by ferrets ranging from 31% to >60% (Ragg *et al.* 2000; Byrom 2004; Caley and Hone 2005).

Acquisition of infection by non-oral routes has also been inferred. Approximately 20% of visible TB lesions in ferrets occur at peripheral lymph node sites in surveys reporting gross pathology (Ragg *et al.* 1995a), suggesting that infection might sometimes occur via skin breakage, possibly through abrasions incurred

during aggressive social contacts with other ferrets. This has also been reported among tuberculous cases in Eurasian badgers (*Meles meles*) and African meerkats (Gallagher and Clifton-Hadley 2000, Drewe *et al.* 2009). Peripheral site infections are more common among males, possibly reflecting the higher rate at which males interact with other ferrets (Ragg *et al.* 1995b; Lugton *et al.* 1997b), which could also explain the greater force of infection (the rate at which susceptible individuals acquire infection) observed in male ferrets (Caley and Hone 2002). Overall, however, non-oral acquisition of infection in feral ferrets in New Zealand is thought to be less important than oral acquisition (Caley and Hone 2002).

More crucially important than the mechanism(s) of intra-specific transmission between ferrets is the purported rate at which this occurs. If each infected ferret could infect (on average) one or more other ferrets during its life-span, elimination of TB from wildlife would necessarily require ferret management. In reality, this could occur wherever ferret populations surpassed the threshold density theoretically required for independent disease maintenance (Caley and Hone 2005). Caley (2002) demonstrated that, among high-density populations, reducing ferret abundance alone could reduce the consequent force of infection in ferrets, indicating some level of intra-specific transmission was occurring. But such transmission in isolation was not sufficient to signify sustained independent maintenance of TB in the ferret population. In order for ferrets to maintain TB independently of other wildlife sources of infection, it was postulated that ferret populations would need to reach a peak density of  $\geq 5.3$  ferrets per km<sup>2</sup>, and a year-round density of  $\geq 2.9$  ferrets per km<sup>2</sup> although these figures have wide confidence limits (Caley 2002). There has been no further research on this, so more precise empirical data, quantifying the rate at which intra-specific transmission naturally occurs in ferrets, are still needed to unequivocally clarify the host status of ferrets in different habitats (Caley and Hone 2005) and for epidemiological models of infection in multi-host complexes (Craft *et al.* 2008).

Thus there seems to be little doubt that ferret-ferret transmission occurs (probably by a variety of mechanisms), and occurs frequently enough to temporarily amplify infection within the species at high population densities, but it appears to only rarely, if ever, occur frequently enough for ferrets to be regarded as true maintenance hosts. Accordingly, ferrets are generally classed as being spillover hosts in New Zealand's multi-host wildlife TB complex (Nugent 2011).

#### **The risk of spillback infection to other wildlife hosts from ferrets**

As ferrets are seen as spillover hosts in most areas, determining the risk of spillback is crucial in assessing whether ferrets should be regarded as an end-host, or whether *M. bovis*-infected ferrets have the potential to cause new TB outbreaks in the other wildlife species from which they, directly or indirectly, originally acquired TB. From the perspective of TB management, the principal risk is that ferrets could re-establish infection in populations of the primary maintenance host (possums) among which *M. bovis* had been eradicated, and after possum control had been discontinued. Ferrets could potentially do this by carrying TB into the area from some distant source, or by acting as a 'go-between' host, transmitting residual infection from long-lived hosts (such as deer) to possums (Nugent 2011). This could occur by interactions between the other wildlife species and live *M. bovis*-infected

ferrets, or through interaction with ferret carcasses. The former seems unlikely, as observations of livestock interactions with simulated terminally ill wildlife have shown that deer are far less likely to make contact with ferrets than with possums (Sauter and Morriss 1995). Further, we are unaware of data or observations showing interaction between live ferrets and possums, and suggest that healthy ferrets would avoid pigs.

