THE EPIDEMIOLOGY OF BOVINE TUBERCULOSIS IN NEW ZEALAND

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Introduction

This article draws on recent tuberculosis research in wildlife, and aims to distill critical conclusions from currently available data Because of the central role of wild mammals in the disease, the lack of an *in vivo* diagnostic test for infection, and the questionable validity of experimentally infected animals as a guide to the field disease process, understanding must come from careful epidemiological inference rather than direct measurement This inference process depends on obtaining multiple sources of supporting evidence for individual parts of the transmission hypothesis, and failing in attempts to deliberately disprove crucial arguments Practical progress comes through development and statement of a hypothesis, rigorous and extensive evaluation of its components leading to refinement of its details, and proof through better control of the disease

Categorization of Hosts

It is generally accepted that the problem of tuberculosis in New Zealand is due to wildlife infection - the issue which has not been fully resolved is the number of species which play a significant role in the disease. There are at least 12 wild and feral species which have been detected as infected with tuberculosis in New Zealand - possum, deer, cattle, sheep, pig, cat, ferret, stoat, weasel, goat, rabbit [(1)] and more recently hare [(2)] and hedgehog [(3)] , although animals such as the rabbit have very rarely shown disease in the wild Some of the species listed are however of little or no consequence in the maintenance of infection in the wild or in transmission of disease to domestic stock

If tuberculosis occurs in one wildlife species which acts as a *reservoir host* or *maintenance host* in a region, infection may pass into certain other species (such as scavengers and predators) as *spillover hosts* , if the challenge is high enough due to widespread infection It is crucial to know which species fall into each category, since maintenance hosts are central to the disease, whereas infection in spillover hosts will disappear progressively if disease is eliminated or reduced in the species which is acting as the main source A spillover host may be a *dead-end host* if it plays

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no significant role in onward transmission of infection, or at the other extreme may be an *amplifying host* which increases prevalence of infection in domestic stock or expands the number of species affected by the disease Thus control directed at a spillover host may be pointless, or may be valuable as an interim measure, whereas control directed at a reservoir host is crucial if progress is to be made

Host Status

There is no single way of deciding the position of a species in this epidemiological spectrum Moreover the situation is not necessarily static In determining the importance of a species, point prevalence of infection assessed through cross-sectional sampling (surveys) is an unreliable guide if used in isolation Infection may also be detectable long before an animal becomes infectious for its own and other species, which further undermines point prevalence data For example, possums mostly die soon after the disease becomes detectable, and so the annual prevalence is about 5 times as high as the point prevalence estimated from cross-sectional (survey) sampling For feral pigs, which are often not seriously affected by the disease and are long-lived, point prevalence may be high - yet incidence may be very low and infection dead-end Moreover, prevalence data may not reflect the true situation - such as if the disease is very seasonal or clustered (as in possums) The role of a particular species also depends on how long individuals are infectious and how infectious each such animal is (which depends on the routes of excretion, and on likelihood of transmission opportunities occurring)

Simple static views of wildlife populations and of diseases such as tuberculosis within them can therefore produce dangerously misleading conclusions, and it is essential to view the overall ecological and epidemiological system in a dynamic way, considering flows of infection within and between species, not just levels of disease as measured crudely by surveys

The evidence is now conclusive that the brushtail possum (*Trichosurus vulpecula*) meets the requirements to be considered a reservoir host

1 There is a clear spatial and temporal association between infection in possums and incidence of infection in domestic stock
2 Infection can persist continuously in possum populations without any evidence that it is being seeded from domestic or wild animals
3 Reduction in the spatial density of tuberculous possums by control methods produces a prolonged reduction in the incidence of infection in indicator species which have been studied (mainly cattle), when it is unlikely that control efforts directed against possums would reduce numbers or tuberculosis prevalence in other infected wildlife to a sufficient degree to mimic this effect
4 The Castlepoint longitudinal study [4] has shown a coherent epidemiological pattern of spatial and temporal distribution of infection with specific restriction endonuclease (REA) types of tuberculosis in possums, consistent with long-term maintenance of infection in groups of possums and transfer of infection to other species - without maintenance in some of those secondary species (such as cattle)

Of the other species, evidence is steadily accumulating to suggest that feral deer do not fulfil the requirements for an important reservoir host, although still being crucial as amplifiers of the disease The data is however much less comprehensive and clear-cut than it is for possums There has been persistent infection in feral deer populations over extended periods, which has carried over to farmed deer in the process of domestication Recent studies in the Hauhungaroa ranges found a prevalence of 37% in wild deer, however none of 10 animals under one year of age showed evidence of infection, even though half their mothers were likely to have been tuberculous[5]) It appears that deer to deer spread in wild populations overseas will only maintain a prevalence of up to 5% in the absence of other significant wildlife vectors The high prevalence of infection in wild deer in New Zealand appears to result from the investigation of tuberculous possums by bold and inquisitive adults, which results in a preponderance of tonsillar and retropharyngeal lymph node lesions Most infected wild deer shed few bacilli, but those that are highly infectious will usually be terminal cases which separate from their cohorts

There is a growing list of cases where deer have infected previously negative possum populations, whereas the specific evidence for cattle having infected possum populations is surprisingly sparse, given the far larger number of infected cattle which have been in contact with possums in both Australia and New Zealand Deer may be highly infectious[6] and have contact with possums under circumstances which would potentially allow transmission to occur, albeit as a rare event However, the evidence from geographical patterns of possum infection is that establishment of infection in additional possum populations from another species is a rare event, which has probably occurred less than 50 times in total over the last 50 years in New Zealand (and certainly not an order of magnitude greater) In contrast, the reverse transfer from possums to cattle and deer has occurred orders of magnitude more commonly

Deer may be the principal initiator of new areas of wildlife infection, which would make them of much greater significance to the maintenance of the national problem than cattle However, once infected, the possum has become a numerically larger and effective reservoir host population, which can now maintain infection in endemic areas without the need for further transfer from deer, and can produce progressive expansion of infection into contiguous areas However, according to the hypothesis, creation of new independent foci of possum tuberculosis in free areas depends principally on feral or domestic deer as an initiating factor It is crucial to future control to clarify the epidemiological role of deer since this hypothesis depends more on inference than on direct evidence, due to the lack of critical data for deer

The role of other wildlife species has not yet been adequately resolved Clearly if one or more additional species are acting as true maintenance hosts, either nationally or locally, we need to know and adjust control strategies However this is not a judgement to be made lightly because it has important consequences, and an erroneous indictment of another species may prove impossible to reverse once it has become dogma - as various other beliefs on tuberculosis have done in the past, without good evidence

It is inevitable that susceptible predator/scavenger species such as ferrets, pigs and cats which consume maintenance hosts will become infected and that the prevalence of disease in such

species may in some cases be substantially higher than in the true maintenance hosts (because they are so frequently exposed by eating other infected animals) - *even if there is little or no transmission within the predator species itself* In certain restricted situations a predator/scavenger species may transmit infection to domestic stock, but what is critical to control policy is whether infection is truly self-maintaining in the predator species for the long term without constant re-introduction of infection

This in turn depends on whether there is direct animal to animal transmission within the predator species at sufficiently high incidence to maintain infection in the predator population of an area for multiple generations after the infected prey have been withdrawn from the diet by control measures. If incidence of tuberculosis in a predator/scavenger species is largely or entirely determined by its consumption of a reservoir host species, and there is little or no onward transmission to other wildlife, then control of its numbers will make little contribution to reducing the wildlife tuberculosis problem Population reduction in such a predator species can then only be justified if that species is acting as a secondary amplifying transmitter to domestic stock on a substantial scale, and will at best be a palliative measure until more fundamental control measures can be implemented

In the Castlepoint longitudinal study[4] of an infected possum population and associated species (which has provided the basis for much of our data on wildlife tuberculosis), intensive investigation has revealed infection in feral deer, sheep, pigs, hedgehogs, and ferrets, but the reservoir of infection appears to be in possums, with the other species apparently of little consequence either in determining whether infection persists on the site, or in determining incidence in cattle