Compared to pigs and deer, the often highly liquefactive lesions observed in ferrets probably contain greater numbers of live bacilli per gram of tissue and potentially present more of an infection risk (Lugton *et al.* 1996), thus tuberculous ferret carcasses may represent a greater risk of spillback infection of *M. bovis* per unit size than do those of pigs and deer (Nugent *et al.* 2003). Accordingly, the risk to other wildlife species of infection from tuberculous ferrets is mostly the risk posed by ferret carcasses, and this has been explored in monitoring studies. Ferret carcasses (13 in summer and autumn, 20 in winter and spring) were monitored using infrared-triggered cameras in the presence of a full complement of potential wildlife hosts (including pigs and deer) and free-ranging livestock (Byrom 2004), in conjunction with a similar study monitoring pig carcasses (Yockney and Nugent 2003). Of the 15 different species visiting the ferret carcass sites, sheep and cattle, Australasian harriers (*Circus approximans*), ferrets, possums, cats, and hedgehogs (*Erinaceus europaeus*) approached to within 1 m of, and interacted with, the carcasses in some way. Many encounters resulted in behaviours that could be considered risky in terms of TB transmission (i.e. rolling on the carcass, sniffing, licking, or scavenging), and the average duration of each interaction was about 30 seconds. Interactions by different species in summer and winter are presented in Table 3. Pigs and deer did not visit the ferret carcasses in either season despite being present in the area. By comparison, the concurrent study on pig carcasses indicated somewhat higher scavenging rates by wildlife, including ferrets (although only during the summer months) (Yockney and Nugent 2003). Based on recorded interactions with ferret carcasses in these studies, the authors concluded that the overall risk of transmission of *M. bovis* infection from dead ferrets to pigs and deer was low, particularly compared with the likelihood of transmission from wild pigs to ferrets.

In the monitoring study of Byrom (2004), possums themselves were not observed directly consuming tissue from ferret carcasses, although other close-contact behaviour, specifically sniffing and licking of carcasses, was noted. In contrast, Ragg *et al.* (2000) documented an instance (from two encounters) of a possum actively scavenging at a ferret carcass, showing at least the feasibility of direct *M. bovis* transmission to possums by scavenging from ferret carcasses. Transmission to possums from ferret carcasses could also occur indirectly, as McAuliffe (2001), Ragg *et al.* (2000) and Byrom (2004) all observed other wildlife species (such as harriers) scavenging from ferret carcasses, sufficient to open up the carcass and scatter potentially infectious material over a wide area. Potentially therefore, any possum subsequently visiting the site (even if it was not actually scavenging on the carcass) could come into contact with infectious material now dispersed beyond the immediate vicinity of the carcass.

#### **Transmission of *M. bovis* infection to livestock from ferrets**

There is little empirical evidence with which to quantify the potential risk of transmission of infection from ferrets to livestock. Ferrets must play some role in livestock infection, because one

**Table 3. Percentage of ferret (*Mustela furo*) carcasses visited by each of five wildlife species and two livestock species in summer and winter, with average time to first discovery for carcasses visited, and the percentage of carcasses touched or sniffed, licked or fed upon. There were 13 ferret carcasses in summer and 20 in winter (from Byrom 2004).**

Species	Season	Carcasses visited (%)	Average time to discovery (hours)	Carcasses touched or sniffed (%)	Carcasses licked (%)	Carcasses scavenged (%)
Possum	Summer	38	21	38	0	0
	Winter	55	49	45	10	0
Ferret	Summer	38	11	38	0	31
	Winter	10	128	10	5	0
Cat	Summer	31	57	31	0	24
	Winter	30	12	30	15	0
Australasian harrier	Summer	31	105	31	0	31
	Winter	15	20	15	0	15
Hedgehog	Summer	31	27	31	0	5
	Winter	25	72	25	5	5
Cattle	Summer	ND	ND	ND	ND	ND
	Winter	25	54	25	0	0
Sheep	Summer	8	100	8	0	0
	Winter	5	97	5	0	0