In the Northern Territory of Australia feral pigs showed over 30% tuberculosis prevalence when the disease was common in feral swamp buffalo, and pigs scavenged carcases of buffalo which died at the end of the dry season[7] However, Corner *et al*[8] concluded from their epidemiological investigations that the feral pig was a dead-end host rather than a reservoir host As a consequence no control measures were carried out against feral pig tuberculosis when the disease was being eradicated from feral buffalo in the Northern Territory, yet the prevalence in pigs has spontaneously dropped to a very low level This demonstrates that high prevalence is not a good guide to the importance of a host, or the necessity for control Tuberculosis has been found in feral pigs in various parts of New Zealand, and Wakelin and Churchman[9] found a prevalence of about 30% in Central Otago, with clusters in families up to 60% All 28 pigs from close to the Castlepoint study site have been infected with tuberculosis, about half having generalized lesions and 4 with draining mandibular abscesses, indicating the possibility of at least some intraspecific transmission However it is assumed that pigs become infected in most instances by scavenging It seems most unlikely on present evidence that the feral pig is a reservoir host, although dead pigs could provide a source of infection for ferrets, thus amplifying the short-term spillover of possum infection into scavengers

Feral ferrets have been touted as potential reservoir hosts, but the credible evidence is quite limited and equivocal The ferret is highly susceptible to infection with *M. bovis*[10] In the wild,

the disease probably develops slowly, and lesions during the infection remain obscure for much of the time Ferrets are widespread in New Zealand, but abundance varies greatly, with rabbit numbers generally believed to be an important determinant Tuberculosis has been found in a number of ferret populations[11,12], and has been closely associated with the occurrence of tuberculosis in cattle herds, although not all cattle herds with infection in an area have necessarily had infected ferrets in the vicinity In the only available study, no relationships were found between cattle tuberculosis reactor incidence and ferret tuberculosis prevalence, total ferret abundance, or abundance of infected ferrets No clear-cut case has yet been found where tuberculosis is persisting in ferrets in the absence of a possum population that could be the underlying reservoir, but as yet the evidence is not extensive enough to make any definitive judgment Possums form part of the diet of ferrets[13] Prevalence of ferret tuberculosis in localities found to have the disease varies, but has been as high as 100%[14], with substantially higher prevalence in adults than juveniles In contrast to possums, the distribution of lesions indicates that the major portal of infection is via the digestive tract Superficial lymph node lesions, possibly arising from bite wounds, were suspected to be the initial site of infection in 21% of 24 animals where a primary site could be identified[14] Positive cultures have been obtained from the pharynx, faeces, urine, tracheal washes and mammary glands of 48%, 24%, 14%, 12% and 17% infected ferrets respectively Lesions in ferrets are much less apparent than in possums, which complicates diagnosis, and surveys based on gross necropsy findings may significantly underestimate infection prevalence It is unclear whether at a population level they excrete on an adequate scale to be capable of transmitting infection to sufficient domestic or wild animals to contribute significantly to the epidemiological pattern seen in the field

There is as yet no substantive evidence to show maintenance of infection within ferret populations in the absence of food sources of *M. bovis* It is clear, however, that horizontal transmission in ferrets can occur through fighting or cannibalism, and pseudo-vertical infection may be possible, but there is no certainty that the flow of infection through these pathways will be sufficient to ensure that ferrets can be considered a reservoir host for *M. bovis* Moreover, it seems likely from behavioural studies that ferrets would be substantially less effective than possums in transmitting infection to domestic stock[15] Of the four lines of evidence used above to support the case that the possum is a reservoir host, there is as yet no equivalent body of data to make a judgement with respect to ferrets Therefore judgement must currently be withheld on the contribution of ferrets to the epidemiology of tuberculosis in New Zealand, because there is as yet no positive evidence that they are either reservoir or secondary transmitter hosts

The hedgehog has recently been confirmed as a host for tuberculosis In heavily infected areas of the Wairarapa there have been six animals diagnosed tuberculous from a sample of 157 examined[16] As hedgehogs will eat almost any animal substance it has been hypothesized that they become infected by ingestion of tuberculous carrion On the available evidence, it would seem that hedgehogs are another spillover host for tuberculosis As the feeding ranges of hedgehogs in New Zealand are small (e g between 1 9 and 3 6 ha), the discovery of a tuberculous hedgehog is indicative that a tuberculous animal has died in the vicinity The moderate prevalence of tuberculosis, limited home range, and ease of capture of these animals in surveys may make them useful as an indicator species for endemic tuberculosis

At present no species other than the possum can be confidently classified as true feral reservoir host in New Zealand Other species may prove to be significant transmitters in at least some situations, but the evidence remains weak and conflicting It is essential that a well-designed process of field investigation be followed to resolve the role of the ferret The situation is no doubt evolving all the time when there is substantial weight of infection in wildlife It is conceivable that new species may become local or widespread reservoirs in much the same way that the possum did in about 1955-60 Conversely, some species may go through waves of acting as temporary transmitters in particular areas without becoming maintenance hosts, especially if there has been a reduction in prey numbers which could provide sufficient population stress to precipitate an epidemic in the species, without prolonged continuation of transmission from that species to cattle and deer We must therefore be alert to the possibility of new reservoir species emerging, but be very cautious about deciding that a species has moved from spillover to reservoir status since the two can be very difficult to distinguish in the short term, and it would be difficult to reverse such a conclusion in people's minds once it became established - regardless of the evidence

Possum Transmission Mechanisms

On the evidence so far available it would appear that transmission between possums occurs through two main pathways and one minor pathway The first major pathway is transmission from mother to joey during the rearing process Some infection may be transferred through milk, since the mammary gland has been found infected in 12% of tuberculous females [17] and *M. bovis* has been isolated from a joey which must have become infected while it was continuously attached to the teat[17] However, the evidence suggests that a much higher proportion than 12% of progeny of tuberculous mothers become infected [6, 18], and most of these additional animals would have become infected through close physical association with their dams through the rearing period The respiratory route is probably the most important for transfer because a high proportion of tuberculous adults have lung lesions [19], and lung lesions have been found in unweaned joeys [20] A substantial proportion of tuberculous adults also have discharging sinuses in advanced disease, and cross-infection could occur by ingestion or across mucous membranes during grooming of the joey It is notable, however, that lesions are uncommon in mesenteric lymph nodes of apparently early cases and appear to typically develop late in the disease [20], so it seems unlikely that joeys are infected commonly by ingestion of organisms This group of mechanisms can be considered a single pathway, "pseudo-vertical transmission", since a large proportion of the progeny of infected females become infected early in life

The second major transmission mechanism is direct horizontal transmission among adult possums Our evidence from the Castlepoint longitudinal study strongly suggests that this takes place mainly in the area around where a possum dens While denning patterns vary somewhat among the small number of sites studied over extended periods in New Zealand (Orongorongo Valley [21], Bridge Pa [22], Castlepoint [18]), there are many similarities At Castlepoint (as in forest at Orongorongo Valley [23]) possums tend to den within a relatively small area, commonly at one extremity of their home range [24], and we define this as the denning range, to distinguish it from the much larger foraging range As a result, within the Castlepoint study site there are sub-populations of

possums which usually den within a predictable small sub-group of the total complement of dens on the site, and have strongly overlapping denning ranges These subpopulations can be considered as "affinity groups" on the grounds that they den in the same vicinity and hence can be inferred to have more frequent potentially infectious social contacts with each other than they do with a random animal in the population of the area Over a period of five years one of these affinity groups at Castlepoint has been continuously infected with tuberculosis of the same REA type, while a second group denning about 200 m away on the fringe of the study site has been infected (probably continuously) with a second REA type, and other areas of the study site with high denning density have not regularly had any tuberculosis-infected animals denning in them Using den site radio-tracking, distinct tightly localised foci or clusters of tuberculosis-infected animals can be identified (by their denning sites), and these clusters persist over extended periods of time These have become known colloquially as "hot spots"