ND=Not assessed at that time

study has shown that the incidence of TB in cattle can be reduced following ferret control in the area (Caley *et al.* 1998). Potential routes of transmission of *M. bovis* from ferrets to domestic stock are similar to those postulated for possum-to-livestock routes, i.e. via the mouth or nasopharynx by licking or sniffing carcasses (Sauter and Morris 1995), although it is possible that oral-route infection of livestock could also occur by contact with live bacilli shed onto the ground by tuberculous ferrets (Ragg *et al.* 1995a, b; Lugton *et al.* 1997a, b) or by bacilli dispersed from a tuberculous carcass by the action of scavenging ferrets. Shedding of potentially infectious bacilli through faeces and urine, or in maternal milk, has been shown for ferrets. About a quarter of tuberculous ferrets in the study by Lugton *et al.* (1997b) provided evidence of oral shedding of viable *M. bovis* bacilli through coughing or sneezing, apparently in ferrets where disease was well advanced.

As noted above, live drugged ferrets exhibiting behaviour similar to late-stage tuberculous animals were nosed and sniffed by cattle and deer in an experimental study, although to a lesser degree than similarly drugged possums (Sauter and Morris 1995). Byrom (2004) also observed that cattle investigated ferret carcasses, sniffing and making nose contact on several occasions, often after environmental exposure of opened lesions could have occurred through scavenging by harriers. Whether such behaviours result in transmission of infection to livestock under natural circumstances, and the frequency with which that might occur, are not known under New Zealand conditions, making it difficult to quantify the management significance of this transmission pathway.

#### The potential for long-distance spread of TB by ferrets

Information on the movements and dispersal patterns of wildlife TB hosts is critical in assessing the role they play not only in disease persistence but also in the geographic spread of the disease, and therefore in determining how much emphasis should be placed on population control as part of vector management programmes (Pech *et al.* 2010). Ferrets are capable of acquiring *M. bovis* infection soon after emerging from the natal

den (Caley and Hone 2002; Yockney and Nugent 2003). However, dispersal does not occur until about 57 days on average after emergence (Byrom 2002), so there is a window in which juveniles could acquire infection and then transport it elsewhere.

The probability of long-distance dispersal of TB by infected ferrets has long been inferred from their general movement behaviour (Livingstone 1996), and such dispersal was implicated as the likely underlying cause of the rapid southward spread of TB in North Canterbury in the early 1990s (O'Hara 1993). Probable cases of such dispersal were recorded in a study in the North Canterbury high country (Byrom 2004) in which 10 juvenile (seven male, three female) ferrets were trapped, radio-collared, and successfully tracked from mid-summer, a time when they were likely to still be in family groups and close to their mothers' home ranges (Byrom 2002), until they were recaptured and killed in autumn, during which time they had moved 0–10.7 (median 2.5) km. By autumn, three of the males were *M. bovis*-infected, with these three having moved straight-line distances of 1.5, 3.5, and 10.7 km. The carcasses of four of the ferrets were video-monitored and two were confirmed as being scavenged by other ferrets.

The geographical scale of the risk of TB spread as a result of host dispersal is critical in determining the boundaries of Vector Risk Areas (VRA), the areas in which there is a risk of TB being persistent in wildlife. To help TB managers delineate boundaries that could minimise the risk of disease spread by wide-ranging spillover hosts, Nugent *et al.* (2003) modelled the relative risk of persistence and spread of TB posed by ferrets, assuming that possum densities within VRA boundaries were kept very low and that TB would therefore eventually disappear from both possum and ferret populations (model input parameters as outlined in Supplementary Information 1<sup>1</sup>). The modelling predicted that, if ferret densities were close to the theoretical disease maintenance threshold (Caley and Hone 2005), TB could persist in the ferret population

<sup>1</sup><http://dx.doi.org/10.1080/00480169.2014.981314>



within a VRA for >20 years, at least when TB-induced mortality in ferrets was set to zero, although it would be <8 years if TB infection significantly increased ferret mortality. Persistence was predicted to reduce substantially if ferret density was reduced (i.e. if ferret control was imposed).