It has so far proved impossible to make extended observations of behaviour in the vicinity of dens However, from both direct observation and indirect inference it would appear that activity during the principal breeding season in autumn and the subsidiary season in spring are central to the transmission process Interaction among possums is greatly increased, and is far more intense than at other times It is common for males to compete actively for oestrous females, and this competition and its expression in threat/agonistic behaviour between males (especially the forceful expiratory sound used to express threat) appears prominent in causing transmission, as is courting and mating activity between males and females Mutual grooming among adults may also contribute to transmission to a minor extent Simultaneous den-sharing can also be responsible for transmission, but at Castlepoint it is very uncommon except between mother and joey Simultaneous sharing is more common at some other sites, such as Bridge Pa in Hawkes Bay Most excretion of organisms appears to be via the respiratory tract , although discharging sinuses also contribute in some animals [17] Faecal and urinary excretion is insignificant [17] Infection appears to be mainly by inhalation, although the distribution of lesions in apparently early cases is somewhat confusing with regard to possible other routes of infection, with axillary lymph nodes infected more commonly than would be expected It is, however, doubtful on the evidence available that this is due to transcutaneous infection The predilection for these specific nodes rather than a broader range of nodes draining skin areas, and for the left nodes in particular, suggest that lymphatic drainage patterns rather than derivation from skin are responsible

The third, and apparently less important pathway is indirect transmission among mature possums This may take place through sequential den-sharing, through sequential chest rubbing of a common marking site, or through contamination of some other commonly shared location such as a track Persistence studies show that organisms last for a maximum of about three weeks in dens, and for a maximum of about two days in sites such as pasture fully exposed to the weather [25] In ruminants, and probably in most other species, the minimum infective dose *via* the respiratory route is about one to ten organisms, whereas the infective dose *via* the alimentary tract is millions of organisms[6] Since these indirect transmission pathways would be likely to produce oral/alimentary rather than nasal/respiratory infection, lesion distribution data [19,20] suggests they are unimportant Evidence from epidemiological patterns of infection in the Castlepoint study

shows that sharing of grazing areas alone does not explain transmission either among possums or from possums to cattle

Necropsy studies of possum populations show that the proportion of possums infected with *M. bovis* is substantially higher than the proportion in which lesions would be found by typical necropsy techniques and on average 8 4% above the prevalence found even in meticulous necropsies[19], a discovery which matches our preliminary finding from computer modelling that infection persists reliably in the population only if there is a substantial pool of sub-clinical animals Continuing lack of a test capable of identifying infected possums during the incubation period prevents the issue being resolved, but the evidence fits best to the hypothesis that of the order of 15 to 20% of possums in an endemic area become infected at some stage of life, a substantial proportion of them as joeys Over half of the early cases found have been in animals below breeding age[19] The lack of any clear age-prevalence association[19] suggests that transmission from adults to juveniles and to other adults are of broadly equal importance, and the lack of any clear sex-prevalence association[19] suggests that male-male interactions are not disproportionately important in transmission

The incubation period from initial infection to clinical (i e externally detectable) disease appears to be very variable, and the majority of new cases occur in temporal waves, indicating some initiating factor for clinical disease Although evidence on initiating factors is limited, it would appear that stress factors, such as weather which produces conditions outside the thermoneutral zone of the possum[26] (rain, fluctuating temperature) or imposes nutritional stress (shortage of suitable feed, or difficulty in grazing due to frequent rain) may be important It is likely that such stress factors act in a cumulative fashion to increase incidence, so no single factor may be responsible The fact that across a full spectrum of stages of infection there is no association between the number of lesion sites and factors such as mesenteric fat weight, body weight, sex or presence of pouch young suggests that it is only the conversion to full clinical manifestation of the disease that is precipitated by external factors, not the underlying development of the infection[19]

Once an animal has developed detectable evidence of disease, median survival time is between two and three months [19], but a small proportion of infected animals last for a year or occasionally more Initially body condition and activity do not seem to suffer, but as animals become terminal they show increasing debility[19] They may den in areas more accessible to feed supplies, on the lower slopes of hills and a number have been found wandering on pasture during daylight hours Tuberculous animals appear to die on pasture far more commonly than the larger proportion of animals which die of other causes[4], supporting the other evidence of behavioural changes

The pattern of excretion of *M bovis* over time (i e when a possum is infectious) has to be inferred from indirect evidence since it cannot be monitored directly and is usually intermittent It would seem that the proportion of possums infectious at any particular moment is very low in the early incubating stages of the disease, but builds up at an escalating rate as the disease advances and once the possums have become clinically detectable most are almost continuously infectious until death