Empirical dispersal data was used from two contrasting case study sites, a typical pastoral habitat, Scargill Valley in North Canterbury (Caley and Morriss 2001) and a sparsely vegetated riverbed habitat, the Mackenzie Basin (Byrom 2002). Modelling predicted that few juvenile ferrets would disperse long distances in the pastoral habitat but would disperse further in the riverbed habitat, and relatively few ferrets were predicted to disperse more than 10 km in either habitat (Nugent *et al.* 2003). Importantly, for both habitat types, the density of *M. bovis*-infected juvenile ferrets settling outside the area containing infected possums was high when the possum population inside the VRA was not controlled. Under some scenarios, more than one *M. bovis*-infected juvenile ferret was predicted to settle beyond the VRA for each kilometre of VRA boundary in the simulation, at a proximity of <1 km to the VRA boundary. Possum control within the VRA was predicted to quickly reduce that figure to one ferret per 15 km of VRA boundary. The density of infected juvenile ferrets settling further outside the VRA boundary was predicted to decline to near zero over just 4 km from the boundary under the Scargill Valley dispersal profile, or 8 km under the McKenzie Basin profile. Overall, the width of a precautionary buffer around the known or suspected locations of TB in possums was therefore predicted to be <10 km. Thus, while it is plausible that ferrets could occasionally carry TB up to 27 km from a VRA (Ragg 2002), the probability of that is extremely low, excluding any area where ferret densities are high enough to be maintenance hosts.

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## Use of ferrets for TB surveillance

Over the last two decades prevalence of TB has fallen dramatically in livestock in New Zealand (Livingstone *et al.* 2015) and in possum populations (Nugent *et al.* 2015a), and the focus of research and management efforts has shifted from predominantly control towards a combination of both control and surveillance of TB in wildlife, particularly with the purpose of confirming absence of TB in response to wildlife control (Anderson *et al.* 2015). This has seen increased use of spillover hosts (in this case ferrets) as a sentinel for detecting persistence of *M. bovis* infection in sympatric possum populations (Nugent *et al.* 2002; Yockney *et al.* 2013; Anderson *et al.* 2013). This concept has now been formally integrated into a national proof of freedom of TB surveillance framework for New Zealand (Nugent *et al.* 2006; Hutchings *et al.* 2011), and also underpins an equivalence-based TB-testing approach developed for game estates (Byrom *et al.* 2004).

The most in-depth comparative analysis of the sensitivity (as sentinels for persistent wildlife TB) of all of the main non-possum hosts of TB in New Zealand was conducted in semi-arid mountain land in the Marlborough region (Molesworth Station 42.2°S, 173.1°E) by Byrom *et al.* (2008), and Nugent and Whitford (2008) (input data parameters are outlined in Supplementary Information 2<sup>1</sup>). The results indicated that 21.7% of 407 ferrets surveyed were infected with *M. bovis*, with infection prevalence