Causes of "Hot Spots" of Tuberculous Possums

Spatial clustering in possum tuberculosis is well-recognized, but as in all clustered diseases there must be identifiable mechanisms which produce this Findings from the Castlepoint site show that one cluster persisted in possums which denned in a single localised part of the site with the same REA type continuously for over five years, and re-emerged in the same area within four months after an intensive possum control operation on the site Extended persistence of clusters has also been found at other sites

One explanation of this would be prolonged persistence of organisms in the environment, although it would have to be in favoured locations such as dens to explain the clustering. However, environmental persistence studies at Castlepoint [25] and the weight of other evidence suggest that organisms in the environment usually remain viable for at most three weeks even in dens, and could not produce persistence over years, as found in diseases such as anthrax Even if persistence in some locations exceeded the measured figures by a substantial margin, there are difficulties with the explanation If there was persistence in an environmental reservoir rather than in possums, the organisms must be readily accessible to other possums over very long periods, which is difficult to envisage as plants which provide the bulk of den cover die and are replaced Moreover, as tuberculous possums use 20 or more dens and annual incidence is in excess of 10%, over time infection should become less patchy in an infected area New clusters could emerge but virtually all existing clusters should be semi-permanent and progressively expand Only if the micro-environmental requirements for persistence were very exacting could clusters persist, and then infection should be far more patchy on a large scale than it appears to be, and the location of clusters should be very predictable from environmental indicators

The alternative explanation for persistence of foci (which on the evidence available so far we consider more plausible) is that infection of a previously negative affinity group of possums commences usually with the arrival of a pseudo-vertically infected juvenile Most dispersers are males, which disperse typically about 2 to 5 km (maximum 11 6) from their natal area between independence at about 10 months and maturity at 2 years [27], in two waves per year If such an animal succeeds in establishing itself as part of a distant affinity group to the point where it has frequent social contact with the other members of the group, and the disease advances to the stage where it becomes infectious, then it will produce horizontally transmitted cases in the group, especially in association with competition between males and mating contacts between males and females However, whether or not disease persists at that site to form a recognizable cluster depends on how rapidly each animal in the infection chain becomes infectious, and the success of each in transmitting infection to other animals in the affinity group We believe that a key factor in determining this is pseudo-vertical transmission to female progeny, which mostly form home ranges overlapping their mother's, so would be very effective in maintaining the cluster in the same location over time.

Our hypothesis is that the disease has a substantial and very variable incubation period, and not only is the temporal pattern of conversion from incubating to infectious precipitated by environmental stress, but also those infected animals which den in environmentally adverse areas will tend to become infectious more rapidly than those denning in protected areas Hence these

factors in combination will produce local incidence sufficient to exceed the threshold rate and maintain a persistent cluster Therefore prolonged maintenance of disease in such "endemic clusters" is common If the environment is less adverse, "sporadic clusters" may arise and then disappear if transmission falls below the threshold These have also occurred at Castlepoint

Persistent clusters explain the maintenance of disease in an area even despite intensive possum control, while sporadic clusters explain herds which have intermittent reactors or single incidents (sometimes with no use of possum control)

The single most important (and difficult) current issue in tuberculosis epidemiology is to identify spatial determinants for persistent clusters, and to assess whether the location of such foci can be predicted with any degree of confidence We are pursuing three options so far A range of environmental variables is being evaluated for known clusters and nearby control sites to seek environmental predictors, the use of indicators of climatic and nutritional stress in the possums themselves is being examined, and the use of scavengers with small home ranges (notably the hedgehog) as indicator species is being explored

Possum to Stock Transmission

Transfer of infection to domestic stock appears to occur largely due to the stock investigating terminally ill possums - which from field observation at Castlepoint fail to show the avoidance behaviour exhibited by healthy possums towards larger animals When we have simulated such behaviour by sedating possums and releasing them some distance from grazing cattle and deer, the more inquisitive among a mob investigate such possums [28], being attracted by the unusual activity In contrast, sheep show little interest in the possum, and although they are experimentally susceptible to tuberculosis, they only rarely contract the disease There is an association between the position of an animal in the dominance hierarchy of its mob of cattle or deer and two indicators of infection risk - its intensity of exploration of a sedated possum, and whether or not it was one of the animals in a group which became infected with tuberculosis [29]