increasing with age (Figure 1). However, there were no clear changes in patterns of the apparent annual incidence (Thrusfield 1995) of infection with age, indicating that acquisition of new infection probably occurred steadily throughout life for these ferrets. It was estimated that the ferret population accumulated new *M. bovis* infections steadily at a rate of 0.6 occasions per year of ferret exposure. Ferret densities were crudely estimated to span 0.9–1.5 per km<sup>2</sup> which, combined with an apparent annual incidence of 0.6, suggested 0.5–0.9 cases of new ferret *M. bovis* infection occurring per km<sup>2</sup> per year (Nugent and Whitford 2008). Using matching data and estimates of possum population density, average home range and prevalence of TB, and assuming a ferret home range size of 1.2 km<sup>2</sup> (Byrom *et al.* 2008), Nugent and Whitford (2008) calculated that there would be 1.7–3.4 infected possum carcasses available annually to be encountered within each ferret range. If the bulk of the observed infection in ferrets was acquired from possums, individual ferrets would have become infected on >30% of occasions during which an infected possum carcass occurred within their range. However, that figure, which is effectively the marginal probability of ferrets detecting TB in possums, ignores the possibility of ferrets being infected from pigs or deer carcasses. Therefore a much lower figure (8%) has been adopted conservatively in the proof of freedom model (Anderson *et al.* 2013) that is now widely used to quantify the probability of true absence of TB in possums.

By comparison with the surveillance sensitivity calculated for ferrets, the probability of detection for pigs on Molesworth was estimated by Nugent and Whitford (2008) to be higher still, whereas those for cattle and deer were predicted to be much lower (as shown in Supplementary Table 1<sup>1</sup>). Taking into account differences in the mean exposure (life span) of each sentinel species and home range size, and the cost of obtaining and testing each sentinel, ferrets on Molesworth Station were predicted to be only about half as cost-effective (per hectare) as pigs, but twice as cost-effective as using tuberculin testing of cattle, and many times more cost-effective than surveying wild deer, or possums directly (Supplementary Table 1<sup>1</sup>). As a result of that moderately high utility, ferrets are now widely used, usually in conjunction with direct surveys of possums, for proving freedom from TB of wildlife during surveillance operations in farmland and unforested semi-arid areas, particularly those where feral pigs and wild deer are scarce (Anderson *et al.* 2015).

In practical terms, the importance of ferrets to effective monitoring and management of TB in New Zealand is borne out in surveillance statistics (Supplementary Information 3 and Supplementary Table 2<sup>1</sup>). Over the 2010/11 period, TBfree New Zealand reported capture and necropsy of 17,397 ferrets for TB surveillance (data sourced from unpublished metadata in the VectorNet database, TBfree Ltd, Wellington, NZ). Most of the ferrets were trapped from management units that had been subject to intensive possum control for many years. Some of these surveys were conducted primarily to help assess the probability of freedom from TB, but others aimed to support preventive control, either to investigate the potential role of wildlife in a recent TB outbreak among livestock, or to confirm reduction of TB within VRA or disease containment in areas close to the perimeter of VRA. These latter surveys yielded 4,447 ferrets, including 3,782 captured inside VRA zones, of which 33 animals were confirmed *M. bovis*-culture-

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<sup>1</sup><http://dx.doi.org/10.1080/00480169.2014.981314>



positive. All 33 infected ferrets were located within the southern South Island VRA, representing an overall prevalence of TB in ferrets of 0.89% within VRA and 0% in perimeter areas (Anonymous 2011a), suggesting that containment of disease in ferrets to the VRA was effective.

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## Conclusions: Management of ferrets for TB control in New Zealand

Ferrets are consummate scavengers of potentially infectious material, with a demonstrated propensity for pig (and presumably possum and deer) remains. They are also cannibalistic. Where scavenging occurs within TB-endemic areas, ferrets are likely to amplify the disease. In the habitats most favourable to ferrets, and in the absence of ferret control, amplification by cannibalism (and/or other means) may be sufficient for ferrets to independently maintain TB. Although feasible (Caley 2002; Caley and Hone 2005), there is, as yet, no confirmation that ferrets do actually achieve true maintenance host status in New Zealand. Rather than maintenance hosts, ferrets might therefore best be characterised as extended spillover hosts in which *M. bovis* infection originally acquired from possums could then occasionally cycle within a ferret population before eventually disappearing. Thus, even when possum control has been sufficient to reduce inter-specific *M. bovis* transmission to ferrets to near zero, ferrets may sometimes provide a persistent reservoir of TB after possum control has been undertaken, particularly if there is no additional mortality attributable to the disease burden (Nugent *et al.* 2003).