Evidence from Castlepoint is difficult to reconcile with the idea that transfer of infection via contamination of pasture with *M. bovis* is significant in the epidemiology of the disease , since stock which were excluded from the half of the area where the majority of possum dens were located have not become infected, whereas a substantial proportion of the cattle which had access to that area have become infected, even though tuberculous possums regularly grazed the first area Transmission from the dead or decomposing carcases of tuberculous animals has also been proposed It is certainly true that a disproportionate number of tuberculous animals die on pasture However, the behavioural interaction theory explains all of the evidence without needing a second mechanism, and as we have accumulated more evidence we have found increasing difficulties with the dead possum theory It requires that dead possums act as point sources of infection for extended periods of time, and the evidence fits poorly to that since it would require different cattle or deer to each ingest infectious doses from a very small area over weeks or months Moreover, the persistence of organisms in a carcase does not appear to be long enough, and persistence on pasture around a carcase far shorter In addition, predator/scavenger species

tend to consume carcases quite quickly, exposing the remainder to weather and hence death of organisms Thus although this mechanism cannot be totally discarded (cattle will also investigate, though far less actively, a recently dead possum introduced to their paddock), it seems to be relatively inconsequential

Analysis of cattle to cattle transmission through reactor data suggests that it accounts for only a low proportion of total incidence in cattle in endemic areas, because of the testing program which detects infected animals before most of them become infectious Multiple cases at a single test can readily be explained by behavioural transmission direct from possums In contrast, while some deer herds show a similar pattern of sporadic incidence, explosive outbreaks involving large numbers of deer are far more common than among cattle, and it is likely that some at least of these are partly due to deer-to-deer transmission during the interval between tests, possibly complicated by failure to detect some animals at a test because of test sensitivity problems

Transmission from possums to deer appears to be by both respiratory and oral routes, and the relative contribution remains to be resolved In both cattle and deer, infection of the retropharyngeal lymph nodes is one of the most common findings, and it is likely that this arises from oro-pharyngeal tonsillar uptake following ingestion The relative importance of various infection routes for predator species is still under investigation, but there appears to be a majority of alimentary tract infections Organisms are most commonly excreted from the pharynx of ferrets, although a small number do have skin contamination from draining sinuses The likelihood of aerosol transmission is far lower than in possums

Possum Infection from Domestic Stock

The final issue in transmission is how new populations of possums become infected This remains uncertain, but the most plausible explanation is that on rare occasions infected deer (and perhaps even more rarely cattle) corner or otherwise come in close contact with a possum under conditions which allow transfer of infection before the possum escapes Deer are far more aggressive and fast-moving in dealing with possums than are cattle, and will confront a possum and even pick it up, so it is not unrealistic for deer to infect possums on rare occasions Feral deer in particular are also more likely to be infectious than cattle Another mechanism would be possums denning in a deer dark house (which is not uncommon), and becoming infected due to sharing the small air space with infectious deer Thus the ingredients are present for deer to be largely responsible for seeding infection into possum populations Whatever the mechanism, it has to be a rare event to fit the national epidemiological evidence - so it will be difficult to ever confirm it conclusively

Conclusion

It would appear that the epidemiology of the disease is determined by behaviour of both wildlife and domestic stock, and environmental influences on the development of the infectious state in wildlife The central cause of persistent disease at a national level remains the possum, although other species may play a subsidiary role in limited situations

The results of the increased investment in research on tuberculosis control in possums over recent years is starting to show fruit in better understanding of how the disease behaves, and how control should be carried out for maximum effectiveness, and what field strategies should be pursued With newly available knowledge it should be possible to begin to make discernible progress in reducing the possum tuberculosis problem, and then to add further control methods as they become available, until the problem is reduced to a minor irritation which no longer puts our trading position and the livelihood of individual farmers at risk

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