The probability of *M. bovis* infection being transmitted from tuberculous ferrets on to, or back to, other species of wildlife through scavenging, particularly by key host species such as pigs and possums, appears to be low (Ragg *et al.* 2000; McAuliffe 2001). Likewise, the risk of transmission from live infectious ferrets to other hosts such as cattle and deer appears to be lower than it would be for possums (Sauter and Morris 1995). Nonetheless, there are indications that some transmission to livestock does occur (Caley *et al.* 1998), so this risk must also be considered and managed as part of the overall TB problem.

As pigs and deer are spillover hosts for TB in New Zealand (Nugent *et al.* 2015b), and because TB in livestock can be prevented from spreading to other livestock by standard diagnostic testing and movement control means (Buddle *et al.* 2015), it is the risk of occasional transmission of *M. bovis* infection from ferrets to possums that is of greatest management significance. This is firstly because it could increase the time required to achieve eradication of TB from possums within a VRA. At densities below the theoretical TB maintenance threshold for ferrets, complementing possum control with ferret control should help to reduce the prevalence TB in ferrets, and therefore risk of transmission of infection from ferrets to livestock, more quickly than possum control alone. The geographical areas where this might apply need to be better defined, which would require field validation of ferret-ferret transmission rates and, ideally, the TB-persistence threshold densities proposed by Caley (2002) and Caley and Hone (2005) for key habitat types containing ferrets.

The second main risk from occasional ferret-possum transmission arises because they are more likely, on average, to move further

than possums, dispersing on a scale equivalent to that of deer and pigs (Yockney *et al.* 2013). Infected ferrets could, therefore, cause new outbreaks of TB in areas previously free of tuberculous wildlife. However, both the empirical dispersal data available and the modelling summarised above suggest that movement of TB by ferrets is restricted to the immediate periphery of a VRA, as the frequency with which dispersing *M. bovis*-infected ferrets might transport TB well beyond VRA (>10 km) appears to be low. The probability of an infected ferret dispersing a long distance and the probability of ferret-possum transmission are both low, so the joint risk of both occurring appears negligible, especially once intensive possum control has been initiated. Adding to that, the prevalence of TB is lowest in the juvenile age class most likely to disperse. For older age classes, ferrets pose little risk of moving outside a VRA unless there is a sudden environmental perturbation, such as a rapid reduction in rabbit numbers (Norbury *et al.* 1998b). There is also a smaller likelihood of ferrets being moved by humans compared to species such as deer and pigs (which are hunted for recreation), so the risk of TB spread by ferrets is likely to be low overall (Nugent *et al.* 2003).

As sentinels for assessing the likelihood of freedom from TB in possums, ferrets are cost-effective largely because, like pigs, they cover much larger areas than possums and appear to have a moderately high chance of finding and feeding on any carcasses of tuberculous possums that occur within their home range. Ferrets rank second in terms of wildlife TB-detection sensitivity behind pigs (Yockney *et al.* 2013), but nonetheless they are now frequently surveyed (and concurrently controlled) in many of the less-developed farmland areas within VRA in order to provide data needed to estimate the probability that that area is free of TB. For this purpose, the number of years of active surveillance of ferrets without any detection of TB becomes a crucial consideration. The other purpose of using ferrets as a survey tool is to determine the prevalence and distribution of residual TB in wildlife in areas where it is known (or suspected) to persist even after possum control. Nugent and Whitford (2008) concluded that where TB is common, both pig and ferret necropsy and culture, as well as diagnostic testing in cattle, would all provide equally inexpensive ways of monitoring trends in prevalence of TB. Overall, ferrets have been an important part of the National Pest Management Plan for bovine TB in New Zealand (Anonymous 2009) and will remain so.

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